

# LETTERS

## EVALUATING EDUCATION AS AN INTERVENTION FOR INJURY CONTROL

In the June 2004 issue of the Journal, Greenberg-Seth et al. raise several generic issues for the field of injury prevention that need to be discussed if we are to understand the potential public health implications of intervention assessments.<sup>1</sup>

Greenberg-Seth et al. demonstrated the effectiveness of an intervention for increasing child rear seating in a short time frame.<sup>1</sup> The improvements were greater in high-income areas, one feature of educational interventions that must be carefully monitored. Interventions that benefit the richer segments of society more than the poorer segments further existing inequalities and do not serve the goal of health equity. Even if such a trend is temporary, it may have a longer-term effect in terms of perceptions of the intervention and an eventual impact on equity between socioeconomic groups.<sup>2</sup>

The impact on equity becomes even more important in the context of interventions in low-income countries where literacy and access to media is far from universal. More than 90% of the burden of injuries is in the developing world, and this burden is marked by

major inequalities in access to preventive and curative health care.<sup>3</sup> It would be critical for interventions implemented in such settings to be modified to ensure that they are pro-poor.<sup>4</sup>

The effect of education as an independent intervention for injury prevention is not clear.<sup>3</sup> It is an effective component when it is accompanied by another intervention or when it is part of a set of specific interventions. It is natural to assume that health education is beneficial. However, health education may use resources that might be better used to fund interventions with proven effectiveness, especially in resource-scarce environments. Maximizing health outcomes in terms of reduction in injury burden per unit of investment is a critical measure for guiding resource allocation.

It is important to note that output indicators such as use of devices is only half of impact assessment. The more important indicator would be a decrease in childhood injury rates as a result of interventions. Outcome measures require larger samples and longer time frames but are essential in determining the true impact of interventions. Outcome evaluation for interventions, especially education-related interventions, is lacking.

Defining a global research agenda for preventing child injuries and leveraging funds should be a priority for injury prevention stakeholders. ■

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### References

1. Greenberg-Seth J, Hemenway D, Gallagher SP, Ross JB, Lissy KS. Evaluation of a community based intervention to promote rear seating for children. *Am J Public Health* 2004;94:1009–1013
2. Evans T, Whitehead M, Diderichsen F, Bhuiya A, Wirth M. *Challenging Inequities in Health: From Ethics to Action*. New York, NY: Oxford University Press; 2001.

3. Peden M, Scurfield R, Sleet D, Hyder AA, Jarawan E. *World Report on Road Traffic Injuries Prevention*. Geneva, Switzerland: World Health Organization; 2004.

4. Bishai D, Koenig M, Ali Khan M. Measles vaccination improves the equity of health outcomes: evidence from Bangladesh. *Health Econ*. 2003;12:415–419.

5. Mohan D. Road safety in less-motorized environments: future concerns. *Int J Epidemiol*. 2002;31: 527–532.

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# LETTERS

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## GREENBERG-SETH ET AL. RESPOND

We agree with the points raised by Hyder. It is important to design programs that effectively communicate injury prevention messages to people with lower incomes, limited literacy, or limited access to media. We think that community participation in program design and evaluation is a key to accomplishing this.

Any intervention, domestic or abroad, in low- or high-income communities, should begin with formative evaluation to determine literacy needs, community resources, and credible and accessible sources of information. Involving members of the population one is trying to reach is critical. Proven strategies should be modified or adapted for different settings or countries and evaluated in those settings.

In our study, the goal was to increase child rear seating in conjunction with proper restraint use by reinforcing educational messages with incentives for positive behavior. We made special attempts to reach lower-income segments of the population through collaboration with existing community organizations, including those that primarily serve low-income residents. The community task force formulated much of the intervention design. Focus groups and interviews with members of the target population were used to test for appropriateness of materials and distribution channels.

The greater improvement at observation sites in higher-income areas was unexpected, given the level of community participation and our formative research. We are unable to assess whether the difference in impact is because our message did not reach as many of the families we observed in lower-income

areas or because the families we observed in higher-income areas were more likely to change their behavior.

However, even if the direct impact of an intervention is greater in higher-income areas, programs that take a participatory approach to design and implementation can have indirect or unanticipated benefits in addition to changing the intended behavior. Organizing the community to address a specific issue allows people and organizations to collaborate in new ways. Building these relationships can strengthen the community's capacity for addressing other issues. Through surveys and informal observations, we learned that members of our community task force began to extend their relationships beyond the project and were collaborating on other injury prevention, health promotion, and community-building projects.

We think that a participatory approach can be effective regardless of the country in which the intervention takes place, the target audience, or the injury topic being addressed. ■

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and poor quality of health care are neither the most important nor the primary reasons for health inequalities. The social determinants of health, including poverty, status of women, and social vulnerability, are more important.<sup>2</sup> This premise is critical, as it frames the context within which people live in the developing world.

2. The authors propose focusing on “methods that communities can implement, if proved successful.”<sup>1(p925)</sup> This concept has been debated within health systems and health research.<sup>3</sup> What information is required to determine which intervention can be implemented? What level of evidence should be required from researchers? Wendler et al. do not operationalize this concept, leaving it a theoretical construct—which does not further the debate from previous discussions.

3. The example of the Glaser Foundation is misplaced, since health *care* allocations are fundamentally different from decisions within health *research*. The foundation was maximizing benefit per unit of investment—one rational way of resource allocation for health services. The same type of clinical example for research appears in Wendler et al.’s discussion of Niverapine.<sup>1</sup> Health research does not (and should not) have a primary aim of provision of health services, and thus cannot be held to principles of health care distribution.

4. Wendler et al. define a ceiling by calling for defense of any standard of care less than the best in the world. A complementary approach is needed to define the floor, by proposing national standards as the minimum for research studies, even if they can defend the use of a standard less than the world’s best. This would prevent the exploitation of the most disadvantaged groups within developing countries (the worst off).

5. Wendler et al.’s article has important implications for ethics committees in the developing world, in terms of their strengthening and their dialogue with those in the developed world.<sup>4</sup> For example, what are the competencies required for making such decisions in institutional review boards? In externally funded projects, which committee has precedence—sponsoring country or host country?

Finally, there is a need for greater participation of developing-country researchers,

ethicists, and health professionals in this debate. ■

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### References

1. Wendler D, Emanuel EJ, Lie RK. The standard of care debate: can research in developing countries be both ethical and responsive to those countries’ health needs? *Am J Public Health*. 2004;94:923–928.
2. Evans T, Whitehead M, Diderichsen F, Bhuiya A, Wirth M. *Challenging Inequities in Health: From Ethics to Action*. New York, NY: Oxford University Press; 2001.
3. *World Health Report 2000: Health Systems*. Geneva, Switzerland: World Health Organization; 2000.
4. Hyder AA, Wali S, Khan AN, Teoh N, Kass N, Dawson L. Ethical review of health research: a perspective from developing country researchers. *J Med Ethics* 2004;30:68–72.

### THE STANDARD OF CARE DEBATE: CONCEPTUAL CLARIFICATIONS

In the June issue of the Journal, Wendler et al. discuss the standard of care for research in developing countries and propose a valuable set of criteria.<sup>1</sup> However, there are conceptual issues with important implications for this debate that require clarification.

1. Wendler et al. assert that “inequalities in health care have contributed to significant inequalities in health.”<sup>1(p923)</sup> Lack of access to

**WENDLER ET AL. RESPOND**

We proposed conditions under which health investigators may provide research participants with less than the worldwide-best methods. Hyder poses a number of questions for this framework, and we consider his points in turn. First, as Hyder points out, current data suggest that social factors have a significant impact on health. Our claim that inequalities in health care have *contributed* to inequalities in health was not meant to deny the importance of the social determinants of health or to imply that inequalities in health care are more important than these factors.

Second, Hyder points out that it is important to develop a way of analyzing when interventions can be implemented in host countries. Our goal, however, was to establish a framework to ground the ethical relevance of the question of *whether* interventions can be implemented in the host country. In our view, this previous work was especially important given the argument, made by many commentators, that researchers have an obligation to provide research subjects with the best methods in all cases.

Third, our use of the Glaser Foundation example was not intended to imply that health

researchers have an obligation to provide subjects with health services. Rather, this example was intended as a counterexample to the argument that researchers have an obligation to provide the best methods in all cases because they are clinicians. Specifically, the Glaser Foundation example reveals that clinicians do *not* have an obligation, in all cases, to provide the best methods in the clinical setting. This conclusion undercuts the argument that researchers' status as clinicians entails such an obligation in the research setting.

Fourth, our framework is intended to facilitate research on interventions that may benefit those living in developing countries. As Hyder points out, standards, such as national research standards, are needed to ensure that such research does not, in practice, exploit its subjects.

Fifth, Hyder rightly points out that institutional review board competencies and the nature of institutional review board review for multinational research are important concerns.

Finally, we agree that individuals from the developing world should be more involved in the planning, conduct, and reporting of research trials. We hope our framework, which requires input from host communities, will help in realizing this important goal. ■

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**ERRATUM**

*In:* Wei M, Wei P. Occupational risk factors for selected cancers. *Am J Public Health*. 2004;94:1078.

A letter was published without this disclaimer in the About the Authors section:

**Note.** *The opinions expressed by this letter are those of the authors alone and do not reflect the views of the Pennsylvania Department of Health.*



## EDITOR'S CHOICE



# Health Disparities: The Importance of Culture and Health Communication

The root causes of health disparities are numerous and relate to individual behaviors, provider knowledge and attitudes, organization of the health care system, and societal and cultural values. Disparities have been well documented, even in systems that provide unencumbered access to health care, such as the VA Healthcare System, suggesting that factors other than access to care (e.g., culture and health communication) are responsible.

Efforts to eliminate health disparities must be informed by the influence of culture on the attitudes, beliefs, and practices of not only minority populations but also public health policymakers and the health professionals responsible for the delivery of medical services and public health interventions designed to close the health gap. There is credible evidence suggesting that cultural norms within Western societies contribute to lifestyles and behaviors associated with risk factors for chronic diseases (e.g., diabetes and cardiovascular disease). This is the context in which smoking cessation, increased physical activity, and dietary regulation are prime targets for intervention.

We believe that matching the cultural characteristics of minority populations with public health interventions designed to affect individuals within the group may enhance receptivity to, acceptance of, and salience of health information and programs. This approach is consistent with the documented evidence that factors such as belief systems, religious and cultural values, life experiences, and group identity act as powerful filters through which information is received. It is important to consider these factors in the development of health communication campaigns.

Since January 2001, the Center for Minority Health in the Graduate School of Public Health at the University of Pittsburgh, led by Stephen B. Thomas, has hosted the Annual National Minority Health Leadership Summit in partnership with the VA Center for Health Equity Research and Promotion. The annual summit provides a forum for assessing the progress of the national effort to eliminate disparities in health and health care. In 2001, the summit's theme was

"Mapping a Course for Community Action and Research"; in 2002, "The Impact of Discrimination on Health Status"; in 2003, "The Role of Community Based Participatory Research"; and in 2004, "The Role of Health Communication." (The theme of the 2005 summit will be "Race, Genes, and the Environment.")

The articles and editorials featured in this issue of the Journal reflect the content and scope of the scientific and community-based interventions presented during the 2004 summit. Collectively, they describe the continuum of observational, explanatory, and interventional research on racial and ethnic health disparities.

Eliminating racial and ethnic health disparities by 2010 will require a sustained sense of urgency over the next 6 years. Our efforts must address social, cultural, and environmental factors beyond the biomedical model. This approach includes breaking the cycle of poverty, increasing access to quality health care, eliminating environmental hazards in homes and neighborhoods, and implementing effective prevention programs tailored to specific community needs. Making health communication programs work requires both the active participation of affected individuals and communities in the creation of health communication interventions and the consideration of culture in message development. If these requirements are met, health communication campaigns can reflect our unyielding commitment to speak up for racial and socioeconomic equality and to reduce inequalities and enhance life for all Americans. ■

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# Communication at the Core of Effective Public Health

Years from now, we may remember the fall of 2001 as the traumatic period in our country's history when public health became visible throughout society. While the dust was still settling from the terrorist attacks and the country grappled with fears of anthrax in our mail, the American public learned what professionals have known for years: the health and security of our nation depend heavily on a robust public health system. An additional revelation from this turbulent time was the critical role of strategic, timely, and effective communication in public health. The realization that both *science* and *communication* are essential to promoting and protecting the health of the public was a major milestone in the emerging discipline of public health communication, reinforcing its vital role as a new core component of public health.

## COMMUNICATION RENAISSANCE

Over the last several decades, the application and study of communication and health have rapidly developed and expanded. Originally conceived in departments and schools of communication and medicine,<sup>1</sup> courses in health communication are now found throughout the academy, from the liberal arts to the health sciences and at many points in between. The recent proliferation of health communication is evident in the field's significant accomplishments, including the establishment of 2 peer-reviewed health communi-

cation journals, the inclusion of a health communication chapter in *Healthy People 2010*,<sup>2</sup> the funding of health communication centers of excellence by the National Cancer Institute, and the publication of 3 books from the Institute of Medicine (IOM) stressing the importance of health communication.<sup>3-5</sup>

Despite these accomplishments, the discipline of communication has until recently operated at the periphery of public health. Perceived as more skill than science, communication was equated only with dissemination of findings by many public health professionals, who assumed that public health information could "speak for itself." Fortunately, many of today's public health leaders realize that promoting health and protecting the public require both sound science and effective public health communication. The Centers for Disease Control and Prevention (CDC), for example, has recognized the importance of public health communication, concluding that "public health research, innovations in information technology, and advanced communications offer unprecedented opportunities for CDC to improve health in America and around the world."<sup>6(p8)</sup>

## WHAT IS PUBLIC HEALTH COMMUNICATION?

*Healthy People 2010* defines health communication as "the art and technique of informing, influencing, and motivating individual, institutional, and public audiences about important health

issues."<sup>2(p11-6)</sup> The IOM defined public health as "what we, as a society, do collectively to assure the conditions in which people can be healthy."<sup>4(p28)</sup> If we integrate these 2 perspectives, the following new definition emerges: Public health communication is the scientific development, strategic dissemination, and critical evaluation of relevant, accurate, accessible, and understandable health information communicated to and from intended audiences to advance the health of the public.

Public health communication draws from numerous disciplines, including mass and speech communication, health education, marketing, journalism, public relations, psychology, informatics, and epidemiology. Although it is transdisciplinary in nature,<sup>5</sup> the core principles of public health communication are firmly anchored in the central tenets of public health.

## Ecological Perspective

Public health recognizes that health is profoundly affected by the social, political, environmental, and behavioral factors with which people live.<sup>5,7</sup> Public health communication embraces this *ecological perspective* by encouraging multilevel communication strategies and interventions, such as tailored messages at the individual level, targeted messages at the group level, social marketing at the community level, media advocacy at the policy level, and media campaigns at the population level. In addition, public health communication strategies are often com-

bined with other intervention efforts, such as community organizing or coalition building, to produce multilevel public health interventions.

### Change Orientation

Like most applied research and practice in public health,<sup>5</sup> public health communication focuses more on improving the health of communities and populations than on deconstructing the underlying mechanisms of communication. Public health communication is inherently interventionist, seeking to promote and protect health through change at all levels of influence. When well conceived, carefully implemented, and sustained over time, public health communication programs have the capacity to elicit change among individuals and populations by raising awareness, increasing knowledge, shaping attitudes, and changing behaviors.<sup>8</sup> Although communication initiatives often target for change those behaviors that contribute directly to morbidity and mortality, public health communication also targets social, physical, and environmental changes that can influence health outcomes.

### Audience-Centered Philosophy

Health communication campaigns have sometimes been criticized as paternalistic, and concerns have been raised about the use of 1-way communication from "beneficent" experts to passive audiences.<sup>9</sup> Public health communication recognizes that for programs to be both ethical and effective, information from and about the intended audience should inform all stages of an intervention, including development, planning, and implementa-

tion, to ensure that the program reflects the audience's ideas, needs, and values.<sup>3,9</sup> Areas of particular interest include the audience's health literacy, culture, and diversity.<sup>3</sup> Furthermore, public health communication programs rely heavily on formative research and 2-way communication between sources and receivers to ensure that messages are accessed and understood, communities are involved and invested, and programs are modified as needed.

### FUTURE DIRECTIONS

The field of public health communication is poised for a period of rapid expansion, fueled in part by 2 major developments. First, the IOM report on public health professional preparation identifies communication as a critical content area for future public health education.<sup>5</sup> Second, through its "Futures Initiative," the CDC is creating a national center focused on public health communication and marketing. These developments, along with continued scientific and technological advances, will dramatically affect future training, research, and practice in public health communication.

The reach and impact of public health communication has never been greater, as numerous campaigns address diverse health issues and audiences throughout the United States and the world. Progress toward the Healthy People 2010 objective of increasing research and evaluation in health communication<sup>2</sup> is evidenced by recent funding opportunities in public health communication research established by several federal agencies. However, for public health communication to reach its full potential, signifi-

cantly more research will be needed, especially in the study of health disparities, communication-access disparities, and the application of electronic health interventions with underserved populations.

Greater support is also needed for research and evaluation in public health communication that is truly transdisciplinary, simultaneously addressing multiple health issues, intervention levels, and communication channels. Further, although there are many challenges for program evaluation in public health communication, conducting comprehensive evaluations and disseminating the results is critical for expanding knowledge, improving programs, and allocating limited resources.

Instructional opportunities in public health communication have been available for many years and are likely to expand in response to the IOM recommendations<sup>5</sup> and future changes in workforce needs. Many schools and programs in public health are developing academic programs in public health communication, including certificate programs and concentrations within traditional and distance-learning MPH programs. To ensure that these programs adequately prepare students for careers in public health communication, however, it is important that disciplinary competencies be collectively developed and disseminated, ideally with support and direction from a federal agency or national organization.

Finally, public health communication professionals have a responsibility to communicate well with each other. To facilitate the exchange of information and the translation of public health communication research to practice,

public health communicators need to become more visible and vocal in our professional organizations. We must also embrace innovative tools for translation and dissemination, such as the TREND Statement (<http://www.trendstatement.org>),<sup>10</sup> the HealthComm Key database (<http://www.healthcommkey.org>), and the Prevention Communication Research Database (<http://www.health.gov/communication>).

### CONCLUSION

With its transdisciplinary nature, ecological perspective, change orientation, and audience-centered philosophy, public health communication has the potential to make significant contributions to the health of the public. I applaud today's public health leaders and visionaries who have recognized this potential and the innovative work conducted by public health communication professionals. Over the coming months and years, the discipline of public health communication will continue to grow and develop, and when the dust has finally settled, public health will be changed and improved to its very core. ■

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## References

1. Ratzan SC. Editor's introduction: communication—the key to a healthier tomorrow. *Am Behav Scientist*. 1994;38: 202–207.
2. *Healthy People 2010*. 2nd ed. Washington, DC: US Dept of Health and Human Services; 2000.
3. Institute of Medicine. *Speaking of Health: Assessing Health Communication Strategies for Diverse Populations*. Washington, DC: National Academy Press; 2002.
4. Institute of Medicine. *The Future of the Public's Health in the 21st Century*. Washington, DC: National Academy Press; 2003.
5. Institute of Medicine. *Who Will Keep the Public Healthy?* Washington, DC: National Academy Press; 2003.
6. *State of the CDC Fiscal Year 2003*. Atlanta, Ga: Centers for Disease Control and Prevention; 2004.
7. Institute of Medicine. *America's Vital Interest in Global Health*. Washington, DC: National Academy Press; 1997.
8. Hornik RC. *Public Health Communication: Evidence for Behavior Change*. Mahwah, NJ: Lawrence Erlbaum; 2002.
9. Guttman N. *Public Health Communication Interventions: Values and Ethical Dilemmas*. Thousand Oaks, Calif: Sage Publications; 2000.
10. Des Jarlais DC, Lyles C, Crepaz N, TREND Group. Improving the reporting quality of nonrandomized evaluations of behavioral and public health interventions: the TREND statement. *Am J Public Health*. 2004;94:361–366.

# The Contributions of Health Communication to Eliminating Health Disparities

The pressing need to eliminate health disparities calls on public health professionals to use every effective tool possible. Health communication, defined as the study and use of methods to inform and influence individual and community decisions that enhance health, was first recognized as a subset of the field of communication in 1975, when the Health Communication Division of the International Communication Association was founded.<sup>1,2</sup> The National Communication Association formed a division of the same name in 1985. In 1997, the Public Health Education and Health Promotion section within the American Public Health Association formally recognized health communication as part of its group. The peer-reviewed journal *Health Communication* began in 1989, followed 7 years later by the *Journal of Health Communication*. Today, while many communication departments and schools of public health offer limited graduate course work in health communication, there are fewer than a dozen comprehensive programs in health communication.

The federal government has recognized the contributions of health communication. The Centers for Disease Control and Prevention developed an office of communication in 1996 with the purpose of diffusing the sci-

ence of health communication throughout the agency. The National Cancer Institute, in 1999, developed an "Extraordinary Opportunity in Cancer Communications," which included awarding Centers of Excellence in Cancer Communication to 4 universities; 2 of the 4 centers explicitly focus on research in health communication aimed at health disparities. In addition, for the first time, health communication is part of the Healthy People 2010 objectives.<sup>3</sup>

## THE SCOPE AND LIMITATIONS OF HEALTH COMMUNICATION

These achievements notwithstanding, the public health community seems to have a limited understanding of what health communication can offer to the elimination of health disparities. According to the National Cancer Institute, health communication can increase the intended audience's knowledge and awareness of a health issue, problem, or solution; influence perceptions, beliefs, and attitudes that may change social norms; prompt action; demonstrate or illustrate healthy skills; reinforce knowledge, attitudes, or behavior; show the benefit of behavior change; advocate a position on a health issue or policy; increase demand or support for health

services; refute myths and misconceptions; and strengthen organizational relationships.<sup>1(p3)</sup>

However, health communication alone, without environmental supports, is not effective at sustaining behavior changes at the individual level. It may not be effective in communicating very complex messages, and it cannot compensate for lack of access to health care or healthy environments.<sup>1(p3)</sup> Nonetheless, we believe that public health professionals should use the full range of health communication strategies in the effort to eliminate health disparities.

## THE RANGE OF HEALTH COMMUNICATION STRATEGIES

Many are familiar with mass media campaigns aimed at stimulating individual behavior change. However, there is less familiarity with other forms of health communication that can be effective in the context of health disparities. Health communicators can bring their expertise to bear in entertainment-education, media advocacy, new technology, and interpersonal communication, including patient-provider communication.

### Entertainment-Education

Entertainment programming in the media is a powerful way

to communicate health information, especially for minority audiences, who are heavy consumers of this type of media. Several research studies have demonstrated that even brief exposure to health information and behaviors through entertainment media can have strong effects. In surveys ( $n=3719$ ) conducted by Porter Novelli during 2001, more than half of regular prime time and daytime drama viewers reported that they learned something about a disease or how to prevent it from a TV show. Among minority viewers who watch regularly, 70% of Hispanic women, 65% of Black women, and 64% of Black men said they took some action after hearing about a health issue or disease on a TV show.<sup>4</sup> More than 50% of Black men and women reported that a storyline helped them to provide information to friends or family, as did 60% of Hispanic women.<sup>4</sup> Entertainment programming has the capacity to reach significant proportions of the populations experiencing health disparities.

### Media Advocacy

Media advocacy is defined as the strategic use of mass media and their tools, in combination with community organizing, for the purpose of advancing healthy public policies.<sup>5(p338)</sup> Because the roots of health disparities extend to social, economic, and political conditions, media advocacy, which moves beyond the focus on the individual, holds promise as one form of health communication to address health disparities. One example of such a campaign is the Uptown Coalition in Philadelphia, which used the media and community organizing to defeat RJ Reynolds's proposed campaign to market Up-

town cigarettes in African American communities.

### Interactive Health Communication

Interactive technology, "computer-based media that enable users to access information and services of interest, control how the information is presented, and respond to information and messages in the mediated environment,"<sup>6(p2)</sup> has created new opportunities for health communication that can overcome barriers such as low literacy and expand opportunities to tailor and personalize information. One of the pioneer applications of such technology is the Comprehensive Health Enhancement Support System (CHES), for which there is impressive research evidence of its potential for reducing disparities. In a study of the use of an HIV CHES application, women and minorities made more use of several information tools than men and nonminorities, and minorities and those with less education used the decision and analysis tools more than nonminorities and people with more education, even though these tools were the most complex in the system.<sup>7</sup> Similar results were found in a pilot study of low-income, African American women with breast cancer.<sup>7</sup> Yet computer access issues prevent these approaches from achieving their potential in reducing health disparities.

### Interpersonal Communication

Interpersonal communication theory helps us understand the provider-client interaction, the role of social support in health, and the ways in which interpersonal relationships influence health behaviors and decision-making. Clearly, the relationship

between patient and provider can exacerbate health disparities. Van Ryn and Fu<sup>8</sup> suggest that providers may contribute to health disparities by influencing clients' views of themselves and their relation to the world, by differentially encouraging health promotion and disease prevention behaviors and services, and by withholding access to treatments or services and denying benefits and rights. They cite evidence of physicians' contributions to racial/ethnic disparities in kidney transplant rates and cardiac procedures, in pain assessment and control, and in mental health services. They argue for interventions to help providers avoid their own biases as one way to reduce disparities. Ashton and colleagues<sup>9</sup> examined communication between providers and minority patients and found that poor communication is linked to health disparities and requires specific interventions to address communication patterns.

Social support is another communication behavior that has profound consequences for mental and physical well-being.<sup>10</sup> Yet there is evidence that kinship support networks are deteriorating in low-income and minority communities because of unemployment, transience, and substance abuse.<sup>11</sup> Virtual support networks are becoming increasingly important, but again, access is an issue in underserved communities. Much more needs to be learned about the impact of culture on both expectations of support and the effects of support.

Cline's<sup>12</sup> argument for shifting the focus of interpersonal communication about health from formal to informal contexts such as everyday talk highlights a rich and untapped dimension of com-

munication that could contribute to reducing disparities. Certainly, the impact of interpersonal communication through the use of lay health advisors, respected in their communities, is well documented. Extensive research on tailoring and targeting health messages promises new opportunities for reaching those who suffer most from health disparities.

### CULTURAL DIFFERENCES AND HEALTH COMMUNICATION

However, in all these efforts, health communicators often struggle to understand the audiences they seek to reach, frequently equating culture in a simplistic fashion with race and ethnicity. The Institute of Medicine<sup>13</sup> argues that culture has been poorly examined in the context of health communication, asserting that to consider culture requires significant exploration beyond the typical variables of race, ethnicity, and socioeconomic status. According to the Institute, health communication campaigns typically address the issue of diverse audiences in 1 of 3 ways: by developing a communication campaign with common-denominator messages relevant to most audiences; by developing a unified campaign with systematic variations in messages to increase relevance for different audience segments, retaining one fundamental message; or by developing distinctly different messages or interventions for each audience segment.<sup>13</sup>

Many health communication interventions address what Resnicow and Braithwaite<sup>14</sup> refer to as the surface structure of a culture. Addressing surface structure includes matching



messages and channels to observable social and behavioral characteristics of a culture, for example, familiar people, foods, music, language, and places. It may be more important to address deep structure, which reflects the cultural, social, psychological, environmental, and historical factors that affect health for a minority community. Resnicow and Braithwaite argue that when health communication appropriately addresses surface structure, it increases receptivity to and acceptance of the campaign, but when it also addresses deep structure, it conveys true salience to the community it seeks to reach. Clearly, there is much to learn about creating health communication interventions that appreciate the complexity of culture, and then evaluating the impact of such programs on eliminating health disparities.

Eliminating health disparities requires that public health professionals expand their use of health communication strategies in comprehensive interventions aimed at effecting individual, community, organizational, and policy change. Such interventions can effectively address the

multiple determinants of health that underlie disparities. However, to design effective interventions, we must understand the complexity of culture and integrate cultural factors into our health communication efforts. Furthermore, we must work collaboratively with communities experiencing disparities to overcome the historical context of distrust and create meaningful, effective health communication interventions. ■

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#### References

1. *Making Health Communication Programs Work*. Bethesda, Md: National Cancer Institute; 2001.
2. Freimuth V, Cole G, Kirby S. Issues in evaluating mass media health communication campaigns. In: Rootman I, Goodstadt M, Brian Hyndman, et al., eds. *Evaluation in Health Promotion: Principles and Perspectives*. Copenhagen, Denmark: WHO Regional Office for Europe; 2001:475–492.
3. *Healthy People 2010: Understanding and Improving Health*. Washington, DC: US Dept of Health and Human Services, Office of Disease Prevention and Health Promotion; 2000.
4. Office of Communication, Centers for Disease Control and Prevention. Entertainment Education: Overview. Available at <http://www.cdc.gov/communication/surveys/surv2001.htm>. Accessed July 21, 2004.
5. Institute of Medicine. *The Future of the Public's Health in the 21st Century*. Washington, DC: National Academies Press; 2003.
6. Street RL Jr, Rimal RN. Health promotion and interactive technology: A conceptual foundation. In: Street RL Jr, Gold WR, Manning T, eds. *Health Promotion and Interactive Technology*. Mahwah, NJ: Lawrence Erlbaum Associates Inc; 1997:1–18.
7. Hawkins RP, Pingree S, Gustafson DH, et al. Aiding those facing health crises: the experience of the CHES project. In: Street RL Jr, Gold WR, Manning T, eds. *Health Promotion and Interactive Technology*. Mahwah, NJ: Lawrence Erlbaum Associates Inc; 1997:79–102.
8. Van Ryn M, Fu S. Paved with good intentions: do public health and human service providers contribute to racial/ethnic disparities in health? *Am J Public Health*. 2003;93:248–255.
9. Ashton C, Haidet P, Paterniti D, et al. Racial and ethnic disparities in the use of health services: bias, preferences or poor communication? *J Gen Intern Med*. 2003;18:146–152.
10. Albrecht T, Goldsmith D. Social support, social networks, and health. In: Thompson T, Dorsey A, Miller K, Parrott R, eds. *Handbook of Health Communication*. Mahwah, NJ: Lawrence Erlbaum Associates Inc; 2003: 263–284.
11. Roschelle A. *No More Kin: Exploring Race, Class, and Gender in Family Networks*. Thousand Oaks, Calif: Sage Publications; 1997.
12. Cline R. Everyday interpersonal communication and health. In: Thompson T, Dorsey A, Miller K, Parrott R, eds. *Handbook of Health Communication*. Mahwah, NJ: Lawrence Erlbaum Associates Inc; 2003:285–318.
13. Institute of Medicine. *Speaking of Health: Assessing Health Communication Strategies for Diverse Populations*. Washington, DC: National Academies Press; 2002.
14. Resnicow K, Braithwaite R. Cultural sensitivity in public health. In Braithwaite R, Taylor S, eds. *Health Issues in the Black Community*. 2nd ed. San Francisco, Calif: Jossey-Bass; 2001: 516–542.

# Confronting Health Inequity: The Global Dimension

Since the days of Hippocrates, health inequities and the role of social and environmental factors in the determination of marked differences in health status have been well recognized. For some time now, the driving force behind public health has been understanding and intervening in the underlying causes of health inequity. The publication of the Black Report<sup>1</sup> in the United Kingdom in 1980 brought a more focused approach to this discourse by identifying specific factors, such as social class, gender,

and race/ethnicity, as the social and economic determinants of health inequities. With this evolution came a conceptual and operational distinction between health disparities/inequalities and health inequity/equity.<sup>2</sup>

These distinctions aside, the issue of health inequity has moved beyond the academic discourse into the arena of policy and action. In the United States, the 2002 Institute of Medicine report *Unequal Treatment: Confronting Health Care Disparities* marked a

turning point.<sup>3</sup> It is, however, important to recognize that like the problem of health inequity itself, the struggle to confront it is neither unique to the United States nor simply a local matter. Many nations, both developed and developing, have adopted strategies to reduce health inequities.

## EFFORTS IN THE DEVELOPED WORLD

Confronting health inequities is increasingly a priority for



health policymakers, both nationally and internationally. There are several recent examples of national governments in developed countries undertaking major initiatives to reduce health inequities. For instance, in the United Kingdom one of the first decisions of the incoming Labor government in 1997 was to commission the "Independent Inquiry into Inequalities in Health." Under the direction of Sir Donald Acheson, the commission's mandate was to establish the facts and suggest why, despite the increase in prosperity and substantial reductions in mortality evinced in the United Kingdom in the previous 2 decades, the gap in health status between those at the top and bottom of the social scale, as well as between various ethnic groups and between the sexes, had continued to widen.<sup>4</sup> On the basis of the commission's recommendations, the government formulated a comprehensive plan that recognizes the structural determinants of health, such as the social environment and the wider community, with the overarching goal of reducing avoidable health disparities.<sup>5,6</sup>

In 1998, the EURO Health for All policy (Health 21) was published.<sup>7</sup> This policy specifies that by 2020 the health gap between countries and between socioeconomic groups within countries should be reduced by at least one fourth in all member states. Since that time, other European countries have undertaken similar comprehensive reviews and action plans at regional, national, and local levels.<sup>8,9</sup> The following EURO Health for All policy recommendations are being implemented, at least partially, in member

states of the European Union and various other neighboring countries, providing a useful model for similar action in other regions<sup>10</sup>:

1. Establish national health inequity targets by identifying and advocating relevant national and regional health targets and by tackling health determinants to reduce health inequalities.
2. Integrate health determinants into other policy areas at national, regional, and local levels, using cross-sectoral approaches.
3. Work at the local level by supporting community development approaches and the integration of local services, multidisciplinary approaches, and partnerships.
4. Reduce barriers to ensure access to and use of effective health care and prevention services by socially disadvantaged and vulnerable groups.
5. Develop indicators and systems for monitoring health inequalities, including systems for collecting data on structural factors and determinants of health, such as social class, gender, and ethnicity.
6. Assess health impact by developing and applying procedures, methods, and tools by which policies, programs, and projects may be judged as to their potential effects on the health of a population and the distribution of those effects within the population.
7. Evaluate financial and human resources to ensure sufficiency and to increase knowledge on how to effectively tackle health inequities.
8. Create and support opportunities to disseminate models of good practice and evidence-based approaches to tackle health in-

equalities, including databases of successful interventions.

Other developed countries, such as Australia, New Zealand, and Canada, are also in the process of incorporating health equity and social determinants of health into regional or national public health policies.<sup>11-13</sup>

In the United States in 1998, the Clinton administration established the Initiative to Eliminate Racial and Ethnic Disparities in Health, which set a national goal of eliminating longstanding racial/ethnic disparities in health status by 2010 and, for the first time, set high national goals for all Americans, ending a practice of separate, lower goals for racial and ethnic minorities.<sup>14</sup> There are mounting public and private coalition efforts aimed at "closing the gap" in health and health care that have continued under the Bush administration.<sup>15</sup>

In contrast to the European approach to health inequities, it is racial and ethnic disparities that are of greater policy relevance in the United States. First, there are obvious historical reasons for the extensive overlap of socioeconomic and racial inequalities in the United States. Second, the predominant use of ethnic and racial group categories in most vital statistics, census, economic, and other population and health related data greatly facilitates monitoring disparities by race instead of by social class.<sup>16</sup> In fact, it has been well demonstrated in the United States that socioeconomic differences between races account for much of the racial differences in health, even though race per se—or rather, the results of societal discrimination based on race—may have an independent effect on health

status and health care access/utilization.<sup>17-19</sup>

## STRATEGIES IN THE DEVELOPING WORLD

The emergence of health equity as a public health issue is also occurring in the developing world. Following the Alma-Ata Primary Health Care Summit in 1979, many national governments in Latin America, Asia, and Africa came together to formulate a strategy for achieving the goal of "Health for All."

The Alma-Ata summit advocated the achievement of greater health equity and the reduction of health disparities as national goals. Prior to the emergence of the HIV/AIDS epidemic in the 1990s, many developing countries achieved noteworthy improvements in national average life expectancy and mortality rates, even though health disparities between socioeconomic and ethnic groups within countries actually increased in most cases. For example, in Latin America and the Caribbean, the region that experienced the highest rate of improvement in health indicators in that period, health disparities were also the greatest. The ratio between the highest and lowest national infant mortality rates in the region of the Americas was 7:1 in 1964 and had risen to 14:1 by 1994. Similarly, within Brazil, even though the national infant mortality rate fell by 40% between 1977 and 1995, the ratio between the rural northeast and the rest of the country actually increased from 1.7:1 to 2.0:1.<sup>20</sup>

In response to these disparities, from 1996 to 2002 the Pan American Health Organization undertook an ambitious effort to promote health equity in its tech-

nical cooperation programs in the Americas by promoting research, benchmarking, strengthening information dissemination, establishing databases, and improving health information analysis for monitoring and reducing health disparities within and between countries in the region.<sup>21,22</sup> In fact, some Latin American countries—for example, Costa Rica, Chile, Peru, Bolivia, and Brazil—have incorporated equity goals into their national public health programs.

## THE ROLE OF THE INTERNATIONAL COMMUNITY

The international community also has a role in the global campaign to confront health inequalities. Some international organizations are already in the forefront of this campaign. For example, the Poverty and Health Network of the World Bank<sup>23,24</sup> has developed a methodology for the analysis of socioeconomic differences in health, nutrition, and population in developing countries that is based on the World Bank's demographic and health surveys. This methodology provides a much needed empirical approach for monitoring intracountry trends and intercountry comparisons of health disparities.<sup>25,26</sup>

In 1996, the Rockefeller Foundation and the Sweden International Development Cooperation Agency established a Global Health Equity Initiative, with a network of more than 100 researchers in more than 15 countries, for the purpose of raising global awareness and building capacity to address health inequities. The most visible product of this effort was the publication in 2001 of a groundbreaking report<sup>27</sup> that established a solid

conceptual and operational framework, based on a global perspective and country-specific analysis, of health equity in which global and national determinants are closely interrelated (via the economic and social consequences of economic and financial globalization, political stability and governance, poverty and development, ethnic conflicts, migration, etc.). The report emphasized the need to strengthen the capacity of the health sector in all countries and provide it with tools for tackling health disparities, in partnership with all potential partners in government and civil society.

Various current global initiatives have emerged from the Global Health Equity Initiative and other aforementioned efforts. One is the Global Equity Gauge Alliance, also supported by the Rockefeller Foundation and the Sweden International Development Cooperation Agency, which was created to participate in and support an active approach to monitoring health inequalities and promoting equity within and between societies. The Alliance currently includes 11 member-teams, called Equity Gauges, located in 10 countries in the Americas, Africa, and Asia.<sup>28</sup> In sub-Saharan Africa, an initiative closely linked to the Global Equity Gauge Alliance is EQUINET, the Regional Network on Equity in Health in Southern Africa. EQUINET involves professionals, civil society members, policymakers, state officials, and academic, government, and civic institutions from Botswana, Malawi, Mozambique, South Africa, Tanzania, Zambia, Zimbabwe, and the South African Development Community who have come together as an

equity catalyst to promote shared values of equity and social justice in health.<sup>29</sup>

The International Society for Health Equity, founded in 2000, has successfully held 3 international conferences with hundreds of participants from all continents; today it constitutes the most authoritative international professional association of health equity researchers, analysts, and advocates.<sup>30</sup> The most recent conference, held in Durban, South Africa, in June 2004, dealt with a myriad of emerging issues for effectively reducing health disparities in the developing and developed world. Some of these issues included insurance and finance, resource allocation, access to care, special population groups, analytical methods for time trends and life-course determinants, community action, social empowerment, gender and health, law and human rights, local governance and planning, and the impact of HIV/AIDS.<sup>31</sup>

The United Nations organizations, such as the World Health Organization (WHO), also have a leadership role to play in the global effort to confront health inequalities. Such action is consistent with the 1998 World Health Assembly resolution, which confirmed that a reduction in socioeconomic inequalities in health was a priority for all countries.<sup>32</sup> In 2000, a special issue of the *Bulletin of the World Health Organization*<sup>33</sup> was devoted to inequalities in health, and the WHO Global Health Survey, initiated in 2001, provides valuable health indicators that can be crossed with socioeconomic data to provide the basis for the monitoring of health disparities.<sup>34</sup>

Since 2003, under the leadership of Director General Lee Jong-Wook, the issue of health

equity has acquired a new place in the priorities of WHO.<sup>35</sup> An equity team has been established within the area of evidence and information for policy, with the objective of supporting innovation and strengthening knowledge sharing on a global level. An expressed goal is to develop new forms of collaboration between health experts and decisionmakers to translate current evidence on the social and environmental determinants of health disparities into effective public policy.<sup>36</sup> In his speech to the 57th World Health Assembly in May of this year, the WHO director general announced his intention to set up and launch a new global commission formed by expert public health scientists and policymakers to gather evidence on the social and environmental causes of health inequities and ways to overcome them, with the purpose of providing guidance for all WHO programs.<sup>37</sup>

In summary, there is a global movement for health equity that began in the last decade of the 20th century and continues to grow. The role of information and knowledge sharing is key in linking this global effort to local actions and challenges; international and national health organizations in the developed and developing countries, be they in the public sector or in civil society, must join hands with local communities and governments if health inequities are to be effectively reduced.

The optimists among us believe that the road toward globalization can lead us to a future in which development becomes freedom<sup>38</sup> and in which all human beings can enjoy complete citizenship, wherever they may be; exercise the right to gainful employment; and fully

share in the benefits of knowledge and information.<sup>39</sup> Such a world is one in which avoidable and unfair differences in the opportunity to lead a healthy life—differences between men and women; among Black, White, and brown; among inhabitants of the North and South, East and West—would cease to exist. The road to this world is a long one, one that will take us far beyond the horizon. Although it begins on our very doorstep, it has global dimensions. ■

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### References

- Black D. *Inequities in Health*. London, United Kingdom: Penguin; 1980.
- Macinko JA, Starfield B. Annotated bibliography on equity in health, 1980–2001. *International Journal for Equity in Health*. 2002;1(1):1–20.
- Unequal treatment: confronting racial and ethnic disparities in health care. A report of the Institute of Medicine*. Washington, DC: National Academy Press; 2002.
- Acheson D. Independent Inquiry into Inequalities in health report. London, United Kingdom: The Stationery Office; 1998. Available at: <http://www.archive.officialdocuments.co.uk/document/doh/ih/ih.htm>. Accessed September 27, 2004.
- Exworthy M, Stuart M, Blane D, Marmot M. *Tackling Health Inequalities Since the Acheson Inquiry*. London, United Kingdom: The Policy Press; 2003.
- Department of Health. Health inequalities. 2004. Available at: <http://www.dh.gov.uk/policyandguidance/healthandsocialcaretopics/healthinequalities>. Accessed September 27, 2004.
- World Health Organization Regional Office for Europe. Health 21: an introduction to the health for all policy framework for the WHO European Region. Available at: <http://www.euro.who.int/document/EHFA5-E.pdf>. Accessed September 27, 2004.
- World Health Organization Regional Office for Europe. Update of the regional Health for All Policy framework. 2003. Available at: <http://www.euro.who.int/document/rc53/edoc08.pdf>. Accessed September 27, 2004.
- Menke R, Streich W, Rossler G, Brand H. *Report on Socio-Economic Differences in Health Indicators in Europe: Health Inequalities in Europe and the Situation of Disadvantaged Groups*. Bilthoven, the Netherlands: National Institute for Public Health and the Environment (RIVM); 2003.
- European Network of Health Promotion Agencies. The role of health promotion in tackling inequalities in health: policy recommendations. Available at: <http://www.eurohealthnet.org/eurohealthnet/ressources/brochure.pdf> (PDF file). Accessed September 27, 2004.
- New South Wales Department of Health. Social determinants of health. Updated October 24, 2002. Available at: [http://www.health.nsw.gov.au/public-health/chorep/soc/soc\\_intro.htm](http://www.health.nsw.gov.au/public-health/chorep/soc/soc_intro.htm). Accessed September 27, 2004.
- Ministry of Health. Social inequalities in health—New Zealand 1999. 2000. Available at: <http://www.moh.govt.nz/moh.nsf/0/65b8566b2ac3a9684c2569660079de90?OpenDocument>. Accessed September 27, 2004.
- Health Canada. The social determinants of health: an overview of the implications for policy and the role of the health sector. 2003. Available at: [http://www.hc-sc.gc.ca/hppb/phdd/pdf/overview\\_implications/01\\_overview\\_e.pdf](http://www.hc-sc.gc.ca/hppb/phdd/pdf/overview_implications/01_overview_e.pdf). Accessed September 27, 2004.
- Ibrahim SA, Thomas SB, Fine MJ. Achieving health equity: an incremental journey. *Am J Public Health*. 2003;93:1619–1621.
- Department of Health and Human Services, Office of Minority Health. Health disparities. Available at: <http://www.omhrc.gov/OMH/Health%20Disparities/index.htm>. Accessed September 27, 2004.
- National Research Council, Panel on DHHS Collection of Race and Ethnic Data. Ver Ploeg M, Perrin E, eds. *Eliminating Health Disparities: Measurement and Data Needs*. Washington, DC: National Academies Press; 2004. Also available at: <http://www.nap.edu/catalog/10979.html>. Accessed September 27, 2004.
- Krieger N, Sidney S. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health*. 1996;86:1370–1378.
- Krieger N, Rowley DL, Herman AA, Avery B, Phillips MT. Racism, sexism, and social class: implications for studies of health, disease, and well-being. *Am J Prev Med*. 1993;9(suppl 6):82–122.
- House J, Williams DR. Understanding and reducing socioeconomic and racial/ethnic disparities in health. In: Hofrichter R, ed. *Health and Social Justice: Politics, Ideology, and Inequity in the Distribution of Disease*. San Francisco, Calif: Jossey-Bass; 2003.
- Casas JA, Dachs NW, Bambas A. Health disparities in Latin America and the Caribbean: the role of social and economic determinants. In: *Equity and Health: Views From the Pan American Sanitary Bureau*. Washington, DC: Pan American Health Organization; 2001:22–49.
- Casas-Zamora JA, Gwatkin DR. The many dimensions of health equity. *Rev Pan Am Salud Publica*. 2002;11(5-6). Available at: [http://www.scielosp.org/scielo.php?script=sci\\_issuetoc&pid=1020-498920020005&lng=en&nrm=iso](http://www.scielosp.org/scielo.php?script=sci_issuetoc&pid=1020-498920020005&lng=en&nrm=iso). Accessed September 27, 2004.
- EQUIDAD electronic mailing list. Available at: <http://equidad.bvsalud.org>.
- Gwatkin DR. Reducing health inequalities in developing countries. In: Detels R, McEwen J, Beaglehole R, Tanaka H, eds. *Oxford Textbook of Public Health*. Vol 3. 4th ed. New York, NY: Oxford University Press; 2002:1791–1810.
- Wagstaff A. Poverty and health sector inequalities. *Bull World Health Organ*. 2002;80:97–105.
- Round I country reports on health, nutrition, population conditions among poor and better-off in 45 countries. 2000. Available at: <http://www1.worldbank.org/prem/poverty/health/data/round1.htm>. Accessed September 27, 2004.
- Round II Country Reports on Health, Nutrition, Population Conditions among Poor and Better-Off in 56 Countries. 2004. Available at: <http://www1.worldbank.org/prem/poverty/health/data/round2.htm>. Accessed September 27, 2004.
- Evans T, Whitehead M, Diderichsen F, Bhuiya A, Wirth M, eds. *Challenging Inequities in Health: From Ethics to Action*. New York, NY: Oxford University Press; 2001.
- McCoy D, Bambas L, Acurio D, et al. Global equity gauge alliance: reflections on early experiences. *J Health Popul Nutr*. 2003;21:273–287.
- EQUINET Africa. Available at: <http://www.equinetfrica.org>. Accessed September 27, 2004.
- Toronto declaration on equity in health. *Pan Am J Public Health*. 2002;12:465–467.
- International Society for Equity in Health, Third International Conference. Conference program. 2004. Available at: <http://www.iseqh.org/documents/Agenda-DETAILEDJune12004-Printer.doc>. Accessed September 27, 2004.
- World Health Organization. Health-for-all policy for the 21st century. Available at: [http://www3.who.int/whosis/discussion\\_papers/pdf/paper43.pdf](http://www3.who.int/whosis/discussion_papers/pdf/paper43.pdf). Accessed September 27, 2004.
- World Health Organization. *Bull World Health Organ*. 2000;78(1).
- Sandana R, Tandon A, Murray CJL, et al. Describing population health in six domains: comparable results from 66 household surveys. March 2002. Available at: [http://www3.who.int/whosis/discussion\\_papers/pdf/paper43.pdf](http://www3.who.int/whosis/discussion_papers/pdf/paper43.pdf). Accessed September 27, 2004.
- Lee JW. Global health improvement and WHO: shaping the future. *Lancet*. 2003;362:2083–2088.
- Vega J, Irwin A. Tackling health inequalities: new approaches in public policy. *Bull World Health Organ*. 2004;82:432–433.
- World Health Organization. Fifty-Seventh World Health Assembly, Address by the Director-General. May 17, 2004. Available at: <http://www.who.int/dg/lee/speeches/2004/wha57/en>. Accessed September 27, 2004.
- Sen AK. *Development as Freedom*. New York, NY: Knopf; 1999.
- Hardt M, Negri A. *Empire*. Cambridge, Mass: Harvard University Press; 2001.

# Asleep at the Switch: Local Public Health and Chronic Disease

Local health departments generally do a good job of monitoring and controlling conditions that killed people in the United States 100 years ago. Yet noncommunicable diseases, which accounted for less than 20% of US deaths in 1900,<sup>1</sup> now account for about 80% of deaths.<sup>2</sup> Our local public health infrastructure has not kept pace with this transition.

Health departments must continue to handle traditional public health priorities as well as emerging infectious diseases. They must also increasingly address terrorism detection, preparedness, and response. But it is even more urgent that they adjust to the epidemiological transition from communicable to chronic disease. All too many are asleep at the switch.

There are many reasons for the relative lack of local public health activity in chronic disease prevention and control. For the public, the fact that there is no urgent crisis with a short-term solution is key. For public health, the foremost reason may be the assumption that chronic diseases are not meaningfully amenable to public health action and that traditional public health strategies are not relevant to chronic diseases. In fact, these strategies hold enormous promise.

## TRADITIONAL PUBLIC HEALTH STRATEGIES CAN HELP CONTROL CHRONIC DISEASE

Traditional public health activities include (1) surveillance, including mandatory reporting; (2) environmental modifications, such as those used to control water- and mosquito-borne illnesses; (3) regulation; (4) clinical care, including direct provision of care and monitoring of care

given by others; (5) outbreak detection, investigation, and control; (6) case management and contact tracing; (7) immunization; and (8) health education.

## Surveillance

Surveillance is essential in monitoring and controlling disease; the lack of robust local systems for chronic disease surveillance is a critical deterrent to progress.<sup>3</sup> Local systems could delineate and galvanize local action on the epidemics of obesity and diabetes; the health care system's failure to effectively treat most cardiovascular disease and diabetes; and the minimal levels of pharmacological and counseling help provided to the majority of smokers who want to quit.

State cancer registries now conduct surveillance for incidence and treatment as well as for deaths. These registries have potentially significant implications for prevention; they include all patients diagnosed and can bridge the gap between surveillance and clinical care, tracking stage at diagnosis and treatment effectiveness communitywide. Los Angeles County began a local telephone survey in 1997 and has found it to be an effective planning tool.<sup>4</sup> New York City has conducted local telephone surveys since 2002, generating information on smoking, mental illness, alcohol use, diabetes, obesity, and more (<http://www.nyc.gov/html/doh/html/data/data.html>) and leading to the establishment of neighborhood health department offices in the city's sickest communities.

New York City is also conducting a health and nutrition examination survey modeled after the National Health and Nutrition Examination Survey (NHANES; <http://www.nyc.gov/>

health/nychanes). This may be the first community survey of its kind in the United States, and it will provide definitive information on the local prevalence and level of control of hypertension, diabetes, hypercholesterolemia, depression, and other health conditions. The Centers for Disease Control and Prevention's (CDC's) recently released Selected Metropolitan/Micropolitan Area Risk Trends (SMART) system provides some information for local action (<http://apps.nccd.cdc.gov/brfss-smart/index.asp>). Medical informatics also holds promise. In theory, a local health officer could and should know how many people locally have high blood pressure, high cholesterol, and diabetes; how many of them are adequately controlling their condition; and which patient and provider characteristics are associated with poor control.

Disease registries (analogous to those used to track treatment, progress, and outcomes of patients with tuberculosis) have enormous potential to improve chronic disease management. Facilities with registries improve management of patients with diabetes, follow-up of people with abnormal screening tests for cancer, and more. These results could be aggregated and analyzed on a population basis or tracked individually if electronic medical records were used.

## Environmental Interventions

Changes in housing, water, and the physical environment were essential to controlling infectious disease. Today, modifications of the physical environment to promote physical activity, or of the food environment to address obesity, are essential for chronic disease prevention and control.



## Regulation

Modern public health practice started with regulation of food and water. Potential roles for local regulation in chronic disease control include establishment of smoke-free workplaces; local requirements on food pricing, advertising, content, and labeling; regulations to facilitate physical activity, including point-of-service reminders at elevators and safe, accessible stairwells; tobacco and alcohol taxation and advertising and sales restrictions; and regulations to ensure a minimal level of clinical preventive services.

## Clinical Care

Traditional public health has moved increasingly from direct provision of clinical care to assurance of care. Similarly, for chronic disease prevention and control, direct provision of care (e.g., smoking cessation clinics, smoking cessation medication distribution programs, and cancer screening) can have strategic importance but will be secondary to broader health care quality issues. Secondary prevention of complications of many chronic diseases (e.g., hypertension, hyperlipidemia, and diabetes) can be achieved with currently available medical interventions; however, most patients with high blood pressure, high cholesterol, and diabetes do not have these conditions adequately controlled.<sup>5-7</sup> Health departments must promote and monitor treatment standards in the local health care system.

## Outbreak Detection, Investigation, and Control

Outbreaks of noncommunicable disease are slower, larger, and more complex than outbreaks of communicable disease—

but they are still outbreaks. Traditional outbreak control methods—case definition, risk factor analysis, and risk factor control—are relevant and are underused for conditions such as diabetes, obesity, and death from cervical cancer.

## Case Management and Contact Tracing

Case management of noncommunicable disease does not have the same societal imperative (prevention of transmission) that case management for infectious disease does. But improved prevention and management of chronic disease also has societal benefits, including reductions in health care costs and increases in productivity. Disease management approaches improve outcomes for people with diabetes, asthma, and other chronic conditions. Contacts of individuals with chronic disease are not generally at risk of contracting the disease directly, but they may be at risk because of shared genetic or environmental factors. Family members of people with colon cancer, breast cancer, and diabetes are at higher risk for these conditions and may benefit from outreach and preventive interventions; the clinical medical system is limited in its capacity to ensure this type of contact tracing and follow-up.

## Immunization

While we do not yet have vaccines against most chronic diseases, influenza vaccination is associated with reductions in cardiovascular mortality.<sup>8</sup> Hepatitis B vaccination will reduce liver disease and hepatoma. Human papilloma virus vaccines are promising candidates for prevention of cervical cancer. Effective education can “immunize” young adults against tobacco

dependency and tobacco company marketing,<sup>9</sup> an approach that could also prevent other chronic diseases.

## Health Education

Local health departments can fulfill a unique role as an honest broker and policy setter by providing unbiased information to legislators, policymakers, and the public. The cacophony of recommendations on personal health topics such as exercise, diet, and depression is reminiscent of the large number of scientifically unfounded recommendations on communicable disease prevention and control that circulated in the late 1800s. This is at least in part a reflection of the failure of federal, state, and local agencies to address these issues clearly, convincingly, and consistently.

## MORE FUNDING NEEDED FOR CHRONIC DISEASE CONTROL

There is a misperception that public health is synonymous with infectious disease control. In fact, public health activities have long encompassed noncommunicable disease prevention programs, such as motor vehicle and workplace safety, maternal and infant health, lead poisoning prevention, water fluoridation, and tobacco control. Just as decades ago public health prevented disease by improving air and food safety, today we must address pathogenic tobacco advertising strategies and toxic food policies. As shown by the reduction in tobacco use and in cardiovascular mortality, success in chronic disease prevention and control is possible.<sup>10,11</sup> But success is not assured, and it is unlikely to be achieved without a substantial increase in funding.

In 2001, the New York City Department of Health and Mental Hygiene received no federal government and minimal state government grant support for prevention and control of cardiovascular disease, diabetes and cancer, and less than 10 cents per capita for tobacco control. It also spent less than 1.5% of its city funds on monitoring and control of these conditions. And although the amount of federal funding received for infectious disease control was far from sufficient, it was more than 100 times the amount received for chronic disease and tobacco control (about \$10 per capita). “Steps to a Healthier US” (<http://www.healthierus.gov/steps>) federal grants are funding surveillance and chronic disease control in some local health departments for the first time. Funding for local public health programs is inversely proportional to the risk of death from the very conditions these programs address.

Chronic disease will not be controlled in weeks or months. This is all the more reason to begin control programs now. There is an urgent need for both structural interventions that change the context for health (e.g., smoke-free workplaces; increased taxes on tobacco and alcohol; sidewalks and parks to promote increased physical activity) and improved clinical management (e.g., preventing cardiovascular disease complications through improved treatment of diabetes, high cholesterol, and hypertension; preventing or detecting cancer; and supporting smoking cessation). The costs of chronic disease—related care are increasing rapidly, and all of society has both an interest in and a responsibility for improving the prevention and control of such

diseases. The federal government must greatly increase its support for local activities in these areas. It is time for state and local health departments to wake up to the challenge. ■

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### References

1. Linder FE, Grove RD. *Vital Statistics Rates in the United States, 1900–1940*. Washington, DC: National Office of Vital Statistics; 1947.
2. Mokdad AH, Marks JS, Stroup DF, Gerberding JL. Actual causes of death in the United States, 2000. *JAMA*. 2004;291:1238–1245.
3. Brownson RC, Bright FS. Chronic disease control in public health practice: looking back and moving forward. *Public Health Rep*. 2004;119:230–238.
4. Simon PA, Wold CM, Cousineau MR, Fielding JE. Meeting the data needs of a local health department: the Los Angeles County Health Survey. *Am J Public Health*. 2001;91:1950–1952.
5. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988–2000. *JAMA*. 2003;290:199–206.
6. Ford ES, Mokdad AH, Giles WH, Mensah GA. Serum total cholesterol concentrations and awareness, treatment, and control of hypercholesterolemia among US adults: findings from the National Health and Nutrition Examination Survey, 1999 to 2000. *Circulation*. 2003;107:2185–2189.
7. Saydah SH, Fradkin J, Cowie CC. Poor control of risk factors for vascular disease among adults with previously diagnosed diabetes. *JAMA*. 2004;291:335–342.
8. Nichol KL, Nordin J, Mullooly J, Lask R, Fillbrandt K, Iwane M. Influenza vaccination and reduction in hospitalizations for cardiac disease and stroke among the elderly. *N Engl J Med*. 2003;348:1322–1332.
9. Farrelly MC, Heaton CG, Davis KC, Messeri P, Hersey JC, Haviland ML. Getting to the truth: evaluating national tobacco countermarketing campaigns [published correction appears in: *Am J Public Health*. 2003;93:703]. *Am J Public Health*. 2002;92:901–907.
10. Fichtenberg CM, Glantz SA. Association of the California tobacco control program with declines in cigarette consumption and mortality from heart disease. *N Engl J Med*. 2000;343:1772–1777.
11. Achievements in public health, 1900–1999: decline in deaths from heart disease and stroke—United States, 1900–1999. *MMWR Morb Mortal Wkly Rep*. 1999;48:649–656.

# The Latina Paradox: An Opportunity for Restructuring Prenatal Care Delivery

Michael S. McGlade, PhD, Somnath Saha, MD, MPH, and Marie E. Dahlstrom, MA

Latina mothers in the United States enjoy surprisingly favorable birth outcomes despite their social disadvantages. This “Latina paradox” is particularly evident among Mexican-born women.

The social and cultural factors that contribute to this paradox are maintained by community networks—informal systems of prenatal care that are composed of family, friends, community members, and lay health workers. This informal system confers protective factors that provide a behavioral context for healthy births. US-born Latinas are losing this protection, although it could be maintained with the support of community-based informal care systems.

We recommend steps to harness the benefits of informal systems of prenatal care in Latino communities to meet the increasing needs of pregnant Latina women. (*Am J Public Health*. 2004; 94:2062–2065)

**ALTHOUGH RATES OF EARLY** prenatal care in the United States rose steadily during the latter part of the 20th century, significant racial/ethnic disparities have persisted.<sup>1</sup> To address this problem, Oregon participants in the National Public Health Leadership Institute (NPHLI) launched a 2-year project in the fall of 2001 that focused on access to prenatal care among Latina women. Data from the 2000 Pregnancy Risk Assessment and Monitoring System Survey showed that only 57% of Mexican-born women who lived in Oregon received first-trimester prenatal care compared with 78% of non-Latina women (Suzanne Yusem, Office of Family Health, Oregon Department of Human Services, written communication, August 2003).

To generate innovative solutions to this disparity, the Oregon NPHLI team held a Latina Prenatal Summit in September 2003 that brought together a diverse group of community leaders and prenatal care experts. In preparation for the summit, a planning committee held a series of meetings to discuss options for improving prenatal care among pregnant Latinas. A recurring theme in these discussions was the “Latina paradox”—the well-documented observation that despite socioeconomic disadvantage, Latinas in the United States have birth outcomes comparable to those of White women<sup>2–6</sup>—and its implications for restructuring prenatal care systems to improve their ef-

fectiveness. These discussions resulted in the development of a conceptual framework that described an “informal system” of prenatal care delivery within Latino communities. This system consists of family, friends, community members, and lay health workers who collectively form a social support system that maintains the protective social and cultural factors responsible for the better-than-expected birth outcomes. In this commentary, we describe the conceptual framework and make recommendations for tapping the informal system of prenatal care to improve prenatal care access and, in turn, birth outcomes in Latino communities.

## THE LATINA PARADOX

Epidemiological research has shown that despite their parents’ socioeconomic disadvantages, Latino infants experience low-birthweight and mortality rates that are generally lower than the national averages.<sup>2–6</sup> Overall, the US Latino population had a low-birthweight incidence of 6.5% in 2002, while the incidence was 6.9% among non-Latino Whites and 13.4% among African Americans.<sup>7</sup> At first this observation was believed to be artifactual and was attributed to the effects of migration or other biases in data collection.<sup>8,9</sup> However, as additional studies reported the same pattern, favorable birth outcomes became accepted as part of the larger Latino paradox of health, which is now known to

also include all-cause mortality rates among most age groups, with the exception of young adult males.<sup>10, 11</sup> Favorable birth outcomes among Latinos are particularly striking because of the strong and consistent association between socioeconomic status and birth outcomes<sup>12</sup> and because Latinos as a group are among the most socioeconomically disadvantaged racial/ethnic populations in the United States.<sup>13</sup> It should be noted, however, that the term Latino refers to a diverse mix of peoples who have roots in the primarily Spanish-speaking regions of North and South America. Favorable birth outcomes are not uniform across these populations. In general, Mexican American and other women of Central American origin have the strongest advantages in birth outcomes, while Puerto Rican women have a less favorable profile.<sup>2,14</sup>

## EXPLAINING THE PARADOX

Proposed explanations for the Latina paradox can be classified, with some overlap, as migratory-selection processes, cultural protective factors, and social support.

The healthy-migrant theory posits that it is generally the healthiest Latinas who immigrate to the United States and that this health advantage is responsible for their relatively positive birth (and other health) outcomes. Landale et al. have provided some evidence that supports the healthy-migrant theory.<sup>15</sup> After they controlled for a variety of



confounding factors, Landale et al. found that infant mortality among Puerto Ricans was lower among recent migrants to the United States than among nonmigrant families in Puerto Rico. This differential between migrants and nonmigrants can be seen in statistics from other countries as well. Overall, Latinas in the United States have a low-birthweight incidence of 6.5%,<sup>7</sup> while the incidence is 9% in Mexico; 10% in Peru; and 13% in Guatemala, Nicaragua, and El Salvador.<sup>16</sup> Although these statistics may indicate that emigrating women are healthier than their compatriots, they also may reflect the general environmental and economic disadvantages of mothers in Latin American countries compared with mothers in the United States.

Most studies that have examined cultural protective factors have focused on the largest subgroup of the Latino population, Mexican Americans. Protective factors include a strong cultural support for maternity, healthy traditional dietary practices, and the norm of selfless devotion to the maternal role (*marianismo*).<sup>17</sup> These protective factors are believed to provide a healthy normative and behavioral context for maternity, and they enable immigrant mothers to resist adopting the negative risk behaviors of the new host society, particularly those related to smoking, alcohol abuse, and diet.<sup>18–20</sup>

Cultural protective factors are interrelated with the role of social support networks, i.e., informal systems of health care.<sup>21</sup> The informal systems of prenatal care that support maternity among Latina mothers are diverse, but many share at least several of the following components. First, there is a strong tradition of intergenera-

tional knowledge transfer through which healthy behaviors are passed down from one generation of mothers to the next. Second, many mothers benefit from the support of other family figures, particularly sisters and extended family members.<sup>22</sup> Third, Mexican women often take responsibility for the health needs of those beyond their nuclear households,<sup>23</sup> and supportive Mexican fathers also play a positive role in birth outcomes, although their effect is generally smaller.<sup>24</sup>

Friends and neighbors also may provide informal prenatal and postpartum support to pregnant Latinas. The tradition of women helping other women in the community is very strong in Latin America, and the high value placed on warm interpersonal relationships (*personalismo*) of Latin American societies persists to the present.<sup>25</sup> Finally, *parteras*—lay midwives—who have various levels of training have always been an important part of the delivery process in Latin America.

The direct mechanisms through which family and social support contribute to positive birth outcomes are not entirely clear. Such support may mitigate the adverse effects of poverty through the pooling of resources.<sup>26</sup> It also may have a stress-buffering effect that improves the psychological and physiological milieu in which pregnancies occur.<sup>27</sup> Whatever the mechanisms, mothers who have this support generally experience better birth outcomes than those who do not.<sup>21</sup>

## ACCULTURATION AND THE LATINA PARADOX

Since birth outcomes among Latina women are generally positive, should poor access to formal (clinical) prenatal care be consid-

ered a problem? Two lines of evidence show that it should be. First, prenatal care helps Latina mothers: in a study of 1.1 million births to Mexican American women, infant mortality was 2.5 times greater among women who did not receive prenatal care compared with those who did.<sup>28</sup>

Second, the social and cultural protective factors responsible for positive birth outcomes among immigrant Latin American women appear to erode in subsequent generations. For example, a study of more than 22 000 Mexican American births in Illinois showed that US-born mothers experienced worse birth outcomes than immigrant women from Mexico.<sup>14</sup> In low-income census tracts, Mexican-born mothers had low-birthweight rates of 3%, while US-born Mexican American mothers had low-birthweight rates of 14%. Notably, maternal age, education, and trimester of prenatal care initiation were associated with the prevalence of low birthweight among US-born Mexican American mothers but not among foreign-born mothers. This finding supports the hypothesis that social and cultural protective factors—maintained in immigrant communities by an informal system of care—are a substitute, at least in part, for formal prenatal care.

This loss of advantage in birth outcomes is caused in part by the process of acculturation to the norms of mainstream American society. In an analysis of data from the Hispanic Health and Nutrition Examination Survey, higher levels of acculturation among Mexican American women, as measured by language preference, ethnic identification, and nativity status, were associated with higher rates of low birthweight.<sup>29</sup> Cobas et al. reanalyzed these data with structural equation

modeling that showed part of the association between acculturation and low birthweight was caused by smoking and nutrition, which reflects the fact that Mexican American women take up more unhealthy behaviors as they assimilate from a Mexican cultural orientation to a US cultural orientation.<sup>30</sup> However, even after they controlled for diet and smoking, acculturation was a still significant predictor of low birthweight, which indicates that other protective health behaviors or social support—factors that were not modeled in the aforementioned studies—may contribute to the Latina paradox.

A study of more than 1 million Southwest-US Mexican American infants showed that both lifestyle factors and social support are important variables in the Latina paradox. In this study, infant mortality ranged from 4.3 in counties that had high proportions of Mexican births to 5.5 in counties that had low proportions of Mexican births.<sup>31</sup> However, this association in a community context was limited to US-born Mexican American mothers, whose rates ranged from 7.0 in low-concentration counties to 4.4 in high-concentration counties. Interestingly, among births to Mexican-born mothers, there was no association between community context and mortality. This suggests that continued exposure to a Mexican cultural orientation may support and reinforce healthy behaviors that Mexican American women, particularly those born in the United States, may otherwise lose through acculturation.<sup>31</sup> Another implication is that areas that have a high concentration of Mexican Americans may provide better access to culturally appropriate prenatal care, e.g., care provided by bilingual bicultural

clinicians, community health workers, or both.

In summary, there is direct and indirect evidence that health behaviors associated with both Mexican cultural norms and the social support systems maintained in Mexican American communities may contribute to paradoxically low rates of low birthweight within these communities. However, this support tends to erode with increases in acculturation. While this erosion bodes poorly for future birth outcomes of the rapidly acculturating Latin American women who currently reside in the United States, it also suggests a solution at the programmatic and public policy level.

## A POLICY PRESCRIPTION

Latinos in the United States are among the most medically underserved populations in the industrialized world. In 2000, 40% of Latino adults aged 19 to 64 years lacked any form of health insurance.<sup>32</sup> In today's political and economic climate, the prospects for expanding insurance coverage seem dim, particularly in light of the crisis many state Medicaid programs are facing because of budget shortfalls. Lack of access to prenatal care threatens the health of an entire generation of Latinos, who have the highest birth rate among racial/ethnic groups in the United States.<sup>7</sup>

We hypothesize that maintaining the positive birth outcomes experienced by first-generation Latinas within existing financial resource limitations can be achieved by harnessing the benefits of the health-promoting cultural and social milieu within Latino communities, that is, the informal systems of prenatal care. While this system of care can thrive in the more collective,

community-based Latin American context, it will likely deteriorate in the United States if it is not supported.

This is not to discount the value of formal (clinical) prenatal care. The benefits of prenatal care services are incontrovertible and apply to both non-Latinas and Latinas, particularly nonimmigrants.<sup>28</sup> Informal systems of care, however, can complement formal systems. We suggest an approach that merges elements of these 2 systems of care by expanding the roles of individuals who participate to some degree in both systems. These individuals include community health workers (*promotoras*), lay midwives (*parteras*), and caregivers who provide support during labor and the postpartum period (*doulas*). These lay practitioners, who are typically members of the communities in which they work and who have had formal or informal training in maternal and child health, can serve several integrating functions. First, they can provide *outreach* to ensure pregnant women are aware of and access formal prenatal care services. Second, they can *deputize* members of the community, particularly older immigrant women, whose experience and knowledge are integral to maintaining the benefits that appear to be lost with acculturation. Deputizing these women validates their beliefs and practices, which may be looked down upon by more acculturated women, and it empowers the women as community leaders, which may help preserve within the community the traditional Latino cultural context that appears to confer positive health effects. Third, lay practitioners can *organize* community mem-

bers to provide the type of social support system for pregnant mothers that exists in most areas of Latin America but often disintegrates in the United States. Finally, the *personal contact* provided by community-based lay practitioners serves some of the functions and roles of the informal system of care.

La Clinica del Cariño in Hood River County, Oregon, provides one example of the potential for integrating formal and informal systems of care. This clinic, which serves a predominantly rural Latino population, including many seasonal farm workers, began its Perinatal Health Promoter Program in 1987. In this program, *promotoras* are recruited from the community served by the clinic and are trained to both communicate the need for and to provide basic clinical prenatal services. The *promotoras* work in the communities and in the clinic. Their knowledge of, and integration within, the communities ensures that they are aware of nearly all pregnancies that occur within their communities. Nearly all pregnant women are or eventually become aware of the *promotoras*, who then become case managers for these women by providing prenatal counseling and by facilitating access to the clinic, which is a federally qualified health center. In addition to prenatal services, the *promotoras* provide early postpartum care and family planning services. They work closely with physicians in the clinic and discuss all cases, particularly high-risk pregnancies. Records from the clinic have shown that more than 85% of Latina mothers who accessed services at the clinic received prenatal care within the first trimester of pregnancy (Helen Bel-

lanca, MD, oral communication, August 2003).

## CONCLUSIONS

The Latina paradox can partly be explained by the functioning of informal prenatal systems of care that confer culturally and socially mediated benefits; however, these benefits disintegrate as Latinas acculturate within the United States. Supporting the functions of these informal systems of care and integrating them with formal prenatal services through the expanded use of lay health practitioners has the potential to both improve prenatal care access and improve birth outcomes at a relatively low cost. ■

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M.S. McGlade originated the framework of the article and was the primary author. S. Saha contributed to refining the framework and to writing/revising the article. M.E. Dahlstrom originated, organized, and implemented the Latina Prenatal Summit and contributed to refining the framework.

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## References

1. *Health, United States, 2003*. Hyattsville, Md: National Center for Health Statistics; 2003.
2. Fuentes-Afflick E, Lurie P. Low birthweight and Latino ethnicity. Examining the epidemiologic paradox. *Arch Pediatr Adolesc Med*. 1997;151:665–674.
3. Fuentes-Afflick E, Hessel NA, Perez-Stable EJ. Testing the epidemiologic paradox of low birthweight in Latinos. *Arch Pediatr Adolesc Med*. 1999;153:147–153.
4. Hessel NA, Fuentes-Afflick E. The perinatal advantage of Mexican-origin Latina women. *Ann Epidemiol*. 2000;10:516–523.
5. James SA. Racial and ethnic differences in infant mortality and low birthweight. A psychosocial critique. *Ann Epidemiol*. 1993;3:130–136.
6. Scribner RA. Infant mortality among Hispanics: the epidemiological paradox. *JAMA*. 1991;265:2065–2066.
7. Martin JA, Hamilton BE, Sutton PD, Ventura SJ, Menacker F, Munson ML. *National Vital Statistics Report. Births: Final Data for 2002*. Vol. 52, No. 10. Hyattsville, Md: National Center for Health Statistics. Available at: [http://www.cdc.gov/nchs/data/nvsr/nvsr52/nvsr52\\_10.pdf](http://www.cdc.gov/nchs/data/nvsr/nvsr52/nvsr52_10.pdf). Accessed February 27, 2004.
8. Selby ML, Lee ES, Tuttle DM, Loe HD Jr. Validity of the Spanish surname infant mortality rate as a health status indicator for the Mexican American population. *Am J Public Health*. 1984;74:998–1002.
9. Powell-Griner E, Streck D. A closer examination of neonatal mortality rates among the Texas Spanish surname population. *Am J Public Health*. 1982;72:993–999.
10. Franzini L, Ribble JC, Keddie AM. Understanding the Hispanic paradox. *Ethn Dis*. 2001;11:496–518.
11. Markides KS, Coreil J. The health of Hispanics in the southwestern United States: an epidemiologic paradox. *Public Health Rep*. 1986;101:253–265.
12. Singh GK, Yu SM. Infant mortality in the United States: trends, differences, and projections, 1950 through 2010. *Am J Public Health*. 1995;85:957–964.
13. Proctor BD, Dalaker J. *Poverty in the United States: 2002*. Washington, DC: US Government Printing Office; 2003.
14. Collins JW Jr, Shay DK. Prevalence of low birth weight among Hispanic infants with United States-born and foreign-born mothers: the effect of urban poverty. *Am J Epidemiol*. 1994;139:184–192.
15. Landale NS, Oropesa RS, Gorman BK. Migration and infant death: assimilation or selective migration among Puerto Ricans? *Am Soc Rev*. 2000;65:888–909.
16. UNICEF. Information by country: Latin America and Caribbean. Available at <http://www.unicef.org/infobycountry/latinamerica.htm>. Accessed February 27, 2004.
17. Stevens EP. Marianismo: the other face of machismo in Latin America. In: Pescatell A, ed. *Female and Male in Latin America*. Pittsburgh, Pa: University of Pittsburgh Press; 1973.
18. Marcus AC, Crane LA. Smoking behavior among US Latinos: an emerging challenge for public health. *Am J Public Health*. 1985;75:169–172.
19. Black SA, Markides KS. Acculturation and alcohol consumption in Puerto Rican, Cuban-American, and Mexican-American women in the United States. *Am J Public Health*. 1993;83:890–893.
20. Guendelman S, Abrams B. Dietary intake among Mexican-American women: generational differences and a comparison with white non-Hispanic women. *Am J Public Health*. 1995;85:20–25.
21. Weigers ME, Sherraden MS. A critical examination of acculturation: the impact of health behaviors, social support, and economic resources on birth weight among women of Mexican descent. *Int Migr Rev*. 2001;34:804–839.
22. Guendelman S, Chavez G, Christianson R. Fetal deaths in Mexican-American, black, and white non-Hispanic women seeking government-funded prenatal care. *J Community Health*. 1994;19:319–330.
23. Clark L. Maternal responsibility for health in the household. *Health Care Women Int*. 1995;16:43–55.
24. Guendelman S, Malin C, Herr-Harthorn B, Vargas PN. Orientations to motherhood and male partner support among women in Mexico and Mexican-origin women in the United States. *Soc Sci Med*. 2001;52:1805–1813.
25. Clark L. Mexican-origin mothers' experiences using children's health care services. *West J Nurs Res*. 2002;24:159–179.
26. Sherraden MS, Barrera RE. Maternal support and cultural influences among Mexican immigrant mothers. *Fam Soc J Contemp Hum Serv*. 1996;77:298–313.
27. Sherraden MS, Barrera RE. Poverty, family support, and well-being of infants: Mexican immigrant women and childbearing. *J Sociol Soc Welfare*. 1996;23:27–54.
28. Zuvekas A, Wells BL, Lefkowitz B. Mexican American infant mortality rate: implications for public policy. *J Health Care Poor Underserved*. 2000;11:243–257.
29. Scribner R, Dwyer JH. Acculturation and low birthweight among Latinos in the Hispanic HANES. *Am J Public Health*. 1989;79:1263–1267.
30. Cobas JA, Balcasar H, Benin MB, Keith VM, Chong Y. Acculturation and low-birthweight infants among Latino women: a reanalysis of HHANES data with structural equation models. *Am J Public Health*. 1996;86:394–396.
31. Jenny AM, Schoendorf KC, Parker JD. The association between community context and mortality among Mexican-American infants. *Ethn Dis*. 2001;11:722–731.
32. Doty MM. *Hispanic Patients' Double Burden: Lack of Health Insurance and Limited English*. New York, NY: Commonwealth Fund; 2003.

# Self-Care Among Chronically Ill African Americans: Culture, Health Disparities, and Health Insurance Status

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Little is known about the self-care practices of chronically ill African Americans or how lack of access to health care affects self-care. Results from a qualitative interview study of 167 African Americans who had one or more chronic illnesses found that self-care practices were culturally based, and the insured reported more extensive programs of self-care. Those who had some form of health insurance much more frequently reported the influence of physicians and health education programs in self-care regimens than did those who were uninsured. It is concluded that the cultural components of self-care have been underemphasized, and further, that the potential to maximize chronic illness management through self-care strategies is not realized for those who lack access to health care. (*Am J Public Health*. 2004;94:2066–2073)

There is widespread consensus that self-care practices play a critical role in the management of chronic illness, yet we know relatively little about the daily self-care practices of chronically ill African Americans. We know even less about the self-care practices of the 23% of African Americans who have no health insurance.<sup>1</sup> African Americans shoulder dramatically disproportionate rates of disease, unemployment, poverty, and premature death.<sup>2,3</sup> Some indicators show that Black–White disparities have made no sustained improvement since the end of World War II.<sup>4</sup> Given the extent and effects of health disparities for African Americans, it is essential to examine a wide range of health-related factors much more closely in order to identify potential avenues especially appropriate for public health efforts.

The topic of self-care has emerged in the last 30 years as a particular focus of health concerns, but it was not widely viewed as a legitimate area of inquiry among health professionals until the 1980s.<sup>5</sup> Prior to that, an ideology of self-care was limited to use in political and health consumer activist contexts such as the feminist movement's demand for sovereignty of the body. Although indigenous traditions of self-care were present among African Americans from their arrival in the United States,<sup>6</sup> civil rights emancipatory projects focused on protesting the exclusionary, dehumanizing, and stratified nature of medical

care.<sup>7</sup> The concept of a “right” to self-care practice never materialized as a galvanizing issue for African Americans. More visible were battles to reverse entrenched patterns of limited access to health care that resulted in segregated, substandard care, and the need for health professionals to care for protesters injured by police in violent civil rights encounters.<sup>7,8</sup> Nevertheless, among African Americans, the maintenance of indigenous traditions of self-care was essential for survival in the South under Jim Crow and in northern, ghettoized cities.

In this article, we examine the social, cultural, and historical roots of African Americans' approaches to self-care, as well as the daily self-care practices of people in our research. Our aim is to demonstrate cultural influences on self-care approaches and how self-care practices are tied to broader social and cultural themes. Culture constitutes a shared system of meaning, the way that people experience, perceive, and interpret their world. Cultural guidelines pass from one generation to the next through a process in which individuals develop a cultural lens for understanding the world.<sup>9(p3)</sup>

Culture is fluid, ever-changing. People may move between cultures while simultaneously inhabiting a relatively unique coculture (or “subculture”) with its own concepts, rules, and social organization, as do African Americans. Cocultures have their own unique and distinct

features, even as other features overlap with those of the larger culture. Members of a coculture may adhere to values specific to their group, such as particular values associated with self-care practices, while at the same time espousing values of the larger society. We will show how African Americans' self-care practices emerge from strategies for survival and long-term efforts to overcome adversity. By addressing African American self-care practices in their cultural context, we aim to facilitate culturally sensitive public health approaches to the health of African Americans.

## SELF-CARE: HISTORY, THEORY, AND DEFINITIONS

Despite the relatively short history of self-care research, there is no shortage of self-care definitions and concepts. Self-care has emerged as a multidimensional construct with definitions varying as to who is involved, why self-care occurs, what is entailed, and how self-care is accomplished.<sup>10</sup> Dean's definition includes “the range of health and illness behavior undertaken by individuals on behalf of their own health.”<sup>11(p34)</sup> The World Health Organization defines self-care as “the activities individuals, families, and communities undertake with the intention of enhancing health, preventing disease, limiting illness, and restoring health,”<sup>12(p181)</sup> thus recognizing how self-care skills and knowledge stem from lay and professional experience. Vickery and Iverson<sup>13</sup> differentiate between medical and health actions: medical self-care deals with medical problems while health self-care is for health maintenance and improvement. Here, as with many self-care models, the individual is seen as the pivotal decisionmaker, a Western assumption that is erroneously construed as a cross-cultural universal.

In Segall and Goldstein's typology,<sup>14</sup> self-care regulates body processes, prevents disease, and alleviates symptoms and illness. Self-



care practices are thus regulatory, preventive, reactive, and restorative. Orem's model<sup>15</sup> is particularly influential and is the subject of ongoing empirical refinement and investigations of cross-cultural applicability.<sup>16–19</sup> Orem defined self-care as learned behavior that was purposeful, with patterned and sequenced actions, and suggested that individuals acquire the capacity for self-care during childhood, principally in the family, where cultural standards are learned and transmitted intergenerationally.<sup>15(p95)</sup> She observed that self-care develops throughout the life course, and that such behavior varies according to an individual's group affiliation in habits, beliefs, and practices that constitute a cultural way of life.<sup>15(p227)</sup>

Despite the breadth of Orem's approach, the frequent focus of empirical research has been on delineating and measuring discrete components of self-care, characterized by a pervasive tendency to examine questions of self-care from a biomedical perspective that is ahistorical and devoid of context. Such an approach has the effect of obscuring the complexity of culture and its relevance for self-care, and how biomedical precepts about self-care layer onto preexisting cultural approaches. Dill and colleagues<sup>20</sup> observe that definitions of self-care are largely composed of immediate responses to symptom experiences and to preventive or health maintenance activities. They propose a synchronic model that examines the repertoire of self-care behaviors that individuals develop over time, identifies the sources of particular symptom responses in the context of that repertoire, and explores diverse linkages among sources of care. Such an approach is best suited to a qualitative, interpretive approach in which personal meanings, unique linkages among forms of care, and the relationship between self-care behavior and the individual's social context can be identified.<sup>20</sup>

## THE CONTEXT OF AFRICAN AMERICAN SELF-CARE

African Americans have a long tradition of health and healing practices that shape, in part, what they do to care for themselves in the present day. African American traditional medicine can be traced back beyond enslavement in the United States to their native cultures in Africa.<sup>6,21,22</sup> They used magical and herbal

cures from their homelands, but over time they also borrowed additional herbal lore and curative practices from Native Americans and adopted colonial European approaches such as purgatives, bleedings, and preventive measures based on classical humoral pathology, leading to an amalgamated ethnomedical system with many regional variants.<sup>23</sup> This system reflects spiritual power in action and is part of a sacred worldview.<sup>6,24</sup>

Forming a foundation for an ethos of resistance in response to slave owners' emphasis on the soundness of slaves for work and sale, African American spirituality transformed itself, creating an internal world resistant to the power of slave owners.<sup>6(p39)</sup> Moreover, this view of health was relational, linking the well-being of the individual to the health of the larger community and the community to its spiritual life, culminating, as well, in a collective version of self-reliance.<sup>6(p197–198)</sup> Continued reliance on this system was further fostered after emancipation for a variety of reasons: access to mainstream care continued to be denied or was of poor quality because of racism, abuse occurred in the name of scientific experimentation such as the Tuskegee experiments, and resistance to the oppressive tactics of the White mainstream persisted in life and in death.<sup>6,7</sup>

African American self-care practices continue to be affected by the overriding struggle for survival in the face of racism and oppression. Survival and efforts to overcome adversity are core themes in historical documents and academic work,<sup>25–27</sup> as well as in the vast array of cultural expressions, such as music, art, oral, and literary traditions, that symbolize the ongoing impact of and resistance to racial oppression. One specific survival response can be seen in the concept of John Henryism, which reflects the African American cultural ethos of overcoming adversity through self-determination and hard work; this construct has been applied to the study of hypertension.<sup>28,29</sup>

Cultural values and practices among African Americans have been called a "survival arsenal."<sup>30</sup> They are embedded in a variety of African American social institutions, including family structure and organization and the church, and encompass spirituality, social support, and traditional, nonbiomedical health and healing practices. These emphases are central to self-

care practices. The family is the repository of specific cultural beliefs and health practices and is a primary source of cultural meaning.<sup>31</sup> Much has been written on the adaptive value of the African American family, and its great importance has been linked to cultural survival.<sup>32–34</sup> Considerable work has also addressed the imparting of Black consciousness and Afrocentric values in the socialization of children, as well as the development and protective function of Black identity throughout the life course.<sup>35,36</sup>

A large body of work now emphasizes the supportive nature of African American extended kin relationships.<sup>37,38</sup> Dilworth-Anderson<sup>39</sup> observes that the mutual aid system is rooted in a larger African and antebellum context, in which group affiliation was promoted over individuality. Mutual aid has been demonstrated to be an important part of self-care strategies in the management of illness.<sup>40,41</sup> Similarly, spirituality is central: the Black church has developed and sustained itself as a vital institution for the survival and advancement of African Americans, who were refused access to the institutional life of White American society, and who rejected the hypocrisy of White Christianity.<sup>42,43</sup>

The church continues to fulfill many functions of social organization, such as education, social welfare, civic duties, and business enterprises, as well as serving as an outlet for social expression, a vehicle for social protest, and a refuge from racism and discrimination.<sup>44</sup> The church has addressed everyday problems of human existence and survival,<sup>45</sup> providing specific services such as health care, housing, violence prevention, financial aid, child care, services for the elderly, family counseling, hospice, the provision of food and clothing, and educational scholarships.<sup>46,47</sup> No wonder the church has been characterized as a healing resource.<sup>48</sup>

The continued strength of self-care practices is affirmed in an analysis of national survey data among African Americans, in which almost 70% reported that their families used home remedies and 35% reported that they used home remedies themselves.<sup>49</sup> Traditional medicine has continued to be an integral part of self-care not only because of continued poverty, institutional racism, and underuse of biomedicine,<sup>50,51</sup> but also because of its centrality

in African American cultural history and its perceived efficacy and benefits. Self-care practices are grounded in these social and cultural practices.<sup>50</sup> Their cultural values, together with responses to racism and structural inequalities, have shaped how African Americans care for their chronic illnesses.<sup>52</sup> These strategies vary widely, depending on people's past and present social history, the availability of economic resources, and access to health care. However, there has been little work that explores self-care among African Americans.<sup>51,53–56</sup>

We report findings from a study of middle-income and low-income African Americans who had 1 or more chronic illnesses. The main objective of this analysis was to explore the cultural factors that underpinned the development of self-care processes and the use of these practices in daily life after diagnosis of a chronic illness. Our analysis found that while cultural factors were at the root of self-care practices, socioeconomic status and health insurance status were also significant because of their role in shaping access to health care resources.

## METHODS

Findings reported in this article were based on 3 large qualitative studies that examined the same questions about daily management of chronic illness but included people from different age groups and with varying health insurance status. They are combined here to illustrate self-care practices across the life span. Respondents were African Americans aged 21 to 91 years who had 1 or more chronic illnesses. The most common illnesses were diabetes mellitus, asthma, and heart disease or hypertension. A total of 167 African Americans were included in the study. Data collection has been completed in 2 of the 3 studies.

Respondents were recruited from a variety of sources in 2 urban counties in California between June 1994 and August 2002; 28% were recruited from field contacts and contacts in social service agencies, 24% from clinics and home care services, 20% from participant referrals, 19% from flyers, 5% from the Internet, and 4% from religious organizations. The criterion for entry into all studies was the self-reported presence of 1 or more chronic illnesses, and in the first study an additional cri-

terion was complete lack of health insurance. In all 3 studies, we sampled across a range of illness severity, from mild to severe, and a range of people, from those recently diagnosed to those who had had a chronic illness for many years. Our aim was to capture people's retrospective views about the development of self-care measures as they looked back on their illnesses, as well as to gain the perspectives of those who were in the process of discovering what illness management entailed. In addition to African Americans, 3 other racial/ethnic groups were studied: Latinos, Filipino Americans, and Cambodian Americans. They are omitted from this analysis in order to focus on patterns particular to African Americans.

Following key tenets of the in-depth interviewing approach,<sup>57,58</sup> we interviewed all respondents 3 times in a 1-year period. Interviews were conducted by the second and third authors, who were of the same ethnicity as the respondents. Gender-based distrust was not observed among men, all of whom knew they would be interviewed by a woman. Interviews were semistructured with many open-ended questions, lasted for approximately 1 to 2 hours, and focused on respondents' health, experiences with their illnesses, self-care practices (what they did to take care of their health), economic situation, and use of and access to health care. Each interview was tape-recorded and transcribed verbatim. All but 2 respondents were born in the United States, and all were interviewed in English.

The data were divided into low-income and middle-income groups. Krieger and colleagues<sup>59</sup> propose a multifaceted analysis of social class for public health research that includes individual, household, neighborhood, and poverty area levels, and this approach was adapted to this primarily qualitative study. In differentiating persons categorized as low-income from those categorized as middle-income, we examined the following categories: income history, occupation and employment history, medical insurance history and current status, and living arrangements. A further step was taken in separating the data by health insurance status: uninsured, Medicaid, Medicare, or privately insured. Each income group was analyzed separately, and cross-group comparisons were then made. A specific data analytic procedure was followed: core categories that

reappeared in the data repeatedly were identified (e.g., spirituality) and compared with other emergent categories. Codes were developed, generated from meanings in the data. We coded the entire data set for specific topics using QSR Nud\*ist (QSR International Pty Ltd, Melbourne, Australia), a data-sorting software program, resulting in over 100 discrete codes.

A case-by-case narrative analysis was also conducted. Narratives are the stories people tell about their experiences; they provide important insights into their perspectives on those experiences.<sup>60</sup> Narrative analysis, which emphasizes the topics that dominate respondents' reports and the way they are addressed, leads to the identification of themes across the data set. For this analysis, transcripts were sorted into privately insured, Medicaid, Medicare, and uninsured, and then analyzed by group. The coded data and the narrative analysis yielded the same themes regarding self-care and served as a cross-check on each other.

## RESULTS

### Demographics

The 167 respondents ranged in age from 21 to 91 years. Respondents reflected diversity in socioeconomic status, ranging from those who were middle-income, worked as professionals, were home owners, and had medical insurance, to those who were low-income, unemployed, lived in public housing or homeless shelters, and had no medical insurance. Educational levels were comparable across all 3 studies, with between 70% and 75% reporting a high school education or less and 25% reporting at least a college degree. Some of those who were unemployed at the time of the study and were categorized as low-income had formerly been middle-income. Downward mobility was especially found among those who were currently uninsured. All respondents were living in the community at the time of the study. See Table 1 for demographic characteristics and health insurance status.

### The Cultural Basis of Self-Care Practices

Self-care practices among African Americans were found to be culturally based. That is, respondents described idea systems and behavioral practices that were shared by the sample with respect to general issues of self-care

**TABLE 1—Demographics and Health Insurance Status of Respondents (N = 167)**

|                                   | Medicare<br>(n=8) | Medicaid<br>(n=20) | Private and<br>HMO (n=39) | Medicare and<br>Medicaid (n=29) | Uninsured<br>(n=71) |
|-----------------------------------|-------------------|--------------------|---------------------------|---------------------------------|---------------------|
| Age, y                            |                   |                    |                           |                                 |                     |
| Range                             | 52–78             | 25–91              | 21–83                     | 41–89                           | 22–63               |
| Mean                              | 65.50             | 47.05              | 51.31                     | 67.17                           | 45.89               |
| Gender, no. (%)                   |                   |                    |                           |                                 |                     |
| Female                            | 4 (50.0)          | 13 (65.0)          | 26 (66.7)                 | 23 (79.3)                       | 24 (33.8)           |
| Male                              | 4 (50.0)          | 7 (35.0)           | 13 (33.3)                 | 6 (20.7)                        | 47 (66.2)           |
| Marital status, no. (%)           |                   |                    |                           |                                 |                     |
| Married                           | 0 (0)             | 5 (25.0)           | 10 (25.6)                 | 2 (6.9)                         | 4 (5.6)             |
| Unmarried <sup>a</sup>            | 8 (100)           | 15 (75.0)          | 29 (74.4)                 | 27 (93.1)                       | 67 (94.4)           |
| Education, no. (%)                |                   |                    |                           |                                 |                     |
| High school                       | 5 (62.5)          | 15 (75.0)          | 9 (23.1)                  | 22 (78.6)                       | 28 (39.4)           |
| College/postgraduate <sup>b</sup> | 3 (37.5)          | 5 (25.0)           | 30 (76.9)                 | 6 (21.4)                        | 43 (60.6)           |
| Work life, no. (%)                |                   |                    |                           |                                 |                     |
| Currently working                 | 0 (0)             | 5 (25.0)           | 19 (48.7)                 | 0 (0)                           | 19 (26.8)           |
| Unemployed                        | 0 (0)             | 6 (30.0)           | 2 (5.1)                   | 0 (0)                           | 44 (62.0)           |
| Other <sup>c</sup>                | 8 (100)           | 9 (45.0)           | 18 (46.2)                 | 29 (100)                        | 8 (11.3)            |
| Occupation, no. (%)               |                   |                    |                           |                                 |                     |
| Business and professional         | 1 (12.5)          | 3 (15.0)           | 19 (48.7)                 | 5 (17.2)                        | 18 (26.1)           |
| Clerical                          | 1 (12.5)          | 6 (30.0)           | 7 (17.9)                  | 1 (3.4)                         | 10 (14.5)           |
| Skilled and unskilled labor       | 6 (75.0)          | 11 (55.0)          | 12 (30.8)                 | 21 (72.4)                       | 40 (58.0)           |
| Other <sup>d</sup>                | 0 (0)             | 0 (0)              | 1 (2.6)                   | 2 (6.9)                         | 1 (1.4)             |

<sup>a</sup>Widowed, divorced, separated, never married.<sup>b</sup>The majority of uninsured reported some college but no degree.<sup>c</sup>Retired, disabled, student.<sup>d</sup>Student, homemaker, never worked.

for protecting health, preventing illness, and promoting healing and recovery from illness. These cultural approaches to self-care formed the basis from which individuals developed strategies specific to the particular parameters of their illnesses. Three culturally based factors that were central to the development of self-care approaches were (1) spirituality, (2) social

support and advice, and (3) nonbiomedical healing traditions. These cultural factors were present regardless of socioeconomic status and encompassed a diverse range of activities.

### Spirituality and Daily Life

Respondents called attention to underlying spiritual philosophies that were important in

how they proceeded to manage their illnesses. Almost all respondents reported that their belief in God or a higher power helped them to manage their illness. The majority were Protestant (Table 2). Those who did not claim a religious affiliation frequently said they were “spiritual,” but claiming a specific religious affiliation did not necessarily mean a person was a member of a church, and people sometimes claimed the denomination associated with their upbringing, whether they attended church currently or not. Spirituality was usually a part of daily practices. For example, a 45-year-old middle-income university administrator had asthma. She observed, “I start my day with a happy moment. I wake up every morning and before my feet hit the floor I say a prayer.”

Spirituality was also used to ameliorate the effects of structural inequalities. For example, a 35-year-old low-income, unemployed, uninsured man who had asthma described how he used his spirituality to shield himself from discriminatory treatment. He reported, “To maintain one’s spirituality, a high sense of spiritual identity is something you work at daily because there are affronts that you experience daily, so you have to keep yourself healed, physically, spiritually, and mentally on a daily basis, daily practice.”

This integration of mind and body was also expressed by people with a more secular outlook for whom illness management obstacles and strategies reflected racial and class consciousness more than spiritual identity. Both passive and active types of self-care responses to racist encounters were identified. Although this is the subject of another report, we give 1 example of an active response: a 40-year-old man who was employed and uninsured verbally challenged what he believed was racist and class-based bias in the emergency room where he received most of his treatment for hypertension. He described himself as direct, participatory, and demanding in medical interactions: “I had to ask for these things, but if I was White or insured I wouldn’t have to.”

Respondents cited the importance of focusing on inner strength derived from their religion and cultural values in order to effectively manage their illnesses. For example, a 60-year-old middle-income man who had heart disease and kidney disease said, “I need to challenge

**TABLE 2—Religious Affiliations of Respondents (N = 167)**

|                                       | Medicare<br>(n=8) |      | Medicaid<br>(n=20) |      | Private and<br>HMO (n=39) |      | Medicare and<br>Medicaid (n=29) |      | Uninsured<br>(n=71) |      |
|---------------------------------------|-------------------|------|--------------------|------|---------------------------|------|---------------------------------|------|---------------------|------|
|                                       | No.               | %    | No.                | %    | No.                       | %    | No.                             | %    | No.                 | %    |
| Protestant                            | 4                 | 50.0 | 6                  | 33.3 | 18                        | 46.2 | 18                              | 62.1 | 30                  | 42.3 |
| Roman Catholic                        | 1                 | 12.5 | 4                  | 22.2 | 5                         | 12.8 | 5                               | 17.2 | 10                  | 14.1 |
| Other <sup>a</sup>                    | 1                 | 12.5 | 1                  | 5.6  | 12                        | 30.8 | 3                               | 10.3 | 12                  | 16.9 |
| No religious affiliation <sup>b</sup> | 2                 | 25.0 | 7                  | 38.9 | 4                         | 10.3 | 3                               | 10.3 | 19                  | 26.8 |

<sup>a</sup>Includes Christian sects and other religions of the world.<sup>b</sup>Not currently a member of a church.



myself, to force myself to just go on because, well, somewhere in my psyche, I doubt if it's machismo or anything like that. But I have this inner spirit and strength that just makes me just tax myself and in doing that, I get better."

The development of spiritual strength was also used to combat the problems of being uninsured. For example, a 50-year-old unemployed and uninsured man who had a chronic back problem reported, "I just tell the Lord, 'Please, I just hope I don't have no problems because I don't know how in the hell I'm going to pay for it.' It's the mind, it's the mind that heals a lot of things on your body. That's how I deal with it. If I can be strong-minded and not cause my mind to break down and cause it to really bring me down, physically or mentally. [Otherwise] I would probably be dead already."

### Social Support and Advice

Most respondents had kin or close friends who lived in the same geographic locale, and, with few exceptions, they were involved with them on an ongoing, often daily, basis. Emotional support was highly valued and multifaceted, coming from a wide variety of sources. Reports of receiving no support were rare, and almost everyone had someone they could turn to for emotional support. Both men and women reported their mother was a major source of support and advice. For example, a 30-year-old low-income, unemployed, uninsured woman who had asthma, bronchitis, and debilitating gynecologic problems said, "It [self-care] has a lot to do with my mother—not a doctor—my mother telling me certain things. Giving me advice. I have done a lot of the things she has told me."

Such social support went from child to parent as well. For example, a 70-year-old middle-income woman who had hypertension and was on kidney dialysis reported how her daughter was a constant source of reminders about self-care: "She fusses, 'Mama, you shouldn't go out at night,' and 'Mama, you know you're supposed to stay off that leg.'"

Other relatives were also an important source of support. For example, a 43-year-old low-income, unemployed man who was uninsured and who had been recently diagnosed with a heart condition said, "I don't want to tell them [family] because Ma will worry herself to

death. I wouldn't want to worry her about her baby. I'm the baby of the family. I have a couple of brothers I will tell, and the next thing I will hear is, 'Boy, do this, do that.'"

Friends were also important in reinforcing self-care. For example, a 45-year-old middle-income African American woman who was an accountant and had diabetes reported, "A lot of times I'll take my insulin and won't eat. So that drops my blood sugar down. And everyone gets on me about that. 'Did you eat?' 'Nope.' My friend's mom downstairs helps me a lot, she feeds me. She's like my mother. She makes sure that I eat. Sometimes I do forget and sometimes I don't want to eat. I'm not hungry."

### Nonbiomedical Healing Traditions

Respondents of all ages reported the use of nonbiomedical healing traditions in their families as children, and some continued with these traditions in adulthood. For example, a 55-year-old unemployed, uninsured, low-income man who had asthma, allergies, and hepatitis C, said, "She [mother] gave us medicine, a lot of medicines—castor oil, cough syrup, aspirin, and eat right—that was what she used to tell me all the time." A 63-year-old, middle-income, retired woman who had hypertension and diabetes said, "There is kitchen medicine. There have been some things that I have used that have worked well—old family recipes that I'm not willing to divulge."

Some respondents continued to be actively influenced by family members who used traditional medicine. For example, a 23-year-old low-income, unemployed and uninsured man had asthma as well as brittle bones from rickets in childhood. He said, "My mother, she's got over a hundred different teas at the house that you can take for every particular cold through anything. This natural foods grocer, she went in there, and she was like, 'This is heaven.' My mom, she teaches me about the stuff, but it is so much [information] that it is a blur."

Not everyone subscribed to the use of traditional medicine at the time they were interviewed, however. While some were noncommittal, a few reported negative experiences. For example, a 32-year-old low-income woman who was employed as a home health aide commented on her grandmother's efforts

to provide a remedy for her asthmatic child: "Don't try to give me this old folks' remedies. Like my grandmother tried to do that and she ended up harming my son more than helping."

Among people aged younger than 50 years, there was also considerable interest in other types of complementary medicine, regardless of income level. For example, a 40-year-old middle-income loan specialist who had asthma reported, "I think I am in love with the holistic type of healing, even though I half-heartedly pursue it. But I like it better [than biomedicine], the thought of it." When a local low-income clinic began offering free acupuncture, uninsured respondents reported they felt positive about it.

### Health Insurance and the Influence of Biomedical Perspectives

African Americans integrated basic cultural approaches to self-care with the development of specific biomedical self-care approaches and applied them to their chronic illnesses. (In anthropology, this process is subsumed within the concept of medical pluralism.) But the development and maintenance of self-care strategies also hinged on socioeconomic and health insurance statuses. Those who had some form of ongoing health insurance much more frequently reported the influence of physicians and health education programs in self-care regimens than did those who were uninsured.

The insured reported more extensive, biomedically informed programs of self-care such as diet and exercise regimens. The case of a retired 48-year-old man who had diabetes and multiple sclerosis and who was insured through a health maintenance organization (HMO) illustrates how cultural approaches to self-care could be integrated with biomedical approaches when adequate private insurance coverage was in place. He described his overall approach to his illnesses: "If I let it get me down, confine me, physically and mentally, then I ain't gonna be worth nothing. But if I can stay positive about it, I'm doing good. I'm definitely praying about it, constantly. I find praying about it helps me focus on the positive. Even if I fail, I'm still going to try. So if I go out on my bike and I scrub, I'm not gonna give it up." He saw his physician frequently: "I talk with him and read the pamphlets he gave me for the diabetes."

His physician also referred him to courses on diabetes run by the HMO: "They signed me up to the diabetes clinic, where I'm required to go every so many days, and they give you nutrition, diets and whatever. It's a packet that you have to sign and agree to. I just have to get out and go. You know, I gave my word, I told 'em I'm coming." He has been to the hospital library twice to read about diabetes. He gets social support and advice from friends and relatives: "A lot of people give me advice. And my thing is, I'm going to do what the doctor says. The doctor says for me, specifically, this is gonna work."

Diagnosed with diabetes 1 year earlier, he was recently hospitalized, at which time it was decided he needed to be on insulin. He was determined not to undergo another hospitalization: "I don't want to go back in there. So you work at it. Just tell yourself, 'No, I ain't going back.' Right now I'm doing proactive as much as I can. I exercise. I walk and I lift weights. I took those two 8-pound weights. I carry them when I walk, which is 16 pounds. It helps, you know, exercise helps keep the sugar down. So I just try to do that and stay focused on that. Because sometimes you don't feel like doing it." He watched his diet carefully, and his family was very supportive: "My wife says she wants to eat what I eat. I figure why should they [wife and children] have to suffer? But she wants to. They're [meals] low sugar, low sodium, more vegetables, less starch, very small portions."

Those who had regular medical care reported how physicians helped to tailor specific approaches to a chronic illness. For example, a 33-year-old low-income woman who was unemployed and received Medicaid had diabetes and high blood pressure. She reported how her physician had educated her about necessary changes to her diet when she was diagnosed with these conditions, which subsequently affected her food shopping and eating habits: "When I grocery shop, I have to watch what I buy, and I have 3 kids, so it's kind of hard because I can't put them on a diet. They need sugar and stuff like that. So I have to really, really help myself and discipline myself . . . not to eat it, or whatever. So since I was diagnosed, I have eaten healthier."

Similarly, a 35-year-old low-income woman, a homemaker who received Medicaid and had

asthma, reported following her physician's advice: "I don't drink, I don't smoke. If I do drink, it's apple cider. I can't walk the hills unless I sit down, I still get short of breath. Two months ago I had so much weight, and my doctor advised me to lose weight. I went from 230 to 202. I changed my diet and it is better. Now with my diet consisting mostly of fish and chicken and salad and stuff, I feel better. The weight problem was part of me not walking. So now that I've lost that, it's a little easier for me to walk places."

However, those who were uninsured reported less continuity in medical input about self-care. One reason they gave was the discrimination they experienced in their efforts to receive basic care. A 42-year-old woman said, "You are treated different when you don't have insurance. Sometimes I think you are treated different based on your color, on your race. They were borderline rude [in a clinic]. Sort of an indifference."

Uninsured people were asked to discuss their self-care practices in detail. A 45-year-old man who had been uninsured for 8 years had had asthma since childhood. He relied on borrowing inhalers from friends to manage his asthma. Summing up his situation he said, "I probably suffer more than I should because I don't have the money to pay for medication." Unemployed after a long career in the travel industry, his daily self-care emphasized trying to avoid pollen and dust. As a child, he and his siblings took a lot of castor oil, Three Sixes (patent cough medicine) for colds, and hot toddies for the flu. He didn't recall any special remedies for asthma.

He assumed there were other approaches for treating asthma but he had been frustrated in his attempts to learn more: "I'm sure there are different ways to treat asthma other than an inhaler. But through the years I don't really know of any other remedies to try and control it myself." He explained what he meant: "I've had a lot of problems with doctors, when I'm trying to talk to them about my problem, and they'll cut you off. You know, like, 'You're not important, you're wasting my time.' That's been a real problem for me. It makes you think that no one really cares, especially when it's done often. It's not like its 1 or 2 doctors, it's a lot of them. I have gone to a lot of different doctors." As part of his overall effort to

stay as healthy as possible, he reported he did 3 hours of exercise a week, such as walking, calisthenics, and stretching exercises.

In the absence of continuity of care, uninsured people tended to rely on cultural approaches to self-care, especially basic precepts they had learned in their families. For example, a 40-year-old unemployed, uninsured man who had hypertension said, "Basically I try to eat a healthy, balanced diet, but I like junk food. Because ever since I was a child my mom always taught me that eating a meat, vegetable, and a starch is a good healthy diet. She said, 'Add a fruit here and there.'"

Regular exercise was reported less often by persons who were uninsured, but when it was, walking was most commonly reported. For example, a 52-year-old middle-income man who was diabetic and employed in temporary clerical jobs was uninsured. He said, "If I take a really, really long walk and walk for hours or miles, then it lowers my blood sugar. Definitely has an effect." Rare were comments such as that of a 50-year-old, middle-income, uninsured man who had high blood pressure, lupus, and arthritis: "I do yoga, and that kinda puts me in a frame of mind where I can accept the pain."

## DISCUSSION

This research demonstrates several important phenomena regarding self-care among African Americans. Key aspects of African American culture are central to the development of self-care strategies. There is a basic approach to self-care that builds on widespread values and practices, including spirituality, social support and advice, and traditional medicine. Each of these cultural practices is important in shaping people's understandings of self-care. While these practices have been separated for purposes of discussion, they are in fact interrelated; for example, social support and advice may emphasize the use of traditional medicine or the importance of spirituality. These practices are part of an overall cultural ethos regardless of social class or income level. Together, they form the basis for self-care activities that are further refined in order to manage specific chronic illnesses.

However, when this basic approach to self-care was applied to specific health concerns,

the development of additional strategies of self-care was influenced by access to health care. Access to health care made a difference in how people managed their chronic illnesses. Those who had some form of health insurance had many more opportunities to discuss their chronic illnesses with physicians and other health professionals, who were important sources of information and reinforcement of activities aimed at both illness management and prevention. The ongoing nature of this interaction between people and their primary care providers was critical to the development of a self-care approach targeted to specific illnesses. Such interactions resulted in a comprehensive approach to self-care by insured people that incorporated both basic cultural approaches to self-care and biomedically influenced approaches. The combination of approaches often led to highly effective self-care for chronic illness.

In contrast, most of those who were uninsured were left to their own devices. Their attempts at self-care vied with other health and social concerns such as their efforts to gain access to health care, find employment, get enough medication, obtain information about their illnesses, and make ends meet in daily life.<sup>61</sup> In the absence of regular health care, they relied even more heavily on self-care precepts gleaned earlier in life, such as taking a basic approach to healthy diet. They were often without the economic means to actually maintain a healthy diet, however. They did not have ready access to health professionals who could suggest and reinforce steps they could take to integrate their cultural approach to self-care with a biomedical approach.

This research has implications for health disparities. In this study, lack of health insurance had a significant, and deleterious, effect on people's ability to develop complex self-care approaches that reflected both cultural and biomedical precepts of self-care. Combined with their low-income, often unemployed status, uninsured people lacked the economic resources to implement self-care regimens that integrated cultural and biomedical approaches. Access to basic health care was extremely limited, and the cursory attention that uninsured people received when they did seek medical care rarely encompassed directions for self-care.<sup>61</sup> Although health disparities are directly

related to social and economic conditions—conditions that must change for health to improve significantly<sup>2</sup>—rectifying disparities in access to health care is 1 starting point. Within that realm, access to biomedical input could facilitate the refinement of self-care practices with respect to illness management.

This study had several limitations. The sample was drawn from 1 geographic location, from volunteers who were recruited through a variety of means such as flyers and referrals. Nevertheless, these qualitative findings have implications for how self-care is conceptualized, demonstrating that when self-care is conceptualized as primarily a biomedically derived approach to health, critically important cultural practices directly germane to self-care are overlooked. Self-care needs to be understood as a process that not only evolves over time but develops in relation to the types of illnesses people experience and their specific concerns about their health.

Underlying culturally based self-care practices are important not only in general; they give rise to the development of illness-specific self-care schemes for chronic illnesses. However, regardless of the sophistication of biomedically influenced self-care schemes that people may evolve, cultural approaches to self-care are ongoing and an intrinsic part of daily life. Those approaches not only form the precursor to the incorporation of biomedically derived self-care approaches, they offer a complementary philosophy that both enhances the incorporation of these approaches and interacts with them, as others have also found.<sup>62</sup>

Studying self-care thus necessitates examining the cultural basis of self-care in a given racial/ethnic group, how biomedically derived constructs are applied, and how the 2 types of self-care approaches are integrated. However, lack of access to health care clearly interferes with this integration and tailoring process. More needs to be learned about how self-care is shaped when people do not have access to health care. Studies are also needed that explore how to build effectively on the cultural basis of self-care in order to help people maximize appropriate management of their illnesses.

In conclusion, although self-care has emerged as an important component of health maintenance, prevention, and illness manage-

ment in recent decades, the cultural components of self-care and their relevance for illness management have been underemphasized. Greater attention to the ways in which culture is implicated in self-care practices could greatly advance our ability to facilitate chronic illness management. At present, with the exception of church-based interventions, public health practice overlooks the feasibility of building on cultural principles and practices of self-care to educate people about management of specific chronic illnesses, an avenue that promises to have great potential. Moreover, comparison of those who are uninsured with those who have some form of health insurance suggests that self-care is an important adjunct to chronic illness management; however, its potential for maximizing that management is not realized for those who lack access to health care. In the face of ongoing health disparities, public health efforts to build on the cultural aspects of self-care would be one step toward reducing morbidity and mortality among racial/ethnic minorities. ■

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### Contributors

G. Becker designed the study, developed the interview schedules, led the data analysis, and drafted the article. R.J. Gates participated in the conceptual development of the article, wrote parts of the article, conducted interviews, and analyzed data. E. Newsom managed the research, conducted interviews, participated in data analysis, and edited the article.

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### Human Participant Protection

The study protocols and consent forms were approved by the institutional review board of the University of California, San Francisco.

### References

1. Ni H, Cohen R. Trends in health insurance coverage by race/ethnicity among persons under 65 years



- of age, 1997–2001. Health E-Stats National Center for Health Statistics, 2000. Available at: <http://www.cdc.gov/nchs>. Accessed January 9, 2003.
2. Williams DR. Race, socioeconomic status, and health: the added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999;896:173–188.
  3. Williams DR, Rucker TD. Understanding and addressing racial disparities in health care. *Health Care Financ Rev*. 2000;21:75–90.
  4. Cooper R, Kennelly J, Durazo-Arvizu R, Oh HJ, Kaplan G, Lynch J. Relationship between premature mortality and socioeconomic factors in black and white populations of US metropolitan areas. *Public Health Rep*. 2001;116:464–473.
  5. DeFries GH, Konrad TR, Woomert A, Kincade Norburn JE, Bernard S. Self-care and quality of life in old age. In: Abeles RP, Gift HC, Ory MG, eds. *Aging and Quality of Life*. New York, NY: Springer; 1994: 99–117.
  6. Fett SM. *Working Cures: Healing, Health, and Power on Southern Slave Plantations*. Chapel Hill: University of North Carolina Press; 2002.
  7. Byrd WM, Clayton LA. *An American Health Care Dilemma: Race, Medicine, and Health Care in the United States, 1900–2000*. New York, NY: Routledge; 2002.
  8. Kotelchuk R, Levy H. The medical committee for human rights: a case study in the self-liquidation of the New Left. In: Reed A, ed. *Race, Politics and Culture: Critical Essays on the Radicalism of the 1960's*. New York, NY: Greenwood Press; 1986:146–180.
  9. Helman CG. *Culture, Health and Illness*. 3rd ed. Oxford, England: Butterworth-Heinemann; 1994.
  10. Health Care Network-Health Canada Online, The Self-Care Project. Chapter 1. Self-care: learning from the literature. Available at: <http://www.hc-sc.gc.ca/hppb/healthcare/pubs/selfcare/ch1en.htm>. Accessed May 31, 2002.
  11. Dean K. Health-related behavior: concepts and methods. In: Ory MG, Abeles RP, Lipman DP, eds. *Aging, Health, and Behavior*. Newbury Park, Calif: Sage; 1992:27–56.
  12. *Health Education in Self-Care: Possibilities and Limitations. Report of a Scientific Consultation*. Geneva, Switzerland: World Health Organization; November 21–25, 1983.
  13. Vickery D, Levinson A. The limits of self-care. *Generations*. 1993;17:53–56.
  14. Segall A, Goldstein J. Exploring the correlates of self-provided health care behavior. *Soc Sci Med*. 1989; 29:153–161.
  15. Orem D. *Nursing: Concepts of Practice*. 5th ed. St. Louis: Mosby; 1995.
  16. Moore JB, Pichler VH. Measurement of Orem's basic conditioning factors: a review of published research. *Nurs Sci Q*. 2000;13:137–142.
  17. Taylor SG, Isaramalai S, Wongvatunyu S. Orem's self-care deficit nursing theory: its philosophic foundation and the state of the science. *Nurs Sci Q*. 2000;13: 104–110.
  18. Villarruel AM, Denyes MJ. Testing Orem's theory with Mexican Americans. *Image J Nurs Sch*. 1997;29: 283–288.
  19. Wang CY. The cross-cultural applicability of Orem's conceptual framework. *J Cult Divers*. 1997;4: 44–48.
  20. Dill A, Brown P, Ciambone D, Rakowski W. The meaning and practice of self-care by older adults: a qualitative assessment. *Res Aging*. 1995;17:8–41.
  21. Mathews HF. Doctors and root doctors: patients who use both. In: Kirkland J, Mathews HF, Sullivan CW III, Baldwin K, eds. *Herbal and Magical Medicine: Traditional Healing Today*. Durham, NC: Duke University Press; 2002:68–98.
  22. Savitt TL. *Medicine and Slavery*. Urbana: University of Illinois Press; 1978.
  23. Puckrein GA. Humoralism and social development in colonial America. *JAMA*. 1981;245: 1755–1757.
  24. Smith TH. *Conjuring Culture: Biblical Formations of Black America*. New York, NY: Oxford University Press; 1994.
  25. Mullings L. *On Our Own Terms: Race, Class and Gender in the Lives of African American Women*. New York, NY: Routledge; 1997.
  26. Stack CB. *All Our Kin: Strategies for Survival in a Black Community*. New York, NY: Harper; 1974.
  27. Dilworth-Anderson P, Burton LM, Johnson LB. Reframing theories for understanding race, ethnicity, and families. In: Boss PG, Doherty WJ, LaRossa R, Schumm WR, Steinmetz SK, eds. *Sourcebook of Family Theories and Methods: A Contextual Approach*. New York, NY: Plenum Press; 1993:627–646.
  28. James SA. John Henryism and the health of African-Americans. *Cult Med Psychiatry*. 1994;18:163–182.
  29. James SA, Thomas PE. John Henryism and blood pressure in black populations: a review of the evidence. *Afr Am Res Perspect*. 2000;6:1–10.
  30. Myers HF. Research on the Afro-American family: a critical review. In: Bass BA, Wyatt GE, Powell GJ, eds. *The Afro-American Family: Assessment, Treatment, and Research Issues*. New York, NY: Grune and Stratton; 1982:35–68.
  31. Johnson CL. Cultural diversity in the late-life family. In: Blieszner R, Bedford V, eds. *Handbook of Aging and the Family*. Westport, Conn: Greenwood; 1995.
  32. Dilworth-Anderson P, Burton LM, Turner WL. The importance of values in the study of culturally diverse families. *Fam Relat*. 1993;42:238–242.
  33. Hill R. *The Strengths of Black Families*. New York, NY: Emerson Hall; 1972.
  34. McAdoo H. Factors related to stability in upwardly mobile black families. *J Marriage Fam*. 1978;40: 761–776.
  35. Richards H. The teaching of Afrocentric values by African American parents. *West J Black Stud*. 1997;21: 42–50.
  36. Harris D. Exploring the determinants of adult black identity: context and process. *Soc Forces*. 1995; 74:227–241.
  37. Sudarkasa N. African and Afro-American family structure. In: Cole J, ed. *Anthropology for the Nineties*. New York, NY: Free Press; 1988:182–210.
  38. Chatters LM, Taylor RJ. Social Integration. In: Harel Z, McKinney EA, Williams M, eds. *Black Aged: Understanding Diversity and Service Needs*. Newbury Park, Calif: Sage; 1990:82–99.
  39. Dilworth-Anderson P. Extended kin networks in black families. *Generations*. 1992;17:29–32.
  40. Becker G, Beyene Y, Newsom E, Mayen N. Creating continuity through mutual assistance: intergenerational reciprocity in four ethnic groups. *J Gerontol Soc Sci*. 2003;58B:S151–S159.
  41. Holder B. Family support and survival among African-American end-stage renal disease patients *Adv Ren Replace Ther*. 1997;4:13–21.
  42. Frazier EF. *The Negro Church in America*. New York, NY: Schocken Books; 1974.
  43. Jones LN. The black churches in historical perspective. *Crisis*. 1982;89:6–10.
  44. Taylor RJ, Chatters LM. Religious life. In: Jackson JS, ed. *Life in Black America*. Newbury Park, Calif: Sage; 1991:105–123.
  45. Kalish RA, Reynolds DK. *Death and Ethnicity: A Psycho-Cultural Study*. Los Angeles: University of Southern California Press; 1976.
  46. Caldwell CH, Greene AD, Billingsley A. The black church as a family support system: instrumental and expressive functions. *Natl J Sociol*. 1992;6:21–40.
  47. Billingsley A, Caldwell C. The church, the family and the school in the African American community. *J Negro Educ*. 1991;60:427–440.
  48. McRae MB, Carey PM, Anderson-Scott R. Black churches as therapeutic systems: a group process perspective. *Health Educ Behav*. 1998;25:778–789.
  49. Boyd EL, Taylor SD, Shimp LA, Semler CR. An assessment of home remedy use by African Americans. *J Natl Med Assoc*. 2000;92:341–353.
  50. Davis LH, McGadney BF. Self-care practices of black elders. In: Barresi CM, Stull D, eds. *Ethnic Elders and Long Term Care*. New York, NY: Springer; 1994: 73–86.
  51. Davis L, Wykle ML. Self-care in minority and ethnic populations: the experience of older black Americans. In: Ory MG, DeFries GH, eds. *Self-Care in Later Life: Research, Program, and Policy Issues*. New York, NY: Springer; 1998:170–192.
  52. Becker G, Newsom E. Socioeconomic status and dissatisfaction with health care among chronically ill African Americans. *Am J Public Health*. 2003;93: 742–748.
  53. Reid BV. "It's like you're down on a bed of affliction": aging and diabetes among black Americans. *Soc Sci Med*. 1992;34:1317–1323.
  54. Kart CS, Engler CA. Predisposition to self-health care: who does what for themselves and why? *J Gerontol Soc Sci*. 1994;49:S301–S308.
  55. Silverman M, Musa D, Kirsch B, Siminoff LA. Self-care for chronic illness: older African Americans and whites. *J Cross Cult Gerontol*. 1999;14:169–189.
  57. Rubinstein RL. In-depth interviewing and the structure of its insights. In: Reinharz S, Rowles G, eds. *Qualitative Gerontology*. New York, NY: Springer; 1987.
  58. Mishler E. *Research Interviewing*. Cambridge, Mass: Harvard University Press; 1986.
  59. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18:341–378.
  60. Becker G. *Disrupted Lives: How People Create Meaning in a Chaotic World*. Berkeley: University of California Press; 1997.
  61. Becker G. Effects of being uninsured on ethnic minorities' management of chronic illness. *West J Med*. 2001;175:19–23.
  62. Whitaker ED. The idea of health: history, medical pluralism, and the management of the body in Emilia-Romagna, Italy. *Med Anthropol Q*. 2003;17:348–375.

# Variations in Health Communication Needs Among Combat Veterans

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In this cross-sectional study of US military combat veterans, we assessed the helpfulness of different media for providing health risk communication messages. We have provided preliminary results from a postal survey of 5000 veterans sampled because of their deployment to Vietnam, the Persian Gulf, or Bosnia–Kosovo. Respondents endorsed the primary care provider as the most helpful source of health information. Access to the Internet and use of this medium for seeking health information differed by race, age, and cohort. (*Am J Public Health*. 2004;94:2074–2076)

Military personnel deployed to war zones face a variety of physical, environmental, and psychological exposures.<sup>1,2</sup> Concern about health consequences related to exposures may lead veterans to visit health care providers for treatment and health risk education.<sup>3,4</sup> Risk communication regarding potential health outcomes is a fundamental task in the successful treatment of combat veterans.<sup>5–7</sup> We have provided preliminary findings from a population-based survey of health concerns and preferred sources for health risk communication among combat veterans.

## METHODS

### Design

This cross-sectional, 206-item postal survey sampled 5000 veterans from the Vietnam War (1964–1973), the Persian Gulf

War (1991), and the Bosnia–Kosovo peacekeeping activity (1995–2002). Approximately equal numbers of veterans from the Vietnam War, the Persian Gulf War, and the Bosnia–Kosovo peacekeeping activity were selected from Department of Defense personnel rosters. Initial findings from the first of 3 survey mailings have been reported here.

### Measures

We asked participants to rate the helpfulness of different media for obtaining health information on a 10-point scale and to answer items regarding access to and use of the Internet.

### Data Analysis

Mean scores for helpfulness ratings were computed and tested to assess differences by race for each communication mode. Proportions that had access to the Internet and used it to find health information were reported by conflict and race. Differences in Internet access by race were tested with the Pearson  $\chi^2$  statistic. Trends for Internet use by age and conflict were tested with regression analysis and the Cochran–Armitage trend test, respectively.<sup>8</sup>

## RESULTS

### Subjects

The overall response rate for the first wave of surveys (N=1432, 29%) showed variation by conflict, with the highest response rate from Vietnam veterans (n=740, 45% of Vietnam sample), followed by veterans from the Persian Gulf War (n=462, 28% of the Persian Gulf sample) and the Bosnia–Kosovo peacekeeping activity (n=230, 14% of the Bosnia–Kosovo sample). A small group of respondents served in more than 1 conflict (n=109).

The respondents were predominantly White (80% overall), which was comparable to the sample distribution (77% overall). Black veterans represented 17% of the sample but only 10% of respondents. The mean age of Vietnam veterans was 60.4 years, of Persian Gulf War veterans was 45.6 years, and of Bosnia–Kosovo peacekeeping activity veterans was 41.7 years.

### Helpfulness of Health Information Sources

When participants rated the helpfulness of health information sources, “own doctor” received the highest mean scores overall and across conflict and race subgroups (Table 1). In contrast, “VA doctor” received the lowest score for helpfulness overall, with significant differences observed by race and cohort. Non-White respondents assigned higher scores than did Whites for the helpfulness of “VA doctor” across all categories, with significant findings for all conflicts combined ( $P=.008$ ) and Persian Gulf War veterans ( $P=.05$ ).

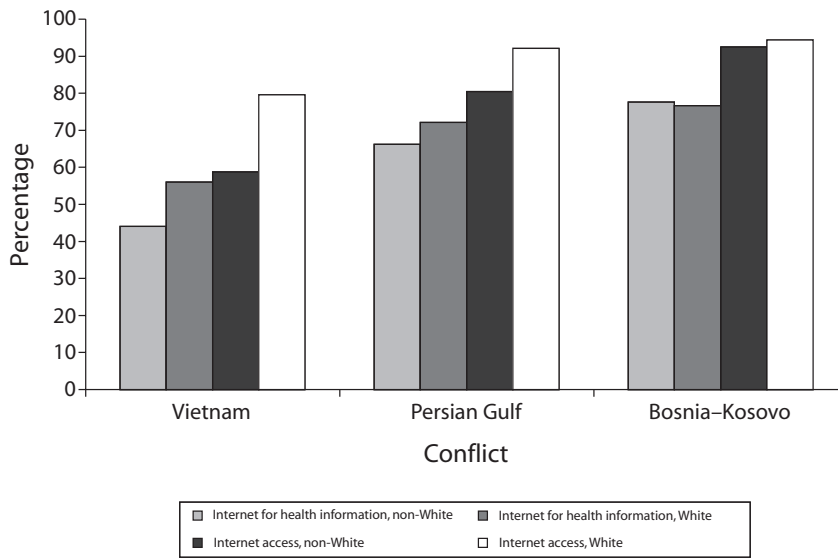
Pamphlets or brochures and Department of Veterans Affairs (VA) newsletters were given the second and third most favorable mean scores for helpfulness (6.6 and 6.5, respectively). Newsletters from Veterans Service Organizations were considered more helpful by non-White Vietnam veterans ( $P=.02$ ) and by non-Whites for the 3 cohorts combined ( $P=.04$ ). Television received a low mean score (5.7) relative to other modes for receiving health information but showed a statistically significant difference by race, with a preference expressed by non-White respondents from the Persian Gulf War ( $P=.01$ ), from Bosnia–Kosovo ( $P=.03$ ), and overall ( $P=.002$ ). Regression analyses found that decreasing age strongly predicted higher ratings for the helpfulness of Web sites as a source of health information ( $\beta=-0.045/\text{year}$ ;  $P<.001$ ;  $R^2=0.03$ ).

### Internet Access

Access to the Internet and use of this medium for obtaining health information have been increasing with successive cohorts (Cochran–Armitage trend test,  $P<.001$ ; Figure 1). Differences in access to the Internet for Whites and non-Whites were largest for Vietnam veterans (nearly 20%,  $P<.001$ ) and narrowed over time (12% for the Persian Gulf War veterans,  $P<.001$ ; and 2% for Bosnia–Kosovo veterans,  $P=.62$ ).

## DISCUSSION

These preliminary findings indicated high Internet use among veterans, suggest-



**FIGURE 1—Percentage of combat veterans from Vietnam War, Persian Gulf War, and Bosnia-Kosovo conflicts who have any access to the Internet and who use the Internet to obtain health information, by race.**

than did Whites, suggesting greater receptivity to a wider array of information sources.

These findings reinforce the role of the primary health care provider as the most helpful resource for health risk communication. For VA health care providers, having access to knowledge resources and supporting materials related to postdeployment health is critical. Recognizing this need, the VA recently created the War Related Illness and Injury Study Centers, with an expressed purpose of addressing the health risk communication needs of combat veterans. These results will help to form the basis of future postdeployment health risk communication activities in the VA. ■

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**Note.** The opinions expressed by this brief are not necessarily those of the Department of Veterans Affairs.

ing that the Internet offers an important channel for delivering health risk information to combat veterans. Internet access among Vietnam veterans (77%; 50–87 years old) was higher than that among the general US population (62% of 50- to 58-

year-olds, 46% of 59- to 68-year-olds).<sup>9</sup> Greater familiarity with the Internet among younger cohorts of veterans makes this medium an especially important tool for risk communication messages. Also, non-Whites rated a variety of media more favorably

**TABLE 1—Helpfulness Ratings<sup>a</sup> of Different Modalities for Receiving Health Risk Information, by Race and Conflict: Mean Scores (SD)**

| Information Source          | Total (N = 1288) | All Conflicts    |                     |      | Vietnam         |                     |     | Persian Gulf    |                     |     | Bosnia-Kosovo   |                    |     |
|-----------------------------|------------------|------------------|---------------------|------|-----------------|---------------------|-----|-----------------|---------------------|-----|-----------------|--------------------|-----|
|                             |                  | White (n = 1034) | Non-White (n = 254) | P    | White (n = 662) | Non-White (n = 109) | P   | White (n = 405) | Non-White (n = 148) | P   | White (n = 158) | Non-White (n = 67) | P   |
| Own doctor                  | 7.2 (2.8)        | 7.3 (2.7)        | 6.8 (3.0)           | .1   | 7.3 (2.8)       | 7.2 (3.1)           | .73 | 7.0 (2.6)       | 6.4 (3.1)           | .11 | 7.5 (2.5)       | 6.7 (3.0)          | .11 |
| Pamphlet or brochure        | 6.6 (2.7)        | 6.6 (2.7)        | 6.7 (2.6)           | .58  | 6.6 (2.8)       | 6.8 (2.7)           | .76 | 6.5 (2.6)       | 6.7 (2.6)           | .49 | 6.3 (2.6)       | 6.8 (2.6)          | .24 |
| VA newsletter <sup>b</sup>  | 6.5 (2.9)        | 6.5 (2.9)        | 6.5 (2.8)           | .97  | 6.5 (3.0)       | 6.6 (2.9)           | .99 | 6.6 (2.7)       | 6.4 (2.8)           | .58 | 5.7 (2.9)       | 6.5 (2.8)          | .09 |
| VSO newsletter <sup>c</sup> | 6.1 (3.0)        | 6.0 (3.0)        | 6.4 (2.9)           | .04  | 6.0 (3.1)       | 6.8 (2.9)           | .02 | 6.2 (2.8)       | 6.4 (2.9)           | .47 | 5.5 (2.8)       | 6.1 (3.1)          | .18 |
| Web site                    | 6.0 (3.1)        | 5.9 (3.0)        | 6.3 (3.0)           | .06  | 5.6 (3.1)       | 5.7 (3.3)           | .66 | 6.3 (2.9)       | 6.1 (3.0)           | .73 | 6.2 (2.9)       | 6.9 (2.9)          | .08 |
| Television                  | 5.7 (2.8)        | 5.6 (2.7)        | 6.1 (2.8)           | .002 | 5.6 (2.8)       | 5.9 (2.9)           | .27 | 5.5 (2.6)       | 6.1 (2.9)           | .01 | 5.5 (2.8)       | 6.5 (2.6)          | .03 |
| Newspaper article           | 5.6 (2.7)        | 5.6 (2.7)        | 5.8 (2.8)           | .15  | 5.7 (2.7)       | 5.8 (2.9)           | .71 | 5.4 (2.5)       | 5.6 (2.9)           | .27 | 5.4 (2.8)       | 6.1 (2.7)          | .09 |
| VA doctor                   | 5.5 (3.2)        | 5.4 (3.2)        | 5.9 (3.2)           | .008 | 5.2 (3.2)       | 5.8 (3.4)           | .12 | 5.4 (3.0)       | 6.0 (3.1)           | .05 | 5.5 (3.3)       | 5.8 (3.1)          | .55 |

Note. Kruskal-Wallis test used to compare distributions of scores for Whites and non-Whites. P for  $\chi^2$  statistic shown. Missing responses for helpfulness ratings ranged from 5% to 32%; Vietnam veterans, 12%–32%; Persian Gulf War veterans, 5%–14%; and Bosnia-Kosovo peacekeeping activity veterans, 5%–12%.

<sup>a</sup>Range: 1 = not very helpful; 10 = very helpful.

<sup>b</sup>VA newsletter = official publication of the Department of Veterans Affairs (VA).

<sup>c</sup>VSO newsletter = publication of a congressionally accredited Veterans Service Organization.

### Contributors

A.I. Schneiderman assisted in originating the study, drafted the brief, and conducted the data analysis and interpretation. A.E. Lincoln assisted in the data analysis and interpretation and in the editing of the brief. B. Curbow assisted in originating the study, interpreting the analysis, and editing the brief. H.K. Kang assisted in originating the study and editing the brief.

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### Human Participant Protection

This study was approved by the institutional review board of the Department of Veterans Affairs Medical Center, Washington, DC.

### References

1. Hyams KC, Wignall FS, Roswell R. War syndromes and their evaluation: from the US Civil War to the Persian Gulf War. *Ann Intern Med.* 1996;125:398–405.
2. A National Obligation: Planning for Health Preparedness and Readjustment of the Military, Veterans, and Their Families After Future Deployments. *Washington, DC: National Science and Technology Council, Executive Office of the President; 1998.*
3. National Research Council. *Improving Risk Communication.* Washington, DC: National Academy Press; 1989.
4. Institute of Medicine. Prevention measures for deployed forces. In: Joellenbeck LM, Russell PK, Guze SB, eds. *Strategies to Protect the Health of Deployed U.S. Forces: Medical Surveillance, Record Keeping, and Risk Reduction.* Washington, DC: National Academy Press; 2000:92–120.
5. Engel CC. Clinical risk communication: explaining causality to Gulf War veterans with chronic multisymptom illnesses. Washington, DC: Dept of Defense; 1999. Available at: [http://www.pdhealth.mil/clinicians/downloads/risk\\_comm.doc](http://www.pdhealth.mil/clinicians/downloads/risk_comm.doc). Accessed March 30, 2004.
6. Lundgren R, McMakin A. *Risk Communication.* Columbus, Ohio: Batelle Press; 1998.
7. DOD/VHA clinical practice guideline for post-deployment health evaluation and management. Washington, DC: Department of Defense (DoD) and Department of Veterans Health Affairs (VHA); 2000 [Updated December 2001]. Available at: [http://www.oqp.med.va.gov/cpg/PDH/PDH\\_Gol.htm](http://www.oqp.med.va.gov/cpg/PDH/PDH_Gol.htm). Accessed March 30, 2004.
8. Stokes ME, Davis CS, Koch GG. *Categorical Data Analysis Using the SAS System.* 2nd ed. New York, NY: John Wiley & Sons Inc; 2001.
9. Fox S. Older Americans and the Internet. Washington, DC: Pew Internet and American Life Project; 2004. Available at: [http://www.pewinternet.org/reports/pdfs/PIP\\_Seniors\\_Online\\_2004.pdf](http://www.pewinternet.org/reports/pdfs/PIP_Seniors_Online_2004.pdf). Accessed March 30, 2004.



# Accounting for Apparent “Reverse” Racial Disparities in Department of Veterans Affairs (VA)—Based Medical Care: Influence of Out-of-VA Care

Andrea D. Gurmankin, PhD, MBe, Daniel Polsky, PhD, and Kevin G. Volpp, MD, PhD

Conclusions regarding racial differences in care following a newly elevated prostate-specific antigen (PSA) test at the Department of Veterans Affairs (VA) may differ depending on whether follow-up care outside the VA is considered. Consecutive Philadelphia, Pa, VA patients with newly elevated PSA tests ( $n = 183$ ) were interviewed 1 year after baseline. Among exclusive VA users, Blacks had higher rates of urology referrals and prostate biopsies compared with Whites. However, these racial differences were attenuated when care obtained outside the VA also was considered. (*Am J Public Health.* 2004;94:2076–2078)

For many conditions, including prostate cancer, studies in US patient populations have found that Blacks have worse health outcomes and lower health care use than do Whites.<sup>1–5</sup> However, studies in the Department of Veterans Affairs (VA) have often shown that outcomes and utilization are as good, if not better, for Blacks as for Whites.<sup>6–14</sup>

One possible explanation for this discrepancy is that VA disparities studies typically measure treatment and outcomes only within the VA,<sup>6–13,15</sup> even though most patients receive some care outside the VA.<sup>16</sup> If Whites receive a greater proportion of care outside the VA than do Blacks, assessments of service use that examine only within-VA services may

show a misleading lack of disparity or “reverse disparity.”

Our study compared the magnitude of racial disparities in knowledge of an elevated prostate-specific antigen (PSA) test, urology referral, and prostate biopsy among patients with a newly elevated PSA test at the Philadelphia Veterans Affairs Medical Center, depending on whether or not care obtained outside the VA was considered.

## METHODS

With approval from the Philadelphia Veterans Affairs Medical Center institutional review board, we conducted a 1-year retrospective cohort study of consecutive patients with newly elevated PSA tests (“index PSA test”) ( $PSA > 4$ ) at the Philadelphia Veterans Affairs Medical Center from June 1, 2001, to April 11, 2002. One year after the index PSA test, we conducted telephone interviews with patients on follow-up care received and medical chart abstractions to validate self-reports (for which we found concordance in 88% cases).

Of the 343 eligible patients identified, patients were excluded if they were deceased at time of enrollment ( $n = 4$ ), uncontactable ( $n = 60$ ), too ill ( $n = 7$ ), or not Black or White ( $n = 5$ ). Of the 267 remaining patients, 183 (69%) participated.

To distinguish between patients who were likely to seek follow-up care outside rather than inside the Philadelphia Veterans Affairs Medical Center, subjects were classified as either “partial VA users” or “exclusive VA users.” Partial VA users reported using the Philadelphia Veterans Affairs Medical Center for prescription medications only or reported seeing a urologist outside the Philadelphia Veterans Affairs Medical Center. All others were classified as exclusive VA users. Subjects who did not know that they had received a PSA test ( $n = 40$ ) were not asked about follow-up care and hence were excluded from initial analyses. Sensitivity analyses were performed by including these subjects in sequence in the exclusive VA or partial VA users’ groups because appropriate group assignment was unclear. In analyses in which these subjects were included, their response to each outcome was coded as “no.”

**TABLE 1—Unadjusted Rates of Knowledge and Follow-Up Care Among Blacks Relative to Whites**

|  | Exclusive VA Users, %    |                         |    |                    | Partial VA Users, %     |                          |     |                    | All Subjects, %          |                          |    |                    |
|--|--------------------------|-------------------------|----|--------------------|-------------------------|--------------------------|-----|--------------------|--------------------------|--------------------------|----|--------------------|
|  | Blacks<br>(n = 33)       | Whites<br>(n = 18)      | Δ  | OR (95% CI)        | Blacks<br>(n = 13)      | Whites<br>(n = 79)       | Δ   | OR (95% CI)        | Blacks<br>(n = 46)       | Whites<br>(n = 97)       | Δ  | OR (95% CI)        |
| Know of elevated PSA test                    | 55                       | 56                      | 1  | 0.96 (0.30, 3.04)  | 31                      | 43                       | -12 | 0.59 (0.17, 2.07)  | 48                       | 45                       | 3  | 1.10 (0.56, 2.23)  |
| Urology referral within 1 y                  | 73                       | 44                      | 29 | 3.33 (1.00, 11.12) | 50                      | 58                       | -8  | 0.69 (0.20, 2.33)  | 67                       | 55                       | 12 | 1.55 (0.74, 3.25)  |
| Biopsy within 1 y                            | 67                       | 28                      | 39 | 5.20 (1.47, 18.30) | 36                      | 31                       | 5   | 1.25 (0.33, 4.74)  | 59                       | 30                       | 29 | 3.28 (1.54, 7.00)  |
| Biopsy within 1 y if had urology appointment | 92 (n = 24) <sup>a</sup> | 63 (n = 8) <sup>a</sup> | 29 | 6.60 (0.86, 50.54) | 80 (n = 6) <sup>a</sup> | 58 (n = 44) <sup>a</sup> | 22  | 2.86 (0.29, 28.19) | 90 (n = 30) <sup>a</sup> | 59 (n = 52) <sup>a</sup> | 31 | 6.00 (1.57, 22.86) |

Note. VA = Department of Veterans Affairs; OR = odds ratio; CI = confidence interval; PSA = prostate-specific antigen.

<sup>a</sup>Sample includes only those subjects who had a urologist appointment, a requirement for undergoing biopsy.

## RESULTS

Compared with partial VA users, exclusive VA users were younger (73.5 vs 65.8 years;  $P < .001$ ) and more likely to be Black (17% vs 65%;  $P < .001$ ) and to earn \$30 000 or less (56% vs 88%;  $P < .001$ ). Education (13.5 vs 13.0 years;  $P = .245$ ) and index PSA test levels (7.1 vs 6.6;  $P = .622$ ) were similar.

In general, Blacks were more likely than Whites to know of their PSA test and to have a urology referral and biopsy among exclusive VA users and were as likely as or less likely than Whites to have these characteristics among partial VA users. The rates of each outcome among all subjects represented the combined rates of the 2 subgroups (Table 1).

For example, Blacks were significantly more likely to have a urology referral than were Whites (73% vs 44%) among exclusive VA users but as likely as Whites among partial VA users (50% vs 58%) and all subjects (67% vs 55%). Among exclusive VA users

and all subjects, Blacks were more likely than Whites to have a biopsy (67% vs 28% and 59% vs 30%, respectively), but there was no difference for partial VA users (36% vs 31%).

Multivariate models adjusted for age and index PSA level, factors that affect the likelihood of prostate cancer and, therefore, the importance of a urology referral and a biopsy. In these models, the odds ratios for race reflected a pattern for each outcome similar to that in the unadjusted analyses (Table 2). In addition, we examined whether the odds ratios for race were different for exclusive VA users and partial VA users by combining the 2 groups into 1 multivariate model and examining an added term of being an exclusive VA user interacted with being Black. The statistical significance of this coefficient for urology referral ( $P = .04$ ) suggested that the racial differences among exclusive VA users were statistically different from those among partial VA users. For the other outcomes, the racial differences between exclusive and partial VA users were not statistically different.

Racial differences in each of the groups were reexamined after including the 40 subjects who did not know that they had received a PSA test. The results were qualitatively similar to analyses that had excluded these subjects.

## DISCUSSION

Among patients who received care exclusively within the VA, Blacks had higher rates of urology referrals and prostate biopsies than did Whites in the year following a newly elevated PSA test. However, when care obtained outside the VA was considered, the rates of these outcomes among Blacks were generally no different from rates among Whites. Therefore, including information about the care received by many VA patients outside the VA<sup>16</sup> markedly affects the assessment of whether racial disparities exist in the rates of urology referral and prostate biopsy.

These findings are likely the result of different rates of use of care outside the VA by Black compared with White VA patients. Because more Whites than Blacks receive care outside the VA, these White patients may receive less care within the VA. As a result, in assessments of racial differences among VA patients in care received at the VA, it appears that Blacks obtain more care than do Whites.

The results should be considered in light of certain limitations. We relied on patient self-reports of care, although these self-reports were largely validated by chart data. Some subjects' PSA tests may not have been newly elevated, despite efforts to exclude

**TABLE 2—Adjusted Racial Differences in Knowledge and Follow-Up Care in Blacks Relative to Whites**

|  | OR (95% CI) for Adjusted Racial Difference <sup>a</sup> |                     |                    |
|--|---|---------------------|--------------------|
|  | Exclusive VA Users                                      | Partial VA Users    | All Subjects       |
| Know of elevated PSA test                    | 0.85 (0.24, 2.96)                                       | 0.52 (0.13, 2.00)   | 0.85 (0.40, 1.83)  |
| Urology referral within 1 y                  | 3.91 (1.05, 14.59)                                      | 0.61 (0.17, 2.28)   | 1.62 (0.73, 3.59)  |
| Biopsy within 1 y                            | 5.16 (1.34, 19.83)                                      | 1.13 (0.26, 4.79)   | 2.63 (1.18, 5.88)  |
| Biopsy within 1 y if had urology appointment | 4.97 (0.58, 42.71)                                      | 7.90 (0.11, 594.35) | 4.82 (1.00, 23.34) |

Note. OR = odds ratio; CI = confidence interval; VA = Department of Veterans Affairs; PSA = prostate-specific antigen.

<sup>a</sup>Adjusted for age 70 or younger (yes or no) and index PSA test result.

these patients. However, this was more likely among partial VA users; their VA records may be less complete, which would bias against finding smaller racial differences in follow-up rates when including partial VA users.

Our results have implications for the VA studies of racial disparities in outcomes and utilization.<sup>6–8,10–13</sup> Exclusive examination of services received by patients within the VA system may overestimate total use by Blacks compared with Whites. Interpretation of VA studies of racial disparities should consider this potential bias. ■

### About the Authors

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### Contributors

A.D. Gurmankin initiated, developed, and implemented all aspects of the study and led the data analysis and writing of the brief. D. Polsky helped to formulate the study aims. K.G. Volpp supervised all aspects of the study, including study design and data analysis. All authors helped to conceptualize ideas, interpret the results, and review drafts of the brief.

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### Human Participant Protection

This study was approved by the institutional review board of the Philadelphia Veterans Affairs Medical Center.

### References

1. *United States Cancer Statistics: 2000 Incidence*. Hyattsville, Md: National Center for Health Statistics, Public Health Service; 2000. Available at: [http://www.cdc.gov/cancer/npcr/uscs/pdf/USCS\\_Report\\_2000.pdf](http://www.cdc.gov/cancer/npcr/uscs/pdf/USCS_Report_2000.pdf).
2. Farkas A, Schneider D, Perrotti M, Cummings KB, Ward WS. National trends in the epidemiology of prostate cancer, 1973 to 1994: evidence for the effectiveness of prostate-specific antigen screening. *Urology*. 1998;52:444–448.
3. Merill R, Lyon JL. Explaining the difference in prostate cancer mortality rates between white and black men in the United States. *Urology*. 2000;55:730–735.
4. Carey T, Garrett JM. The relation of race to outcomes and use of health care services for acute low back pain. *Spine*. 2003;28:390–394.
5. Egede L, Zheng D. Racial/ethnic differences in adult vaccination among individuals with diabetes. *Am J Public Health*. 2003;93:324–329.
6. Bennett C, Horner RD, Weinstein RA, et al. Racial differences in care among hospitalized patients with *Pneumocystis carinii* pneumonia in Chicago, New York, Los Angeles, Miami and Raleigh-Durham. *Arch Intern Med*. 1995;155:1586–1592.
7. Goldstein L, Matchar DB, Hoff-Lindquist J, Samsa GP, Horner RD. Veterans Administration Acute Stroke (VAST) study: lack of racial/ethnic-based differences in utilization of stroke-related procedures or services. *Stroke*. 2003;34:999–1004.
8. Jha A, Shlipak MG, Hosmer W, Frances CD, Browner WS. Racial differences in mortality among men hospitalized in the Veterans Affairs health care system. *JAMA*. 2001;285:297–303.
9. Washington D, Harada ND, Damron-Rodriguez J, Dhanani S, Shon H, Makinodan T. Racial variations in Department of Veterans Affairs ambulatory care use and unmet health care needs. *Mil Med*. 2002;167:235–241.
10. Petersen L, Wright SM, Peterson ED, Daley J. Impact of race on cardiac care and outcomes in veterans with acute myocardial infarction. *Med Care*. 2002;40(suppl 1):186–196.
11. Akerley W, Moritz TE, Ryan LS, Henderson WG, Zacharski LR. Racial comparisons of outcomes of male Department of Veterans Affairs patients with lung and colon cancer. *Arch Intern Med*. 1993;153:1681–1688.
12. Smith TJ, Penberthy L, Desch CE, et al. Differences in initial treatment patterns and outcomes of lung cancer in the elderly. *Lung Cancer*. 1995;13:235–252.
13. Petersen LA, Wright SM, Peterson ED, Daley J. Impact of race on cardiac care and outcomes in veterans with acute myocardial infarction. *Med Care*. 2002;40(suppl 1):186–196.
14. Freeman VL, Durazo-Arvizu R, Arozullah AM, Keys LC. Determinants of mortality following a diagnosis of prostate cancer in Veterans Affairs and private sector health care systems. *Am J Public Health*. 2003;93:1706–1712.
15. Bach P, Cramer LD, Warren JL, Begg CB. Racial differences in the treatment of early-stage lung cancer. *N Engl J Med*. 1999;34:1198–1205.
16. Shen Y, Hendricks A, Zhang S, Kazis LE. VHA enrollees' health care coverage and use of care. *Med Care Res Rev*. 2003;60:253–267.

# The Health Impact of Resolving Racial Disparities: An Analysis of US Mortality Data

Steven H. Woolf, MD, MPH, Robert E. Johnson, PhD, George E. Fryer Jr, PhD, MSW, George Rust, MD, MPH, and David Satcher, MD, PhD

The US health system spends far more on the “technology” of care (e.g., drugs, devices) than on achieving equity in its delivery. For 1991 to 2000, we contrasted the number of lives saved by medical advances with the number of deaths attributable to excess mortality among African Americans. Medical advances averted 176 633 deaths, but equalizing the mortality rates of Whites and African Americans would have averted 886 202 deaths. Achieving equity may do more for health than perfecting the technology of care. (*Am J Public Health*. 2004;94:2078–2081)

Much of the billions of dollars<sup>1</sup> spent in the United States to improve health outcomes is directed at the “technology” of care—the race among private industries and academia to develop better drugs, devices, and procedures. Far less money and infrastructure is devoted to improving health by enhancing *equity*—achieving equal care for equal need—and eliminating disparities in the treatment and outcomes of those with similar conditions.<sup>2</sup>

Whether this asymmetry is prudent is best determined by comparing the degree to which the population benefits from each endeavor. Does society save more lives by enhancing the technology of care or by resolving disparities? The answer would take years to determine (data and statistical methods for sound projections are lacking), but today’s policymakers need some guidance, albeit approximate, to judge whether the current balance of effort is best for the population. We performed a “thought experi-

ment<sup>3</sup> to compare the number of lives saved through the 2 strategies.

## METHODS

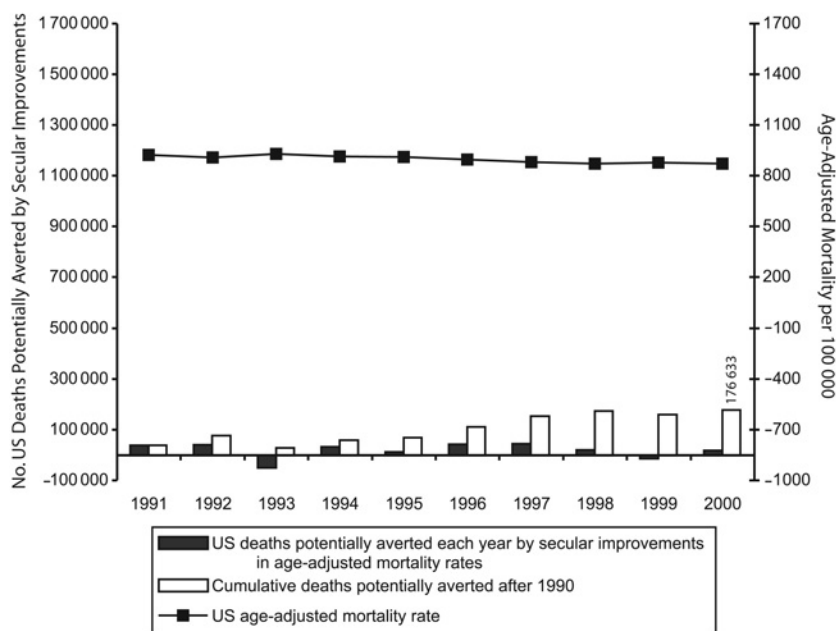
We obtained mortality data for 1991 to 2000 from the National Center for Health Statistics<sup>4–8</sup> to estimate the maximum number of deaths averted by improving the technology of care and the number of avoidable deaths had African Americans experienced the age-adjusted mortality rates of Whites. Our crude measure of the benefit of medical advances was declines in age-adjusted mortality rates. Such declines stem from multiple factors, not just improved technology, but we gave full credit to the latter to define the maximum number of averted deaths that could be attributed to this endeavor.

For this estimate, we performed an indirect standardization of mortality rates,<sup>9</sup> multiplying the population by the difference between the crude mortality rate for each calendar year and a recalculated age-adjusted rate reflecting no improvement in mortality rates. The latter was derived by multiplying age-specific populations by the corresponding age-specific mortality rates from the prior year and dividing by the total population.

To determine the number of deaths among African Americans attributable to higher mortality rates, we performed an indirect standardization of mortality rates and used African Americans as the reference population. For each calendar year, by gender, we multiplied the White age-specific mortality rate by the population of African Americans in the corresponding age groups. We divided the total calculated deaths by the population of African Americans to arrive at a gender-specific mortality rate. This hypothetical crude mortality rate was subtracted from the actual African American crude mortality rate and multiplied by the total population of African Americans to estimate the number of avertable deaths in that calendar year. (Our calculations and methods are detailed at <http://www.vcu.edu/fp/research/AJPHaddendum.pdf>.)

## RESULTS

Age-adjusted mortality rates showed some year-to-year increases but declined an aver-



Source. Age-adjusted death rates are from Table 35: Death rates for all causes, according to sex, race, Hispanic origin, and age: United States, selected years 1950–2001. National Center for Health Statistics. Available at: [ftp.cdc.gov/pub/Health\\_Statistics/NCHS/Publications/Health\\_US/hus03/Table035.xls](ftp.cdc.gov/pub/Health_Statistics/NCHS/Publications/Health_US/hus03/Table035.xls). Potentially avoidable deaths were calculated as described in: <http://www.vcu.edu/fp/research/AJPHaddendum.pdf>.

**FIGURE 1—Potential number of lives saved by improvements in age-adjusted mortality rates in the United States, 1991–2000.**

age of 0.7% per year. Our calculations suggested that these declines averted 176 633 deaths in 1991 to 2000 (Figure 1). During the same years, age-adjusted mortality rates for White males and females were an average of 29% and 24% lower, respectively, than those for African Americans. As of 2000, the mortality rate for African American infants and adults aged 25 to 54 years was more than double that of Whites. Had the age-specific mortality rates of the 2 races been comparable during 1991 to 2000, our calculations suggested that 886 202 deaths could have been averted (Figure 2).

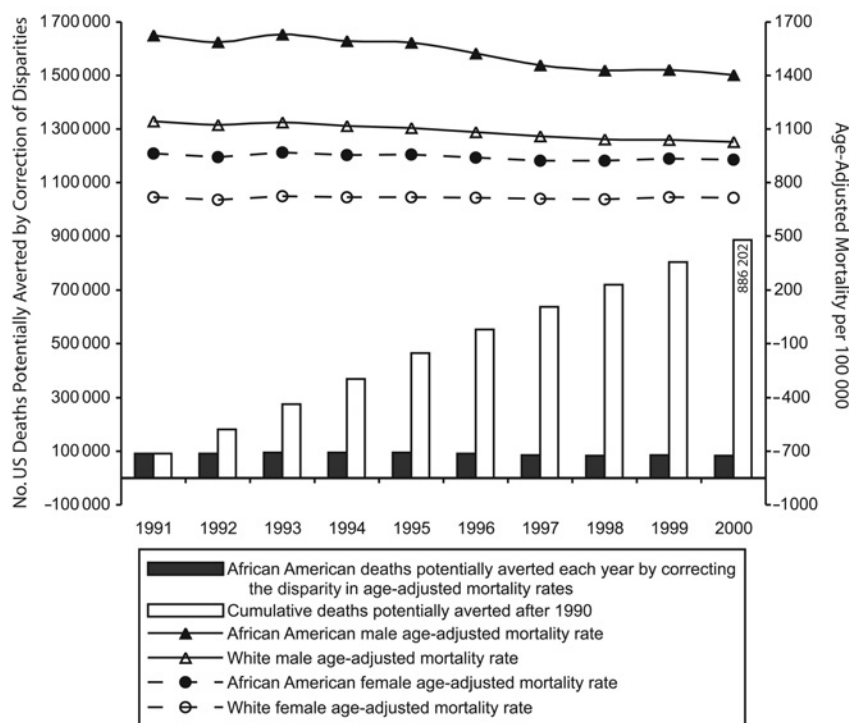
## DISCUSSION

Improvements in the technology of care did save lives during 1991 to 2000, but the deaths averted were considerably fewer than the potential lives saved by reducing the mortality rate of African Americans to the rate of Whites. Five deaths could have been averted for every life saved by medical advances.

This contention assumes that racial disparities could be abolished, a formidable premise. Elsewhere, we discuss the immense societal challenges such an effort must overcome.<sup>10</sup> Here, our intent was to offer policymakers a sense of perspective about how the potential gains from overcoming these challenges would compare with continued investment in the technology of care.

Because we observed a 5-fold difference in averted deaths, more precise calculations would be unlikely to change the direction of our findings. Our estimates are consistent with others.<sup>11,12</sup> We acknowledge important limitations, however. First, we focused on mortality, and racial disparities encompass morbidity and other domains. Second, mortality is influenced by variables other than medical care (e.g., demographics, lifestyle, environment). Modeling techniques can clarify the contribution of medical interventions,<sup>13</sup> but the requisite interactive terms are lacking. Third, the absence of a reduction in mortality does not exclude a benefit from improved care, which





Source. Age-adjusted death rates are from Table 35: Death rates for all causes, according to sex, race, Hispanic origin, and age: United States, selected years 1950–2001. National Center for Health Statistics. Available at: [ftp.cdc.gov/pub/Health\\_Statistics/NCHS/Publications/Health\\_US/hus03/Table035.xls](ftp.cdc.gov/pub/Health_Statistics/NCHS/Publications/Health_US/hus03/Table035.xls). Potentially avoidable deaths were calculated as described in: <http://www.vcu.edu/fp/research/AJPHaddendum.pdf>.

**FIGURE 2—Difference in age-adjusted mortality rates of Whites and African Americans in 1991–2000 and potential number of lives saved if the rates had been comparable.**

might avert a rise in mortality. Our calculations assumed that medical advances would lower mortality in the same decade, but benefits might occur years later<sup>14</sup> or might accrue more in population subgroups.

Fourth, our calculations modeled a sudden disappearance of disparities. A graduated model would be more realistic, projecting benefits from partial reductions in disparities over time. Fifth, we treated efforts to improve technology and reduce disparities as mutually exclusive, when one can enhance the other. Sixth, our analysis dealt with only 2 races, excluding the disparities experienced by others (e.g., Native Americans). Lives also might be saved by reducing the mortality rate of Whites to that of Hispanics or Asian Americans.<sup>15</sup> Socioeconomic conditions represent a more pertinent cause of disparities than race.<sup>10,16</sup> An intriguing question is whether more lives are saved by medical ad-

vances or by resolving social inequities in education and income.

Future work will explore these issues but is unlikely to alter our fundamental finding: resolving the causes of higher mortality rates among African Americans can save more lives than perfecting the technology of care. Policymakers could act on this information without waiting for more precise projections. The prudence of investing billions in the development of new drugs and technologies while investing only a fraction of that amount in the correction of disparities deserves reconsideration. It is an imbalance that may claim more lives than it saves. ■

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#### Contributors

S.H. Woolf originated the study and was chiefly responsible for writing the brief. R.E. Johnson provided methodological and statistical advice, conducted calculations, and developed related figures and tables. G.E. Fryer Jr, G. Rust, and D. Satcher provided advice on content, data interpretation, and policy implications.

#### Human Participant Protection

No human subjects were involved in this study.

#### References

1. Hefler S, Smith S, Keehan S, Clemens MK, Won G, Zezza M. Health spending projections for 2002–2012. *Health Aff (Millwood)*. 2003;(suppl W3):54–65.
2. Smedley BD, Stith AY, Nelson AR, eds. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care, Board on Health Sciences Policy, Institute of Medicine. Washington, DC: National Academy Press; 2003.
3. Arthur R. On thought experiments as a priori science. *Int Stud Philosophy Sci*. 1999;13:215–229.
4. National Center for Health Statistics. Bridged-race intercensal estimates of the July 1, 1990–July 1, 1999, United States resident population by state, county, age, sex, race, and Hispanic origin, prepared by the US Census Bureau with support from the National Cancer Institute, 2003. Available at: <http://www.cdc.gov/nchs/about/major/dvs/popbridge/datadoc.htm#inter5>. Accessed December 6, 2003.
5. National Center for Health Statistics. Estimates of the April 1, 2000, United States resident population by age, sex, race, and Hispanic origin, prepared under a collaborative arrangement with the US Census Bureau, 2003. Available at: <http://www.cdc.gov/nchs/about/major/dvs/popbridge/popbridge.htm>. Accessed December 6, 2003.
6. Table 290A: Deaths for 72 selected causes, by 10-year age groups, race, and sex: United States, 1979–98. National Center for Health Statistics. Available at: <http://www.cdc.gov/nchs/data/statab/gmwk290a.pdf>. Accessed December 6, 2003.
7. Table 250A: Deaths from 113 selected causes, by 10-year age groups, race, and sex: United States, 1999. National Center for Health Statistics. Available at: [http://www.cdc.gov/nchs/data/statab/V500199\\_TBL250F.pdf](http://www.cdc.gov/nchs/data/statab/V500199_TBL250F.pdf). Accessed December 6, 2003.
8. Table 250A: Deaths from 113 selected causes, by 10-year age groups, race, and sex: United States, 2000. National Center for Health Statistics. Available at: <http://www.cdc.gov/nchs/data/dvs/tab1250a.pdf>. Accessed December 6, 2003.
9. Lilienfeld AM, Lilienfeld DE. *Foundations of Epidemiology*. 2nd ed. New York, NY: Oxford University Press; 1980.
10. Woolf SH. Society's choice: the tradeoff between

efficacy and equity, and the lives at stake. *Am J Prev Med.* 2004;27:49–56.

11. Levine RS, Foster JE, Fullilove RE, et al. Black-white inequalities in mortality and life expectancy, 1933–1999: implications for Healthy People 2010. *Public Health Rep.* 2001;116:474–483.
12. Report of the Secretary's Task Force on Black and Minority Health. *MMWR Morb Mortal Wkly Rep.* 1986;35:109–112.
13. Nolte E, McKee M. Measuring the health of nations: analysis of mortality amenable to health care. *BMJ.* 2003;327:1129–1130.
14. Halfon N, Hochstein M. Life course health development: an integrated framework for developing health, policy, and research. *Milbank Q.* 2002;80:433–479.
15. *Health, United States, 2003 With Chartbook on Trends in the Health of Americans.* Hyattsville, Md: National Center for Health Statistics; 2003.
16. Kawachi I, Kennedy BK. *The Health of Nations: Why Inequality Is Harmful to Your Health.* New York, NY: New Press; 2002.

# Association of Retail Tobacco Marketing With Adolescent Smoking

Lisa Henriksen, PhD, Ellen C. Feighery, RN, MS, Yun Wang, MS, and Stephen P. Fortmann, MD

A survey of 2125 middle-school students in central California examined adolescents' exposure to tobacco marketing in stores and its association with self-reported smoking. Two thirds of sixth-, seventh-, and eighth-grade students reported at least weekly visits to small grocery, convenience, or liquor stores. Such visits were associated with a 50% increase in the odds of ever smoking, even after control for social influences to smoke. Youth smoking rates may benefit from efforts to reduce adolescents' exposure to tobacco marketing in stores. (*Am J Public Health*. 2004;94:2081–2083)

The tobacco industry spends more on point-of-purchase marketing than on all other forms of cigarette advertising combined (\$9.5 billion vs \$1.7 billion in 2001).<sup>1</sup> Since the ban on billboard advertising, promotional materials for to-

bacco have increased significantly on store windows and around the counter.<sup>2,3</sup> Three out of 4 teenagers shop at a convenience store at least once a week,<sup>4</sup> staying an average of 16 minutes per visit—twice as long as adults.<sup>5</sup> In recent surveys, all 15- and 16-year-olds reported seeing point-of-purchase marketing for cigarettes,<sup>6</sup> and teenaged smokers preferred whichever brand (Marlboro or Camel) was advertised most heavily in the convenience store nearest their school.<sup>7</sup> Experimental studies suggest that retail tobacco marketing exposure distorts adolescents' perceptions about the availability, use, and popularity of tobacco<sup>8,9</sup>—normative beliefs that are precursors of smoking.<sup>10–12</sup>

The only study to correlate adolescents' self-reported smoking with exposure to retail tobacco marketing found that experimentation was 38% more likely for seventh-grade students who said that they visited a convenience, liquor, or small grocery store at least weekly and remembered seeing advertisements for cigarettes sometimes or a lot.<sup>13</sup> Because cigarette advertising is more noticeable to experimenters than to abstinent youths,<sup>14,15</sup> this finding may be attributable to perceived exposure alone. Alternatively, store exposure may be associated with smoking for reasons other than the influence of advertising, such as a propensity for risk taking or lack of after-school supervision, which are established risk factors for smoking.<sup>11,16,17</sup> After we controlled for such risk factors and social influences to smoke, we examined whether adolescent smoking is associated with exposure to stores that contain widespread tobacco marketing.

## METHODS

We administered the Survey of Teen Opinions about Retail Environments (STORE Study) in all 3 middle schools in Tracy, Calif (population 62 500), in spring 2003. Of the initial sample of sixth-, seventh-, and eighth-grade students (N=2731), 396 students did not return parental consents, 95 were absent for the survey, and 115 parents refused permission, yielding a response rate of 78%. The final sample (N=2125) was 53% female and 42% Hispanic, 27% White, 11% Asian, 6% African American, 1% American Indian, 1% Pacific Islander, and 12% multiethnic.

Each survey included photographs and addresses of 12 retail tobacco outlets in the school

catchment area that were identified by student focus groups as popular destinations for purchasing snacks. Exposure to retail tobacco marketing was coded for students who reported at least weekly visits to convenience, liquor, or small grocery stores, either in response to questions about the pictured stores or in response to questions about visiting any such stores in the past month. Exposure to other forms of tobacco marketing was coded for students who reported owning a cigarette promotional item, sometimes or often seeing tobacco advertisements in magazines in the past week, or sometimes or often seeing people smoking on television or in movies in the past week. Exposure to family and peer smoking was coded, separately, for students who reported having a parent or other household member who currently smokes and for students who reported having at least one friend who currently smokes. The survey also included self-reported grades, a 3-item risk-taking measure,<sup>11</sup> and 4 items of the Authoritative Parenting Index<sup>18</sup> that measure maternal supervision.

We used GLIMMIX for SAS, Version 8.0 (SAS Institute Inc, Cary, NC), to examine the potential effect of school clustering with multi-level modeling, but results did not differ from the standard multiple regression reported here. Odds ratios were computed to test the association between adolescents' exposure to retail tobacco marketing and ever smoking a cigarette, even just a puff. This was the primary study outcome because the prevalence of current smoking was quite small: 2.6% of sixth-, 6.0% of seventh-, and 7.6% of eighth-grade students reported any cigarette smoking in the past 30 days.

## RESULTS AND DISCUSSION

Two thirds of the students said that they visited a convenience, liquor, or small grocery store on their way to or from school at least weekly (Table 1); about one fourth of the students reported visiting such stores practically every day. Exposure to retail tobacco marketing was more prevalent among boys; Latino youths; and students who reported low maternal supervision, high risk taking, exposure to social influences to smoke, and exposure to other forms of tobacco marketing.

Table 2 summarizes the bivariate and multivariate associations of ever smoking with de-

mographic attributes and psychosocial risk factors. After we controlled for all other variables in the model, weekly or more frequent exposure to retail tobacco marketing was associated with a 50% increase in the odds of ever smoking—nearly as much as the effect of exposure to a parent or household member who smokes. Although Latino youths were more likely to be exposed to retail tobacco marketing, they had

no greater risk for smoking when other variables were controlled.

Because we controlled for confounders such as risk taking, maternal supervision, and self-reported grades, the association between adolescents' store visits and their smoking behavior appears more likely an effect of advertising exposure than an artifact of idle hands or hanging out with the wrong crowd. Relative to

other forms of tobacco marketing, retail marketing exposure was second only to owning a cigarette promotional item in increasing the odds of ever smoking. However, the smaller association observed for exposure to cigarette advertisements in magazines and no association for depictions of smoking on television or in movies may be an artifact of redundancy within exposure measures.

This cross-sectional study could not confirm a causal role for retail tobacco marketing in the uptake of smoking but provided stronger evidence for causality than previous studies have by controlling several potential confounds.

The effect of retail tobacco marketing on adolescent smoking may be particularly potent in states with comprehensive tobacco control programs where tobacco advertisements and promotions are more numerous at the point of sale.<sup>19,20</sup> Because the tobacco industry has relatively few regulations on advertising in stores,<sup>21–25</sup> further research, including longitudinal studies, is needed to determine how the proliferation of tobacco marketing in stores affects youths. ■

**TABLE 1—Characteristics of Study Participants and Prevalence of Exposure to Retail Tobacco Marketing: 2125 Middle-School Students, Tracy, Calif, 2003**

|   | % of Full Sample<br>(N = 2125) | Prevalence of Exposure to<br>Retail Tobacco Marketing, % | Unadjusted OR (95% CI) |
|---|--------------------------------|--|------------------------|
| Grade                                     |                                |  |                        |
| 6th <sup>a</sup>                          | 33.2                           | 68.0   |                        |
| 7th                                       | 29.8                           | 66.1   | 0.9 (0.7, 1.2)         |
| 8th                                       | 37.0                           | 65.2   | 0.9 (0.7, 1.1)         |
| Gender                                    |                                |  |                        |
| Female <sup>a</sup>                       | 53.3                           | 63.9   |                        |
| Male                                      | 46.7                           | 69.1   | 1.3 (1.1, 1.5)         |
| Ethnicity                                 |                                |  |                        |
| Not Latino or Hispanic <sup>a</sup>       | 57.9                           | 59.6   |                        |
| Latino or Hispanic                        | 42.1                           | 76.0   | 2.2 (1.8, 2.6)         |
| Self-reported grades                      |                                |  |                        |
| Above median <sup>a</sup>                 | 52.6                           | 60.2   |                        |
| Below median                              | 47.4                           | 73.1   | 1.8 (1.5, 2.2)         |
| Parent or household smoking               |                                |  |                        |
| No <sup>a</sup>                           | 55.5                           | 60.3   |                        |
| Yes                                       | 44.5                           | 74.0   | 1.9 (1.6, 2.3)         |
| At least 1 friend smokes                  |                                |  |                        |
| No <sup>a</sup>                           | 72.3                           | 61.9   |                        |
| Yes                                       | 27.7                           | 77.9   | 2.2 (1.7, 2.7)         |
| Maternal supervision                      |                                |  |                        |
| Above mean <sup>a</sup>                   | 50.6                           | 62.7   |                        |
| Below mean                                | 49.4                           | 70.0   | 1.4 (1.2, 1.7)         |
| Risk taking                               |                                |  |                        |
| Below mean <sup>a</sup>                   | 46.3                           | 56.6   |                        |
| Above mean                                | 53.7                           | 74.6   | 2.3 (1.9, 2.7)         |
| Own cigarette promotional item            |                                |  |                        |
| No <sup>a</sup>                           | 84.8                           | 63.5   |                        |
| Yes                                       | 15.2                           | 82.8   | 2.8 (2.0, 3.7)         |
| See cigarette advertisements in magazines |                                |  |                        |
| Never or rarely <sup>a</sup>              | 48.8                           | 55.4   |                        |
| Sometimes or often                        | 51.2                           | 76.9   | 2.7 (2.2, 3.2)         |
| See smoking on television or in movies    |                                |  |                        |
| Never or rarely <sup>a</sup>              | 32.0                           | 55.9   |                        |
| Sometimes or often                        | 68.0                           | 71.4   | 2.0 (1.6, 2.4)         |
| Exposure to retail tobacco marketing      |                                |  |                        |
| <Once/week <sup>a</sup>                   | 33.8                           | ...  | ...                    |
| At least once/week                        | 66.2                           | ...  | ...                    |

Note. OR = odds ratio; CI = confidence interval.

<sup>a</sup>Reference category.

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### Contributors

L. Henriksen helped design the study and supervised all aspects of its implementation. E. C. Feighery helped design the study and supervised store advertising surveys. Y. Wang assisted with the study and data analyses. S. P. Fortmann helped design the study. All authors helped to interpret findings and review drafts of the brief.

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### Human Participant Protection

All students were required to return signed parental consent forms in order to participate in this study.

**TABLE 2—Unadjusted and Adjusted Odds Ratios (ORs) From Stepwise Logistic Regression for Ever Smoking: 2125 Middle-School Students, Tracy, Calif, 2003**

|   | Unadjusted OR | CI       | Adjusted OR <sup>a</sup> | CI       |
|---|---------------|----------|--------------------------|----------|
| Grade                                     |               |          |                          |          |
| 6th <sup>b</sup>                          |               |          |                          |          |
| 7th                                       | 2.7           | 2.0, 3.7 | 2.0                      | 1.4, 2.9 |
| 8th                                       | 4.5           | 3.3, 6.1 | 3.0                      | 2.1, 4.3 |
| Gender                                    |               |          |                          |          |
| Female <sup>b</sup>                       |               |          |                          |          |
| Male                                      | 1.1           | 0.9, 1.4 | NS                       |          |
| Ethnicity                                 |               |          |                          |          |
| Not Latino or Hispanic <sup>b</sup>       |               |          |                          |          |
| Latino or Hispanic                        | 1.4           | 1.1, 1.7 | NS                       |          |
| Self-reported grades                      |               |          |                          |          |
| Above median <sup>b</sup>                 |               |          |                          |          |
| Below median                              | 2.7           | 2.2, 3.4 | 1.5                      | 1.2, 2.0 |
| Parent or household smoking               |               |          |                          |          |
| No <sup>b</sup>                           |               |          |                          |          |
| Yes                                       | 2.6           | 2.1, 3.2 | 1.8                      | 1.4, 2.3 |
| At least 1 friend smokes                  |               |          |                          |          |
| No <sup>b</sup>                           |               |          |                          |          |
| Yes                                       | 6.6           | 5.2, 8.3 | 3.1                      | 2.4, 4.0 |
| Maternal supervision                      |               |          |                          |          |
| Above mean <sup>b</sup>                   |               |          |                          |          |
| Below mean                                | 1.9           | 1.5, 2.4 | 1.5                      | 1.1, 1.9 |
| Risk taking                               |               |          |                          |          |
| Below mean <sup>b</sup>                   |               |          |                          |          |
| Above mean                                | 6.7           | 5.1, 8.9 | 3.4                      | 2.5, 4.7 |
| Own cigarette promotional item            |               |          |                          |          |
| No <sup>b</sup>                           |               |          |                          |          |
| Yes                                       | 6.0           | 4.7, 7.8 | 2.7                      | 2.0, 3.6 |
| See cigarette advertisements in magazines |               |          |                          |          |
| Never or rarely <sup>b</sup>              |               |          |                          |          |
| Sometimes or often                        | 2.5           | 2.0, 3.1 | 1.4                      | 1.1, 1.8 |
| See smoking on television or in movies    |               |          |                          |          |
| Never or rarely <sup>b</sup>              |               |          |                          |          |
| Sometimes or often                        | 2.2           | 1.7, 2.8 | NS                       |          |
| Exposure to retail tobacco marketing      |               |          |                          |          |
| <Once/week <sup>b</sup>                   |               |          |                          |          |
| At least once/week                        | 2.8           | 2.1, 3.6 | 1.5                      | 1.1, 2.1 |

Note. CI = confidence interval; NS = not selected by stepwise selection method.

<sup>a</sup>Adjusted OR controls for school differences and all other model variables simultaneously.

<sup>b</sup>Reference category.

The parental consent forms explained the study protocol and participant rights. Students also were given a consent form to read and sign before survey administration. The use of human subjects was approved by the human subjects panel of Stanford University.

## References

1. *Cigarette Report for 2001*. Washington, DC: Federal Trade Commission; 2003.
2. Celebucki CC, Diskin K. A longitudinal study of externally visible cigarette advertising on retail storefronts

in Massachusetts before and after the Master Settlement Agreement. *Tob Control*. 2002;11(suppl 2):ii47–ii53.

3. Wakefield MA, Terry-McElrath YM, Chaloupka FJ, et al. Tobacco industry marketing at point of purchase after the 1998 MSA billboard advertising ban. *Am J Public Health*. 2002;92:937–940.
4. Point-of-Purchase Advertising Institute. *The Point-of-Purchase Advertising Industry Fact Book*. Englewood, NJ: The Point of Purchase Advertising Institute; 1992.
5. Chanil D. Profile of the convenience store customer. *Convenience Store News*. February 11, 2002:54–70.
6. MacFadyen L, Hastings G, MacKintosh AM. Cross

sectional study of young people's awareness of and involvement with tobacco marketing. *BMJ*. 2001;322:513–517.

7. Wakefield MA, Ruel EE, Chaloupka FJ, Slater SJ, Kaufman NJ. Association of point-of-purchase tobacco advertising and promotions with choice of usual brand among teenage smokers. *J Health Commun*. 2002;7:113–121.
8. Donovan RJ, Jancey J, Jones S. Tobacco point of sale advertising increases positive brand user imagery. *Tob Control*. 2002;11:191–194.
9. Henriksen L, Flora JA, Feighery E, Fortmann SP. Effects on youth of exposure to retail tobacco advertising. *J Appl Soc Psychol*. 2002;32:1771–1789.
10. Evans RI, Dratt LM, Raines BE, Rosenberg SS. Social influences on smoking initiation: importance of distinguishing descriptive versus mediating process variables. *J Appl Soc Psychol*. 1988;18(11, pt 1):925–943.
11. Flay BR, Hu FG, Richardson J. Psychosocial predictors of different stages of smoking among high school students. *Prev Med*. 1998;27(5, pt 3):A9–A18.
12. Graham JW, Marks G, Hansen WB. Social influence processes affecting adolescent substance use. *J Appl Psychol*. 1991;76:291–298.
13. Schooler C, Feighery E, Flora J. Seventh graders' self-reported exposure to cigarette marketing and its relationship to their smoking behavior. *Am J Public Health*. 1996;86:1216–1221.
14. Botvin EM, Botvin GJ, Michela JL, et al. Adolescent smoking behavior and the recognition of cigarette advertisements. *J Appl Soc Psychol*. 1991;21:919–932.
15. Unger JB, Cruz TB, Schuster D, Flora JA, Johnson CA. Measuring exposure to pro- and anti-tobacco marketing among adolescents: intercorrelations among measures and associations with smoking status. *J Health Commun*. 2001;6:11–29.
16. Flannery DJ, Williams LL, Vazsonyi AT. Who are they with and what are they doing? Delinquent behavior, substance use, and early adolescents' after-school time. *Am J Orthopsychiatry*. 1999;69:247–253.
17. Mott JA, Crowe PA, Richardson J, Flay B. After-school supervision and adolescent cigarette smoking: contributions of the setting and intensity of after-school self-care. *J Behav Med*. 1999;22:35–58.
18. Jackson C, Henriksen L, Foshee VA. The Authoritative Parenting Index: predicting health risk behaviors among children and adolescents. *Health Educ Behav*. 1998;25:319–337.
19. Chaloupka F, Slater S, Wakefield M. USA: price cuts and point of sale ads follow tax rise. *Tob Control*. 1999;8:242.
20. Slater S, Chaloupka FJ, Wakefield M. State variation in retail promotions and advertising for Marlboro cigarettes. *Tob Control*. 2001;10:337–339.
21. *A Bill to Protect the Public Health by Providing the Food and Drug Administration With Certain Authority to Regulate Tobacco Products*, S2626, 107th Cong, 2nd Sess (2002).
22. *Food and Drug Administration et al. v. Brown & Williamson Tobacco Corp*, F3d 153,155 (US 2000).
23. Gardiner P. A reversal of fortune: the framework convention and federal tobacco policy. *Burning Issues*. 2001;8–11.
24. *Lorillard Tobacco Co. v. Reilly*, 533 US 525, F Supp 2d 76,124 (US 2001).
25. Master Settlement Agreement. Washington, DC: National Association of Attorneys General; December 8, 1998.



# Patient Race/Ethnicity and Quality of Patient–Physician Communication During Medical Visits

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The existence of racial/ethnic health disparities in health care in the United States is largely undisputed.<sup>1</sup> However, the mechanisms through which race/ethnicity contribute to these disparities are complex and are often obscure.<sup>2</sup> The Institute of Medicine report *Unequal Treatment*<sup>3</sup> confirmed that racial/ethnic disparities in health care are not entirely explained by differences in access to care, clinical appropriateness, or patient preferences. Studies have documented differential receipt of technical aspects of care, such as tests, therapies, and procedures, among racial/ethnic minorities compared with Whites, even after control for insurance status and access to a regular primary care provider.<sup>2,3</sup> Access to appropriate treatments and screening tests is less than optimal among all patients who navigate the complex US health system<sup>4</sup>; however, such deficits are magnified among racial/ethnic minorities.<sup>1,2,5,6</sup>

Access to and receipt of appropriate diagnostic, preventive, and therapeutic services and modalities are not the only measures of health care quality. A 1999 report by the Institute of Medicine<sup>7</sup> included patient-centered care prominently among indicators of health care quality. One indicator of patient-centered care—patient–physician communication—has been postulated as a mechanism for racial/ethnic health disparities.<sup>3</sup> It has been associated with patients' perceptions of finding common ground with their physicians<sup>8</sup> and with better health outcomes.<sup>9–11</sup>

Racial/ethnic minorities rate the quality of interpersonal care by physicians and within the health care system in general more negatively than Whites.<sup>5,12–20</sup> However, research that uses objective measures of patient–physician interactions, rather than patient reports, is lacking. We conducted a study in which patient–physician communication during primary care visits of African American and White patients was directly observed. We hypothesized that African American patients

**Objectives.** We examined the association between patient race/ethnicity and patient–physician communication during medical visits.

**Methods.** We used audiotape and questionnaire data collected in 1998 and 2002 to determine whether the quality of medical-visit communication differs among African American versus White patients. We analyzed data from 458 African American and White patients who visited 61 physicians in the Baltimore, Md–Washington, DC–Northern Virginia metropolitan area. Outcome measures that assessed the communication process, patient-centeredness, and emotional tone (affect) of the medical visit were derived from audiotapes coded by independent raters.

**Results.** Physicians were 23% more verbally dominant and engaged in 33% less patient-centered communication with African American patients than with White patients. Furthermore, both African American patients and their physicians exhibited lower levels of positive affect than White patients and their physicians did.

**Conclusions.** Patient–physician communication during medical visits differs among African American versus White patients. Interventions that increase physicians' patient-centeredness and awareness of affective cues with African American patients and that activate African American patients to participate in their health care are important strategies for addressing racial/ethnic disparities in health care. (*Am J Public Health*. 2004;94:2084–2090)

would experience poorer-quality medical-visit communication than White patients.

## METHODS

### Study Design and Population

We compiled data from 2 brief cohort studies conducted between July 1998 and June 1999 and between January 2002 and November 2002. Data collection for the 1998 study has been described in detail elsewhere.<sup>21</sup> The study procedures were reviewed and approved by the institutional review board of the Johns Hopkins Medical Institutions. Physicians were recruited from group practices and from federally qualified health centers that served both managed care and fee-for-service patients in the Baltimore, Md/Washington, DC/Northern Virginia metropolitan area. After meetings with practice leaders, physicians who delivered primary care to patients at least 30 hours per week were invited to participate in the studies via letters and follow-up telephone calls. Both studies attempted to target practices that had a high percentage of African American physi-

cians and patients. Hispanic and Asian physicians were not included in the 1998 study, which focused specifically on issues of patient–physician race concordance and communication, but they were included in the 2002 study, which examined the association between race/ethnicity and patient–physician communication in broader terms.

Patient recruitment took place over an average of 1 to 2 days for each physician. Research assistants attempted to approach all patients during each recruitment day, with a target of 10 patient participants per physician. Patients were eligible to participate if they (1) were aged 18 years or older, (2) were seeing their physician on the recruitment days, and (3) self-identified their race/ethnicity as White or African American. When patients appeared or reported themselves to be too acutely ill or cognitively impaired to participate in the interview, they were not recruited.

### Data Collection

Patients and physicians provided informed consent and were told that the goal of each

study was to learn more about how doctors and patients communicate with one another. Patients in both cohorts completed a 5-minute survey that included questions about health status and demographics. Research assistants set up a tape recorder in the physician's office, started the recording, and left the room; physicians and patients were instructed that they could turn off the tape recorder or pause the recording at any time during the visit. Physicians in both studies completed a background questionnaire about their demographics and a post-visit questionnaire that included a question about how well the physician knew a given patient.

Audiotaped medical visits were coded with the Roter Interaction Analysis System (RIAS), a widely used coding system for medical encounters that has documented reliability and predictive validity.<sup>22–24</sup> The RIAS provides a framework for understanding the communication dynamics between patients and physicians during a medical visit. The 37 exhaustive and mutually exclusive RIAS categories capture a complete thought that is expressed by either the patient or the physician (referred to as an *utterance* or *unit of talk*). These categories group elements of exchange that reflect socioemotional communication (i.e., positive, negative, emotional, partnership building, and social exchanges) and task-focused communication (i.e., asking questions, giving instruction and direction, and giving information).<sup>25</sup> In this way, the system captures 4 primary functions of the medical visit: data gathering, patient education and counseling, responding to patient emotions, and partnership building.<sup>22</sup> In addition to the categorization of verbal communication, coders are asked to rate the global affect (emotional context) of the patient and the physician on each audiotape across several dimensions on a numeric scale of 1 (low/none) to 6 (high).

The same 2 experienced RIAS coders were responsible for coding all medical-visit data collected between 1998 and 1999 and during 2002. The coders, both of whom were White women, were not told the race/ethnicity of patients or physicians, but they may have made assumptions about race/ethnicity and other characteristics on the basis of auditory cues. In all cases, they were unaware of the study hypotheses and did not have access to

questionnaire data or demographics about patients or physicians.

Reliability for the RIAS coding was assessed separately for the 2 studies. Inter-coder reliability for talk categories and percent agreement for affect ratings in the 2002 study were similar to those for the 1998/99 study.<sup>21</sup> The overall average intercoder reliability across all categories was 0.88 for physician talk (range=0.32–1.00) and 0.79 for patient talk (range=0.06–1.00). Only communication categories that occurred at low frequencies (i.e., with an average of <1 statement per visit) had reliability coefficients that fell below 0.70. Coder agreement within 1 point on each dimension of patient and physician positive affect (9 dimensions for patients and 8 for physicians) ranged from 78% to 100%.

### Measures of Communication

Patient–physician communication during medical visits was the main study outcome and was assessed with measures derived from analysis of RIAS-coded audiotape data. We evaluated 3 aspects of the medical visit (measures are listed in parentheses): (1) overall process (duration of visit and average speech speed), (2) patient-centered orientation (physician verbal dominance and physician patient-centeredness scores), and (3) overall emotional tone (patient and physician positive-affect scores).

*Visit duration* was the amount of time in minutes that transpired from the start of the medical visit until its conclusion. *Speech speed* reflected the average number of complete statements expressed per minute and was calculated by dividing the total number of patient and physician statements by the duration of the medical visit in minutes.

The *physician verbal dominance* score was calculated by dividing the total number of physician statements by the total number of patient statements.<sup>23,24</sup> A value greater than 1 denoted relatively more physician than patient talk, while a value less than 1 denoted relatively more patient than physician talk. The *physician patient-centeredness* score was calculated by dividing the total amount of socioemotional talk and questions asked by the patient by all the biomedical elements of talk during the course of a medical visit. Socioemotional talk included all partnership-

building, emotional, and psychosocial elements of exchange (i.e., physician open-ended questions and psychosocial information and counseling and relationship building, positive talk, negative talk, and social talk by physicians and patients), while biomedical talk reflected the task-oriented elements of the exchange (i.e., physician and patient talk aimed at conveying biomedical information and counseling, statements of orientation, and physician closed-ended questions).<sup>24,26</sup>

Positive-affect scale scores were calculated by summing coders' ratings for patients and physicians (separately) on several dimensions. The *patient positive-affect score* was the sum of coders' ratings of dominance/assertiveness, interest/attentiveness, friendliness/warmth, responsiveness/engagement, and sympathy/empathy exhibited by the patient during the visit. The *physician positive-affect score* was the sum of coders' ratings of interest/attentiveness, friendliness/warmth, responsiveness/engagement, and sympathy/empathy exhibited by the physician during each medical visit and the degree to which the physician sounded hurried or rushed was subtracted. Interitem reliability (Cronbach  $\alpha$ ) for both scales was high (patient positive affect  $\alpha$ =0.83; physician positive affect  $\alpha$ =0.91).

### Patient and Physician Characteristics

The independent variable in our study was patient race/ethnicity. While patients self-identified as a member of 1 of 6 racial/ethnic groups (Asian, Latino/Latin American or Hispanic, Native American/American Indian or Indigenous People, Pacific Islander, Black/African American, and White), there were insufficient numbers of patients who identified themselves as anything other than White or Black/African American for meaningful statistical analysis, and those patients were excluded from the final sample ( $n=21$ ). Several demographic characteristics of both patients and their physicians were identified for inclusion as possible covariates in multivariate analyses. Patient characteristics included age, gender, educational attainment, self-rated health status (a 1-item question adapted from the Medical Outcomes Study Short-Form),<sup>27</sup> and whether the patient was part of the 1998 or the 2002 cohort. Physician characteristics included age, gender, race/ethnicity, medical

specialty, location of medical school training (US vs international), time since completing postgraduate training (i.e., residency training and fellowship training, if applicable), reported exposure to communication skills training, and the physician's appraisal of how well he or she knew a patient.

### Statistical Analysis

We performed exploratory analyses that associated all covariates with outcome variables. We then performed univariate and multivariate linear regressions to determine the degree to which patient race/ethnicity was associated with medical-visit communication. To identify potential confounders, we performed descriptive analyses with  $\chi^2$  tests for categorical variables and with analyses of variance for continuous variables to associate all patient and physician characteristics with patient race/ethnicity; univariate linear regression analyses were performed to associate all characteristics with outcome measures. Patient and physician characteristics were included in the multivariate models if they were statistically significantly associated with patient race/ethnicity and at least 1 of the outcome measures or if there was substantial evidence from the existing literature that these factors were potential confounders of the relationships under investigation.

We used the generalized estimating equation method for correlated data<sup>28</sup> in all regression analyses to account for nonindependence across observations, because the same physician was involved in several patients' audiotaped medical visits. An exchangeable correlation structure was assumed with strongly consistent estimation, which was likely to yield more accurate or valid coefficient estimates, even if the correct correlation structure was specified incorrectly.<sup>29</sup> There were 29 sites that had an average of only 2 physicians per site, and some physicians practiced at more than 1 site. Therefore, analyses were designed to account for intraclass correlation within physicians but not within sites. We present 1 multivariate model that included both physician and patient demographic characteristics as covariates, because models in which these characteristics were entered in separate blocks yielded similar results.

## RESULTS

### Recruitment and Sample Characteristics

Of the 132 physicians invited to participate in the 1998 and 2002 studies, 63 (48%) agreed to do so. Two physicians (3%) were dropped from the study because of scheduling and patient recruitment difficulties. Data were collected for 30 White, 21 African American, 9 Asian or Indian American, and 1 other race/ethnicity physicians ( $n=61$ ).

Seven hundred and eighty-nine patients were approached in physician waiting rooms, 22 (3%) of whom were ineligible. Of the 767 eligible patients, 197 (26%) declined participation or were too ill to complete the survey. Of the 570 (74%) patients who were eligible and who were willing to participate, 70 (12%) had inadequate or missing audiotape data, 21 (4%) were missing data for 1 or more questionnaires, and 21 (4%) reported their race/ethnicity to be other than African American or White and thus were excluded from analyses ( $n=112$ ).

Data for 458 patients who were seen by 1 of 61 physicians recruited during the 1998 ( $n=252$  patients,  $n=31$  physicians) and 2002 ( $n=206$  patients,  $n=30$  physicians) studies were included in our analyses (Table 1). The mean age of patients was approximately 50 years. The mean educational attainment was roughly equivalent to receiving a high school diploma. Two thirds of study patients were female; two thirds reported their health status as good, very good, or excellent; more than one third visited male physicians; half visited White physicians; 72% visited internists; 78% visited US medical graduates; and half visited physicians who reported having some communication skills training. On average, patients visited physicians who had finished training more than 9 years before the study period.

There were statistically significant differences between the White and African American patients. Compared with White patients, African American patients were younger, more likely to be seen by female and family physicians, and less likely to see physicians of their same race/ethnicity. African American patients also were more likely to be seen by physicians who had less experience (i.e.,

fewer years since completing training) (Table 1).

### Quality of Medical-Visit Communication

Table 2 shows the results, associating patient race/ethnicity with measures of medical-visit communication (visit duration and speech speed), patient-centered orientation (physician verbal dominance and patient-centeredness scores), and emotional tone (physician and patient positive-affect scores).

*Communication process.* There were no statistically significant differences in duration or speech speed when medical visits of African American and White patients were compared.

*Patient-centered orientation.* Physicians were more verbally dominant with African American than with White patients; they talked 43% (95% confidence interval [CI]=34, 53) more than African American patients and only 24% (95% CI=16, 32) more than White patients. Similarly, physicians' medical visits with African American patients were less patient-centered than their visits with White patients (1.02 [95% CI=0.89, 1.14] for African Americans vs 1.31 [95% CI=1.02, 1.60] for Whites). Racial/ethnic differences in physician verbal dominance remained statistically significant after we controlled for patient and physician demographic characteristics and how well the physician knew the patient (1.73 [95% CI=1.20, 2.26] for African American patients vs 1.50 [95% CI=0.98, 2.01] for White patients). Racial/ethnic differences in the patient-centeredness of medical visits showed the same pattern in both adjusted and unadjusted analyses (patient-centeredness score=1.91 [95% CI=0.76, 3.07] for White patients vs 1.58 [95% CI=0.68, 2.48] for African American patients) but were not statistically significant ( $P=.08$ ) after adjustment for demographic characteristics.

*Emotional tone.* In the univariate analyses, coders' average ratings of positive affect for African American patients were lower than those for White patients (16.50 [95% CI=16.09, 16.92] vs 17.59 [95% CI=17.23, 17.96], respectively). Similarly, coders rated physicians' affective tone as less positive during medical visits with African American patients than with White patients (11.90 [95% CI=11.26, 12.55] vs 12.68 [95%

**TABLE 1—Patient and Physician Demographics by Patient Race/Ethnicity: Baltimore, Md–Washington, DC–Northern Virginia Metropolitan Area, July 1998–June 1999 and January–November 2000**

|   | Total<br>(N = 458) <sup>a</sup> | White Patients<br>(n = 202) <sup>a</sup> | African American<br>Patients (n = 256) <sup>a</sup> | P <sup>b</sup> |
|---|---------------------------------|--|---|----------------|
| <b>Time of medical visit and questionnaire completion</b> |                                 |  |   |                |
| 1998 cohort (%)   | 252 (55)                        | 110 (54)                                 | 142 (55)  |                |
| 2002 cohort (%)   | 206 (45)                        | 92 (46)                                  | 114 (45)  | .83            |
| <b>Patients</b>   |                                 |  |   |                |
| Mean age (SD)   | 49.26 (16.53)                   | 53.03 (17.77)                            | 46.56 (15.04)                                       | <.01           |
| Gender (%)  |                                 |  |   |                |
| Male  | 152 (33)                        | 72 (36)                                  | 80 (31)   | .32            |
| Female  | 306 (67)                        | 130 (64)                                 | 176 (69)  |                |
| Mean years of education (SD)                              | 12.39 (2.62)                    | 12.61 (2.79)                             | 12.22 (2.47)  | .11            |
| Self-rated health status (%)                              |                                 |  |   |                |
| Poor/fair   | 138 (30)                        | 52 (26)                                  | 86 (34)   | .16            |
| Good  | 180 (39)                        | 87 (43)                                  | 93 (36)   |                |
| Very good/excellent                                       | 138 (30)                        | 62 (31)                                  | 76 (30)   |                |
| How well physician knows patient (%)                      |                                 |  |   |                |
| Very well   | 167 (44)                        | 83 (46)                                  | 84 (41)   | .57            |
| Somewhat  | 144 (38)                        | 63 (35)                                  | 81 (40)   |                |
| Not at all (new patient)                                  | 72 (19)                         | 33 (18)                                  | 39 (19)   |                |
| <b>Physicians</b>   |                                 |  |   |                |
| Gender (%)  |                                 |  |   |                |
| Male  | 176 (38)                        | 94 (47)                                  | 82 (32)   | <.01           |
| Female  | 282 (62)                        | 108 (53)                                 | 174 (68)  |                |
| Race/ethnicity (%)  |                                 |  |   |                |
| White   | 234 (51)                        | 141 (70)                                 | 93 (36)   | <.01           |
| African American  | 157 (34)                        | 35 (17)                                  | 122 (48)  |                |
| Asian   | 63 (14)                         | 26 (13)                                  | 37 (14)   |                |
| Other   | 4 (1)                           | 0 (0)                                    | 4 (2)   |                |
| Specialty (%)   |                                 |  |   |                |
| Internal medicine   | 313 (72)                        | 153 (78)                                 | 160 (66)  | <.01           |
| Family practice   | 124 (28)                        | 42 (22)                                  | 82 (34)   |                |
| Location of medical training (%)                          |                                 |  |   |                |
| United States   | 310 (78)                        | 142 (83)                                 | 168 (75)  | .07            |
| International   | 86 (22)                         | 30 (17)                                  | 56 (25)   |                |
| Has had communication skills training                     |                                 |  |   |                |
| Yes   | 230 (53)                        | 103 (53)                                 | 127 (52)  | .94            |
| No  | 207 (47)                        | 92 (47)                                  | 115 (48)  |                |
| Mean (SD) time since completed training (y)               | 9.5 (8.1)                       | 11.21 (8.27)                             | 8.48 (6.76)   | <.01           |

<sup>a</sup>The sample sizes reflect the total number of patient participants. The actual sample size was lower for certain characteristics because of patient nonresponse (patient age, educational attainment, and self-rated health status) and physician nonresponse (previous communication skills training, time since completing training, location of medical school training, and self-rated appraisal of how well physician knows a patient).

<sup>b</sup>Differences across patient groups were analyzed with  $\chi^2$  statistics for categorical variables and with analysis of variance for continuous variables. Some numbers may not add up to the total number owing to missing data for certain variables.

respectively), and the overall positive affect of physicians was rated lower when they were with African American patients than when they were with White patients (13.19 [95% CI= 10.56, 15.82] vs 14.12 [95% CI= 11.48, 16.75], respectively).

## DISCUSSION

Our study showed objective differences in the quality of physician–patient communication among African American and White patients that mirror previously documented differences in patients' perceptions of their quality of health care.<sup>5,12–20</sup> Physicians were more verbally dominant and tended to be less patient centered in their approach with African American patients than with White patients. Previous studies have shown that both verbal dominance and patient centeredness are sensitive markers of interaction dynamics; patient-centered visits are more consistently marked by active patient participation in the medical dialogue and by less physician verbal dominance.<sup>23,30</sup> Our study suggests that patient engagement and participation, rather than overall time spent, during medical visits may be contributing to health disparities. Current concerns about the ever-increasing time pressure on physicians make this our finding particularly relevant.<sup>31</sup>

Our finding that visits with African American patients showed less positive affect—on the part of both patients and physicians—than visits with White patients provides some insight into the subtle nature of emotional reciprocity. Affect is conveyed primarily through voice tone and can be considered the unspoken subtext of the medical dialogue.<sup>32</sup> A 2002 study in the Netherlands similarly reported lower levels of positive affect among both patients and physicians during the visits of racial/ethnic minority patients compared with the visits of native-born Dutch patients.<sup>33</sup>

Patient-centered communication, including greater patient input into the medical dialogue, has been associated with better patient recall of information, treatment adherence, satisfaction with care, and health outcomes.<sup>11,23,25</sup> While less is known about the association between medical-visit affective tone and health outcomes, there is evidence that global affect ratings are a reliable indica-

CI= 11.91, 13.45], respectively). Even after we controlled for demographic characteristics, the overall positive affect of African

American patients was rated lower than that of White patients (15.77 [95% CI= 13.47, 18.06] vs 16.65 [95% CI= 14.31, 18.99],



**TABLE 2—Association Between Patient Race/Ethnicity and Medical-Visit Communication Quality: Baltimore, Md–Washington, DC–Northern Virginia Metropolitan Area, July 1998–June 1999 and January–November 2000**

|  | White Patients<br>(n = 202) <sup>a</sup><br>Mean (95% CI) | African American<br>Patients (n = 256) <sup>a</sup><br>Mean (95% CI) | P <sup>b</sup> |
|--|---|--|----------------|
| Medical-visit communication process measures |   |  |                |
| Duration of visit, minutes                   |   |  |                |
| Univariate model                             | 15.91 (14.36, 17.47)                                      | 15.27 (13.84, 16.71)   | .46            |
| Multivariate model <sup>c</sup>              | 9.64 (2.01, 17.28)  | 9.01 (1.97, 16.05)   | .59            |
| Speech speed <sup>d</sup>                    |   |  |                |
| Univariate model                             | 23.22 (22.17, 24.28)                                      | 22.81 (12.71, 23.90)   | .38            |
| Multivariate model <sup>c</sup>              | 19.91 (14.96, 24.86)                                      | 19.90 (15.08, 24.72)   | .98            |
| Measures of patient-centered communication   |   |  |                |
| Physician verbal dominance <sup>e</sup>      |   |  |                |
| Univariate model                             | 1.24 (1.16, 1.32)   | 1.43 (1.34, 1.53)  | <.001          |
| Multivariate model <sup>c</sup>              | 1.50 (0.98, 2.01)   | 1.73 (1.20, 2.26)  | <.001          |
| Physician patient-centeredness <sup>f</sup>  |   |  |                |
| Univariate model                             | 1.31 (1.02, 1.60)   | 1.02 (0.89, 1.14)  | <.05           |
| Multivariate model <sup>c</sup>              | 1.91 (0.76, 3.07)   | 1.58 (0.68, 2.48)  | .08            |
| Measures of medical-visit affective tone     |   |  |                |
| Patient positive-affect score                |   |  |                |
| Univariate model                             | 17.59 (17.23, 17.96)                                      | 16.50 (16.09, 16.92)   | <.001          |
| Multivariate model <sup>c</sup>              | 16.65 (14.31, 18.99)                                      | 15.77 (13.47, 18.06)   | <.001          |
| Physician positive-affect score              |   |  |                |
| Univariate model                             | 12.68 (11.91, 13.45)                                      | 11.90 (11.26, 12.55)   | .02            |
| Multivariate model <sup>c</sup>              | 14.12 (11.48, 16.75)                                      | 13.19 (10.56, 15.82)   | .02            |

Note. CI = confidence interval.

<sup>a</sup>The sample sizes reflect the number of observations included in most univariate analyses (to within 1%). Multivariate analyses included approximately 28% fewer cases than univariate analyses.

<sup>b</sup>Derived from generalized estimating equations.

<sup>c</sup>The multivariate model included patient demographics (age, gender, years of education, and self-rated health status) and physician demographics (gender, race, time since completing training, and self-rated appraisal of how well physician knows a patient) as covariates.

<sup>d</sup>Speech speed was computed by summing the total number of patient and physician utterances and dividing by the length of the visit in minutes.

<sup>e</sup>Verbal dominance score was a ratio of amount of physician talk to amount of patient talk.

<sup>f</sup>Patient-centered interviewing score was a ratio of amount of psychosocial and socioemotional talk to amount of biomedical talk.

patients who presented for care from participating physicians on recruitment days, different information might have been obtained if all the patients of a practice had been enrolled. To the extent that findings from our research are consistent with other work that has associated patient race/ethnicity with measures of health care quality, our confidence that the findings reflect truly observed phenomena was increased. Furthermore, insofar as one might expect patients and physicians who are willing to participate in this type of research to be more interested in communication issues or to be more likely to have had positive experiences with the health care system in the past, it is possible that disparities evident under “best scenario” conditions would be even greater among the general population of patients and physicians.

The third potential limitation is that it is possible that confounding by demographic characteristics of physicians (i.e., specialty), patients (i.e., reason for visit), or audiotape coders (i.e., race/ethnicity) could account for our findings. Although African American patients in our sample were more likely than White patients to see family physicians, physician specialty was not related to any of the communication outcomes. One recent study showed that family physicians were more patient-centered with minority patients than general internists were; however, a bias based on specialty in our sample would act to diminish the observed difference between African American and White patients rather than enhance it.<sup>36</sup> We did not collect information about the reason for each medical visit; however, we did control for patients’ self-reported health status, which likely minimized any communication differences associated with the reason for medical visits. Finally, audiotape coders’ interpretation of auditory cues may have introduced systematic bias into affect ratings or categorization of communication content. Both coders were White women who may not have been sensitive to cultural differences in expressions of positive affect by the racial/ethnic minority patients or the physicians. The demonstrated interrater reliability in our study—and across studies that have used the RIAS—and the fact that the coders were not aware of the study hypotheses minimizes the likelihood of such bias.

tor of the emotional context of the medical visit. Furthermore, these ratings are associated with patient satisfaction and return visits.<sup>32</sup> Positive affective evaluations of physician behavior also have been associated with mutual liking<sup>33</sup> between the physician and the patient and with a lower likelihood that a patient would consider changing physicians over a 1-year period.<sup>34</sup>

There are some potential limitations to our study. First, the generalizability of the physician and patient populations may be limited. Approximately 50% of the recruited physicians participated, and these physicians may

be different from their peers in important ways. Compared with a statewide sample of primary care physicians in Maryland (in which minorities were oversampled), the physicians in our study were similar with regard to practice settings but were younger, had fewer years in clinical practice, were somewhat more likely to be trained in the United States, and were more likely to be women.<sup>35</sup> The second potential limitation is that only patients who were willing to complete questionnaires and have their medical visit audiotaped participated in the study. Although research assistants attempted to recruit all



Our study is among the few<sup>23,37</sup> that have assessed the association of patient race/ethnicity with the empirical measures of communication during medical visits in primary care practices. Our findings show that racial/ethnic differences are evident within communication domains that have been associated with clinical outcomes in previous studies. Therefore, racial/ethnic differences in these processes of care may contribute to disparities in health. However, this question will be better addressed when researchers can better quantify the effect of medical-visit communication differentials on clinical outcomes among racial/ethnic minority and White patient populations.

Our study also demonstrates the need for further examination of the mechanisms through which race/ethnicity has an impact on communication during medical visits. For example, future research should identify important similarities between physicians and patients on the basis of the social and personal meaning attributed to race/ethnicity. Such research should employ novel methods for evaluating the role of physician bias and should include measures of patient expectations, beliefs, and preferred roles. Future research also should include assessments of nationally representative samples of patients and physicians and should be expanded to include adequate numbers of Hispanic, Asian, and other racial/ethnic minority patients and physicians.

Our study also has implications for the education and training of health care professionals and the interventions targeted at patients. Along with previous studies, our findings show that communication skills programs for medical students, residents, and practicing physicians that focus on patient-centeredness and affective dimensions of care (i.e., building rapport) will benefit patients in general and racial/ethnic minority patients in particular. We have built on previous research that has associated patient activation with improved health outcomes,<sup>11</sup> and our work suggests that empowering African American patients through interventions that build confidence and competence for active participation in health care may be an important strategy for overcoming racial/ethnic disparities in health care and, subsequently, in health status. ■

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### Contributors

The study was originated and designed by R.L. Johnson and L.A. Cooper, with substantial input from D. Roter and N.R. Powe. R.L. Johnson performed the analyses described, and prepared the article. L.A. Cooper obtained the funding and served as the principal investigator for both studies that provided data for analysis. All authors contributed substantially to editing the article.

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### Human Participant Protection

The research protocols for both the 1998 and 2002 studies included in these analyses were reviewed and approved by the institutional review board of the Johns Hopkins Medical Institutions. The protocol for the secondary data analysis presented in this article was reviewed and approved separately by the committee for human research of the Johns Hopkins Bloomberg School of Public Health.

### References

1. LaVeist T, ed. *Race, Ethnicity, and Health: A Public Health Reader*. San Francisco, Calif: Josey-Bass; 2002.
2. Mayberry RM, Mili F, Ofili E. Racial and ethnic differences in access to medical care. *Med Care Res Rev*. 2000;57 (suppl 1):108-145.
3. Smedley BD, Stith AY, Nelson AR. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care*. Washington, DC: National Academy Press; 2002.
4. McGlynn EA, Asch SM, Adams J, et al. The quality of health care delivered to adults in the United States. *N Engl J Med*. 2003;348:2635-2645.
5. Collins KS, Hughes DL, Doty MM, Ives BL, Edwards JN, Tenney K. *Diverse Communities, Common Concerns: Assessing Health Care Quality for Minority Americans. Findings from the Commonwealth Fund 2001*

*Health Care Quality Survey*. New York, NY: The Commonwealth Fund; 2002.

6. van Ryn M, Fu SS. Paved with good intentions: do public health and human service providers contribute to racial/ethnic disparities in health? *Am J Public Health*. 2003;93:248-255.
7. Committee on Health Care in America Institute of Medicine. *Crossing the Quality Chasm: A New Health System for the 21st Century*. Washington, DC: National Academy Press; 2001.
8. Stewart M, Brown JB, Donner A, et al. The impact of patient-centered care on outcomes. *J Fam Pract*. 2000;49:796-804.
9. Little P, Everitt H, Williamson I, et al. Observational study of effect of patient centredness and positive approach on outcomes of general practice consultations. *BMJ*. 2001;323:908-911.
10. Kaplan SH, Greenfield S, Ware JE Jr. Assessing the effects of physician-patient interactions on the outcomes of chronic disease. *Med Care*. 1989; 27:S110-S127.
11. Stewart MA. Effective physician-patient communication and health outcomes: a review. *CMAJ*. 1995; 152:1423-1433.
12. Blendon RJ, Scheck AC, Donelan K, et al. How White and African Americans view their health and social problems. *JAMA*. 1995;273:341-346.
13. Gross DA, Zyzanski SJ, Borawski EA, Cebul RD, Stange KC. Patient satisfaction with time spent with their physician. *J Fam Pract*. 1998;47:133-137.
14. Cooper-Patrick L, Gallo JJ, Gonzales JJ, et al. Race, gender, and partnership in the patient-physician relationship. *JAMA*. 1999;282:583-589.
15. Saha S, Komaromy M, Koepsell TD, Bindman AB. Patient-physician racial concordance and the perceived quality and use of health care. *Arch Intern Med*. 1999; 159:997-1004.
16. Carrasquillo O, Orav EJ, Brennan TA, Burstin HR. Impact of language barriers on patient satisfaction in an emergency department. *J Gen Intern Med*. 1999;14: 82-87.
17. Malat J. Social distance and patients' rating of healthcare providers. *J Health Soc Behav*. 2001;42: 360-372.
18. Collins TC, Clark JA, Petersen LA, Kressin NR. Racial differences in how patients perceive physician communication regarding cardiac testing. *Med Care*. 2002; 40:127-134.
19. Cooper LA, Roter DL. Patient-provider communication: the effect of race and ethnicity on process and outcomes of healthcare. In: Smedley BD, Stith AY, Nelson AR, eds. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Healthcare*. Washington, DC: National Academy Press; 2002:552-593.
20. Johnson RL, Saha S, Arbelaez JJ, Beach MC, Cooper LA. Racial and ethnic differences in patient perceptions of bias and cultural competence in health care. *J Gen Intern Med*. 2004;19:101-110.
21. Cooper LA, Roter DL, Johnson RL, Ford DE, Steinwachs DM, Powe NR. Patient-centered communication, ratings of care, and concordance of patient and physician race. *Ann Intern Med*. 2003;139:907-915.
22. Roter DL, Larson S. The relationship between residents' and attending physicians' communication during

primary care visits: an illustrative use of the Roter Interaction Analysis System. *Health Commun.* 2001;13:33–48.

23. Roter DL, Stewart M, Putnam SM, Lipkin M Jr, Stiles W, Inui TS. Communication patterns of primary care physicians. *JAMA.* 1997;277:350–356.

24. Mead N, Bower P. Measuring patient-centredness: a comparison of three observation-based instruments. *Patient Educ Couns.* 2000;39:71–80.

25. Hall JA, Roter DL, Katz NR. Meta-analysis of correlates of provider behavior in medical encounters. *Med Care.* 1988;26:657–675.

26. Wissow LS, Roter D, Bauman LJ, et al. Patient–provider communication during the emergency department care of children with asthma. The National Cooperative Inner-City Asthma Study, National Institute of Allergy and Infectious Diseases, NIH, Bethesda, MD. *Med Care.* 1998;36:1439–1450.

27. Ware J Jr, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care.* 1996;34:220–233.

28. Liang KY, Zeger SL. Regression analysis for correlated data. *Annu Rev Public Health.* 1993;14:43–68.

29. Diggle PJ, Heagerty P, Liang K, Zeger SL. *Analysis of Longitudinal Data*. 2nd ed. Oxford, UK: Oxford University Press; 2002.

30. Roter DL, Hall JA. *Doctors Talking to Patients/Patients Talking to Doctors: Improving Communication in Medical Visits*. Westport, Conn: Auburn House. 1992.

31. Mechanic D, McAlpine DD, Rosenthal M. Are patients' office visits with physicians getting shorter? *N Engl J Med.* 2001;334:198–204.

32. Hall JA, Roter DL, Rand CS. Communication of affect between patient and physician. *J Health Soc Behav.* 1981;22:18–30.

33. van Wieringen JC, Harmsen JA, Bruijnzeels MA. Intercultural communication in general practice. *Eur J Public Health.* 2002;12:63–68.

34. Hall JA, Horgan TG, Stein TS, Roter DL. Liking in the physician–patient relationship. *Patient Educ Couns.* 2002;48:69–77.

35. *The Maryland Study on Physician Experience with Managed Care*. Baltimore, Md: University of Maryland Baltimore County, Center for Health Program Development and Management; 2001.

36. Paasche-Orlow M, Roter D. The communication patterns of internal medicine and family practice physicians. *J Am Board Fam Pract.* 2003;16:485–493.

37. Oliver MN, Goodwin MA, Gotler RS, Gregory PM, Stange KC. Time use in clinical encounters: are African American patients treated differently? *J Natl Med Assoc.* 2001;93:380–385.



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PHIn12J1

# Racial Differences in Cardiac Catheterization as a Function of Patients' Beliefs

Nancy R. Kressin, PhD, Bei-Hung Chang, ScD, Jeff Whittle, MD, MPH, Eric D. Peterson, MD, MPH, Jack A. Clark, PhD, Amy K. Rosen, PhD, Michelle Orner, MPH, Tracie C. Collins, MD, MPH, Linda G. Alley, PhD, RN, and Laura A. Petersen, MD, MPH

Racial disparities in invasive cardiac procedure use have been widely documented, yet the reasons for these disparities remain unclear.<sup>1–4</sup> Numerous studies have examined this issue in multiple settings, carefully controlling for sources of variation, but the differences remain unexplained.<sup>1</sup> Thus, the Institute of Medicine report *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care* called for further research to identify the sources of disparities in health care,<sup>2</sup> specifically mentioning the need for further studies to understand patient-level influences on care and the contribution of physicians' perceptions of patients to the care provided.

Indeed, many researchers have speculated that patients' attitudes and health-related beliefs may be associated with racial disparities in the use of procedures.<sup>5–9</sup> African Americans are less likely than Whites to report hypothetical willingness to undergo revascularization,<sup>10</sup> and African American cardiac patients are less satisfied with their care and more likely to perceive racism and to mistrust the medical care system than are White patients.<sup>11</sup> By contrast, in our own prior work with a subset of the sample used in the present study, we found few racial differences in numerous beliefs and attitudes related to cardiac catheterization (C-CATH).<sup>12</sup> Others have examined whether patients' preferences, as evidenced by their refusing to use invasive cardiac procedures, were associated with racial disparities in the use of procedures but found conflicting results.<sup>13–17</sup>

Thus, the literature lacks comprehensive studies that simultaneously examine patients' and physicians' beliefs and attitudes and their potential association with actual racial variations in invasive cardiac procedure use while also controlling for sociodemographic and clinical variables. The purpose of this study was to examine whether

**Objectives.** We examined racial differences in cardiac catheterization rates and reviewed whether patients' beliefs or other variables were associated with observed disparities.

**Methods.** We did a prospective observational cohort study of 1045 White and African American patients at 5 Veterans Affairs (VA) medical centers whose nuclear imaging studies indicated reversible cardiac ischemia.

**Results.** There were few demographic differences between White and African American patients in our sample. African Americans were less likely than Whites to undergo cardiac catheterization. African Americans were more likely than Whites to indicate a strong reliance on religion and to report racial and social class discrimination and were less likely to indicate a generalized trust in people but did not differ from White patients on numerous other attitudes about health and health care. Neither sociodemographic or clinical characteristics nor patients' beliefs explained the observed disparities, but physicians' assessments of the procedure's importance and patients' likelihood of coronary disease seemed to account for differences not otherwise explained.

**Conclusions.** Patients' preferences are not the likely source of racial disparities in the use of cardiac catheterization among veterans using VA care, but physicians' assessments warrant further attention. (*Am J Public Health.* 2004;94:2091–2097)

there were racial disparities in the use of C-CATH in our cohort, and if so, to comprehensively examine whether patients' attitudes and beliefs or physicians' assessments and perceptions about patients could explain this racial disparity when we controlled for the effects of clinical and sociodemographic characteristics.

## METHODS

### Study Setting and Sample

The study was conducted at 5 Department of Veterans Affairs (VA) Medical Centers (Houston, Tex, Pittsburgh, Pa, Atlanta, Ga, Durham, NC, and St. Louis, Mo), chosen because of their high percentage of African American patients. These hospitals are large, urban, academically affiliated tertiary care facilities with on-site C-CATH facilities. We focused our study on patients who might be candidates for invasive cardiac procedures, establishing a prospective cohort of patients likely to have coronary artery disease by screening the results of all cardiac nuclear im-

aging studies performed between August 1999 and January 2001.

We considered the nuclear imaging study results positive if there was any evidence of reversible cardiac ischemia (evidenced by reversible defects or redistribution). Patients were ineligible if they had no reversible ischemia; had received C-CATH, any revascularization, or heart transplant surgery in the 6 months before their nuclear imaging study; had participated in a clinical trial that determined their cardiac treatment; were of a race that was not White or African American; were not veterans; or were unable to complete the survey because of cognitive impairment.

We screened 5278 patients, and 2335 (44%) had a positive nuclear imaging study. Of these, 456 patients (20%) were excluded because we were unable to contact them to enroll in the study, 78 (3%) because they had impaired mental status, 32 (1%) because they were in another research study determining their cardiac treatment, 102 (4%) because they were not African American or White,

189 (8%) because they had had a cardiac procedure in the preceding 6 months, 5 (0.02%) because they were not veterans, and 99 (4%) because of miscellaneous other reasons (e.g., the nuclear imaging studies were conducted for compensation and pension evaluation, the patients' hearing was impaired, or the patients died before we could enroll them). After these exclusions, 1374 patients with positive imaging studies remained; of these, 329 refused participation, failed to return their informed consent, or failed to return mailed questionnaires. Thus, 1045 patients were included in the final cohort, representing a 76% response rate from those 1374.

To examine the representativeness of the sample, we compared the 1045 patients with positive nuclear imaging study results who were included in the cohort to those who were not ( $n=1290$ ) and found that there were disproportionately fewer African Americans in the cohort (23% African Americans in the cohort vs 29% in the excluded patients,  $P=.003$ ). There were no differences in marital status or age between these groups.

### Data Collection

**Procedure.** The patient health attitude and belief questions were taken from 2 serially administered questionnaires that included nonoverlapping content: 1 completed within 4 weeks after the patients' nuclear imaging study and 1 completed after the patients reported that they had received the study results. Patients were contacted by the study research assistant either in person or by telephone. For each enrolled patient, the physician who ordered the nuclear study was also asked to complete a survey.

**Patient questionnaire measures.** The study questionnaires assessed self-reported demographic information including the patient's age, race, education, income, and marital status. The questionnaire also included the Seattle Angina Questionnaire, which assesses patients' perceptions of several dimensions of coronary artery disease including anginal stability and frequency.<sup>18</sup> In our previous work with the first 854 patients enrolled in the cohort,<sup>12</sup> we developed psychometrically valid scales to assess patients' beliefs and attitudes about their health and the health

care they received. These scales assess the following specific dimensions: patients' evaluations of physicians' capabilities and interpersonal style (e.g., how well the physician knows the patient both medically and personally, the degree of respect and caring evidenced by physicians, trust in physicians' judgments and qualifications); patients' evaluations of VA care (e.g., degree of respect with which one is treated in the VA system, the quality of care in VA and satisfaction with it), and patients' attitudes toward religion and its role in dealing with cardiac disease and attendant treatment decisionmaking. We included the 4 items that constitute the medical skepticism scale: "I can overcome most illness without help from a medically trained professional," "Home remedies are often better than drugs prescribed by a doctor," "If I get sick, it is my own behavior that determines how soon I get well again," and "I understand my health better than most doctors do."<sup>19</sup> We did not combine these items into a scale because earlier analyses indicated that the scale did not have acceptable psychometric properties in this setting.<sup>12</sup> Finally, in addition to the scales we previously developed, for the present analyses we also included several published scales to assess other dimensions of patients' attitudes and experiences thought to be relevant to racial differences in patients' perceptions of their care: a generalized trust in people,<sup>20</sup> optimism,<sup>21</sup> and prior experiences of racial and social class discrimination.<sup>22</sup> To the racial and social class discrimination scale, which included an item about experiences of discrimination when getting medical care, we added an item referencing VA medical care.

Although all patients were asked to complete both the study questionnaires, the instruments included several planned skip patterns, so that, for example, patients who had not been offered C-CATH were not asked about their beliefs about the procedure. Similarly, patients who denied having heart disease were not asked about their perceptions of its severity. Thus, although in our earlier work we reported data on these latter 2 dimensions from the subset of the sample answering them (approximately 40% of the full cohort), we did not include these scales in the

present article because of the significant amount of missing data in these dimensions, related to the skip patterns.

**Physician questionnaire measures.** We asked each patient's physician to provide overall clinical assessments, including, "How important it is for [your patient] to have cardiac catheterization now?" (response categories: lifesaving or crucial, procedure is not crucial but the benefits are greater than the risks, equivocal, risks are greater than the benefits). To assess the physician's perception of the probability of the patient's having coronary artery disease, we asked, "On a scale from 0%–100%, please estimate the probability of coronary artery disease in this patient (70% or more narrowing of an epicardial artery)."<sup>23</sup> Then we assessed physicians' perceptions about patients' personal characteristics,<sup>24</sup> asking physicians to indicate, on a scale of 1 to 5 (1 indicating the greatest amount of the characteristic, and 5 indicating the least), whether their patient is a good communicator, has high socioeconomic status, is independent, is intelligent, is knowledgeable, is likely to underreport pain or comfort, is likely to show up for follow-up appointments, is likely to comply with medication treatment, and is likely to participate in cardiac rehabilitation, if recommended.

### Clinical and Treatment Variables.

We reviewed the medical records of each study respondent, obtaining records for non-VA care where possible. Trained nurses abstracted patients' demographics, cardiac symptoms, past medical history (including prior myocardial infarction, diabetes, hypertension, congestive heart failure, and renal or lung disease), laboratory values, test findings, procedure utilization, and hospital course, if admitted. As an indication of the extent to which medical therapy had been maximized for each patient, we used the American College of Cardiology/American Heart Association guidelines for coronary angiography and the management of patients with chronic stable angina.<sup>25, 26</sup> Thus, we defined maximal medical therapy as antiplatelet therapy, sublingual nitroglycerin, and at least 1 of the following: beta blockers, calcium channel blockers, or long-acting nitrates. We also included patient-reported indicators of anginal fre-



**TABLE 1—Demographic and Clinical Characteristics, Physicians' Assessments and Perceptions of Patients, and Study Outcome of the Study Cohort, by Race: United States, August 1999–January 2001**

|  | African Americans,<br>n = 236 <sup>a</sup> | Whites,<br>n = 809 <sup>a</sup> | P      |
|--|--|---------------------------------|--------|
| <b>Demographic variables</b>                                       |  |                                 |        |
| Age, y   |  |                                 |        |
| < 65, %  | 57.2                                       | 53.7                            | .59    |
| 65–74, %   | 30.5                                       | 33.9                            |        |
| ≥ 75, %  | 12.3                                       | 12.5                            |        |
| Education  |  |                                 |        |
| < 12 y, %  | 32.8                                       | 27.9                            | .15    |
| 12 y/high school, %  | 30.2                                       | 36.7                            |        |
| > 12 years, %  | 37.0                                       | 35.4                            |        |
| Married, % yes   | 47.9                                       | 62.2                            | .001   |
| <b>Clinical variables</b>  |  |                                 |        |
| Prior revascularization, % yes                                     | 15.0                                       | 34.8                            | <.0001 |
| Prior MI, % yes  | 25.9                                       | 34.0                            | .02    |
| Hypertension, % yes  | 85.4                                       | 76.2                            | .003   |
| Angina, % yes  | 65.8                                       | 64.8                            | .79    |
| Congestive heart failure, % yes                                    | 17.1                                       | 17.9                            | .78    |
| Diabetes, % yes  | 35.6                                       | 31.1                            | .20    |
| Lung disease, % yes  | 18.5                                       | 27.4                            | .006   |
| Renal dysfunction, % yes   | 17.5                                       | 10.2                            | .002   |
| Maximal medical therapy, % yes                                     | 33.5                                       | 36.2                            | .44    |
| SAQ anginal frequency <sup>b</sup>                                 | 72.8                                       | 75.4                            | .19    |
| SAQ anginal stability <sup>b</sup>                                 | 64.9                                       | 69.8                            | .04    |
| <b>Physician variables</b>   |  |                                 |        |
| Physicians' assessments <sup>c</sup>                               |  |                                 |        |
| Importance of catheterization <sup>d</sup>                         | 2.76                                       | 2.59                            | .11    |
| Probability of coronary artery disease, % <sup>e</sup>             | 65.79                                      | 73.81                           | .005   |
| Physicians' perceptions  |  |                                 |        |
| Patient is good communicator <sup>f</sup>                          | 2.36                                       | 2.15                            | .04    |
| Patient has high socioeconomic status <sup>f</sup>                 | 3.34                                       | 3.08                            | .003   |
| Patient is independent <sup>f</sup>                                | 2.10                                       | 2.15                            | .61    |
| Patient is intelligent <sup>f</sup>                                | 2.29                                       | 2.29                            | .93    |
| Patient is knowledgeable <sup>f</sup>                              | 2.58                                       | 2.48                            | .30    |
| Patient underreports pain/discomfort <sup>f</sup>                  | 2.81                                       | 2.71                            | .20    |
| Patient is likely to show up for appointments <sup>f</sup>         | 1.94                                       | 1.78                            | .14    |
| Patient is likely to comply with medication treatment <sup>f</sup> | 2.08                                       | 1.86                            | .04    |
| Patient is likely to participate in cardiac rehab <sup>f</sup>     | 2.07                                       | 2.06                            | .97    |
| <b>Outcome variable</b>  |  |                                 |        |
| Received cardiac catheterization, %                                | 33   | 47                              | .0002  |

Note. MI = myocardial infarction; SAQ = Seattle Angina Questionnaire.

<sup>a</sup> n = all patients for whom data are available.

<sup>b</sup> Variables obtained through patient self-report; higher scores indicate better functional status: less anginal frequency and greater anginal stability.

<sup>c</sup> We included in the model 591 patients with physician data available.

<sup>d</sup> Answer categories as follows: 1 = lifesaving or crucial; 2 = benefits > risks; 3 = equivocal; 4 = risks > benefits.

<sup>e</sup> Answers could range from 0% to 100%.

<sup>f</sup> Answer categories: 1 = to a very great extent; 5 = to a small extent.

quency and anginal stability from the Seattle Angina Questionnaire.<sup>18</sup>

### Statistical Analysis

We examined bivariate associations between race and each of the sociodemographic, clinical, health belief, physicians' assessment, and perception variables using  $\chi^2$  or *t* tests. We then employed 5 sequential logistic regression models to examine the effects of distinct blocks of variables on racial disparities in the use of C-CATH, while accounting for the effect of clustering of patients within the 5 study sites with the SAS (SAS Institute Inc, Cary, NC) macro GLIMMIX to run a mixed effects model. We included site as a random effect in the logistic regression model to account for the site cluster effect such that patients within the same site might share similar characteristics that are associated with C-CATH.<sup>27,28</sup>

The race indicator variable, sociodemographic variables, clinical variables, health belief variables, and physician assessment/perception variables were added into the models in sequence as fixed effect independent variables. The change in the magnitude of the odds ratio of receiving C-CATH for Whites versus African Americans after the inclusion of each set of independent variables indicates the confounding effect of each set of independent variables on the association between race and C-CATH (racial disparity).<sup>29</sup>

## RESULTS

### Characteristics of the Patients

Most of the sample were male (98%); most were younger than 65 years and had at least a high school education (Table 1). African American and White patients did not differ in age or education, but African Americans were less likely to be married. There were no racial differences in income (not shown), and because this variable was not associated with either race or the use of C-CATH, we did not include it in our multivariable analyses.

The majority of patients had not previously received revascularization (70%) and had not had a prior myocardial infarction (68%). However, most patients did have hypertension (78%) or angina (65%). We ex-



**TABLE 2—Patients' Attitudes and Beliefs, by Race<sup>a</sup>**

|  | African Americans,<br>n = 188 |       | Whites, n = 686 |       | P      |
|--|-------------------------------|-------|-----------------|-------|--------|
|  | Mean                          | SD    | Mean            | SD    |        |
| <b>Health attitudes/belief scales<sup>b</sup></b>  |                               |       |                 |       |        |
| Positive evaluations of physicians                 | 75.59                         | 14.85 | 76.84           | 15.82 | .33    |
| Positive evaluation of VA care                     | 75.63                         | 13.47 | 75.56           | 15.43 | .94    |
| Reliance on religion                               | 78.44                         | 19.37 | 68.93           | 22.70 | <.0001 |
| Self-reported disease severity                     | 44.48                         | 37.58 | 46.32           | 37.24 | .55    |
| Trust in people <sup>c</sup>                       | 41.76                         | 38.79 | 60.28           | 39.79 | <.0001 |
| Optimism   | 59.22                         | 16.29 | 59.81           | 17.64 | .68    |
| Class discrimination <sup>d</sup>                  | 19.68                         | 30.20 | 13.82           | 23.55 | .01    |
| Racial discrimination <sup>d</sup>                 | 36.42                         | 32.44 | 5.01            | 12.83 | <.0001 |
| <b>Health belief items<sup>e</sup></b>             |                               |       |                 |       |        |
| I can overcome illness without a professional      | 2.16                          | 1.05  | 2.40            | 1.05  | .006   |
| Home remedies are better                           | 2.28                          | 1.00  | 2.36            | .94   | .29    |
| If I'm sick, my behavior determines the outcome    | 3.37                          | 1.24  | 3.23            | 1.07  | .18    |
| I understand my health better than most doctors do | 2.69                          | 1.23  | 2.69            | 1.07  | .99    |

Note. VA = Department of Veterans Affairs.

<sup>a</sup>Sample (n = 874) includes all patients with complete data on race, sociodemographics, clinical variables, and health belief items and scales. The results using all available data were similar to the results reported here.

<sup>b</sup>Scales range from 0 to 100 with higher values indicating a higher degree of what the statement described.

<sup>c</sup>Higher scores indicate greater trust.

<sup>d</sup>Higher scores indicate more discrimination.

<sup>e</sup>Higher scores indicate greater likelihood of agreeing with the statement, on a scale of 1 to 5.

[OR] = 1.43). After we adjusted for sociodemographic variables, the odds ratio remained almost identical (1.44) in model 2, indicating that sociodemographic variables are not confounded with (do not explain) the racial disparity in the use of C-CATH.

After we added clinical variables into model 2, the odds of Whites versus African Americans receiving C-CATH increased slightly (to 1.64), with increased statistical significance (from  $P < .04$  to  $P < .009$ ).

After we added the health belief variables into model 3, the odds ratio increased slightly (from 1.64 to 1.79) and remained highly significant ( $P = .015$ ). These results indicate that health beliefs do not explain racial variation in the use of C-CATH in our cohort, consistent with the lack of bivariate associations between health belief variables and C-CATH (results not shown).

Finally, we examined whether the physicians' variables explained racial disparities in C-CATH. Because of the limited response rate from physicians (67%), only 591 patients and their physicians (56% of the full sample) were included in this analysis. In model 5, the odds ratio decreased to 1.20 and was no longer significant ( $P = .647$ ). The drop in odds ratio after adding physician variables into the model indicates that physician variables explain some of the racial disparity in C-CATH rates. Physicians' ratings of patients' probabilities of coronary artery disease and the importance of C-CATH both significantly predicted C-CATH. To determine how much of this decrease in odds ratio was due to the inclusion of the physician variables but not to the different analysis sample used, we reran the 5 models with the subsample for which we had physician data and obtained similar results.

## DISCUSSION

In this study, we examined racial differences in the use of cardiac catheterization among VA patients with documented reversible cardiac ischemia. Then we explored whether patients' attitudes or beliefs, or physicians' assessments or perceptions, could explain observed differences, after we controlled for clinical and sociodemographic characteristics.

Patients' health beliefs did not explain the observed racial differences, but physicians'

examined other relevant comorbid conditions, finding a minority of patients with congestive heart failure (18%), diabetes (32%), lung disease (25%), or renal dysfunction (12%). Only slightly more than one third of the sample (36%) were receiving maximal medical treatment.

African American and White patients differed clinically in that African American patients were less likely to have had a prior myocardial infarction ( $P < .05$ ) or to have had revascularization in the past ( $P < .0001$ ) but were more likely to have hypertension ( $P < .01$ ). African Americans were less likely to have lung disease ( $P < .01$ ) but were more likely to experience renal dysfunction than Whites ( $P < .01$ ). African Americans were less likely than Whites to undergo C-CATH (33% vs 47%, respectively;  $P < .001$ ).

### Patients' Attitudes and Beliefs

African Americans more frequently indicated reliance on religion as a way of coping with their heart disease and treatment decisionmaking and indicated less trust in people (Table 2). African American patients were

more likely to report experiences with racial and social class discrimination and were less likely to agree that they could overcome illness without help from a medically trained professional. All  $P$  values were significant at  $\leq .01$ .

### Physicians' Assessments and Perceptions

Physicians felt the necessity (or importance) of C-CATH was slightly greater for White than for Black patients (2.59 vs 2.76;  $P = .11$ ; lower values indicate more importance) (Table 1). Doctors also rated Whites' pretest probability of coronary artery disease higher than African Americans' (74% vs 66%). Physicians rated White patients as better communicators, of higher socioeconomic status, and as more likely to comply with medication treatment than Black patients. All  $P$  values were significant at  $< .05$ .

### Variables Associated With C-CATH

We examined variables that might explain the racial difference in C-CATH through sequential regression models (Table 3). Whites had a higher C-CATH rate than African Americans (model 1) (unadjusted odds ratio

**TABLE 3—Odds Ratios (for Whites Compared With African Americans) of Receiving Cardiac Catheterization After Nuclear Imaging Study, Accounting for Patient Clustering Within Site**

| Model   | Variables  | OR (95% CI)                    | P    |
|---------|--|--------------------------------|------|
| Model 1 | Race only  | 1.43 (1.02, 2.02)              | .040 |
| Model 2 | Race + demographic   | 1.44 <sup>a</sup> (1.01, 2.03) | .041 |
| Model 3 | Race + demographic + clinical  | 1.64 <sup>b</sup> (1.13, 2.39) | .01  |
| Model 4 | Race + demographic + clinical<br>+ patients' health attitudes/beliefs  | 1.79 <sup>c</sup> (1.12, 2.86) | .015 |
| Model 5 | Race + demographic + clinical + patients'<br>health attitudes/beliefs + physicians'<br>assessments and perceptions | 1.20 <sup>d</sup> (0.55, 2.62) | .647 |

Note. Odds ratio = OR; CI = confidence interval. Individual variables included in each block of adjuster variables are listed in Tables 1 and 2.

<sup>a</sup>Significant covariates from this model other than race included age older than 75 years (OR = 0.63,  $P = .05$ ); > 12 years of education (OR = 0.69,  $P = .04$ ).

<sup>b</sup>Significant covariates from this model other than race included hypertension (OR = 1.45,  $P < .05$ ); anginal stability (OR = 0.99,  $P < .04$ ); maximal medical therapy (OR = 1.74,  $P = .001$ ).

<sup>c</sup>Significant covariates from this model other than race included hypertension (OR = 1.46,  $P < .05$ ); anginal stability (OR = 0.99,  $P = .05$ ); maximal medical therapy (OR = 1.73,  $P = .001$ ).

<sup>d</sup>Significant covariates from this model included hypertension (OR = 2.17,  $P < .05$ ); maximal medical therapy (OR = 1.78,  $P < .05$ ); physicians' ratings of patients' probability of coronary artery disease (OR = 1.02,  $P < .01$ ); and importance of receiving cardiac catheterization (OR = 0.23,  $P < .0001$ ).

assessments of patients did explain some of the variation. In particular, physicians' ratings of coronary artery disease and the importance of C-CATH for a patient (both higher for White patients) contributed to the observed racial disparities in C-CATH use beyond what could be attributed to clinical differences identified by chart review. These assessments may have captured the effects of other unmeasured clinical variables, but our inclusion of numerous relevant clinical indicators that physicians rely on to make decisions to send patients to C-CATH minimized this possibility.

We also observed several racial differences in patients' beliefs and attitudes. African American patients indicated a greater reliance on their religion or God in coping with and making decisions about their cardiac treatment and in their lives in general than did White patients. African American patients indicated less generalized trust in people and more experiences of racial and class discrimination. Contrary to prior findings about African American patients not trusting their doctors or the health care system,<sup>11,30</sup> in our study we detected no such differences. However, African American patients were less likely to believe that they could overcome

illness without a professional, suggesting greater reliance on health professionals, but notably, neither this item nor any of the other health beliefs explained the observed racial differences in C-CATH use.

The continued existence of racial disparities in the use of C-CATH is troubling, especially when observed in an equal access system such as the VA. Two decades of research documenting such racial disparities in cardiac procedure use have failed to identify the causes of such disparities,<sup>1</sup> yet our findings are among the first to address an oft-hypothesized source: patient attitudes and beliefs. However, our results significantly extend those of other studies, which have documented racial disparities in cardiac care, by simultaneously examining patient- and physician-based variables that might be associated with such disparities, while controlling for clinical and sociodemographic variables. Although previous studies have documented racial differences in patients' preferences<sup>10</sup> or trust in the medical care system,<sup>11</sup> none have examined the association of such dimensions with actual use of C-CATH, nor has this variety of factors been examined simultaneously. Our findings also echo others regarding racial disparities in renal transplantation, where adjustment for

patients' preferences regarding transplantation did not account for the racial disparities in referral for the procedure.<sup>31</sup>

Because we studied patients cared for in VA facilities, the effects of ability to pay for care, or physicians' financial incentives to recommend or deny procedures, were diminished. Further, the sociodemographic gap between White and African American patients is minimized in this setting.<sup>32</sup> Both variables, as well as different attitudes among veterans or different practice patterns in the VA, may affect patients' and physicians' perceptions and thus limit the generalizability of our findings to non-VA patient populations; however, numerous previous studies have detected similar racial disparities in care in the VA as found in other environments. Further, the VA system cares primarily for male patients, so our results may not be generalizable to women. Strengths of our study include the fact that all study sites had on-site C-CATH facilities, an important determinant of procedure use in the VA and elsewhere.<sup>33,34</sup> Further, unlike many prior studies, we included individual-level controls for sociodemographic characteristics.

African American patients were less well represented in our study cohort than in the eligible population, and some patients did not have complete data for all study measures and were thus not included in some analyses. Thus, the more distrustful patients may not have been included in the sample, minimizing the effects of patients' beliefs on receipt of C-CATH. However, 22% of the sample was African American, which represents a significant oversampling of nearly twice the proportion of African American patients using the VA nationwide, where 13.2% of patients are self-described African Americans.<sup>35</sup> Further, to examine any effects of bias due to sampling issues, we conducted additional regression analyses comparing patients with complete data with those who had partial data and observed similar patterns of findings. Thus, although selection bias may have affected the absolute value of the racial disparities in C-CATH use, it did not affect the conclusions about the confounding effects of sociodemographic, clinical, health belief, or physician variables on racial disparities in C-CATH. Also, our analy-

ses using physician data were limited to a reduced sample size. However, our physician response rate was comparable to that of other studies of physicians' attitudes about cardiac patients<sup>24</sup> and did not vary by patients' race.

What do these findings suggest for potential clinical interventions to decrease racial disparities in C-CATH? They suggest that educational interventions aimed at altering patients' perceptions of cardiac procedures or the health care system may not be successful, because we found few such racial differences and they did not explain disparities in care in this setting. Similarly, others have suggested that racial differences in patients' trust of individual physicians or the health care system may differentially affect patients' acceptance of recommended procedures, and that interventions aimed at increasing trust may help, but our results do not support that notion. To the extent that physicians evaluate White and African American patients' clinical presentation differently, computerized decision aids (e.g., computerized clinical reminders or decisionmaking algorithms) provided to physicians at the point of care that provide objective and accurate estimates of the prior probability of disease might help reduce this source of disparity. Raising physicians' consciousness about the possibility of bias through cultural competency training may also help decrease the use of racially based clinical stereotypes, which are one kind of cognitive "shortcut" busy clinicians may use to help order their world.<sup>24</sup> Future research should examine the contribution of other potential sources of disparities in care, especially process issues such as deficiencies in doctor-patient communication or limitations in patients' health literacy, while controlling for the effects of clinical, sociodemographic, reimbursement, and financing variables and the availability of cardiac procedure technology.

Racial disparities in cardiac care are widespread, but before they can be addressed and eliminated, their cause must be identified. These results suggest that patients' attitudes and beliefs may not play an important role, and that researchers should focus on other possible etiologic variables. Future research needs to validate our findings in other set-

tings and to examine other possible mechanisms by which racial disparities in care are enacted, so that carefully targeted interventions can foster the availability of C-CATH to all patients who can benefit from it. ■

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### Contributors

N.R. Kressin originated and supervised the study, obtained funding, and led the writing. B.-H. Chang supervised the data analysis and interpretation. J. Whittle assisted with writing and data interpretation. E.D. Peterson helped with the study design and data interpretation. J.A. Clark helped with data analysis and interpretation. A.K. Rosen helped with the data interpretation and provided feedback on drafts. M.B. Orner completed the analyses. T.C. Collins and L.G. Alley supervised the data collection and provided feedback on drafts. L.A. Petersen assisted with obtaining funding, designing the study design, supervising data collection, and providing feedback on data interpretation and drafts.

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### Human Participant Protection

This study was approved by the human studies subcommittee of the 5 Department of Veterans Affairs medical centers where data collection took place, and by the study coordinating center site.

### References

1. Kressin NR, Petersen LA. Racial variations in cardiac procedures: a review of the literature and prescription for future research. *Ann Intern Med.* 2001; 135:352-366.
2. Smedley B, Stith A, Nelson A, Committee on Understanding and Eliminating Racial and Ethnic Disparities in Health Care, Board on Health Sciences Policy, eds. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care.* Washington, DC: National Academy Press; 2002.
3. Mayberry RM, Mili F, Ofili E. Racial and ethnic differences in access to medical care. *Med Care Res Rev.* 2000;57(suppl 1):108-145.
4. Ford ES, Cooper RS. Racial/ethnic differences in health care utilization of cardiovascular procedures: a review of the evidence. *Health Serv Res.* 1995;30 (1, part 2):237-252.
5. Whittle J, Conigliaro J, Good CB, Lofgren RP. Racial differences in the use of invasive cardiovascular procedures in the Department of Veterans Affairs medical system. *N Engl J Med.* 1993;329:621-627.
6. Peterson ED, Jollis JG, Bechuk JD, et al. Changes in mortality after myocardial revascularization in the elderly. The national Medicare experience. *Ann Intern Med.* 1994;121:919-927.
7. Escarce JP, Epstein KR, Colby DC, Schwartz JS. Racial differences in the elderly's use of medical procedures and diagnostic tests. *Am J Public Health.* 1993; 83:948-954.
8. Conigliaro J, Whittle J, Good CG, et al. Understanding racial variation in the use of coronary, revascularization procedures: the role of clinical factors. *Ann Intern Med.* 2000;160:1329-1336.
9. Peterson ED, Shaw LK, DeLong ER, Pryor DB, Califf RM, Mark DB. Racial variation in the use of coronary-revascularization procedures. Are the differences real? Do they matter? *N Engl J Med.* 1997;336: 480-486.
10. Whittle J, Conigliaro J, Good CB, Joswiak M. Do patient preferences contribute to racial differences in cardiovascular procedure use? *J Gen Intern Med.* 1997; 12:267-273.
11. LaVeist T, Nickerson K, Bowie J. Attitudes about racism, medical mistrust, and satisfaction with care among African American and white cardiac patients. *Med Care Res Rev.* 2000;57(suppl 1):146-161.
12. Kressin N, Clark J, Whittle J, et al. Racial differences in health-related beliefs, attitudes and experiences of VA cardiac patients: scale development and application. *Med Care.* 2002;40:172-185.
13. Hannan EL, van Ryn M, Burke J, et al. Access to coronary artery bypass surgery by race/ethnicity and gender among patients who are appropriate for surgery. *Med Care.* 1999;37:68-77.
14. Laouri M, Kravitz RL, French WJ, et al. Underuse of coronary revascularization procedures: application of a clinical method. *J Am Coll Cardiol.* 1997;29: 891-897.



15. Sedlis SP, Fisher VJ, Tice D, Esposito R, Madmon L, Steinberg EH. Racial differences in the performance of invasive cardiac procedures in a Department of Veterans Affairs Medical Center. *J Clin Epidemiol.* 1997;50: 899–901.
16. Ferguson JA, Adams TA, Weinberger M. Racial differences in cardiac catheterization use and appropriateness. *Am J Med Sci.* 1998;315:302–306.
17. Petersen L, Wright S, Peterson E, Daley J. Impact of race on cardiac care and outcomes in veterans with acute myocardial infarction. *Med Care.* 2002;40: 186–196.
18. Spertus JA, Winder JA, Dewhurst TA, et al. Development and evaluation of the Seattle Angina Questionnaire: a new functional status measure for coronary artery disease. *J Am Coll Cardiol.* 1995;25:333–341.
19. Fiscella K, Franks P, Clancy C. Skepticism toward medical care and health care utilization. *Med Care.* 1998;36:180–189.
20. Survey Research Center. *1964 Election Study.* Ann Arbor, Mich: Inter-University Consortium for Political Research, University of Michigan; 1969.
21. Scheier M, Carver C, Bridges M. Distinguishing optimism from neuroticism (and trait anxiety, self-mastery, and self-esteem): a reevaluation of the life orientation test. *J Pers Soc Psychol.* 1994;67:1063–1078.
22. Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med.* 1990;30: 1273–1281.
23. Schulman KA, Berlin JA, Harless W, et al. The effect of race and sex on physicians' recommendations for cardiac catheterization. *N Engl J Med.* 1999;340: 618–626.
24. Van Ryn M, Burke J. The effect of patient race and socio-economic status on physicians' perceptions of patients. *Soc Sci Med.* 2000;50:813–828.
25. Scanlon PJ, Faxon DP, Audet AM, et al. ACC/AHA guidelines for coronary angiography: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee on Coronary Angiography). *J Am Coll Cardiol.* 1999;33:1756–1824.
26. Gibbons R, Chatterjee K, Daley J, et al. ACC/AHA/ACP-ASIM guidelines for the management of patients with chronic stable angina: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Chronic Stable Angina). *J Am Coll Cardiol.* 1999;33:735–1097.
27. Localio A, Berlin J, Ten Have T, Kimmel S. Adjustments for center in multicenter studies: an overview. *Ann Intern Med.* 2001;135:112–123.
28. Donner A. Some aspects of the design and analysis of cluster randomization trials. *Appl Stat.* 1998;47: 95–113.
29. Hennekens C, Buring JE. Analysis of epidemiologic studies: evaluating the role of confounding. In: Mayrent SL, ed. *Epidemiology in Medicine.* Boston: Little Brown & Co; 1987.
30. Blendon RJ, Aiken LH, Freeman HE, Corey CR. Access to medical care for black and white Americans. *JAMA.* 1989;261:278–281.
31. Ayanian J, Cleary P, Weissman J, Epstein A. The effect of patients' preferences on racial differences in

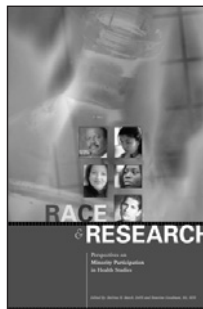
access to renal transplantation. *N Engl J Med.* 1999; 341:1661–1669.

32. Wolinsky FD, Coe RM, Mosely RR 2nd, Homan SM. Veterans' and non-veterans' use of health services: a comparative analysis. *Med Care.* 1985;23:1358–1371.

33. Every N, Larson E, Litwin P. The association between on-site cardiac catheterization facilities and the use of coronary angiography after acute myocardial infarction. *N Engl J Med.* 1993;329:546–551.

34. Petersen L, Normand S, Leape L, McNeil B. Regionalization and the underuse of angiography in the Veterans Affairs Health Care System as compared with a fee-for-service system. *N Engl J Med.* 2003;348: 2209–2217.

35. Kressin N, Chang B, Hendricks A, Kazis L. Agreement between administrative data and patients' self-reports of race. *Am J Public Health.* 2003;93: 1734–1739.



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## Race and Research Perspectives on Minority Participation in Health Studies

Edited by Bettina Beech, DrPH, MPH, and Maurine Goodman, MA, MPH

**R**ace and Research: Perspectives on Minority Participation in Health Studies is a teaching text and resource guide for students, health professionals, public health researchers, and the general public that extends the discussion of environmental factors that influence ethnic minority participation in health studies. This book examines the lack of minority participation in health studies from social, historical, and scientific perspectives.

This book is divided into three main sections: 1) The Meaning of Race, Culture and Ethnicity in Research; 2) Health Studies and Ethnic Minority Populations and 3) The Impact of Revolutionary Changes in Medicine and Health Care on Minority Participation in Health Studies.

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# Health Care Disparities and Cervical Cancer

Cathy J. Bradley, PhD, Charles W. Given, PhD, and Caralee Roberts, PhD

Disparities in cancer diagnosis and survival among racial and income groups are well documented,<sup>1–9</sup> and programs to reduce disparities have become a national priority. Former director of the National Cancer Institute Richard Klausner has noted the “discontinuity between what we have already established as effective in reducing the burden of cancer and the practice and availability of that hard-won knowledge for *all* people regardless of where they live, whether they are rich or poor, or what their cultural backgrounds are” (emphasis in original).<sup>10</sup> Cervical cancer is an excellent example of a disease for which disparities in outcomes could be overcome, because it is easily detected, the means for detection are inexpensive, and treatment is effective if the disease is detected in early stages.<sup>11</sup> Yet the relevant literature reports that disparities in detection and survival persist between African Americans and Whites,<sup>6,12–14</sup> between persons of low socioeconomic status and persons of higher socioeconomic status,<sup>15</sup> between users of public hospitals compared with users of private hospitals,<sup>12</sup> and between uninsured and Medicaid-insured persons compared with privately insured persons.<sup>9,16,17</sup> Elderly women also appear to be disproportionately vulnerable to late-stage diagnosis for cervical cancer and to poor survival.<sup>12,18</sup> We investigated disparities in cervical cancer incidence, detection, and survival in a low-income, Medicaid-insured population.

Since 1991, the Michigan Department of Community Health has administered a comprehensive breast and cervical cancer control program (BCCCP) funded by the US Centers for Disease Control and Prevention. The overarching goal of the BCCCP is to reduce disparate cancer outcomes among low-income women by providing screening services free of charge to women whose incomes are at or below 250% of the federal poverty level.<sup>19</sup> Insured women may also be screened as part of the BCCCP, provided that they are not enrolled in Medicare Part B or a managed care

**Objectives.** We compared cervical cancer incidence, stage at diagnosis, and survival in Medicaid-insured and non-Medicaid-insured populations.

**Methods.** We stratified the sample by age and used ordered logistic regression to predict stage at diagnosis and used Cox proportional hazards regression to predict survival.

**Results.** Medicaid insured nearly one quarter of women diagnosed with cervical cancer. The likelihood of late-stage disease was greatest for women who enrolled in Medicaid after diagnosis. Women younger than 65 years who enrolled in Medicaid after diagnosis were more likely to die from cervical cancer than were women who were not insured by Medicaid (hazard ratio=2.40, 95% confidence interval=1.49, 3.86).

**Conclusions.** Our study underscores the importance of cervical cancer screening programs targeted at low-income women. (*Am J Public Health.* 2004;94:2098–2103)

or health maintenance organization and that they are underinsured for screening services and meet income eligibility requirements. As of 2001, any woman who is diagnosed with breast or cervical cancer in Michigan is automatically enrolled in Medicaid, which will pay for all treatment costs until her physician indicates that she is cancer free.<sup>19</sup> Before 2001, women diagnosed through the BCCCP were enrolled in Medicaid only if they met disability or Aid to Families with Dependent Children eligibility criteria as well as asset and income requirements.

Using linked statewide Cancer Registry and Medicaid enrollment databases, we compared cervical cancer incidence rates, cancer stage at detection, and chances of survival between women insured by Medicaid and women not insured by Medicaid. Our study identified a low-income, insured population and distinguished between women who enrolled in Medicaid after being diagnosed with cancer and women who were diagnosed with cancer while enrolled in Medicaid. Women enrolled in Medicaid before their diagnosis had an opportunity to benefit from covered health care services that may have resulted in early-stage cancer detection and treatment. By contrast, women enrolling in Medicaid after diagnosis may have been previously uninsured or underinsured, with limited access to health care. Our analysis provides information about the

population of women served by the BCCCP who may subsequently enroll in Medicaid. This inquiry is particularly relevant now that many states are considering curtailing Medicaid services to resolve the budgetary crises they are facing.

## METHODS

In 1999, using the Michigan Cancer Registry, analysts from the Michigan Department of Community Health selected all women diagnosed with an incident primary cancer of the cervix in 1996 and 1997 ( $n=5076$ ). During the selected study period (1996–1997), the BCCCP had been in place for 5 years but did not automatically enroll women into Medicaid. Date of death, if it occurred before December 1998, was obtained from the Michigan Death Registry for all patients. Women whose date of diagnosis and date of death were in the same month and year were removed from the sample ( $n=27$ ). The Michigan Cancer Registry has been estimated to be from 95% to 99% complete and is reviewed annually by the North American Association of Central Cancer Registries.

Michigan Department of Community Health analysts matched incident cervical cancer cases to the 1996 and 1997 Medicaid enrollment file. Patients who matched on all variables, on all variables except address, or



on name (first and last), date of birth, gender, and either the first 5 digits or the last 4 digits of the Social Security Number were considered valid matches.<sup>9</sup> This process identified 1125 women who were insured by Medicaid. The Medicaid enrollment file contained patients' complete historical enrollment information extending before the 1996–1997 study period.

Because Medicaid is provided only to individuals who are medically indigent, Medicaid insures the lowest socioeconomic stratum of the population—one that is associated with complex medical conditions and low use of preventive services.<sup>20</sup> Many low-income persons who do not meet the categorical or financial qualifications for Medicaid, as well as uninsured individuals, were present in the control population. Although imperfect, our method correctly identified an important segment of low-income, insured women for whom Medicaid policy changes and programs such as the BCCCP can greatly influence health outcomes.

We stratified the sample by patients younger than 65 years and patients aged 65 years and older. This distinction is important, because the 2 groups of women differ systematically in health status and alternative sources for health insurance. To enroll in Medicaid, recipients must either qualify under the Aid to Families with Dependent Children program or have a disabling condition expected to last 1 or more years. The majority of women younger than 65 years qualified for Medicaid under the Aid to Families with Dependent Children program (66%); therefore, many of these patients were of childbearing age and were likely to be reasonably healthy. Medicaid enrollment for younger women varied from month to month, depending on recipients' family status, age of dependent children, and assets and income. In contrast, Medicare insures nearly every US citizen 65 years and older and may be associated with greater access to health care providers and continuous coverage relative to Medicaid insurance alone. In addition, women 65 years and older who were dually eligible for Medicaid and Medicare were likely to be disabled; 95% of the women who were 65 years or older qualified for Medicaid because of a disability.

We distinguished between patients who enrolled in Medicaid after they were diagnosed with cancer, who were enrolled in Medicaid before diagnosis, and who were non-Medicaid-insured. If a patient was enrolled in Medicaid during the same or later month and year as the month and year of diagnosis, we coded this individual as “enrolled after diagnosis.” If the patient had been enrolled in Medicaid for 1 or more months before the date of cancer diagnosis, we considered the patient “enrolled at diagnosis.” Patients were “non-Medicaid” if they were not enrolled in Medicaid at any time before or during the study period. Enrollment in the same month of diagnosis, in many cases, indicates that once the beneficiary was determined to meet enrollment criteria, which likely occurred some months following a cancer diagnosis, Medicaid enrollment was made retroactive to the date of diagnosis.

To estimate cervical cancer incidence in the Medicaid and non-Medicaid samples, we aggregated female Medicaid enrollees and all women residing in Michigan into 5-year age groups. The number of cancer cases was then calculated for female Medicaid enrollees and all women residing in Michigan for 1996 and 1997 and divided by the total number of female Medicaid enrollees and the total female population, respectively, by age group, for 1996 and 1997. We multiplied the resulting quotients by 1000 to obtain an incidence rate per 1000 women for each age group. In this analysis, we could not subtract Medicaid enrollees from the general population; thus, differences observed in cervical cancer incidence between the 2 groups underestimated the true difference in incidence. Although the actual difference in incidence is likely to be greater than we estimated, our estimates are informative because these incidence rates help to demonstrate the relative cancer burden in the 2 populations.

We used ordered logistic regression to analyze cervical cancer stage at diagnosis (in situ, local, regional, and distant). Patients whose cancers were not staged were excluded from the analysis ( $n=86$ , women younger than 65 years; and  $n=24$ , women aged 65 years and older). Using predicted probabilities computed from the coefficients derived in the ordered logistic regression, we estimated the

likelihood of cancer detection at each stage for non-Medicaid-insured women, women insured by Medicaid at the time of diagnosis, and women enrolled in Medicaid after diagnosis. We used a multivariate Cox proportional hazards model to estimate the risk of death from cervical cancer during the study period. We also estimated the risk of death from all-cause mortality (results are not shown but are available on request). We tested the proportional hazard assumption for individual variables and performed a global test of proportionality with the Schoenfeld residuals.<sup>21</sup> Age and race/ethnicity (White, African American, and Other) were controlled in all regression models, and early-stage diagnosis (defined as either in situ or local cancer) was controlled in the Cox model. All statistical analyses were performed with Stata version 7.0 (Stata Corp, College Station, Tex).

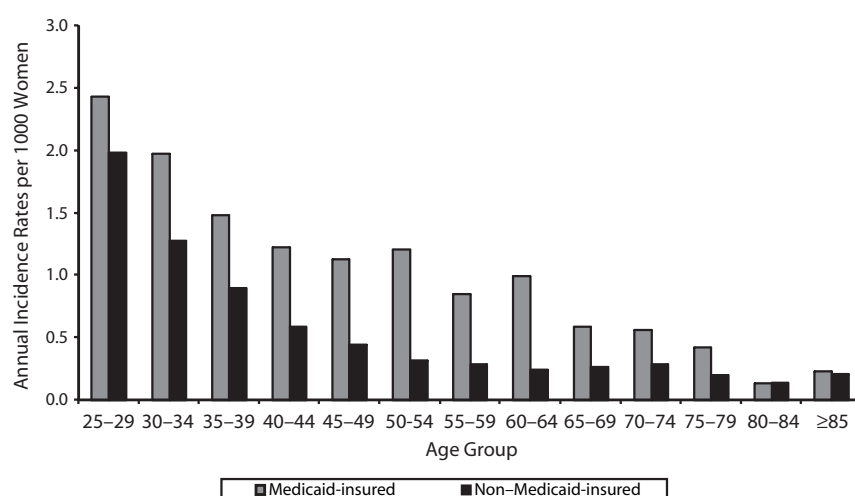
## RESULTS

### Incidence

Figure 1 shows that compared with the incidence in Michigan's general population, the age-adjusted incidence of cervical cancer among Medicaid-insured women was much higher, particularly among women in younger age groups. The highest incidence for both Medicaid (2.43 per 1000 women) and the general population (1.98 per 1000 women) was in women aged 25–29 years. The age group with the largest difference in incidence was women 50–54 years old (1.21 per 1000 Medicaid-insured women vs 0.32 per 1000 women in the general population). Overall, the cervical cancer incidence rate was higher in the Medicaid population in every age group up to 80 years of age. Because women insured by Medicaid were included in the general population of women, the true difference in incidence rates is higher than that reported here.

### Descriptive Statistics

Table 1 shows the descriptive statistics for the cervical cancer sample. During 1996 and 1997, 5049 cases of cervical cancer were diagnosed in Michigan, and Medicaid insured 22% of these women ( $n=1125$ ). For women younger than 65 years, the non-Medicaid- and Medicaid-insured samples were comparable in age (mean: 36.6



**FIGURE 1—Cervical cancer incidence rates for Michigan women, by Medicaid status, 1996–1997.**

**TABLE 1—Descriptive Characteristics of Women in Cervical Cancer Sample (N = 5049): Michigan, 1996–1997**

|  | Women Younger Than 65 Years <sup>a</sup> |                                | Women Aged 65 Years and Older     |                              |
|--|--|--------------------------------|-----------------------------------|------------------------------|
|  | Non-Medicaid-Insured<br>(n = 3647)       | Medicaid-Insured<br>(n = 1063) | Non-Medicaid-Insured<br>(n = 277) | Medicaid-Insured<br>(n = 62) |
| Mean age, y (SD)   | 36.63 (9.16)                             | 34.94 (9.35)                   | 74.03 (6.92)                      | 74.13 (7.70)                 |
| Race/ethnicity, no. (%)                                  |  |                                |                                   |                              |
| White  | 2571 (70.50)                             | 608 (57.20)                    | 209 (75.45)                       | 36 (58.06)                   |
| African American   | 374 (10.26)                              | 301 (28.32)                    | 52 (18.77)                        | 21 (33.87)                   |
| Other  | 702 (19.25)                              | 147 (13.83)                    | 16 (5.78)                         | 5 (8.06)                     |
| Stage at diagnosis, no. (%)                              |  |                                |                                   |                              |
| In situ  | 3166 (86.81)                             | 844 (79.40)                    | 140 (50.54)                       | 18 (29.03)                   |
| Local  | 303 (8.31)                               | 110 (10.35)                    | 58 (20.94)                        | 16 (25.81)                   |
| Regional   | 96 (2.63)                                | 69 (6.49)                      | 44 (15.88)                        | 15 (24.19)                   |
| Distant  | 23 (0.63)                                | 13 (1.22)                      | 17 (6.14)                         | 7 (11.29)                    |
| Invasive/unknown   | 59 (1.62)                                | 27 (2.54)                      | 18 (6.50)                         | 6 (9.68)                     |
| Deaths, no. (%)  |  |                                |                                   |                              |
| Total deaths   | 67 (1.84)                                | 65 (6.11)                      | 51 (18.41)                        | 18 (29.03)                   |
| Cancer deaths  | 62 (1.70)                                | 54 (5.08)                      | 42 (15.16)                        | 15 (24.19)                   |
| Deaths in women who enrolled in Medicaid after diagnosis | ...                                      | 279 (26.25)                    | ...                               | 14 (22.58)                   |

<sup>a</sup>The number of women younger than 65 years (n = 4710) differs from the number (n = 3863) appearing in our published article (Bradley C, Given C, and Roberts C. Late stage cancers in a Medicaid-insured population. *Med Care*. 2003;41(6):722–728). The difference is the result of inclusion of women classified as “other/unknown” race/ethnicity (n = 849) and exclusion of 2 women with missing information in this analysis.

(≥90%), although there was a higher percentage of regional, distant, and invasive/unknown cancers in the Medicaid sample (10%) relative to the non-Medicaid-insured sample (5%). As would be expected given the more severe stage of disease, a higher percentage of women in the Medicaid sample died relative to women in the non-Medicaid sample (6% vs 2%). In both samples, the vast majority of deaths were caused by cervical cancer. In the Medicaid sample, approximately one quarter of the women were enrolled in Medicaid after the detection of cervical cancer.

Medicaid insured 18% of women aged 65 years and older (Table 1, column 4). The average age of the women was 74 years. Nearly 34% of the older women insured by Medicaid were African American. Later-stage cancers and deaths were far more common in women aged 65 years and older relative to women younger than 65 years. Although 71% of older, non-Medicaid-insured women received diagnoses of cervical cancer at in situ or local stage, only 55% of older, Medicaid-insured women received diagnoses at an early stage. Approximately 18% and 29% of non-Medicaid-insured women and Medicaid-insured women, respectively, died during the study period, and as found with the younger women, the majority of these deaths were related to cervical cancer.

On the basis of univariate analysis alone, Medicaid insured a substantial proportion of the total cervical cancer cases, with one quarter of Medicaid recipients enrolled in Medicaid after diagnosis. In addition, women who were insured by Medicaid were more likely to have late-stage disease when diagnosed and were more likely to die compared with women who were not insured by Medicaid. The descriptive analysis also revealed a heightened vulnerability for late-stage cervical cancer among women aged 65 years and older relative to younger women. However, these older women represented fewer than 7% of the total cervical cancer population.

### Cervical Cancer Detection

In an ordered logistic regression predicting cancer stage at diagnosis, age, “other or unknown” race/ethnicity, and enrollment in Medicaid both before and after diagnosis

years and 35 years, respectively). Although both samples had a high percentage of White women (71% and 57%, respectively), only 10% of non-Medicaid-insured women

were African American, whereas 28% of the Medicaid-insured women were African American. In both samples, most cancers were detected at the in situ or local stage

**TABLE 2—Prediction of Cervical Cancer Stage at Diagnosis, Ordered Logistic Regression (In Situ, Local, Regional, Distant): Michigan, 1996–1997**

|  | $\beta$ Coefficient (SE)               |   |
|--|--|---|
|  | Women Younger Than 65 Years (n = 4624) | Women Aged 65 Years and Older (n = 315) |
| Age  | .10 (.01)*                             | .05 (.02)*                              |
| African American (reference group: White)                                  | -.21 (.13)                             | -.50 (.28)                              |
| Other race/ethnicity (reference group: White)                              | -1.59 (.25)*                           | -1.96 (.79)*                            |
| Medicaid-enrolled at diagnosis (reference group: non-Medicaid-enrolled)    | .51 (.13)*                             | .77 (.30)*                              |
| Medicaid-enrolled after diagnosis (reference group: non-Medicaid-enrolled) | 1.49 (.15)*                            | 1.63 (.55)*                             |

\* $P < .05$ .**TABLE 3—Predicted Probabilities of Cancer Stage: Michigan, 1996–1997**

|                                   | Cancer Stage |       |          |         |
|-----------------------------------|--------------|-------|----------|---------|
|                                   | In Situ      | Local | Regional | Distant |
| Women younger than 65 years       |              |       |          |         |
| Non-Medicaid-enrolled             | 0.92         | 0.06  | 0.02     | 0.003   |
| Medicaid-enrolled at diagnosis    | 0.88         | 0.09  | 0.03     | 0.01    |
| Medicaid-enrolled after diagnosis | 0.73         | 0.19  | 0.07     | 0.01    |
| Women aged 65 years and older     |              |       |          |         |
| Non-Medicaid-enrolled             | 0.54         | 0.24  | 0.16     | 0.05    |
| Medicaid-enrolled at diagnosis    | 0.36         | 0.28  | 0.26     | 0.11    |
| Medicaid-enrolled after diagnosis | 0.19         | 0.23  | 0.36     | 0.22    |

Note. Predicted probabilities were estimated with the mean values for age and race/ethnicity.

were associated with later-stage disease for both women younger than 65 years and women aged 65 years and older (Table 2). Table 3 shows the estimated predicted probabilities by cancer stage for women who were not insured by Medicaid, enrolled in Medicaid before a cervical cancer diagnosis, and enrolled in Medicaid after a cervical cancer diagnosis. We used the mean values for age and race/ethnicity in these estimations. A consistent pattern emerged from the data: Women who were not insured by Medicaid received diagnoses at early stages, whereas women who were enrolled in Medicaid at the time of diagnosis had a slightly lower chance of early-stage detection, and women enrolled in Medicaid after diagnosis were much less likely to have an early-stage cancer at detection. This pattern held for both younger and older women (Table 3). In younger women, for example, the probability of receiving a

cervical cancer diagnosis in situ was 92% for those who were not insured by Medicaid, 88% for those who were enrolled in Medicaid at the time of diagnosis, and 73% for those who enrolled in Medicaid after diagnosis.

sis. The most dramatic differences in the predicted cancer stage probabilities were observed in older women with in situ cancer, for whom the predicted probabilities were 54%, 36%, and 19% for the non-Medicaid-enrolled, Medicaid-enrolled at the time of diagnosis, and Medicaid-enrolled after diagnosis categories, respectively.

### Mortality

Table 4 shows the results of a Cox proportional hazards model predicting death from cervical cancer after control for age, early-stage disease (in situ or local), race/ethnicity, and Medicaid enrollment status. Age and cancer stage were associated with survival, whereas race/ethnicity was not. For women younger than 65 years, the hazard ratio for age was 1.06 (95% confidence interval [CI]=1.04, 1.09). The hazard ratio for women enrolled in Medicaid at the time of diagnosis was 1.77 (95% CI=1.02, 3.07), and the hazard ratio for women enrolled in Medicaid after diagnosis was 2.40 (95% CI=1.49, 3.86). This result is striking, given that cancer stage was controlled in the model. Age was positively associated with an increased risk of death for women aged 65 years and older. Medicaid enrollment, however, was not associated with an increased risk of death in older women. This was partly a result of the collinearity between Medicaid enrollment and cancer stage at diagnosis (Table 3). When early-stage cancer at diagnosis was removed from the model, the hazard ratio for women enrolled in Medicaid after diagnosis was 3.20 (95% CI=1.25, 8.17; results not shown), and

**TABLE 4—Prediction of Death From Cervical Cancer: Michigan, 1996–1997**

| Variable (Reference Group)  | Hazard Ratio (95% Confidence Interval) |   |
|---|--|---|
|   | Women Younger Than 65 Years (n = 4624) | Women Aged 65 Years and Older (n = 315) |
| Age   | 1.06 (1.04, 1.09)                      | 1.04 (1.01, 1.08)                       |
| African American (reference: White)   | 0.93 (0.58, 1.48)                      | 0.72 (0.34, 1.53)                       |
| In situ or local-stage cancer at diagnosis (reference: regional and distant stages) | 0.04 (0.02, 0.06)                      | 0.09 (0.05, 0.18)                       |
| Medicaid-enrolled at diagnosis (reference: non-Medicaid-insured)                    | 1.77 (1.02, 3.07)                      | 1.15 (0.55, 2.40)                       |
| Medicaid-enrolled after diagnosis (reference: non-Medicaid-insured)                 | 2.40 (1.49, 3.86)                      | 1.24 (0.48, 3.19)                       |

the hazard ratio for women enrolled in Medicaid at the time of diagnosis was not statistically significant ( $P < .05$ ).

## DISCUSSION

Our findings are consistent with what is known about disparities in health care. Cervical cancer incidence was much higher among low-income women—a considerable public health concern, because Medicaid insured nearly one quarter of all women diagnosed with cervical cancer. This finding underscores the importance of programs that, like the BCCCP, are targeted to low-income women, and it also highlights the need for Medicaid to promote screening in its enrolled population. For example, the use of personalized, tailored letters that contain generic cancer risk information has been shown to increase cancer screening rates among low-income women.<sup>22</sup> Cervical cancer is more likely to be diagnosed at advanced stages in low-income, Medicaid-insured women relative to women who are not insured by Medicaid, and once the disease is diagnosed, low-income women have higher-than-expected mortality. Our study did not find racial/ethnic differences in cancer survival. This finding is a deviation from the Institute of Medicine report on health care disparities<sup>7</sup> but is consistent with previous findings concerning breast cancer outcomes, race/ethnicity, and socioeconomic status.<sup>23</sup>

Our findings with regard to women enrolled in Medicaid after diagnosis suggest that expanding Medicaid insurance to include uninsured women is one way to reduce, although not eliminate, disparities in cervical cancer. The economic challenge of providing health care through the Medicaid program has been widely discussed.<sup>24</sup> Nevertheless, our study indicates that reducing coverage may have adverse economic consequences, because low-income women are likely to enroll in Medicaid after a cancer has progressed and is more expensive to treat. In 1996 and 1997, the Michigan Medicaid program spent nearly \$2.5 million dollars for the treatment of confirmed cervical cancer cases. A question to examine in future research is whether fewer women with late-stage disease (which is less expensive to treat) enrolled in Medicaid after 2001, when

Medicaid eligibility became automatic for women diagnosed through the BCCCP.

Our study demonstrates that there are 2 distinct populations of women with cervical cancer—those younger than 65 years and those aged 65 years and older—who require different strategies for cervical cancer screening and treatment. Promotion of more intensive screening among elderly women, particularly if they are nursing home residents, presents a unique set of challenges. Residents in long-term care facilities might not benefit from improvements in cervical cancer detection and treatment. These women may have comorbid conditions (e.g., dementia, reduced physical functioning) that negatively influence a physician's willingness to recommend screening or treatment. The average age of women in the sample who were older than 65 years was 74 years. Nonetheless, the utility of increased cervical cancer screening and treatment for elderly women should be investigated. However, Fahs found that triennial screening reduced mortality by 74% at a cost of \$7345 per year of life saved for women 65 years and older.<sup>25</sup> Other studies have shown that a strategy of combined Pap smears and human papillomavirus testing every 2 years up to age 100 years can be implemented at \$70347 per quality-adjusted life-year saved.<sup>26</sup> Such investigations assist providers and policymakers in understanding the benefits of intensive screening efforts relative to their costs.

A limitation of our study is that we could not identify uninsured individuals within our sample. We could identify only those who were insured by Medicaid, and thus, the younger, non-Medicaid-insured group contained individuals who were uninsured. We hypothesized, but could not test, that the outcomes (e.g., stage at detection, mortality) of uninsured patients are similar to those of patients insured by Medicaid; thus the presence of uninsured women in the non-Medicaid group underestimates the true difference between women who are Medicaid insured and women who are privately insured. Although it is likely that women who enrolled in Medicaid after diagnosis were previously uninsured, we cannot be certain that this is the case—we only know they were not insured by Medicaid. In addition, the findings from our study

of a single state may not be generalizable to other geographical areas.

Our research suggests that several measures can be taken to reduce disparities in cervical cancer. Medicaid insurance, for example—although it was not comparable to other forms of insurance—improved stage at diagnosis and survival for women who were enrolled at the time of diagnosis. Because disparities in diagnosis and survival were observed in the sample of women enrolled in Medicaid at the time of diagnosis, further investigation regarding the quality of care provided to women insured by Medicaid is warranted. As the medical community considers how best to address disparities in health care, it must face the financial and programmatic commitment required to establish regular patterns of care with vulnerable populations and to ensure that the care delivered is of high quality. Some of the costs to provide the needed care to low-income women may be offset by a reduction in future Medicaid enrollment after the disease has advanced to a stage in which it is expensive to treat and recovery is less likely. ■

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## Contributors

C.J. Bradley and C. W. Given conceptualized the study and supervised all aspects of its implementation. C. Roberts assisted with the study and completed the analyses. C.J. Bradley synthesized analyses and led the writing. All authors helped to conceptualize ideas, interpret findings, and review drafts of the article.

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## Human Participant Protection

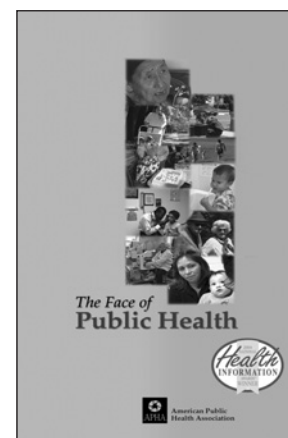
This study was approved by Michigan State University's institutional review board.



## References

1. Wingo PA, Ries LA, Giovino GA, et al. Annual report to the nation on the status of cancer, 1973–1996, with a special section on lung cancer and tobacco smoking. *J Natl Cancer Inst.* 1999;91:675–690.
2. Shinagawa SM. The excess burden of breast carcinoma in minority and medically underserved communities. *Cancer.* 2000;88(5 suppl):1217–1223.
3. Lannin DR, Mathews HF, Mitchell J, et al. Influence of socioeconomic and cultural factors on racial differences in late-stage presentation of breast cancer. *JAMA.* 1998;279:1801–1807.
4. Figueroa JB, Breen N. Significance of underclass residence on the stage of breast or cervical cancer diagnosis. *Am Econ Rev.* 1995;85:112–116.
5. Greenwald HP, Borgatta EF, McCorkle R, et al. Explaining reduced cancer survival among the disadvantaged. *Milbank Q.* 1996;74:215–238.
6. Roetzheim RG, Pal N, Tennant C, et al. Effects of health insurance and race on early detection of cancer. *J Natl Cancer Inst.* 1999;91:1409–1415.
7. Haynes MA, Smedley BD, eds. *The Unequal Burden of Cancer: An Assessment of NIH Research and Programs for Ethnic Minorities and the Medically Underserved.* Washington, DC: National Academy Press; 1999.
8. Eley JW, Hill H, Chen VW, et al. Racial differences in survival from breast cancer. Results of the National Cancer Institute Black/White Cancer Survival Study. *JAMA.* 1994;272:947–954.
9. Bradley CJ, Given CW, Roberts C. Disparities in cancer diagnosis and survival. *Cancer.* 2001;91:178–188.
10. National Cancer Institute. *The Nation's Investment in Cancer Research: A Budget Proposal for Fiscal Year 2001.* Bethesda, Md: National Institutes of Health; 2001. Publication 02–4373.
11. National Institutes of Health Consensus Conference on Cervical Cancer. Bethesda, Maryland, April 1–3, 1996. *J Natl Cancer Inst Monogr.* 1996;(21):1–148.
12. Mandelblatt J, Andrews H, Kerner J, et al. Determinants of late stage diagnosis of breast cancer and cervical cancer: the impact of age, race, social class, and hospital type. *Am J Public Health.* 1991;81:646–649.
13. Mundt AJ, Connell PP, Campbell T. Race and clinical outcomes in patients with carcinoma of the uterine cervix treated with radiation therapy. *Gynecol Oncol.* 1998;71:151–158.
14. Grisby PW, Hall-Daniels L, Baker S, et al. Comparison of clinical outcomes in black and white women treated with radiotherapy for cervical carcinoma. *Gynecol Oncol.* 2000;79:357–361.
15. Lundin FE, Christopherson WM, Mendez WM, et al. Morbidity from cervical cancer: effects of cervical cytology and socioeconomic status. *J Natl Cancer Inst.* 1965;35:1015–1025.
16. Howell EA, Chen YT, Concato J. Differences in cervical cancer mortality among black and white women. *Obstet Gynecol.* 1999;94:509–515.
17. Jones WB, Shingleton HM, Russell A, Chmiel JS, et al. Patterns of care for invasive cervical cancer. *Cancer.* 1995;76:1934–1947.
18. Serur E, Fruchter RG, Maiman M, et al. Age, substance abuse, and survival of patients with cervical carcinoma. *Cancer.* 1995;75:2530–2538.
19. Michigan BCCCP Web page. Available at: <http://www.michigancancer.org/michiganbcccp.htm>. Accessed October 24, 2003.
20. DeVoe JE, Fryer GE, Phillips R, Green L. Receipt of preventive care among adults: insurance status and usual source of care. *Am J Public Health.* 2003;93:786–791.
21. Grambsch P, Therneau T. Proportional hazards tests and diagnostics based on weighted residuals. *Biometrika.* 1994;81:515–526.
22. Weiss-Jibaja ML, Volk RJ, Kingery P, et al. Tailored messages for breast and cervical cancer screening of low-income and minority women using medical records data. *Patient Educ Couns.* 2003;50:123–132.
23. Bradley CJ, Given CW, Roberts C. Race, socioeconomic status, and breast cancer treatment and survival. *J Natl Cancer Inst.* 2002;94:493–496.
24. Boyd DJ. The bursting state fiscal budget and state Medicaid budgets. *Health Aff.* 2003;22:46–61.
25. Fahs MC, Mandelblatt J, Schechter C, et al. Cost effectiveness of cervical cancer screening for the elderly. *Ann Intern Med.* 1992;117:520–527.
26. Mandelblatt JS, Lawrence WF, Womack SM, et al. Benefits and costs of using HPV testing to screen for cervical cancer. *JAMA.* 2002;287:2372–2381.

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# Endometrial Cancer: Socioeconomic Status and Racial/Ethnic Differences in Stage at Diagnosis, Treatment, and Survival

Terri Madison, PhD, MPH, David Schottenfeld, MD, MSc, Sherman A. James, PhD, Ann G. Schwartz, PhD, MPH, and Stephen B. Gruber, MD, PhD, MPH

Cancer of the uterine corpus, of which 95% is classified as endometrial carcinoma,<sup>1,2</sup> is the most common US female genital-tract malignancy. Although the age-adjusted incidence is 31% lower among African American women than among White women, the age-adjusted mortality among African American women is 84% higher.<sup>3</sup> Moreover, the disparity in 5-year relative survival between African American and White women who had uterine corpus cancer (27% in the 1992–1997 cohort) was one of the largest observed in the Surveillance, Epidemiology, and End Results (SEER) program.<sup>3</sup>

One important determinant of the racial/ethnic difference in endometrial cancer survival is stage at diagnosis, where 40% of the survival difference is attributable to African American women who present with more advanced-stage disease.<sup>4</sup> Advanced stage at diagnosis of endometrial cancer has been associated with increasing age, higher tumor grade, and more aggressive histology.<sup>5–9</sup> However, after adjusting for these predictors, African American women are still more likely to present with advanced stage.<sup>2,5</sup>

The role of biological factors (e.g., more aggressive tumors) versus nonbiological factors (e.g., impediments to access to and utilization of quality medical care) in explaining racial/ethnic differences in endometrial cancer stage at diagnosis and survival has been examined in several studies.<sup>4,5,8,10</sup> Whether these racial/ethnic differences reflect true biological variation or differences in lifestyle and sociocultural risk factors is not clear. In the United States, African Americans are more likely to have lower socioeconomic status (SES).<sup>11</sup> Decreased access to or utilization of medical care among those who have lower SES can delay seeking treatment and thus result in greater risk for advanced-stage disease. Although delay in seeking treatment has not been

**Objective.** We evaluated the association between socioeconomic status and racial/ethnic differences in endometrial cancer stage at diagnosis, treatment, and survival.

**Methods.** We conducted a population-based study among 3656 women.

**Results.** Multivariate analyses showed that either race/ethnicity or income, but not both, was associated with advanced-stage disease. Age, stage at diagnosis, and income were independent predictors of hysterectomy. African American ethnicity, increased age, aggressive histology, poor tumor grade, and advanced-stage disease were associated with increased risk for death; higher income and hysterectomy were associated with decreased risk for death.

**Conclusions.** Lower income was associated with advanced-stage disease, lower likelihood of receiving a hysterectomy, and lower rates of survival. Earlier diagnosis and removal of barriers to optimal treatment among lower-socioeconomic status women will diminish racial/ethnic differences in endometrial cancer survival. (*Am J Public Health.* 2004;94:2104–2111)

shown to explain racial/ethnic differences in endometrial cancer stage at diagnosis,<sup>5,12–15</sup> it has been shown that being poor, having no health insurance, and having no usual source of care are associated with lower medical-consultation rates.<sup>13</sup> In univariate analyses, studies have shown several SES factors are associated with early-stage disease<sup>5,7</sup>; however, in the National Cancer Institute's (NCI) Black/White cancer survival study, SES was not shown to have an independent association with endometrial cancer stage at diagnosis.<sup>4</sup>

Several studies have shown racial/ethnic differences in the availability of treatment options or the quality of cancer treatment.<sup>16</sup> Among women who had advanced-stage endometrial cancer, White women were more likely than African American women to receive surgery and radiation therapy.<sup>10</sup> In the NCI's Black/White cancer survival study, substantially more White women had hysterectomies as their primary treatment than African American women did (95.4% vs 70.5%), and this racial/ethnic difference was apparent within each disease stage.<sup>4</sup> SES differences may have contributed to these observed racial/ethnic differences; however, this was not evaluated in the Bain et al. and NCI studies. The

objective of our population-based retrospective cohort study was to evaluate the association between SES (measured at the aggregate census tract level) and racial/ethnic differences in stage at diagnosis, treatment received, and survival.

## METHODS

### Study Population and Eligibility

The study population was selected from the Detroit-area SEER cancer registry, a population-based registry of all incident cancers that occurred among residents of Macomb, Oakland, and Wayne counties in the state of Michigan. Registry data included information about demographics and tumor characteristics, first course of cancer-directed treatment, and time from diagnosis until death or last follow-up. African American and White women who were diagnosed with a primary cancer of the uterine corpus between January 1, 1990, and December 31, 1998, were eligible for our study; only women who had in situ cancers, uterine sarcomas, or a previous history of cancer other than basal or squamous cell carcinoma of the skin were excluded. The final study population of 3168

White and 488 African American women included all the eligible women.

We conducted a validation substudy to estimate the validity of ecologically assigned SES variables. This information was obtained from a separate project where all African American cases from 1998 and a randomly selected subset of White cases from 1998 were selected from the same registry on the basis of the same eligibility criteria. There were 107 women (27 African American and 80 White) who had information available for the validation substudy (response rate=48.4%), which represented 26% of the eligible cases diagnosed in 1998. The women's SES information was collected via telephone interviews or mailed questionnaires.

### Measures

Outcome variables were stage at diagnosis, treatment, and survival. Stage at diagnosis was classified as localized, regional, distant, or unstaged,<sup>17</sup> and it was dichotomized as advanced stage (regional or distant) and not advanced stage (localized). Primary treatment via hysterectomy and/or radiation therapy was obtained from SEER records. Receipt of chemotherapy, hormonal therapy, and immunotherapy was not evaluated, because SEER data are generally captured from hospital records and these treatments are frequently given outside the hospital setting. Survival time was measured from date of diagnosis to date of death. For patients who were still alive, data were censored on the basis of the date of last follow-up visit.

Tumor histology was classified in accordance with the *International Classification of Diseases for Oncology*.<sup>18</sup> Categories for less aggressive adenocarcinomas (endometrioid adenocarcinoma, mucinous adenocarcinoma) and more aggressive adenocarcinomas (clear cell adenocarcinoma, serous papillary adenocarcinoma, squamous carcinoma, undifferentiated carcinoma) also were created.<sup>1,19–21</sup> Tumor grade was classified as well differentiated, moderately differentiated, poorly differentiated, undifferentiated, and unknown.

SEER cancer registry data do not include individual-level SES information; however, each case's registry data are geocoded to a census tract on the basis of residence at diagnosis. For our study, census tract was used to

link each study case to census tract-level SES variables in the 1990 US Census of Population and Housing Summary Tape File 3A.<sup>22</sup> The 3656 women in our study lived in 966 unique census tracts in the Detroit metropolitan area. Census tract information was missing for 17 women, who composed less than 0.5% of the study population.

Median census tract household income was ecologically assigned on the basis of residence at diagnosis after reported income was inflated to 1998 dollars with the consumer price index (CPI). For statistical analyses, we used the natural logarithm of median household income. Mean years of education was estimated by multiplying the race-specific number of individuals at each educational level by the midpoint of that level and then summing over all levels and dividing by the total. On the basis of previous studies of socioeconomic effects on health, we used a derived variable for "lives in an undereducated tract," which was defined as women who lived in census tracts where 25% or more of the adults aged 25 years and older who were of the same race/ethnicity did not have a high school diploma.<sup>23</sup>

### Statistical Methods

The validity of ecologically assigned SES variables was ascertained by comparing these variables with information obtained during the interview. The validity of mean years of education was evaluated with the Pearson product moment correlation coefficient. The validity of median household income, which was collapsed into the same categories obtained during the interview, was evaluated with the Spearman rank order correlation coefficient.

Differences in proportions were evaluated with the  $\chi^2$  test; differences between means were evaluated with the *t* test. Survival was modeled with the Kaplan-Meier method, and racial/ethnic differences were evaluated with the log-rank test. Univariate associations were evaluated with the Wald  $\chi^2$  test; parameters found to be significant ( $P<.05$ ) were retained for multivariate analysis. Interactions that were found to be significant at the 10% level (Wald  $\chi^2$ ) were retained for multivariate analyses, where stepwise logistic regression was used to evaluate advanced stage and treatment, and Cox proportional hazards re-

gression was used to evaluate survival. The criteria for retaining variables were  $P<.05$  for main effects and  $P<.10$  for interactions. All analyses were 2-sided at a level of .05 and were performed with SAS software.<sup>24,25</sup>

## RESULTS

### Demographic and Socioeconomic Characteristics

Demographic and socioeconomic characteristics are shown in Table 1. More than half the study population resided in Wayne County, and more than 90% of the African American women resided in Wayne County. The median age at diagnosis (65 years) did not differ between African American and White women. African American women were less likely to be married at the time of diagnosis than White women were.

More than 80% of the African American women lived in undereducated tracts compared with only 34% of the White women ( $P=.001$ ). Median household income among African American women was about half that among White women (\$22 829 vs \$51 275, respectively;  $P=.0001$ ). Notably, 78% of the African American women compared with 17% of the White women ( $P=.001$ ) lived in census tracts where the median household incomes were in the lowest quartile.

### Validation Substudy

The validation substudy found reasonably good correspondence<sup>26</sup> between ecologically assigned income and self-reported income (Spearman correlation coefficient=0.52). The self-reported mean years of education was 13.4 (SD=2.10), and the ecologically assigned mean years of education was 12.5 (SD=1.13); the correlation of these 2 measures of education was weak (Pearson product moment correlation coefficient=0.37).<sup>26</sup> Because of the poor validity of ecologically assigned education, we did not include education in predictive models for stage at diagnosis, treatment, and survival.

### Stage at Diagnosis

African American women were significantly more likely to have aggressive endometrial cancer in terms of histology ( $P=.001$ ) and tumor grade ( $P=.001$ ) (Table 1). Among women who had known stage at diagnosis,

**TABLE 1—Demographic, Socioeconomic, and Tumor-Related Characteristics Among African American and White Women Who Had Endometrial Cancer: Detroit, Mich, Tri-County Area, 1990–1998**

|   | African American  | White             | P <sup>a</sup> |
|---|-------------------|-------------------|----------------|
| County, no. (%)   |                   |                   | <.001          |
| Macomb  | 7 (1.4)           | 801 (25.3)        |                |
| Oakland   | 35 (7.2)          | 997 (31.5)        |                |
| Wayne   | 446 (91.4)        | 1370 (43.2)       |                |
| Age   |                   |                   |                |
| Mean, y (SD)  | 64.4 (12.8)       | 64.1 (12.3)       | .5662          |
| Aged < 65 y, no. (%)  | 231 (47.3)        | 1537 (48.5)       |                |
| Aged ≥ 65 y, no. (%)  | 257 (52.7)        | 1631 (51.5)       | .627           |
| Marital status, no. (%)   |                   |                   | <.001          |
| Single  | 92 (18.9)         | 340 (10.7)        |                |
| Married   | 150 (30.7)        | 1674 (52.8)       |                |
| Separated/divorced  | 65 (13.3)         | 236 (7.4)         |                |
| Widowed   | 173 (35.5)        | 876 (27.7)        |                |
| Unknown   | 8 (1.6)           | 42 (1.3)          |                |
| Education   |                   |                   |                |
| Adults in tract with highest educational level in category shown, mean % (SD) |                   |                   |                |
| No high school diploma  | 36.3 (13.75)      | 20.8 (11.44)      | <.0001         |
| High school diploma   | 20.5 (6.81)       | 30.2 (8.38)       | <.0001         |
| Some college  | 26.4 (9.06)       | 26.9 (5.86)       | .2226          |
| ≥ 4-year college degree   | 10.0 (12.44)      | 20.1 (15.49)      | <.0001         |
| ≥ High school diploma   | 62.7 (13.80)      | 78.2 (11.44)      | <.0001         |
| Lived in undereducated census tract, <sup>b</sup> no., (%)                    | 394 (81.4)        | 1063 (33.7)       | <.001          |
| Mean years education attained   |                   |                   |                |
| Median  | 11.4              | 12.5              |                |
| Mean (SD)   | 11.6 (1.13)       | 12.6 (1.18)       | <.0001         |
| Income  |                   |                   |                |
| Median household <sup>c</sup>   |                   |                   |                |
| Median  | 22,829            | 51,275            |                |
| Mean (SD)   | 27,008 (15,463.4) | 54,888 (22,149.5) | <.0001         |
| Lived in lowest income quartile, <sup>d</sup> no., (%)                        | 382 (78.3)        | 541 (17.1)        | <.001          |
| Histology, no. (%)  |                   |                   | <.001          |
| Endometrioid  | 350 (71.7)        | 2844 (89.8)       |                |
| Clear cell  | 27 (5.5)          | 52 (1.6)          |                |
| Serous papillary  | 69 (14.1)         | 123 (3.9)         |                |
| Mucinous  | 8 (1.6)           | 59 (1.9)          |                |
| Undifferentiated  | 16 (3.3)          | 57 (1.8)          |                |
| Squamous  | 10 (2.1)          | 16 (0.5)          |                |
| Other   | 8 (1.6)           | 17 (0.5)          |                |
| Nonaggressive histology   | 358 (74.6)        | 2903 (92.1)       |                |
| Aggressive histology  | 122 (25.4)        | 248 (7.9)         | <.001          |
| Stage of disease, no. (%)   |                   |                   | <.001          |
| Localized   | 267 (54.7)        | 2303 (72.7)       |                |
| Regional  | 109 (22.3)        | 530 (16.7)        |                |
| Distant   | 66 (13.5)         | 254 (8.0)         |                |
| Unknown   | 46 (9.4)          | 81 (2.6)          |                |
| Not advanced  | 267 (60.4)        | 2303 (74.6)       |                |
| Advanced  | 175 (39.6)        | 784 (25.4)        | <.001          |

*Continued*

approximately 40% of the African American women presented with advanced-stage disease compared with only 25% of the White women ( $P=.001$ ).

In univariate analyses, the risk for African American women to present with advanced-stage disease was approximately twice that for White women (odds ratio [OR]=1.93; 95% confidence interval [CI]=1.57, 2.37) (Table 2). Increasing age, aggressive histology, and poor tumor grade were associated with increased risk for advanced-stage disease, whereas higher family income was associated with decreased risk for advanced-stage disease. In multivariate analysis (stepwise logistic regression), after we adjusted for age, tumor grade, and histology, African American women were still 41% more likely to present with advanced-stage disease ( $P=.0079$ ).

On the basis of previous studies that showed collinearity of race with income,<sup>27</sup> an alternate model was created to force median family income to be retained in the stepwise logistic regression model. With this model, race/ethnicity was no longer a significant independent predictor of advanced-stage disease, whereas higher median family income was inversely associated with advanced-stage disease (OR=0.83; 95% CI=0.69, 0.99). A third model was created to force both race/ethnicity and median family income in the stepwise logistic regression model. When we used the likelihood ratio test, this third model was inferior to both the other models. Notably, estimates of the odds ratios for age, tumor grade, and histology remained consistent across the 3 models, which suggests it would be difficult to distinguish the independent contributions of race/ethnicity and income level to advanced-stage disease.

Because of the strong association between tumor biology and stage at diagnosis, we performed analyses stratified by “aggressive” and “nonaggressive” histology to determine the association between race/ethnicity and stage at diagnosis among these subgroups. Among women who had aggressive endometrial tumors, race/ethnicity ( $P=.8487$ ), age ( $P=.1117$ ), and median family income ( $P=.9224$ ) were not associated with stage at diagnosis; only poor tumor grade was associated with advanced stage at diagnosis (OR=13.37; 95% CI=1.64, 108.87). However, among

TABLE 1—Continued

| Tumor grade, no. N(%)     |            |             | <.001 |
|---------------------------|------------|-------------|-------|
| Well differentiated       | 108 (22.1) | 1447 (45.7) |       |
| Moderately differentiated | 118 (24.2) | 905 (28.6)  |       |
| Poorly differentiated     | 118 (24.2) | 410 (12.9)  |       |
| Undifferentiated          | 49 (10.0)  | 80 (2.5)    |       |
| Unknown                   | 95 (19.5)  | 326 (10.3)  |       |

<sup>a</sup>Means were compared using the *t* test; proportions were compared with the Mantel-Haenszel  $\chi^2$  test.

<sup>b</sup>≥25% in census tract did not have a high school diploma.

<sup>c</sup>Inflated to 1998 dollars in accordance with the Consumer Price Index (CPI) on the basis of the patient's census tract at the time of diagnosis.

<sup>d</sup>Lowest quartile = \$6613–\$38378 inflated to 1998 dollars in accordance with the CPI on the basis of the patient's census tract at the time of diagnosis.

women who had endometrial tumors with nonaggressive histology, the results were nearly identical to those shown in Table 2. Similar to the multivariate analysis for the full study population, where either race or median family income, but not both, was independently associated with stage at diagnosis, the multivariate analysis of the subgroup of women who had nonaggressive endometrial tumors showed that either race or median family income, but not both, was associated with stage at diagnosis.

### Treatment

The predominant treatment for endometrial cancer was hysterectomy—89.4% of all women received a hysterectomy as their first course of cancer-directed therapy (Table 3). When we controlled for stage at diagnosis, African American women were less likely to receive a hysterectomy ( $P=.001$ ). This difference in hysterectomy was most pronounced among women who had localized disease, where 95.6% of the White women received a hysterectomy compared with 89.1% of the African American women ( $P=.001$ ). The most common reason for not having a hysterectomy was “contraindicated/not recommended.” There was not a significant racial/ethnic difference in reason for not having a hysterectomy; further analysis of reason for not having a hysterectomy was not performed because there was a lack of standard methods for recording these data in hospital records from which the data were abstracted.

In the univariate analyses, African American women were 61% less likely than White

women to have a hysterectomy as primary treatment (OR=0.39; 95% CI=0.30, 0.50) (Table 2). In the multivariate analysis, women of increased age and women who presented with advanced-stage disease were less likely to receive a hysterectomy, whereas higher median household income was associated with increased likelihood of having a hysterectomy. Notably, race/ethnicity was not an independent predictor of hysterectomy in the multivariate analysis. Because hysterectomy is most frequently recommended for women who have local-stage disease, the analysis of predictors of hysterectomy was repeated among the subgroup of women who had local-stage disease only. The results were nearly identical (Table 2).

African American women were more likely than White women to receive radiation therapy (36.1% vs 30.7%) (Table 3). When we controlled for stage at diagnosis, this difference was not statistically significant ( $P=.204$ ). When compared with White women, African American women who had local-stage disease tended to receive radiotherapy more frequently (28.1% vs 23.7%), yet African American women who had distant-stage disease tended to receive radiotherapy less frequently (27.3% vs 34.6%). These differences were not statistically significant.

### Survival

During the 9-year study period, 1066 (29.2%) women died. The proportion of African American women who died (233 women, 47.7%) was higher than that among

White women (833 women, 26.3%) ( $P<.001$ ). Overall survival time was shorter among African American women (Wilcoxon  $\chi^2=130.8$ ,  $df=1$ ,  $P=0.0001$ ), and this racial/ethnic difference in survival time was apparent within each disease stage (Figure 1). The median survival time among African American women was 61.1 months (95% CI=48.8–71.6 months); among White women, the median survival time was greater than 121 months, more than twice that for African American women.

In general, older women (aged ≥65 years) had shorter survival time compared with younger women (aged <65 years) (mean=75.7 months vs 103.5 months; logrank  $P<.0001$ ). African American women were more likely to die at younger ages (<65 years vs ≥65 years) compared with White women (32.6% vs 23.3%;  $P=.038$ ), and the survival time among African American women was shorter than that among White women within each of these age strata ( $P<.001$ ).

In the univariate analysis, African American women who had endometrial cancer were 2.33 times more likely to die than White women ( $P<.0001$ ) (Table 2). In the best-fitting multivariate model, African American ethnicity, increased age, aggressive histology, advanced-stage disease, and poor tumor grade were independently associated with an increased risk for death, and higher median household income and having had a hysterectomy were independently associated with a decreased risk for death. Additionally, there were 2 significant interactions: advanced-stage disease was more strongly associated with increased risk for death among younger women (<65 years) than among older women (≥65 years), and African American women had a higher risk for death at younger ages compared with White women.

Racial/ethnic differences in survival time also were evaluated by cause of death. For deaths caused by noncancer causes, there was a substantial racial/ethnic difference in survival time (median=16.7 months among African American women vs 32.3 months among White women; logrank  $P=.0002$ ). However, racial/ethnic differences in survival time were not observed for deaths caused by endometrial cancer (median survival=14.2 months among African American women vs



**TABLE 2—Variables Associated With Advanced-Stage Disease, Hysterectomy, and Risk for Death Among African American and White Women Who Had Endometrial Cancer: Detroit, Mich, Tri-County Area, 1990–1998**

|   | OR or RR <sup>a</sup> | 95% CI     | P                   |
|---|-----------------------|------------|---------------------|
| <b>Variables associated with advanced-stage disease</b> |                       |            |                     |
| Univariate results                                      |                       |            |                     |
| Race/ethnicity (African American vs White)              | OR = 1.93             | 1.57, 2.37 | <.0001 <sup>b</sup> |
| Age (5-year increase in age)                            | OR = 1.12             | 1.09, 1.16 | <.0001 <sup>b</sup> |
| Histology (aggressive vs nonaggressive)                 | OR = 4.88             | 3.86, 6.16 | <.0001 <sup>b</sup> |
| Tumor grade   | OR = 5.19             | 4.29, 6.26 | <.0001 <sup>b</sup> |
| Median family income                                    | OR = 0.65             | 0.56, 0.76 | <.0001 <sup>b</sup> |
| Multivariate results                                    |                       |            |                     |
| Model selected:   |                       |            | <.0001 <sup>c</sup> |
| Race/ethnicity (African American vs White)              | OR = 1.41             | 1.09, 1.81 | .0079 <sup>b</sup>  |
| Age   | OR = 1.01             | 1.00, 1.02 | .0073 <sup>b</sup>  |
| Tumor grade   | OR = 4.28             | 3.52, 5.20 | <.0001 <sup>b</sup> |
| Histology (aggressive vs nonaggressive)                 | OR = 2.62             | 1.93, 3.55 | <.0001 <sup>b</sup> |
| Alternate model that forced income:                     |                       |            | <.0001 <sup>c</sup> |
| Median family income                                    | OR = 0.83             | 0.69, 0.99 | .0456 <sup>b</sup>  |
| Age   | OR = 1.01             | 1.00, 1.02 | .0123 <sup>b</sup>  |
| Tumor grade   | OR = 4.31             | 3.55, 5.24 | <.0001 <sup>b</sup> |
| Histology (aggressive vs nonaggressive)                 | OR = 2.73             | 2.02, 3.69 | <.0001 <sup>b</sup> |
| <b>Variables associated with hysterectomy</b>           |                       |            |                     |
| Univariate results for all stages of disease            |                       |            |                     |
| Race/ethnicity (African American vs White)              | OR = 0.39             | 0.30, 0.50 | <.0001 <sup>b</sup> |
| Age (5-year increase in age)                            | OR = 0.77             | 0.73, 0.81 | <.0001 <sup>b</sup> |
| Stage (advanced vs not advanced)                        | OR = 0.27             | 0.21, 0.34 | <.0001 <sup>b</sup> |
| Median family income                                    | OR = 2.16             | 1.77, 2.63 | <.0001 <sup>b</sup> |
| Multivariate results for all stages of disease          |                       |            |                     |
| Model selected:   |                       |            | <.0001 <sup>c</sup> |
| Median family income                                    | OR = 1.70             | 1.34, 2.15 | <.0001 <sup>b</sup> |
| Age   | OR = 0.95             | 0.94, 0.96 | <.0001 <sup>b</sup> |
| Stage (advanced vs not advanced)                        | OR = 0.31             | 0.24, 0.40 | <.0001 <sup>b</sup> |
| Univariate results for local stage                      |                       |            |                     |
| Race/ethnicity (African American vs White)              | OR = 0.38             | 0.25, 0.59 | <.0001 <sup>b</sup> |
| Age (5-year increase in age)                            | OR = 0.78             | 0.72, 0.85 | <.0001 <sup>b</sup> |
| Median family income                                    | OR = 2.27             | 1.64, 3.14 | <.0001 <sup>b</sup> |
| Multivariate results for local stage                    |                       |            |                     |
| Model selected:   |                       |            | <.0001 <sup>c</sup> |
| Median family income                                    | OR = 2.13             | 1.53, 2.97 | <.0001 <sup>b</sup> |
| Age   | OR = 0.95             | 0.94, 0.97 | <.0001 <sup>b</sup> |
| <b>Variables associated with risk for death</b>         |                       |            |                     |
| Univariate results                                      |                       |            |                     |
| Race/ethnicity (African American vs White)              | RR = 2.33             | 1.75, 3.10 | <.0001 <sup>b</sup> |
| Age   | RR = 1.07             | 1.06, 1.07 | <.0001 <sup>b</sup> |
| Histology (aggressive vs nonaggressive)                 | RR = 3.64             | 3.14, 4.21 | <.0001 <sup>b</sup> |
| Tumor grade   | RR = 2.84             | 2.44, 3.29 | <.0001 <sup>b</sup> |
| Stage (advanced vs not advanced)                        | RR = 4.67             | 4.12, 5.30 | <.0001 <sup>b</sup> |
| Median family income                                    | RR = 0.51             | 0.45, 0.57 | <.0001 <sup>b</sup> |
| Hysterectomy (yes vs no)                                | RR = 0.19             | 0.17, 0.22 | <.0001 <sup>b</sup> |

Continued

14.7 months among White women; logrank  $P = .7953$ ). The multivariate analysis was repeated among the subgroup of women who died as a result of endometrial cancer. Among this subgroup, increased age and advanced-stage disease were still associated with an increased risk for death, and having had a hysterectomy was still associated with decreased risk for death. Race/ethnicity was not associated with risk for death.

## DISCUSSION

Among this population-based study group, higher median household income was associated with a decreased likelihood of presenting with advanced-stage disease, an increased likelihood of having had a hysterectomy as primary treatment, and a decreased risk for death. In the multivariate analyses, the results suggested that income level and race/ethnicity were somewhat interchangeable when we examined differences in stage at diagnosis and treatment.

In the multivariate analysis, African American ethnicity and decreased median household income were associated with advanced-stage endometrial cancer independently but not simultaneously. When the analysis was stratified into subgroups of aggressive versus nonaggressive endometrial tumors, a notable pattern emerged: among women who had aggressive endometrial tumors, neither race/ethnicity nor median family income was associated with stage at diagnosis, which suggests that sociocultural factors may not differentiate prognostic outcomes among women who had aggressive endometrial tumors. However, among women who had nonaggressive endometrial tumors, race/ethnicity or median family income, but not both, was independently associated with stage at diagnosis. This finding suggests that race/ethnicity and SES similarly influence the risk for presenting with advanced-stage endometrial cancer among women who have nonaggressive endometrial cancer, who usually have an excellent prognosis when tumors are detected early.

Our study showed that higher income was associated with an increased likelihood of receiving a hysterectomy as the primary treatment for endometrial cancer. Although



TABLE 2—Continued

|  |           |            |  |                     |
|--|-----------|------------|--|---------------------|
| Multivariate results                         |           |            |  |                     |
| Model selected:                              |           |            |  |                     |
| Race/ethnicity (African American vs White)   | RR = 1.73 | 1.26, 2.37 |  | <.0001 <sup>c</sup> |
| Age (in quartiles)                           | RR = 1.84 | 1.69, 2.01 |  | <.0001 <sup>b</sup> |
| Histology (aggressive vs nonaggressive)      | RR = 1.71 | 1.39, 2.09 |  | <.0001 <sup>b</sup> |
| Stage (advanced vs not advanced)             | RR = 4.74 | 3.72, 6.03 |  | <.0001 <sup>b</sup> |
| Tumor grade                                  | RR = 1.60 | 1.36, 1.88 |  | <.0001 <sup>b</sup> |
| Median family income                         | RR = 0.80 | 0.68, 0.94 |  | .0065 <sup>b</sup>  |
| Hysterectomy (yes vs no)                     | RR = 0.23 | 0.19, 0.28 |  | <.0001 <sup>b</sup> |
| Interaction between race/ethnicity and age   | RR = 0.64 | 0.45, 0.91 |  | .0141 <sup>b</sup>  |
| Interaction between age and stage of disease | RR = 0.54 | 0.41, 0.71 |  | <.0001 <sup>b</sup> |

Note. OR = odds ratio; RR = relative risk; CI = confidence interval.

<sup>a</sup>ORs derived from the logistic regression model; RRs derived from the Cox proportional hazards model.

<sup>b</sup>Derived from Wald  $\chi^2$ .

<sup>c</sup>Derived from score statistic.

TABLE 3—Treatment for Endometrial Cancer Among African American and White Women Who Had Endometrial Cancer: Detroit, Mich, Tri-County Area, 1990–1998

|                                   | Total       | African American | White       | P                    |
|-----------------------------------|-------------|------------------|-------------|----------------------|
| Hysterectomy, no. (%)             | 3270 (89.4) | 388 (79.5)       | 2882 (91.0) | <.001 <sup>a,b</sup> |
| Local stage                       | 2439 (94.9) | 238 (89.1)       | 2201 (95.6) | <.001 <sup>b</sup>   |
| Regional stage                    | 568 (88.9)  | 94 (86.2)        | 474 (89.4)  | .334 <sup>b</sup>    |
| Distant stage                     | 230 (71.9)  | 45 (68.2)        | 185 (72.8)  | .455 <sup>b</sup>    |
| Unknown stage                     | 33 (26.0)   | 11 (23.9)        | 22 (27.2)   |                      |
| No hysterectomy, no. (%)          | 386 (10.6)  | 100 (20.5)       | 286 (9.0)   |                      |
| Reason for no hysterectomy        |             |                  |             | .176 <sup>b</sup>    |
| Other surgery (e.g., biopsy, D&C) | 16 (4.2)    | 6 (6.0)          | 10 (3.5)    |                      |
| Contraindicated/not recommended   | 249 (64.5)  | 58 (58.0)        | 191 (66.8)  |                      |
| Unknown reason                    | 50 (13.0)   | 12 (12.0)        | 38 (13.3)   |                      |
| Refused                           | 36 (9.3)    | 13 (13.0)        | 23 (8.0)    |                      |
| Planned                           | 31 (8.0)    | 11 (11.0)        | 20 (7.0)    |                      |
| Unknown if performed              | 4 (1.0)     | 0 (0.0)          | 4 (1.4)     |                      |
| Radiation Therapy, no. (%)        |             |                  |             |                      |
| No                                | 2503 (68.5) | 312 (63.9)       | 2191 (69.3) |                      |
| Yes                               | 1149 (31.5) | 176 (36.1)       | 973 (30.7)  | .204 <sup>b,c</sup>  |
| Local stage                       | 621 (24.2)  | 75 (28.1)        | 546 (23.7)  | .113 <sup>b</sup>    |
| Regional stage                    | 392 (61.3)  | 68 (62.4)        | 324 (61.1)  | .807 <sup>b</sup>    |
| Distant stage                     | 106 (33.1)  | 18 (27.3)        | 88 (34.6)   | .258 <sup>b</sup>    |
| Unknown stage                     | 30 (24.4)   | 15 (32.6)        | 15 (19.5)   | .102 <sup>b</sup>    |

<sup>a</sup>For hysterectomy vs no hysterectomy, after stage of disease was controlled.

<sup>b</sup>Mantel-Haenszel  $\chi^2$  test.

<sup>c</sup>For radiotherapy vs no radiotherapy, after stage of disease was controlled.

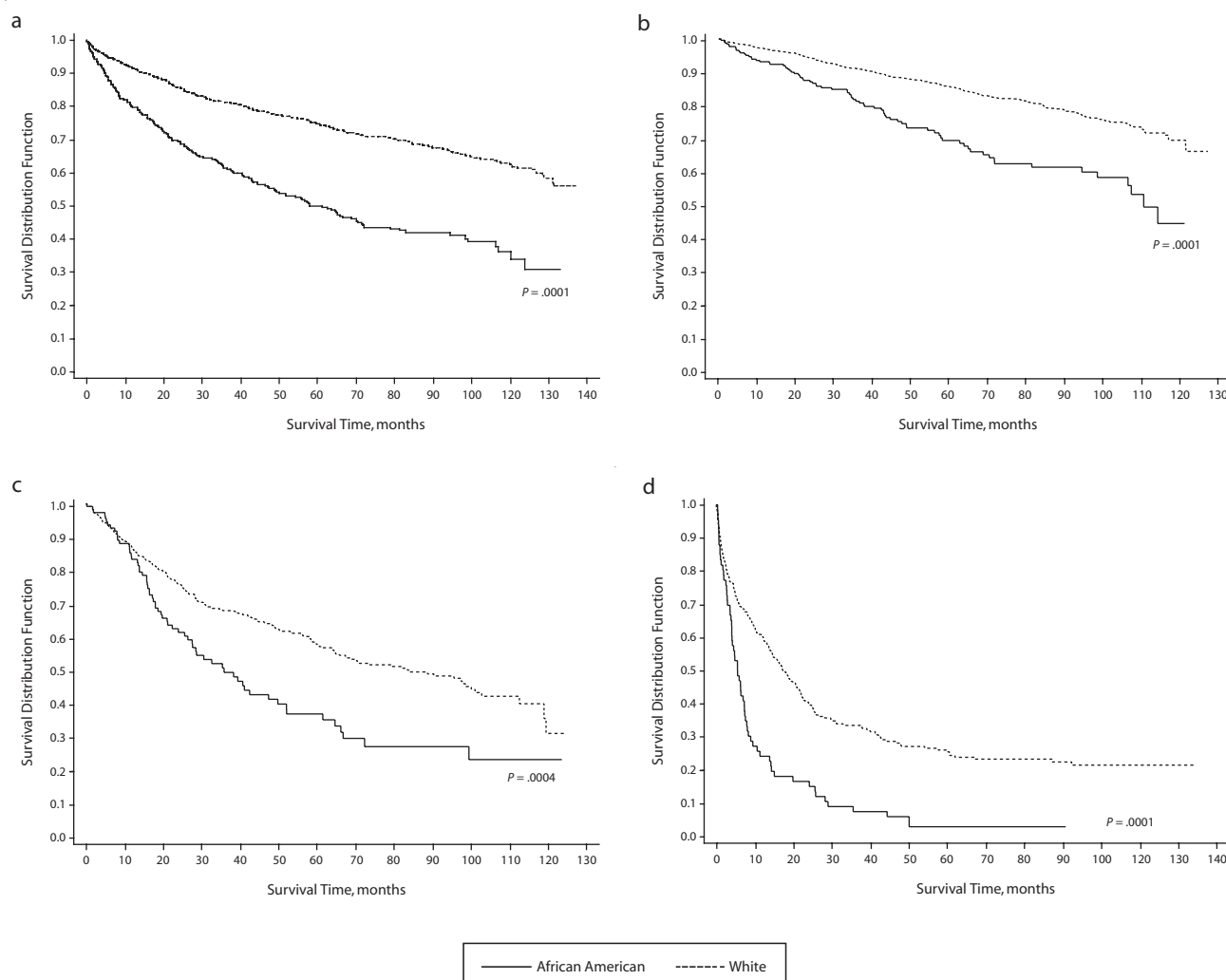
African American women had a decreased likelihood of receiving a hysterectomy. This association was apparent even within the subgroup of women who had local-stage disease, where hysterectomy is usually recommended, and is consistent with findings from a recent literature review that found evidence of racial/ethnic disparities in the receipt of definitive primary-cancer therapy.<sup>16</sup> However, in our multivariate analysis, income, but not race/ethnicity, was independently associated with having had a hysterectomy. Therefore, the racial/ethnic disparities in treatment for endometrial cancer may be mediated by differences in SES. Additionally, the prevalence of comorbid factors may represent contraindications to radical hysterectomy and may potentially confound the associations we found between treatment and race/ethnicity and SES. However, the availability of alternative options, such as vaginal hysterectomy with laparoscopic lymphadenectomy among medically compromised patients, emphasizes the importance of availability of quality medical care.<sup>32,33</sup>

When compared with White women, the African American women in our study had an increased risk for death after we controlled for aggressive tumor biology, advanced age, treatment, and income; this association was particularly apparent among younger African American women ( $P=0.0001$ ). However, when survival was evaluated by cause of death, racial/ethnic differences were observed only among noncancer-related deaths. This result is different than that reported by Bach et al., where the risk for death caused by uterine cancer among African Americans was still twice that among Whites after they controlled for stage of disease, treatment, and other causes of mortality (hazard ratio=2.08; 95% CI=1.34, 3.21).<sup>34</sup> Among women diagnosed with endometrial cancer, this might suggest that the racial/ethnic differences subsequently observed in survival are influenced by variables unrelated to endometrial cancer, including competing causes of mortality. However, these results need to be interpreted with caution because cause of death was not validated in our study.

A limitation to our study was the use of census data collected in 1990 for assigning SES variables to cases diagnosed between

epidemiological studies of all-cause hysterectomies have shown that lower SES is associated with increased receipt of hysterectomy,<sup>28</sup> only 11% of the hysterectomies in the United States are performed to treat uterine can-

cer.<sup>29,30</sup> Variables such as age, race/ethnicity, geographic residence, and medical history<sup>31</sup> also may influence whether hysterectomies are performed to treat women who have endometrial cancer. In the univariate analysis,



**FIGURE 1—Survival, overall and by stage at diagnosis, among African American and White women who had endometrial cancer in the Detroit tri-county area, 1990–1998: (a) all stages combined, (b) local, (c) regional, (d) distant.**

1990 and 1998, thus allowing census data to be extrapolated over an 8-year period. However, a recent methodological study showed little impact when census data up to 10 years were used.<sup>27</sup> Also, census block group data are preferable to the use of census tract data because the former minimizes heterogeneity in the unit of aggregation as a result of the smaller number of persons included in the aggregate.<sup>35</sup> However, a recent methodological study showed little gain in accuracy when census block group data were used in place of census tract data.<sup>36</sup>

A major strength of our study was the generalizability that was a result of the large study population we obtained from a population-based registry that had complete ascertainment of cases. Very few data items were missing, and the registry provided a range of 2.7 to 10.6 years of follow-up.

## CONCLUSIONS

The relationship between SES and cancer survival is complex because SES has an impact on multiple risk factors associated with neoplastic transformation, progression, and

death. Our study is unique in its examination of the impact of SES, independently of race/ethnicity, among a geographically defined patient population that had endometrial cancer. Although age and tumor biology were strongly associated with prognosis, women at lower income levels were more likely to manifest advanced-stage disease, less likely to receive a hysterectomy as their primary treatment, and had poorer survival rates. Because of the strong association between SES and race/ethnicity, improving access to quality health care among low-SES women to facilitate earlier diagnosis and optimal treatment may serve to

diminish the racial/ethnic difference in endometrial cancer survival. ■

### About the Authors

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### Contributors

T. Madison was responsible for originating the study, analyzing and interpreting the data, and writing the article. D. Schottenfeld, S.A. James, A.G. Schwartz, and S.B. Gruber assisted with originating the study and its design. All the authors originated ideas, interpreted results, and reviewed drafts of the article.

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### Human Participant Protection

This study was approved by the institutional review boards at Wayne State University and the University of Michigan Medical School. Informed consent was obtained from each individual in the validation study, which was approved by both the Wayne State University Human Investigation Committee and the University of Michigan Medical School institutional review board.

### References

- Shingleton HM, Fowler WC, Jordan JA, Lawrence WD. *Gynecologic Oncology: Current Diagnosis and Treatment*. London, UK: W.B. Saunders; 1996.
- Platz CE, Benda JA. Female genital tract cancer. *Cancer*. 1995;75(Suppl 1):270-294.
- Ries LAG, Eisner MP, Kosary CL, et al., eds. *SEER Cancer Statistics Review, 1973-1998*. Bethesda, Md: National Cancer Institute; 2001.
- Hill HA, Eley JW, Harlan LC, Greenberg RS, Barrett RJ II, Chen VW. Racial differences in endometrial cancer survival: the black/white cancer survival study. *Obstet Gynecol*. 1996;88:919-926.
- Barrett RJ, Harlan LC, Wesley MN, et al. Endometrial cancer: stage at diagnosis and associated factors in black and white patients. *Am J Obstet Gynecol*. 1995;173:414-423.
- Madison TM, Schottenfeld D, Baker V. Cancer of the corpus uteri in white and black women in Michigan, 1985-1994: an analysis of trends in incidence and mortality and their relation to histologic subtype and stage. *Cancer*. 1998;83:1546-1554.
- Hill HA, Coates RJ, Austin H, et al. Racial differences in tumor grade among women with endometrial cancer. *Gynecol Oncol*. 1995;56:154-163.
- Cronje HS, Fourie S, Doman MJ, Helms JB, Nel JT, Goedhals L. Racial differences in patients with adenocarcinoma of the endometrium. *Int J Gynaecol Obstet*. 1992;39:213-218.
- Aziz H, Hussain F, Edelman S, et al. Age and race as prognostic factors in endometrial carcinoma. *Am J Clin Oncol*. 1996;19:595-600.
- Bain RP, Greenberg RS, Chung KC. Racial differences in survival of women with endometrial cancer. *Am J Obstet Gynecol*. 1987;157:914-923.
- Statistical Abstract of the United States*. 117th ed. Washington, DCS: US Bureau of the Census; 1997.
- Liu JR, Conaway M, Rodriguez GC, Soper JT, Clarke-Pearson DL, Berchuck A. Relationship between race and interval to treatment in endometrial cancer. *Obstet Gynecol*. 1995;86:486-490.
- Coates RJ, Click LA, Harlan LC, et al. Differences between black and white patients with cancer of the uterine corpus in interval from symptom recognition to initial medical consultation (United States). *Cancer Causes Control*. 1996;7:328-336.
- Matthews RP, Hutchinson-Colas J, Maiman M, et al. Papillary serous and clear cell type lead to poor prognosis of endometrial carcinoma in black women. *Gynecol Oncol*. 1997;65:206-212.
- Pirog EC, Heller DS, Westhoff C. Endometrial adenocarcinoma—lack of correlation between treatment delay and tumor stage. *Gynecol Oncol*. 1997;67:303-308.
- Shavers V, Brown ML. Racial and ethnic disparities in the receipt of cancer treatment. *J Natl Cancer Inst*. 2002;94:334-357.
- National Cancer Institute. *Summary Staging Guide, SEER Program*. Bethesda, Md: National Institute of Health; 1981. NIH Publication No. 81.
- International Classification of Diseases for Oncology*. 2nd edition. Geneva, Switzerland: World Health Organization; 1990.
- Rose PG. Endometrial carcinoma. *N Engl J Med*. 1996;335:640-649.
- Hoskins WJ, Perez CA, Young RC. *Principles and Practice of Gynecologic Oncology*. Philadelphia, Pa: J.B. Lippincott; 1992.
- World Health Organization. Histologic typing of female genital tract tumors. In: Scully RE, Bonfiglio TA, Kurman RJ, et al., eds. *International Histologic Classification of Tumours*. 2nd edition. New York, NY: Springer-Verlag; 1994.
- US Bureau of the Census. *Census of Population and Housing, 1990*. Summary Tape File 3 on CD-ROM [machine-readable data files]. Washington, DC: US Bureau of the Census; 1992.
- Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health*. 1997;18:341-378.
- SAS Language Reference, Version 6*. 1st edition. Cary, NC: SAS Institute Inc; 1990.
- SAS Procedures Guide, Version 6*. 3rd edition. Cary, NC: SAS Institute Inc; 1990.
- Landis JR, Koch GG. The measurement of observer agreement for categorical data. *Biometrics*. 1977;33:159-174.
- Geronimus AT, Bound J. Use of census-based aggregate variables to proxy for socioeconomic group: evidence from national samples. *Am J Epidemiol*. 1998;148:475-486.
- Kjerulff K, Langenberg P, Guzinski G. The socioeconomic correlates of hysterectomies in the United States. *Am J Public Health*. 1993;83:106-108.
- Pokras R, Hufnagel VG. Hysterectomies in the United States, 1965-84. *Vital Health Stat*. 1987;92:1-32.
- Lepine LA, Hillis SD, Marchbanks PA, et al. Hysterectomy Surveillance—United States, 1980-1993. *MMWR Morb Mortal Wkly Rep*. 1997;46:1-14.
- Palmer JR, Sowmya RR, Adams-Campbell LL, Rosenberg L. Correlates of hysterectomy among African-American women. *Am J Epidemiol*. 1999;150:1309-1315.
- Chan JK, Lin YG, Monk BJ, Tewari K, Bloss JD, Berman ML. Vaginal hysterectomy as primary treatment of endometrial cancer in medically compromised women. *Obstet Gynecol*. 2001;97:707-711.
- Eltabbakh GH, Shamonki MI, Moody JM, Garafano LL. Hysterectomy for obese women with endometrial cancer: laparoscopy or laparotomy? *Gynecol Oncol*. 2000;78:329-335.
- Bach PB, Schrag D, Brawley OW, Galaznik A, Yakren S, Begg CB. Survival of blacks and whites after a cancer diagnosis. *JAMA*. 2002;287:2106-2113.
- Krieger N. Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am J Public Health*. 1992;82:703-710.
- Soobader M, LeClere FB, Hadden W, Maury B. Using aggregate geographic data to proxy individual socioeconomic status: does size matter? *Am J Public Health*. 2001;91:632-636.

# Is Lipid-Lowering Therapy Underused by African Americans at High Risk of Coronary Heart Disease Within the VA Health Care System?

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Coronary heart disease causes more than 500 000 deaths annually in the United States.<sup>1</sup> Although studies indicate a reduction in coronary heart disease mortality across all ethnic groups, this decline has been less significant in African Americans.<sup>2</sup> Researchers have examined numerous potential causes of this disparity, including the more frequent occurrence of coronary heart disease risk factors seen in the African American population.<sup>3–5</sup> One of the most significant of these coronary risk factors is hypercholesterolemia. The association between hypercholesterolemia and coronary heart disease has been well established and is consistent across gender, race/ethnicity, and age.<sup>6–13</sup> Several clinical trials conclusively demonstrated that lowering total cholesterol and low-density lipoprotein (LDL) levels in patients with coronary heart disease resulted in substantial reductions in recurrent ischemia, mortality, and need for revascularization procedures.<sup>14–19</sup> These findings and clinical guidelines established by the National Cholesterol Education Program (NCEP)<sup>20</sup> indicate that vigorous cholesterol management is required in patients with coronary heart disease.

Despite widespread dissemination of the NCEP guidelines and clear evidence that appropriate cholesterol management favorably affects coronary heart disease morbidity and mortality, hypercholesterolemia remains inadequately diagnosed and treated. Studies have indicated that 33–50% of patients with known coronary heart disease do not receive screening with comprehensive lipid panels.<sup>21–23</sup> Rates of treatment with cholesterol-lowering therapy are similarly low, with only one third of appropriate patients receiving lipid-lowering medications.<sup>24</sup> Other studies have revealed that 25% or fewer patients achieve target LDL levels.<sup>21,22,25,26</sup>

Studies of the influence of patient race on the management of hypercholesterol-

emia have provided inconsistent results. In population-based surveys conducted from 1985–1994, African Americans reported lower rates of awareness, screening, and treatment of high cholesterol than Whites.<sup>27–29</sup> By contrast, more recent data from the 1996 Medical Expenditure Study and the Cardiovascular Health Study showed no differences between African American and White patients in rates of cholesterol screening and treatment.<sup>30,31</sup> Although these studies provided valuable information about recent trends in the diagnosis and treatment of hypercholesterolemia, they largely focused on primary prevention. Therefore, they do not adequately reflect treatment of patients with coronary heart disease who are at highest risk for recurrent events. Given that African Americans have a greater risk of death from coronary heart disease than Whites and that cholesterol lowering confers substantial survival benefits, it is imperative to identify whether disparities in cholesterol monitoring and treatment exist, particularly among the high-risk group of patients with established disease.

We examined a cohort of 1045 veterans with established coronary heart disease to determine whether racial differences exist in cholesterol management for secondary prevention

**Objectives.** We examined whether racial differences exist in cholesterol monitoring, use of lipid-lowering agents, and achievement of guideline-recommended low-density lipoprotein (LDL) levels for secondary prevention of coronary heart disease.

**Methods.** We reviewed charts for 1045 African American and White patients with coronary heart disease at 5 Veterans Affairs (VA) hospitals.

**Results.** Lipid levels were obtained in 67.0% of patients. Whites and African Americans had similar screening rates and mean lipid levels. Among the 544 ideal candidates for therapy, rates of treatment and achievement of target LDL levels were similar.

**Conclusions.** We found no disparities in cholesterol management. This absence of disparities may be the result of VA quality improvement initiatives or prescription coverage through the VA health care system. (*Am J Public Health.* 2004; 94:2112–2117)

of coronary heart disease. Importantly, prior studies of racial disparities in health care use may have been confounded by unequal access to health care services, such as preventive health screening and prescription medications. Hyperlipidemia is asymptomatic, and lipid-lowering agents are expensive. Thus, patients with limited incomes may choose not to comply with lipid-lowering therapy. Therefore, because the effects of medication costs and insurance access are minimized in the Veterans Affairs (VA) health care system, VA hospitals provide an ideal setting to examine racial disparities in treatment.<sup>32</sup>

## METHODS

### Study Design and Population

Data were collected from baseline surveys and medical records of patients who participated in a study that examined differences between African American and White patients in attitudes regarding use of invasive cardiac procedures.<sup>33</sup> To establish a prospective cohort of patients who were likely to have coronary heart disease, we screened the results of all cardiac nuclear imaging studies performed at 5 VA hospital sites (Atlanta/Decatur, Ga, Durham, NC, Houston, Tex, Pittsburgh, Pa,



and St. Louis, Mo) between August 1, 1999, and January 31, 2001. We initially identified 5278 patients who underwent a nuclear imaging study. Of these, 2335 (44%) met criteria for a positive study. A study was positive if there was any evidence of reversible cardiac ischemia. We excluded 961 patients for the following reasons: 456 (20%) could not be contacted to enroll them in the study; 78 (3%) had impaired mental status; 32 (1%) were in another research study determining their cardiac treatment; 102 (4%) were not African American or White; 189 (8%) underwent a cardiac procedure in the preceding 6 months; 5 (<1%) were not veterans; and 99 (4%) were excluded for miscellaneous other reasons (e.g., patient died before study enrollment). After these exclusions, 1374 patients with positive imaging studies remained. Of these, 329 refused to participate, did not return their informed consent, or requested questionnaires but did not return them. Thus, 1045 patients were included in the final cohort, representing a 76% response rate ( $1045/1374=76\%$ ). Of the 1045 participants, 236 were African American and 809 were White. Ninety-eight percent of patients in our study were men, reflecting the predominantly male population served by the Department of Veterans Affairs. This sample size provided 80% power to detect a 10% difference in the use of lipid-lowering agents between African American and White patients.

We examined racial differences in cholesterol monitoring and treatment for secondary prevention of coronary heart disease. Because every participant had a diagnosis of coronary heart disease, all were deemed appropriate for secondary prevention. We examined computerized medical records to determine documented lipid levels, appropriateness for treatment with lipid-lowering therapy, medication use, contraindications to lipid-lowering medications, and coexisting medical conditions. Total cholesterol and LDL levels documented within 3 months before enrollment or during any inpatient admission after enrollment were included in this analysis. Participants completed baseline surveys to provide demographic data.

### Guidelines for Cholesterol Management

Appropriate diagnosis and treatment of hypercholesterolemia was based on the

NCEP Adult Treatment Panel II guidelines, which were in effect at the time of study enrollment.<sup>20</sup> These guidelines stated that for secondary prevention in patients with coronary heart disease, lipid levels should be obtained for all patients, and therapy should be initiated on the basis of LDL cholesterol levels. The target of therapy in these patients is an LDL level of 100 mg/dL or less. Liver disease and allergy to lipid-lowering agents are absolute contraindications to the use of hydroxymethylglutaryl-CoA reductase inhibitor (statin) therapy for management of hypercholesterolemia, and age younger than 35 years old, dementia, and terminal illness are relative contraindications. Therefore, candidates were considered ideal for treatment if they had an LDL cholesterol level exceeding 100 mg/dL or were receiving lipid-lowering therapy during the study period, were at least 35 years old, and did not have a diagnosis of dementia, alcohol abuse (a significant contributor to liver disease), cirrhosis, terminal illness, or lipid-lowering agent allergy.

### Study Variables

The independent variable was self-reported race. The dependent variables were cholesterol monitoring, treatment with lipid-lowering agents, and achievement of target LDL levels. We ascertained use of the following classes of lipid-lowering agents: hydroxymethylglutaryl-CoA reductase inhibitors, fibrates, bile acid resins, and niacin. These medication classes were combined into a dichotomous (treatment yes or no) variable.

### Data Collection

Registered nurses with extensive cardiology and chart review experience abstracted data from the medical records. Patient demographics; documented lipid values; relevant laboratory values; and data on lipid-lowering agent use, adverse reaction to lipid-lowering agents, and comorbid medical conditions, including coronary heart disease risk factors, were collected on all patients.

### Data Analysis

We used Statistical Analysis Software (SAS), Version 8.2 (SAS Institute Inc., Cary, NC) to perform statistical analyses. Simple descriptive statistics were used to describe the study pop-

ulation. We used  $\chi^2$  and *t* tests to assess racial differences in cholesterol monitoring and the use of lipid-lowering agents where appropriate. We assessed lipid-lowering agent use in the entire population and in ideal candidates only. For the ideal candidate analysis, patients with contraindications to lipid-lowering agent use were excluded regardless of whether they were already receiving therapy.

## RESULTS

### Patient Characteristics

The cohort consisted of 236 African American and 809 White patients. Table 1 displays the characteristics of these patients by race. African Americans were more likely than Whites to be younger and unmarried (both  $P<.01$ ), but there was no difference between the two groups in educational level attained. African American patients were more likely than White patients to have a history of hypertension (85.7% vs 76.2%, respectively;  $P=.002$ ). African American participants were less likely than White participants to have had a prior myocardial infarction or cerebrovascular accident, hypercholesterolemia, chronic obstructive pulmonary disease, prior coronary artery bypass graft surgery, or prior percutaneous coronary intervention (all  $P<.05$ ). The two groups had similar rates of smoking, angina, and peripheral arterial disease.

### Cholesterol Screening

Table 2 displays cholesterol screening information for the two groups. African American and White patients were equally likely to receive cholesterol monitoring. There were no differences between the two groups in rates of documented total cholesterol and LDL values. Similarly, there were no differences between African Americans and Whites in the percentage of patients receiving any cholesterol (total cholesterol or LDL) monitoring (64.8% vs 67.6%, respectively;  $P=.42$ ). These results indicate that, although rates of cholesterol screening are similar between African Americans and Whites, approximately one third of all patients with established coronary heart disease did not receive appropriate cholesterol monitoring in the time interval we examined. Of those patients receiving cholesterol screening, African



**TABLE 1—Characteristics of African American and White Patients With Coronary Heart Disease Who Underwent Cardiac Nuclear Imaging Studies (n = 1045): 5 VA Hospitals, August 1, 1999, through January 31, 2001**

|  | White (n = 809) | African American (n = 236) | P      |
|--|-----------------|----------------------------|--------|
| Mean age (SD), y                           | 63.3 (9.7)      | 61.3 (11.1)                | .008   |
| Mean education (SD), y                     | 12.1 (2.7)      | 11.9 (2.8)                 | .32    |
| Unmarried, %                               | 37.8            | 52.1                       | <.0001 |
| History of disease, %                      |                 |                            |        |
| Congestive heart failure                   | 17.8            | 17.2                       | .85    |
| Diabetes                                   | 31.1            | 35.1                       | .25    |
| Hypertension                               | 76.2            | 85.7                       | .002   |
| Hypercholesterolemia                       | 65.0            | 54.8                       | .005   |
| Chronic obstructive pulmonary disease      | 27.3            | 18.7                       | .008   |
| Smoking status, %                          |                 |                            |        |
| Former smoker                              | 62.9            | 63.8                       | .81    |
| Current smoker                             | 30.9            | 33.2                       | .51    |
| Cardiovascular disease diagnosis, %        |                 |                            |        |
| Prior myocardial infarction                | 33.9            | 25.7                       | .02    |
| Angina                                     | 64.8            | 65.5                       | .84    |
| Prior coronary artery bypass graft surgery | 24.2            | 8.2                        | <.0001 |
| Prior percutaneous coronary intervention   | 23.7            | 11.6                       | <.0001 |
| Prior cerebrovascular accident             | 17.7            | 9.5                        | .003   |
| Peripheral arterial disease                | 17.7            | 16.0                       | .55    |

Note. VA = Veterans Affairs.

**TABLE 2—Comparison of Cholesterol Measurements in African American and White Patients With Coronary Heart Disease Who Underwent Cardiac Nuclear Imaging Studies (n = 1045): 5 VA Hospitals, August 1, 1999, through January 31, 2001**

|  | White (n = 809) | African American (n = 236) | P   |
|--|-----------------|----------------------------|-----|
| Lipid level documented, %                        |                 |                            |     |
| Total cholesterol                                | 66.0            | 62.3                       | .29 |
| Low density lipoprotein                          | 57.2            | 55.1                       | .56 |
| Total cholesterol or LDL                         | 67.6            | 64.8                       | .42 |
| Total cholesterol, mean (SD), mg/dL <sup>a</sup> | 188.7 (48.8)    | 194.0 (63.8)               | .13 |
| LDL, mean (SD), mg/dL <sup>b</sup>               | 112.4 (37.8)    | 118.2 (41.1)               | .27 |

Note. VA = Veterans Affairs; LDL = low-density lipoprotein.

<sup>a</sup>For those patients who had total cholesterol levels documented (n = 681).

<sup>b</sup>For those patients who had LDL levels documented (n = 593).

American and White patients had similar mean total cholesterol values (194.0 mg/dL vs 188.7 mg/dL, respectively;  $P=.13$ ) and mean LDL values (118.2 mg/dL vs 112.4 mg/dL, respectively;  $P=.27$ ).

### Use of Lipid-Lowering Therapy

Table 3 displays the use of lipid-lowering therapy in the two groups. We initially as-

sessed overall use of lipid-lowering medications in the entire sample, without exclusions for ideal candidates. Among all patients in our cohort, African Americans were less likely than Whites to receive treatment with lipid-lowering agents (46.2% vs 59.6%, respectively;  $P=.0003$ ). We also assessed the use of lipid-lowering medications in subsets of patients with specific cardiovascular disease

diagnoses. Among patients with a history of angina or prior cerebrovascular accident, African Americans were less likely than Whites to receive lipid-lowering therapy. However, when the comparison was restricted to the 544 patients who met the definition of ideal candidate for treatment with lipid-lowering agents, African Americans and Whites were equally likely to receive treatment (96.9% vs 98.9%, respectively;  $P=.96$ ).

### Achieving LDL Goals

Table 4 displays the mean LDL levels and low rates of achieving guideline-recommended LDL goals among ideal patients receiving lipid-lowering medication. Of those patients who had documented LDL levels, only 40% reached the target LDL of 100 mg/dL or less. However, of these, African American and White patients were equally likely to achieve target LDL levels (32.8% vs 41.4%, respectively;  $P=.21$ ). Mean LDL levels of African American and White patients who successfully reached the LDL goal of 100 mg/dL or less were 81.9 mg/dL versus 76.7 mg/dL, respectively;  $P=.20$ . Mean LDL levels for those who failed to achieve target LDL levels were significantly higher than guideline-recommended levels of 100 mg/dL overall but did not differ by race (141.9 mg/dL vs 134.7 mg/dL, respectively;  $P=.18$ ).

### DISCUSSION

We assessed cholesterol management in African American and White patients with coronary heart disease who were receiving care within the VA health care system. African American and White patients were equally likely to receive cholesterol screening, although overall rates of screening did not reach the level recommended by guidelines in either group. Although White patients were more likely to have a documented diagnosis of hypercholesterolemia, there were no significant differences in measured lipid levels between the two groups. In the subset of 544 patients who were ideal candidates for treatment according to an accepted national guideline, African American patients were equally likely to receive lipid-lowering therapy, and nearly all of the patients identified as appropriate for treatment

**TABLE 3—Use of Lipid-Lowering Agents by African American and White Patients With Coronary Heart Disease Who Underwent Cardiac Nuclear Imaging Studies: 5 VA Hospitals, August 1, 1999, through January 31, 2001**

|  | White (n = 809) | African American (n = 236) | P     |
|--|-----------------|----------------------------|-------|
| All patients (n = 1045), %                           | 59.6            | 46.2                       | .0003 |
| Ideal candidates for treatment only (n = 544), %     | 98.9            | 96.9                       | .96   |
| Cardiovascular disease diagnosis, %                  |                 |                            |       |
| Prior myocardial infarction (n = 328)                | 71.4            | 64.4                       | .29   |
| Angina (n = 669)                                     | 65.7            | 52.7                       | .004  |
| Prior coronary artery bypass graft surgery (n = 211) | 76.0            | 63.2                       | .22   |
| Prior percutaneous coronary intervention (n = 161)   | 78.7            | 75.0                       | .71   |
| Prior cerebrovascular accident (n = 164)             | 70.4            | 45.5                       | .02   |
| Peripheral arterial disease (n = 178)                | 70.2            | 59.5                       | .21   |

**TABLE 4—Comparison of LDL Levels Among Ideal African American and White Candidates Receiving Lipid-Lowering Therapy: 5 VA Hospitals, August 1, 1999, through January 31, 2001**

|   | White        | African American | P   |
|---|--------------|------------------|-----|
| No. of ideal candidates receiving lipid-lowering therapy            | 442          | 94               |     |
| Patients successfully achieving target LDL levels, %                | 41.4         | 32.8             | .21 |
| LDL for ideal candidates achieving target LDL, mean (SD), mg/dL     | 76.7 (17.5)  | 81.9 (13.5)      | .20 |
| LDL for ideal candidates not achieving target LDL, mean (SD), mg/dL | 134.7 (29.8) | 141.9 (37.5)     | .18 |

Note. LDL = low-density lipoprotein.

received medications. However, among ideal patients receiving lipid-lowering medications, only 40% of patients overall achieved target LDL levels. Thus, our findings highlight the need for improvement in cholesterol screening and the achievement of guideline-recommended LDL levels for all patients once screening is performed.

Our study extended prior work, which examined the provision of guideline-based treatment of hypercholesterolemia in two important ways. First, we assessed the association of patient race with receiving cholesterol screening and treatment in patients with established coronary heart disease. Given the high mortality rate in this population, the survival benefit associated with appropriate use of lipid-lowering therapy was particularly large in this group of patients. Second, we conducted this study in the VA health care system, where access to care is not limited by insurance status, thereby diminishing the effect of insurance coverage for medical care or prescription drug costs as a potential confounder in the analysis. Because hydrox-

ymethylglutaryl-CoA reductase inhibitors may be one of the most costly prescription medications, cost as a potential barrier is an important confounder in prior studies of racial disparities in such treatment.

Consistent with other health care settings, our findings indicated that rates of cholesterol monitoring were inappropriately low in the VA system between 1999 and 2001, with approximately one third of coronary heart disease patients not receiving any form of cholesterol screening, and 44% not receiving LDL screening. Although screening rates were low in the VA health care system, some studies have demonstrated that rates in non-VA settings are even lower, ranging from 44% to 50%.<sup>22,23</sup> These findings suggest that NCEP guidelines recommending lipid panels in all patients with coronary heart disease are not followed in clinical practice and may be an area in which future quality improvement efforts are warranted.

Although the reasons for low rates of screening have not been fully elucidated, prior work has suggested that cholesterol

management practices may be influenced by a variety of factors, including physician specialty, physician and patient age, insurance status, comorbid conditions, perception of cardiovascular risk, and knowledge and acceptance of NCEP guidelines.<sup>34</sup> These factors contribute to the widely documented underuse of cholesterol screening. Obviously, low screening rates are a significant barrier to identifying candidates for therapy and to initiating treatment when appropriate. Given that documentation of cholesterol levels is a significant predictor of treatment with lipid-lowering therapy,<sup>22</sup> the effect of suboptimal screening practices is heightened.

In contrast to earlier studies showing significant underuse of lipid-lowering therapy in patients with coronary heart disease,<sup>21,22,24</sup> nearly all patients in our cohort who were deemed ideal for treatment had received cholesterol-lowering medications. This finding suggests that when appropriate candidates are identified in the VA health care setting, lipid-lowering therapy is initiated in accordance with NCEP guidelines. One reason for the absence of disparities in our findings may relate to determinants of health care access, such as education and insurance status. Lower levels of education and lack of insurance have been shown to predict poorer rates of cholesterol screening.<sup>30</sup> Patients in our sample had similar education levels and received medical care within the VA system where barriers such as inadequate access to care and medication costs are minimized. Other potential reasons are the dissemination of practice guidelines, current quality monitoring, and centralized quality standards in the VA health care system that may promote high quality care.<sup>35–38</sup> Although patients were generally receiving treatment, only 40% of ideal patients receiving lipid-lowering medications achieved target LDL levels. Although low, this treatment success rate exceeds that seen in non-VA settings where success rates of 9–25% have been documented.<sup>21,22,25,26</sup>

Our results differ somewhat from those of prior studies that have documented disparities in other forms of care for coronary heart disease patients treated in the VA health care system.<sup>39–43</sup> The absence of consistent disparities in our findings, in contrast to those of studies that have documented racial

differences in invasive cardiac procedure use,<sup>39–44</sup> suggests that disparities may vary according to the type of care provided. Our findings may reflect that national guidelines regarding the management of hypercholesterolemia are more widely disseminated compared with guidelines for invasive cardiac procedure use or that quality improvement efforts within the VA health care system have been more successful in primary and preventive care.

Several limitations should be considered when interpreting our results. First, data were collected during the 3 months before study enrollment and during any inpatient admission that followed enrollment. Thus, cholesterol levels for patients who may have been screened outside of this defined period were not included in the analysis. However, we examined treatment of patients with coronary heart disease who were actively accessing the health care system for evaluation of ischemic symptoms. Therefore, we believe this setting is one in which a comprehensive risk factor assessment, including measurement of lipid levels, was warranted. Second, data used in this analysis were obtained for a study assessing racial differences in patient attitudes regarding use of invasive cardiac procedures. Therefore, patients who underwent cardiac procedures during the 6 months before study enrollment were not included in our cohort. Third, 98% of our study cohort was male, reflecting the predominantly male patient population receiving treatment within the VA system. Thus, our findings are not necessarily generalizable to women. Finally, because we examined patients with documented coronary heart disease, our findings cannot be generalized to patients who have coronary heart disease that has not yet been diagnosed.

In summary, in this assessment of cholesterol monitoring and treatment of African American and White patients with coronary heart disease in the VA health care system, we found that African American and White patients were equally likely to receive cholesterol monitoring, although rates of screening were low in both groups. When examining ideal candidates for therapy, African American and White patients were equally likely to receive treatment and to achieve tar-

get LDL levels. Quality improvement efforts directed at increasing rates of screening and achievement of guideline-recommended cholesterol levels may prove beneficial in ensuring that patients with coronary heart disease receive maximal benefit from lipid-lowering therapy. ■

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#### Contributors

L.D. Woodard originated the study, developed the data analysis plan, and led the writing of the article. N.R. Kressin obtained project funding, participated in data acquisition, and supervised the data analyses. L.A. Petersen participated in data acquisition, assisted with study design, and helped develop the data analysis plan. All authors helped to conceptualize ideas, analyze and interpret the findings, and review drafts of the article.

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#### Human Participant Protection

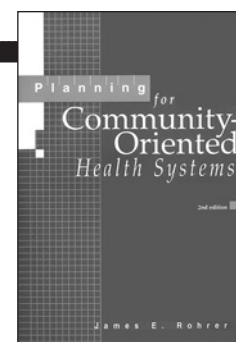
This study was approved by the institutional review board for Human Subject Research at Baylor College of Medicine and the Department of Veterans Affairs Research and Development Committee at all participating Veterans Affairs Medical Centers. All patients gave informed consent to participate in the study.

#### References

1. American Heart Association. 2002 Heart and Stroke Statistical Update. Dallas, Tex: American Heart Association; 2002.
2. Cooper R, Cutler J, Desvigne-Nickens P, et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular disease in the United States: findings of the National Conference on Cardiovascular Disease Prevention. *Circulation*. 2000;102:3137–3147.
3. Clark LT, Emerole O. Coronary heart disease in African Americans: primary and secondary prevention. *Cleve Clin J Med*. 1995;62:285–292.
4. Lewis CE, Raczynski JM, Oberman A, Cutter GR. Risk factors and the natural history of coronary heart disease in blacks. *Cardiovasc Clin*. 1991;21:29–45.
5. Giles WH, Kittner SJ, Hebel JR, Losoncy KG, Sherwin RW. Determinants of black-white differences in the risk of cerebral infarction. *Arch Intern Med*. 1995;155:1319–1324.
6. Kannel WB, Castelli WP, Gordon T, McNamara PM. Serum cholesterol, lipoproteins, and the risk of coronary heart disease: the Framingham Study. *Ann Intern Med*. 1971;74:1–12.
7. Neaton JD, Blackburn H, Jacobs D, et al. Serum cholesterol level and mortality findings for men screened in the Multiple Risk Factor Intervention Trial. *Arch Intern Med*. 1992;152:1490–1500.
8. Wong ND, Cupples LA, Ostfeld AM, Levy D, Kannel WB. Risk factors for long-term coronary prognosis after initial myocardial infarction: the Framingham Study. *Am J Epidemiol*. 1989;130:469–480.
9. Pekkanen J, Linn S, Heiss G, et al. Ten-year mortality from cardiovascular disease in relation to cholesterol level among men with and without preexisting cardiovascular disease. *N Engl J Med*. 1990;322:1700–1707.
10. Newman WP, Freedman DS, Voors AW, et al. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis: the Bogalusa Heart Study. *N Engl J Med*. 1986;314:138–144.
11. Aronow WS, Ahn C. Correlation of serum lipids with the presence or absence of coronary artery disease in 1,793 men and women aged >62 years. *Am J Cardiol*. 1994;73:702–703.
12. Neaton JD, Kuller LH, Wentworth D, Borhani NO. Total and cardiovascular mortality in relation to cigarette smoking, serum cholesterol concentration, and diastolic blood pressure among black and white males followed up for five years. *Am Heart J*. 1984;108:759–769.
13. Klag MJ, Ford DE, Mead LA, et al. Serum cholesterol in young men and subsequent cardiovascular disease. *N Engl J Med*. 1993;328:313–318.
14. Scandinavian Simvastatin Survival Study Group. Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). *Lancet*. 1994;344:1383–1389.



15. The Long-Term Intervention with Pravastatin in Ischaemic Disease (LIPID) Study Group. Prevention of cardiovascular events and death with pravastatin in patients with coronary heart disease and a broad range of initial cholesterol levels. *N Engl J Med.* 1998;339:1349–1357.
16. Sacks FM, Pfeffer MA, Moye LA, et al. The effect of pravastatin on coronary events after myocardial infarction in patients with average cholesterol levels. *N Engl J Med.* 1996;335:1001–1009.
17. The Post Coronary Artery Bypass Graft Trial Investigators. The effect of aggressive lowering of low-density lipoprotein cholesterol levels and low-dose anti-coagulation on obstructive changes in saphenous-vein coronary-artery bypass grafts. *N Engl J Med.* 1997;336:153–162.
18. Pitt B, Waters D, Brown WV, et al. Aggressive lipid-lowering therapy compared with angioplasty in stable coronary artery disease. *N Engl J Med.* 1999;341:70–76.
19. Gotto AM. Results of recent large cholesterol-lowering trials and implications for clinical management. *Am J Cardiol.* 1997;79:1663–1666.
20. Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *JAMA.* 1993;269:3015–3023.
21. McBride P, Schrott HG, Plane MB, Underbakke G, Brown R. Primary care practice adherence to National Cholesterol Education Program guidelines for patients with coronary heart disease. *Arch Intern Med.* 1998;158:1238–1244.
22. Sueta CA, Chowdhury M, Boccuzzi SJ, et al. Analysis of the degree of undertreatment of hyperlipidemia and congestive heart failure secondary to coronary artery disease. *Am J Cardiol.* 1999;83:1303–1307.
23. Frolkis JP, Zyzanski SJ, Schwartz JM, Suhan PS. Physician noncompliance with the 1993 National Cholesterol Education Program (NCEP-ATPII) guidelines. *Circulation.* 1998;98:851–855.
24. Majumdar SR, Gurwitz JH, Soumerai SB. Undertreatment of hyperlipidemia in the secondary prevention of coronary artery disease. *J Gen Intern Med.* 1999;14:711–717.
25. Schrott HG, Bittrner V, Vittinghoff E, Herrington DM, Hulley S. Adherence to National Cholesterol Education Program treatment goals in postmenopausal women with heart disease: the Heart and Estrogen/Progestin Replacement Study (HERS). *JAMA.* 1997;277:1281–1286.
26. Pearson TA, Laurora I, Chu H, Kafonek S. The Lipid Treatment Assessment Project (L-TAP): a multicenter survey to evaluate the percentages of dyslipidemic patients receiving lipid-lowering therapy and achieving low-density lipoprotein cholesterol goals. *Arch Intern Med.* 2000;160:459–467.
27. Sprafka JM, Burke GL, Folsom AR, Hahn LP. Hypercholesterolemia prevalence, awareness, and treatment in blacks and whites: the Minnesota Heart Survey. *Prev Med.* 1989;18:423–432.
28. Nieto FJ, Alonso J, Chambless LE, et al. Population awareness and control of hypertension and hypercholesterolemia: the Atherosclerosis Risk in Communities Study. *Arch Intern Med.* 1995;155:677–684.
29. Nelson K, Norris K, Mangione CM. Disparities in the diagnosis and pharmacologic treatment of high serum cholesterol by race and ethnicity: data from the Third National Health and Nutrition Examination Survey. *Arch Intern Med.* 2002;162:929–935.
30. Stewart SH, Silverstein MD. Racial and ethnic disparity in blood pressure and cholesterol measurement. *J Gen Intern Med.* 2002;17:405–411.
31. Lemaitre RN, Furberg CD, Newman AB, et al. Time trends in the use of cholesterol-lowering agents in older adults: the Cardiovascular Health Study. *Arch Intern Med.* 1998;158:1761–1768.
32. Oddone EZ, Petersen LA, Weinberger M, Freedman J, Kressin NR. Contribution of the Veterans Health Administration in understanding racial disparities in access and utilization of health care: a spirit of inquiry. *Med Care.* 2002;40(suppl 1):I3–I13.
33. Kressin NR, Clark JA, Whittle J, et al. Racial differences in health-related beliefs, attitudes, and experiences of VA cardiac patients: scale development and application. *Med Care.* 2002;40(suppl 1):I72–I85.
34. Stafford RS, Blumenthal D, Pasternak RC. Variations in cholesterol management practices of U.S. physicians. *J Am Coll Cardiol.* 1997;29:139–146.
35. Petersen LA, Normand SL, Leape LL, McNeil BJ. Comparison of the use of medications after acute myocardial infarction in the Veterans Health Administration and Medicare. *Circulation.* 2001;104:2898–2904.
36. Petersen LA, Normand SL, Daley J, McNeil BJ. Outcome of myocardial infarction in Veterans Health Administration patients as compared with Medicare patients. *N Engl J Med.* 2000;343:1934–1941.
37. Petersen LA, Normand SL, Druss BG, Rosenheck RA. Process of care and outcome after acute myocardial infarction for patients with mental illness in the VA healthcare system: are there disparities? *Health Serv Res.* 2003;38:1:41–63.
38. Demakis JG, McQueen L, Kizer KW, Feussner JR. Quality Enhancement Research Initiative (QUERI): a collaboration between research and clinical practice. *Med Care.* 2000;38(6 suppl 1):I17–I25.
39. Petersen LA, Wright SM, Peterson ED, Daley J. Impact of race on cardiac care and outcomes in veterans with acute myocardial infarction. *Med Care.* 2002;40(suppl 1):I86–I96.
40. Whittle J, Conigliaro J, Good CB, Lofgren RP. Racial differences in the use of invasive cardiovascular procedures in the Department of Veterans Affairs medical system. *N Engl J Med.* 1993;329:621–627.
41. Peterson ED, Wright SM, Daley J, Thibault GE. Racial variation in cardiac procedure use and survival following acute myocardial infarction in the Department of Veterans Affairs. *JAMA.* 1994;271:1175–1180.
42. Mirvis DM, Burns R, Gaschen L, Cloar FT, Graney M. Variation in utilization of cardiac procedures in the Department of Veterans Affairs health care system: effect of race. *J Am Coll Cardiol.* 1994;24:1297–1304.
43. Kressin NR, Petersen LA. Racial differences in the use of invasive cardiovascular procedures: review of the literature and prescription for future research. *Ann Intern Med.* 2001;135:352–366.
44. Conigliaro J, Whittle J, Good CB, et al. Understanding racial variation in the use of coronary revascularization procedures: the role of clinical factors. *Arch Intern Med.* 2000;160:1329–1335.



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# Experiences of Racism Among African American Parents and the Mental Health of Their Preschool-Aged Children

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Living in racialized societies such as the United States<sup>1,2</sup> can involve adverse health consequences for members of subordinate racial groups,<sup>3</sup> including negative effects of interpersonal racial discrimination and of characteristics of place of residence on both physical and mental health.<sup>2,4-9</sup> More than a dozen surveys of African Americans have shown an inverse association between self-reported experiences of interpersonal racial discrimination and mental or physical health status.<sup>7,10-15</sup>

The identification of pathways by which experiences of interpersonal racism lead to adverse health outcomes is still an exploratory area of research.<sup>13</sup> The measurement of racism is also in its infancy, and currently no comprehensive measures capture all aspects of experiences of interpersonal racial discrimination.<sup>14</sup> Although reporting discriminatory experiences is associated with greater levels of adverse outcomes, denying actual discrimination may result in equal or greater levels of adverse health effects as those associated with reporting discrimination.<sup>2,10,15</sup> Denial of racism (e.g., denying the existence of racism experience by oneself or by others of one's own racial group<sup>6,16</sup>) is a way of coping that, it has been suggested, leads to poor physical and mental health.<sup>6,15-17</sup> Evidence suggests that more than 90% of African Americans who report having no experiences of discrimination have nevertheless been discriminated against, if discrimination is measured with objective indicators.<sup>18</sup> Thus, research on experiences of discrimination should examine those who report racism experiences as well as those who report no racism experiences.<sup>15</sup>

Because the study of racism is new to the field of public health, previous research into questions of how parental experiences of racism might affect the well-being of African American children is nonexistent. One mechanism by which such parental experiences may affect the well-being of preschool-age African American children is through racial socializa-

**Objectives.** We examined the relationship between parents' experiences of racism and children's well-being and the influence of the residential neighborhood characteristics on this relationship.

**Methods.** African American families were recruited from Baltimore neighborhoods. Parental measures included racism experiences and coping. Neighborhood measures included demographic characteristics, social cohesion, and social climate. Children's mental health was assessed with the Child Behavior Checklist. Analysis was performed with multilevel modeling.

**Results.** Parents who denied experiences of racism also reported higher rates of behavior problems among their preschool-aged children. For families living in neighborhoods characterized by fear of victimization, parents who actively coped with racism experiences by confronting the person involved or taking some sort of action in response to racism reported lower rates of anxiety and depression for their preschool-aged children.

**Conclusions.** Experiences of and responses to racism among African American parents have important effects on the well-being of their young children. (*Am J Public Health*. 2004;94:2118-2124)

tion strategies,<sup>19-20</sup> defined as "specific messages and practices that provide information concerning the nature of race status."<sup>20(pp401-402)</sup> African American parents who emphasize the development of cultural pride in their preschoolers report lower levels of problem behaviors, especially anxiety or withdrawal, in these children.<sup>21</sup> Because racial socialization is how African Americans educate their children to perceive themselves as part of the larger society, the degree to which denial of racism reflects a specific perspective of African American identity might be expected to produce differences in racial socialization practices and, in turn, to differences in child mental health status.

One limitation of current racism research is that it has yet to integrate psychosocial mechanisms with neighborhood characteristics. Analytic techniques integrating mechanisms involving 2 levels have been applied to child behavioral outcomes.<sup>22-26</sup> Kalff et al.<sup>25</sup> reported that after parental socioeconomic status (SES) was taken into account, relative neighborhood poverty was associated with higher rates of child behavioral problems. A study of Chinese Americans found that interpersonal

and neighborhood racial discrimination were predictive of poor health status.<sup>27</sup> However, no studies have used a multilevel framework to integrate self-perceived and residential-area characteristics in the prediction of mental health outcomes among African Americans adults or children.<sup>7</sup>

We sought to explore the relation between children's mental health and neighborhood characteristics, parental experiences of interpersonal racism, parental coping behaviors, and parental racial socialization practices. We addressed the following questions: (1) What is the prevalence of reported racism experiences among African Americans living in an urban setting? (2) What is their typical response to these experiences? (3) Are differences in behavioral and emotional responses to acknowledged racism experiences among African American parents associated with differences in the socioemotional well-being of their preschool-aged children?

We hypothesized that parental self-reported experiences of and responses to racism would affect the child's socioemotional development through effects on the parents' racial socialization strategies. We also aimed to address an-

other research question: Do neighborhood conditions affect the prevalence of reported racism experiences, responses to racism, or racism's effect on the socioemotional well-being of children?

## METHOD

### Participants

Baltimore census tracts were stratified by average household wealth, derived from 1997 projected data (Claritas/National Planning Data Corporation, Ithaca, NY), and according to racial composition ( $\geq 80\%$  African American,  $\geq 80\%$  European American, racially mixed). Two census tracts were chosen from each stratum, and 1–3 block groups were chosen as study neighborhoods from each selected tract. We used census block groups to define neighborhoods, because block groups are more homogeneous than census tracts.<sup>28</sup> The number of block groups chosen was not constant, because the number of block groups varied per tract, and the racial composition of block groups was not always consistent with the composition of the tract. Block groups resembled Baltimore overall, except for racial composition. The average proportion of African Americans was higher among study block groups than in the city (85.7% vs 61.9%;  $t=6.76$ ,  $P<.01$ ). African American families with children 3–4 years old were recruited through door-to-door canvassing, targeted mailings, day-care centers, and Head Start programs. Participants had lived in their block group for at least 6 months. Refusals were minimal ( $<5\%$ ), and the number of participants per block group averaged 5.13 (range 1–16). Two home visits with 200 families were conducted, each lasting 2.5 hours on average. In each family, the primary caregiver of the target child (hereafter referred to as the “parent”) was interviewed.

### Measures

Data were collected at both the block group and the family level. Block group variables included SES, proportion African American, crime density, social cohesion, and negative social climate. Using all block groups in the city, we standardized each variable, and the component variables were averaged.

Socioeconomic status was assessed with a measure of neighborhood impoverishment<sup>29</sup>

(combined poverty, unemployment, and vacant housing rates) and the proportion of single-headed households with children younger than 5 years, as derived from 1997 projections of 1990 census data provided by Claritas/National Planning Data Corporation. African American density—the proportion of the population that was African American—was also based on these data. Crime density was defined as the number of crimes per square mile, as recorded by the police department.

Neighborhood social cohesion and negative social climate were assessed with the Neighborhood Environment for Children Rating Scales (NECRS)<sup>30</sup> and with a measure of psychological sense of community.<sup>31–32</sup> Three subscales of the NECRS were used as indicators of neighborhood social cohesion: willingness of adults in the neighborhood to intervene in acts of delinquency (stop delinquency), to intervene in acts of child misbehavior (stop misbehavior), and to assist children in need (assist). The internal reliability coefficients for these subscales are .90, .85, and .81, respectively. Three additional subscales of the NECRS were used as indicators of neighborhood negative social climate: perceived physical/social disorder, fear of retaliation, and fear of victimization. The internal reliability coefficients for these subscales are .92, .90, and .94, respectively.

Psychological sense of community was used as a measure of social cohesion. A 13-item scale assessed the respondent's perceived sense of membership, shared emotional connection, and degree of mutual influence in the neighborhood. The reliability and validity of this instrument is reported elsewhere.<sup>33–34</sup> In previously published research, we reported a factor analysis resulting in 2 factors: general psychological sense of community and social knowledge of one's neighbors.<sup>35</sup> Findings indicated that very low knowledge of one's neighbors, as represented by the lowest quartile, was an important predictor of child behavior problems. Therefore, we used this binary variable in the current analysis.

Family variables included family SES, parental denial of racism, racism coping strategies, and child behavior problems. SES measures included family poverty level and parental education. Family poverty was defined by family income adjusted for family size as a proportion of the federal poverty level. Pa-

rental education was categorical: less than high school, high school diploma or equivalent, and more than high school.

Parent racial socialization strategies were measured with the Parent's Experience of Racial Socialization (PERS) Scale (Stevenson H, PhD, Pennsylvania State University; unpublished material, 2001), adapted for use with parents of preschoolers. The scale consists of 40 items that asked parents how often they communicate particular messages to their children. We limited this analysis to the racial pride factor, because our previous work has demonstrated that racial pride alone is associated with child behavioral well-being after adjustment for general level of parent involvement.<sup>21</sup> The racial pride factor has an internal reliability coefficient of .76.

Assessment of racism involved 2 measures: denial of experience of racial discrimination and parental coping strategies in response to racism. These measures were assessed with 2 components of the Racism and Life Experiences Scales (RaLES).<sup>36</sup> We used the 4 RaLES-B questions that provided the most direct assessment of racism experiences to create an overall index of denial of racism. (The instruments used are available from the corresponding author.) The response set for each question was a 5-point Likert scale ranging from 1 (“not at all,” “never”) to 5 (“extremely,” “every time”). We counted the number of items to which the respondent answered “not at all,” resulting in an index ranging from 0 to 4, with higher scores reflecting greater denial of racism. We further divided this overall index into 2 indexes that differed in terms of referent. Denial of racism to self counted the number of “not at all” responses to the first 2 questions (lifetime racism experience and experiences during the past year). Denial of racism toward other African Americans counted the number of “not at all” responses to the third and fourth questions (effect of racism on others of one's same racial or ethnic group and on friends or family). Nine items from the RaLES were used to measure coping responses to racism experiences. Emotional coping included the first 6 items, and behavioral coping included the remaining 3 items. These scales have internal reliability coefficients of .68 and .53, respectively.

Child mental health status was assessed with the Child Behavior Checklist.<sup>37</sup> The Child Behavior Checklist yields scores for internalizing

**TABLE 1—Characteristics of Neighborhoods and Respondents: African American Primary Caregivers of Preschool-Aged Children, 1998–1999**

|  | No. (%)                   |
|--|---------------------------|
| <b>Neighborhoods (n = 39)</b>                                  |                           |
| Predominant race/ethnicity <sup>a</sup>                        |                           |
| African American   | 29 (74.4)                 |
| European American  | 1 (2.6)                   |
| Racially mixed (no predominant race/ethnicity)                 | 9 (23.1)                  |
| Average household wealth, quartile (\$ thousands)              |                           |
| Lowest ( $\leq 62$ )   | 11 (28.2)                 |
| Lower middle ( $> 62$ to 102)                                  | 9 (23.1)                  |
| Upper middle ( $> 102$ to 144)                                 | 10 (25.6)                 |
| Highest ( $> 144$ )  | 9 (23.1)                  |
| Number respondents, mean (SD); range                           | 5.13 (3.08); 1–16         |
| Neighborhood impoverishment z score, mean (SD); range          | 0.092 (0.754); -0.87–2.62 |
| Social cohesion  |                           |
| Stop delinquency   | 3.98 (0.58); 2.90–5.00    |
| Stop misbehavior   | 3.50 (0.71); 2.25–5.00    |
| Assist children in need  | 3.74 (0.71); 2.20–5.00    |
| Negative social climate  |                           |
| Physical/social disorder                                       | 2.52 (0.86); 1.00–4.15    |
| Fear of retaliation  | 2.71 (0.80); 1.00–4.14    |
| Fear of victimization  | 2.84 (0.74); 1.29–4.15    |
| <b>Respondents (n = 200)</b>                                   |                           |
| Neighborhood predominant race/ethnicity <sup>a</sup>           |                           |
| African American   | 174 (87.0)                |
| European American  | 1 (0.5)                   |
| Racially mixed (no predominant race/ethnicity)                 | 25 (12.5)                 |
| Neighborhood average household wealth, quartile (\$ thousands) |                           |
| Lowest ( $\leq 62$ )   | 65 (32.5)                 |
| Lower middle ( $> 62$ to 102)                                  | 45 (22.5)                 |
| Upper middle ( $> 102$ to 144)                                 | 52 (26.0)                 |
| Highest ( $> 144$ )  | 38 (19.0)                 |
| Respondent's relationship to child                             |                           |
| Mother   | 173 (86.5)                |
| Father   | 4 (2.0)                   |
| Grandparent  | 18 (9.0)                  |
| Other relative   | 5 (2.5)                   |
| Poverty status, % of federal poverty level                     |                           |
| $< 100$  | 89 (44.5)                 |
| 100–179  | 50 (25.0)                 |
| $\geq 180$   | 61 (30.5)                 |
| Educational attainment   |                           |
| $< \text{high school}$   | 47 (23.5)                 |
| High school or equivalent                                      | 82 (41.0)                 |
| $> \text{high school}$   | 71 (35.5)                 |
| Gender of target child   |                           |
| Male   | 93 (46.5)                 |
| Female   | 107 (53.5)                |

*Continued*

problems (e.g., anxiety, depression, withdrawal) and externalizing problems (e.g., aggression), as well as a score for total problem behaviors.

Raw scores were converted to *t* scores; higher scores indicate a greater number and more severe problem behaviors.

## RESULTS

### Sample Characteristics

Characteristics of respondents and block groups are shown in Table 1. Approximately three fourths of the block groups are African American. Of the 200 participants, 173 (86.5%) were mothers of the target child. The sample was economically heterogeneous, with 44.5% living below the federal poverty level and 30.5% with incomes above 180% of the federal poverty level. Most participants (87%) were living in an African American block group, consistent with the segregated nature of Baltimore. All participants had lived in their block group for 1 year or more, with 32% having resided in their block group for 10 years or longer.

### Prevalence of Reported Racism Experiences

The distribution of the racism denial indexes are also displayed in Table 1. A little more than half of participants agreed with all 4 questions regarding experiences of racism. Because of the skewed distribution of the index, 3 groups were created to represent both acknowledgment and denial of racism: agreed with all items (102, or 51%), denied 1–2 items (75, or 37.5%), and denied 3–4 items (13, or 6.5%). Furthermore, we observed differences in respondents' reporting of their own versus others' experiences of racism. Respondents were much less likely to report racism experienced personally than they were to acknowledge that racism had affected their friends and family or that it affected African Americans in general (this phenomenon is known as personal-group discrimination discrepancy).<sup>38</sup> Only 13 (6.5%) participants denied that racism affected their friends or family, compared with 35 (17.5%) participants who denied personally experiencing racism in the past year or during their lifetime.

We observed no differences in the prevalence of reported racism by family poverty status and only moderate differences by parent

TABLE 1—Continued

| Distribution of racism denial indexes (n = 200) <sup>a</sup>     |            |
|--|------------|
| Overall index of denial: no. of items denied                     |            |
| 0  | 102 (51.0) |
| 1  | 54 (27.0)  |
| 2  | 21 (10.5)  |
| 3  | 12 (6.0)   |
| 4  | 1 (0.5)    |
| Denial of racism to self: no. of items denied                    |            |
| 0  | 101 (50.5) |
| 1  | 57 (28.5)  |
| 2  | 35 (17.5)  |
| Denial of racism to other African Americans: no. of items denied |            |
| 0  | 166 (83.0) |
| 1  | 17 (8.5)   |
| 2  | 13 (6.5)   |

<sup>a</sup>At least 80% any single racial/ethnic group.<sup>b</sup>Balance of frequency distributions represents missing data.

education or employment status ( $\chi^2=8.97$  and  $8.01$ , respectively;  $P<.10$ ). The rate of denial was slightly higher among individuals with a high school education and among individuals who were currently unemployed but had been employed within the past 5 years. In both cases, this difference appeared to be a function of denying personal racism experiences, as op-

posed to denying racism experiences of friends or family or of African Americans in general.

### Responses to Racism Experiences

We examined variations in coping responses to racism among respondents who reported personal experiences of racism within the past year or sometime during their lifetimes

( $n=155$ ). The average score for the emotional response scale was  $2.59$  ( $SD=0.63$ ), and the average behavioral response scale score was  $2.99$  ( $SD=0.80$ ). No differences in responses to racism experiences were associated with poverty, educational, or employment status.

### Parental Racism Experiences and Child Behavior Problems

Average Child Behavior Checklist scores for each level of racism denial are shown in Table 2. Higher denial was associated with higher total problem behavior scores and higher externalizing problem scores. Post hoc comparisons indicated that this association was driven primarily by a higher rate of behavior problems among children whose parents denied 3–4 items than among children whose parents denied none of the items. The pattern of results differed according to the referent (self vs other African Americans). No differences in the rate of behavior problems in children were associated with the parents' report of personal racism experiences. By contrast, the rate of behavior problems (especially internalizing problems such as depression and anxiety) was significantly higher among children whose parents denied that racism affected those close to them or African Americans in general.

Child behavior problems were less common if parents reported taking an active behavioral response to racism. Among parents who reported racism experiences at some point in their lives, the correlation between total child problem behaviors and parental behavioral responses was marginally significant ( $r=-.14$ ,  $P=.08$ ). Specifically, there was a negative association between parental active behavioral responses to racism and child symptoms of depression and anxiety ( $r=-.22$ ,  $P<.01$ ).

### Racism and Child Behavior Problems in the Neighborhood Context

Multilevel linear regression analysis was used to examine joint influence of parental responses to racism, parental racial socialization practices, and block group conditions on child behavior problems. Among Child Behavior Checklist problem behaviors, only internalizing behavior showed significant between-block group variance. After we adjusted for between-block group differences in family

TABLE 2—Average Child Behavior Problem Scores, by Level of Parental Denial of Racism: African American Preschool-Aged Children, 1998–1999

|   | Total Problem Behaviors, Mean (SD) | F test  | Internalizing Problems, Mean (SD) | F test  | Externalizing Problems, Mean (SD) | F test |
|---|------------------------------------|---------|-----------------------------------|---------|-----------------------------------|--------|
| Overall index of denial: no. of items denied    |                                    |         |                                   |         |                                   |        |
| 0   | 42.82 (10.76)                      | 3.40**  | 45.21 (11.03)                     | 2.00    | 43.53 (8.62)                      | 4.10** |
| 1–2   | 45.84 (10.68)                      |         | 47.25 (9.15)                      |         | 46.58 (10.79)                     |        |
| 3–4   | 49.77 (9.64)                       |         | 50.54 (8.83)                      |         | 50.23 (8.52)                      |        |
| Denial of racism to self: no. of items denied   |                                    |         |                                   |         |                                   |        |
| 0   | 42.87 (10.99)                      | 3.23**  | 45.11 (11.06)                     | 2.46*   | 46.61 (8.89)                      | 3.41** |
| 1   | 46.93 (10.73)                      |         | 47.87 (8.89)                      |         | 47.56 (11.11)                     |        |
| 2   | 46.80 (10.43)                      |         | 49.03 (9.58)                      |         | 46.71 (9.05)                      |        |
| Denial of racism to others: no. of items denied |                                    |         |                                   |         |                                   |        |
| 0   | 43.66 (10.48)                      | 6.52*** | 45.64 (10.16)                     | 6.03*** | 44.51 (9.49)                      | 3.67** |
| 1   | 48.47 (12.73)                      |         | 50.76 (10.81)                     |         | 48.24 (10.67)                     |        |
| 2   | 53.54 (6.32)                       |         | 54.31 (6.81)                      |         | 50.92 (7.23)                      |        |

\* $P<.10$ , \*\* $P<.05$ , \*\*\* $P<.01$ . All  $P$  values are 2-tailed.



**TABLE Multilevel Linear Regression of Child Behavior Checklist Internalizing *t* Scores on Measures of Parental Responses to Racism and Neighborhood Characteristics: African American Preschool-Aged Children, 1998–1999**

|   | Individual-Level<br>Model b (SE) | Neighborhood-Level Models, b (SE) |              |              |              |              |              |
|---|----------------------------------|-----------------------------------|--------------|--------------|--------------|--------------|--------------|
|   |                                  | Model 1                           | Model 2      | Model 3      | Model 4      | Model 5      | Model 6      |
| Intercept   | 56.27 (3.08)                     | 56.29 (3.18)                      | 46.90 (6.86) | 59.36 (3.42) | 60.99 (4.02) | 48.43 (4.25) | 30.67 (8.49) |
| Individual-level variables                            |                                  |                                   |              |              |              |              |              |
| Does not deny that others experience racism           | -1.20 (1.58)                     | ...                               | ...          | ...          | ...          | ...          | ...          |
| Takes active behavioral response to racism            | -2.84 (1.02)                     | -3.33 (1.02)                      | -3.26 (1.01) | -3.27 (1.01) | -3.58 (1.01) | -3.03 (1.04) | 2.72 (2.63)  |
| Knows few neighbors                                   | -1.52 (1.97)                     | -.53 (2.14)                       | ...          | ...          | ...          | ...          | ...          |
| Neighborhood-level variables                          |                                  |                                   |              |              |              |              |              |
| Neighborhood impoverishment                           | ...                              | 1.65 (1.60)                       | ...          | ...          | ...          | ...          | ...          |
| Proportion African American                           | ...                              | ...                               | .10 (.06)    | ...          | ...          | ...          | ...          |
| Crime density   | ...                              | ...                               | ...          | -.001 (.001) | ...          | ...          | ...          |
| Social cohesion—to stop delinquency                   | ...                              | ...                               | ...          | ...          | -.88 (.77)   | ...          | ...          |
| Negative social climate—fear of victimization         | ...                              | ...                               | ...          | ...          | ...          | 2.29 (.78)   | 8.65 (2.88)  |
| Cross-level interactions                              |                                  |                                   |              |              |              |              |              |
| Knows few neighbors × neighborhood impoverishment     | ...                              | -5.16 (3.08)                      | ...          | ...          | ...          | ...          | ...          |
| Behavioral response to racism × fear of victimization | ...                              | ...                               | ...          | ...          | ...          | ...          | -2.16 (.96)  |
| $\chi^2$ for model improvement                        | ...                              | 17.49*                            | 2.57         | 2.55         | 15.58*       | 173.26*      | 178.15*      |

Note. All models were adjusted for family income.  $\chi^2$  for model improvement compared each neighborhood model with an individual-level model that includes only family income and behavioral response to racism.

\* $P < .001$ . All  $P$  values are 2-tailed.

SES, the intraclass correlation for internalizing behaviors was .17 ( $t = 2.12$ ,  $P < .05$ )—that is, approximately 17% of the variance in internalizing problems was between block groups, with the remaining 83% between children.

Because depression or anxiety was the only outcome showing significant between-block group variance, multilevel regression analyses were limited to this outcome. In addition to variables significant in preliminary analyses, we included an interaction between the psychological sense of community variable “knows few neighbors” (very low knowledge of neighbors) and neighborhood impoverishment, because our previous work had indicated that the interaction between these 2 variables was an important predictor of symptoms of depression and anxiety.<sup>35</sup> The results of the multilevel regression analysis are displayed in Table 3. In the first model, the individual-level variables (denial of racism to others, behavioral responses to racism, and “knows few neighbors”) were entered. Only behavioral response to racism was significant, with parents who took an active behavioral response to racism reporting fewer child problems with anxiety and depression.

Models 1–6 reflect the addition of each block group variable. In the first model, we re-

tained the “knows few neighbors” variable and added both neighborhood impoverishment and the cross-level interaction with impoverishment on the basis of previous research.<sup>35</sup> When behavioral response to racism was included, the interaction between the “knows few neighbors” variable and neighborhood impoverishment was no longer significant.

After control for individual variables, only fear of victimization was associated with more internalizing problems. In model 6, we tested the interaction between neighborhood victimization and parental behavioral response to racism; this interaction was significant. As can be seen in Figure 1, in block groups in which fear of victimization was low, parental behavioral responses to racism were unrelated to symptoms of depression or anxiety among children. However, in high-fear block groups, behavioral response by parents to racism appeared to protect children against anxiety and depression.

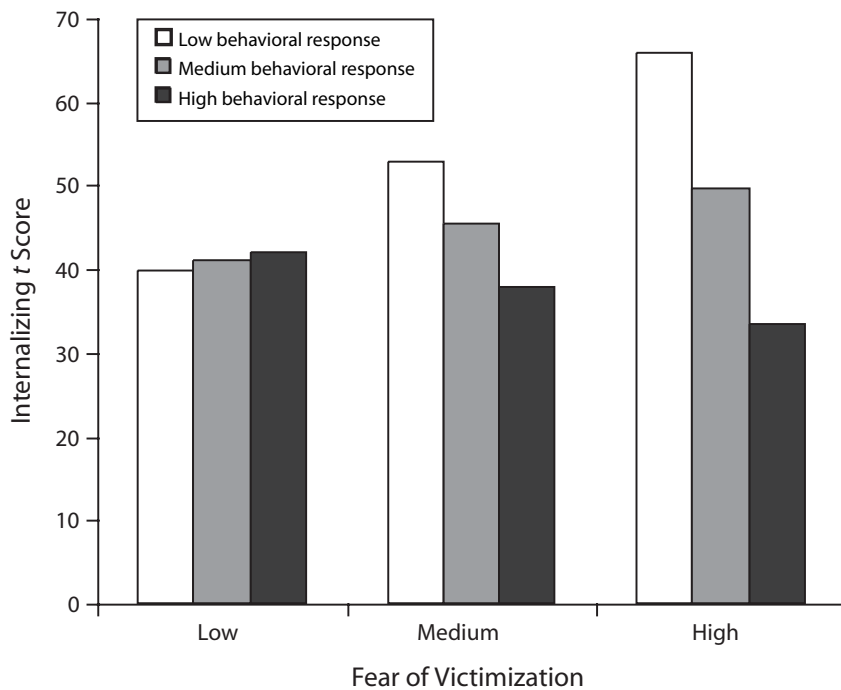
In the final model in Table 3 (model 6), we tested whether the effects of parental responses to racism are mediated by racial socialization strategies. Even when socialization of racial pride was included in the model, the interaction between behavioral responses to racism and neighborhood social climate was

still significant. Therefore, the hypothesis of mediation was not supported.

## DISCUSSION

In this study, we examined the ways in which racism experiences affect the mental health of preschool-aged children and how these effects might be moderated by neighborhood characteristics. A large proportion of parents reported that they and their friends and family had experienced racism. In fact, our study participants reported higher levels of racism among friends and family than among themselves. This personal–group discrimination discrepancy phenomenon has been observed in other studies, although detecting it may be affected by the explicitness of measures of discrimination used.<sup>38</sup> Those in our sample who reported experiencing racism were likely to take action during these incidents. Experiences of racism and responses to these experiences varied little by demographic characteristics of participants.

Some respondents reported not experiencing racial discrimination, a response that has been associated with poor outcomes. Although it is possible that respondents who denied ex-



**FIGURE 1—Influence of neighborhood levels of fear of victimization on parental behavioral response to racism and child internalizing behavior problems.**

perceiving racism never did encounter racism, this possibility does not seem a likely explanation for the majority of respondents.<sup>39</sup> In fact, parents who denied experiencing racism had the highest behavioral problems among their children. On the other hand, parents who reported actively coping with racism experiences by confronting the person involved or taking some sort of action in response to racism also reported fewer behavioral problems in their children. Our results support Krieger's hypothesis of the harmful effects of denial of racism on health, first tested with hypertension,<sup>2,10,39</sup> by testing the hypothesis with child mental health outcomes.

Our hypothesis that the effect of parental experiences of and coping with racism on child mental health status would be mediated by parental racial socialization strategies was not supported. Future research should include a wide variety of parenting factors to elucidate the pathways through which experiences of racism affect children's mental health.

We examined several neighborhood characteristics in relation to child mental health: neighborhood impoverishment, proportion Af-

rican American, crime density, social cohesion, and negative social climate. Only negative social climate, especially fear of victimization, was associated with child symptoms of depression and anxiety. We examined psychological sense of community as reported by respondents and found that this factor was not associated with child mental health after other individual factors were controlled. This result is in contrast to our previous findings,<sup>35</sup> specifically that lack of knowledge of one's neighbors was associated with better mental health outcomes for children in low-income neighborhoods. This difference in findings may have resulted from the fact that our previous study had not considered joint effects of behavioral responses to racism and psychological sense of community.

We found that behavioral responses to racial discrimination were protective only in neighborhoods characterized by high levels of fear of victimization. We can only speculate about possible psychosocial mechanisms behind this association. Some studies of African American women living in urban areas have found evidence of joint effects of interpersonal discrimination and neighborhood stressors on

poor health.<sup>40–42</sup> Therefore, active coping in the face of interpersonal discrimination might be particularly protective for parents exposed to stressful neighborhood environments, such as those identified by our fear of victimization indicator.

Limitations in our analyses should be noted. First, given the cross-sectional design of the study, the timing of racism experiences relative to the occurrence of adverse child mental health could not be established. Future research should collect information on the timing of the onset of children's mental health problems, as well as on experiences of racial discrimination in specific narrow periods (e.g., while the child was an infant) corresponding to key developmental periods of child development.

More comprehensive measures are needed in this area. Although we included a variety of neighborhood characteristics beyond economic indicators,<sup>43</sup> our measures did not include indicators of discrimination at the neighborhood level, such as segregation and redlining, which have been associated with self-reported health.<sup>27</sup> Such indicators may have added information regarding the types of social environments that have an impact on individual-level discrimination or child mental health.

The strengths of our study, however, are numerous. We studied a sample of African American families of diverse socioeconomic backgrounds and neighborhoods. We included a wide selection of information on both individual and neighborhood correlates of child mental health, minimizing the possibility that our results would be affected by confounding of unmeasured factors. In addition, the multilevel analysis design allowed us to demonstrate the contextual nature of the health effects of experiencing racism: parental responses to racism experiences are important for children's mental health, but neighborhood social environment matters as well.

This study contributes to our understanding of how we might improve the mental health of preschool-aged children.<sup>44</sup> Acknowledging racism is related to better child mental health outcomes, as is acting on experiences of racial discrimination. Furthermore, the fact that this association was dependent on characteristics of the neighborhoods in which families lived underscores the importance of incorporating

characteristics of the community into all studies of child mental health. ■

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### Contributors

M.O. Caughy and P.J. O'Campo designed and implemented the data collection for the project. They and C. Muntaner conceptualized the study aims. M.O. Caughy conducted all analyses, and all authors contributed to writing.

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### Human Participant Protection

The project protocol was reviewed and approved by the institutional review boards at both the University of Texas Health Sciences Center, Houston, and the Johns Hopkins Bloomberg School of Public Health.

### References

- Bonilla-Silva E. Rethinking racism. *Am Soc Rev*. 1996;62:465-480.
- Krieger N. Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int J Health Serv*. 1999;29:295-352.
- Krieger N. Discrimination and health. In Berkman L, Kawachi I, eds. *Social Epidemiology*. Oxford, England: Oxford University Press; 2000:36-75.
- Williams DR. Race and health. *Ann Epidemiol*. 1997;7:322-333.
- Muntaner C, Nieto J, O'Campo P. The bell curve: on race, social class and epidemiologic research. *Am J Epidemiol*. 1996;144:531-536.
- Jones JM. *Prejudice and Racism*. New York: McGraw-Hill; 1997.
- Williams DR, Williams-Morris R. Racism and mental health. *Ethn Health*. 2000;5:243-268.
- Acevedo-Garcia D. Zip code-level risk factors for tuberculosis: neighborhood environment and residential segregation in New Jersey, 1985-1992. *Am J Public Health*. 2001;91:734-741.
- Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep*. 2001;116:404-416.
- Krieger N, Sidney S. Racial discrimination and blood pressure: the CARDIA Study of young black and white adults. *Am J Public Health*. 1996;86:1370-1378.
- Williams D, Yu Y, Jackson J, Anderson N. Racial differences in physical and mental health: socioeconomic status, stress, and discrimination. *J Health Psychol*. 1997;2:335-351.
- Dressler W. Lifestyle, stress, and blood pressure in a southern black community. *Psychosom Med*. 1990;52:182-198.
- Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. *Am J Public Health*. 2003;93:200-209.
- Meyer IH. Prejudice as stress: conceptual and measurement problems. *Am J Public Health*. 2003;93:262-265.
- Harrell JP, Hall S, Taliaferro J. Physiological responses to racism and discrimination: an assessment of the evidence. *Am J Public Health*. 2003;93:243-248.
- Cose E. *The Rage of a Privileged Class*. New York, NY: HarperCollins; 1993.
- Williams DR, Neighbors H. Racism, discrimination and hypertension: evidence and needed research. *Ethn Dis*. 2001;11:800-816.
- Darity WA. Employment discrimination, segregation, and health. Paper presented at: "Racial/Ethnic Bias and Health," Conference of the National Institutes of Health; April 18-19, 2002; Vienna, Va.
- Garcia Coll C, Lamberty G, Jenkins R, et al. An integrative model for the study of developmental competencies in minority children. *Child Dev*. 1996;67:1891-1914.
- Thornton MC, Chatters LM, Taylor RJ, Allen WR. Sociodemographic and environmental correlates of racial socialization by black parents. *Child Dev*. 1990;61:401-409.
- Caughy MO, O'Campo PJ, Randolph SM, Nickerson K. The influence of racial socialization practices on the cognitive and behavioral competence of African American preschoolers. *Child Dev*. 2002;73:1611-1625.
- Leventhal T, Brooks-Gunn J. The neighborhoods they live in: the effects of neighborhood residence on child and adolescent outcomes. *Psychol Bull*. 2000;126:309-337.
- Duncan GJ, Raudenbush SW. Assessing the effects of context in studies of child and youth development. *Educational Psychology*. 1999;34:29-41.
- Kohen DE, Brooks-Gunn J, Leventhal T, Hertzmann C. Neighborhood income and physical and social disorder in Canada: associations with young children's competencies. *Child Dev*. 2002;73:1844-1860.
- Kalff A, Kroes M, Vles J, et al. Neighborhood-level and individual-level SES effects on child problem behavior: a multilevel analysis. *J Epidemiol Community Health*. 2001;55:246-250.
- Sampson RJ. Family management and child development: insights from social disorganization theory. In: McCord J, ed. *Facts, Frameworks, and Forecasts. Advances in Criminological Theory*. New Brunswick, NJ: Transaction Publishers; 1992:63-93.
- Gee GC. A multilevel analysis of the relationship between institutional and individual racial discrimination and health status. *Am J Public Health*. 2002;92:615-623.
- Krieger N. Women and social class: a methodological study comparing individual, household, and census measures as predictors of black/white differences in reproductive history. *J Epidemiol Community Health*. 1991;45:35-42.
- Korbin J, Coulton CJ. Understanding the neighborhood context for children and families: combining epidemiological and ethnographic approaches. In: Brooks-Gunn J, Duncan GJ, Aber JL, eds. *Neighborhood Poverty: Policy Implications in Studying Neighborhoods*. Vol 2. New York, NY: Russell Sage Foundation; 1997:65-79.
- Coulton CJ, Korbin JE, Su M. Measuring neighborhood context for young children in an urban area. *Am J Community Psychol*. 1996;24:5-33.
- Linney JA, Wandersman A. *Prevention Plus III—Assessing Alcohol and Other Drug Prevention Programs at the School and Community Level: A Four-Step Guide to Useful Program Assessment*. Washington, DC: US Dept of Health and Human Services; 1991. Publication ADM 91-1871.
- McMillan D, Chavis D. Sense of community: a definition and theory. *Am J Community Psychol*. 1986;14:6-23.
- Chavis DM, Pretty GMH. Sense of community: advances in measurement and application. *J Community Psychol*. 1999;27:635-642.
- Chipuer HM, Pretty GMH. A review of the sense of community index: current uses, factor structure, reliability and further development. *J Community Psychol*. 1999;27:643-658.
- Caughy MO, O'Campo PJ, Muntaner C. When being alone might be better: neighborhood poverty, social capital, and child behavior. *Soc Sci Med*. 2003;57:227-237.
- Harrell SP, Merchant MA, Young SA. Psychometric properties of the Racism and Life Experiences Scales (RaLES). Paper presented at: the American Psychological Association; April 15-19, 1997; Chicago, IL.
- Achenbach TM. *Manual for the Child Behavior Checklist*. Burlington, VT: University of Vermont Department of Psychiatry; 1991.
- Ruggiero KM, Mitchell JP, Krieger N, Marx DM, Lorenzo ML. Now you see it, now you don't: explicit versus implicit measures of the personal/group discrimination discrepancy. *Psychol Sci*. 2000;11:511-514.
- Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med*. 1990;30:1273-1281.
- Schulz A, Israel B, Williams D, et al. Social inequalities, stressors and self-reported health status among African American and white women in the Detroit metropolitan area. *Soc Sci Med*. 2000;51:1639-1653.
- Schultz A, Israel B, Williams D, et al. Unfair treatment, neighborhood effects, and mental health in the Detroit metropolitan area. *J Health Soc Behav*. 2000;41:314-332.
- Israel BA, Farquhar SA, Schulz AJ, James SA, Parker EA. The relationship between social support, stress, and health among women on Detroit's East Side. *Health Educ Behav*. 2002;29:342-360.
- Sampson RJ. Linking the micro and macrolevel dimensions of community social organization. *Social Forces*. 1991;70:43-64.
- Van Ausdale D, Feagin J. *The First R: How Children Learn Race and Racism*. Lanham, Md: Rowman & Littlefield; 2001.

# Self-Reported Experiences of Racial Discrimination and Black–White Differences in Preterm and Low-Birthweight Deliveries: The CARDIA Study

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Despite decades of public health and medical initiatives designed to improve birth outcomes, risks of preterm (less than 37 weeks gestation) and low-birthweight (LBW; less than 2500 g) deliveries remain substantially higher for Black than for White women in the United States.<sup>1</sup> In 2001, national preterm delivery rates among Black and White women were 17.5 and 10.8 per 100 live births, respectively; in the case of LBW, the corresponding rates were 13.1 and 6.8.<sup>2</sup> Extant research indicates that this Black–White gap is only partially explained by major identified determinants of these adverse birth outcomes such as tobacco, alcohol, and drug use; use of prenatal care; genetics; and socioeconomic position.<sup>3–6</sup>

Specifically, studies have shown that although economic deprivation contributes to the higher risk of LBW among Black than White infants, it does not fully account for this risk, given that Black–White disparities remain even within socioeconomic strata.<sup>4</sup> Casting doubt that alleged genetic differences could explain the disparity, moreover, is research demonstrating that recent immigrants, both Black and White, tend to give birth to higher birthweight babies than women of the same ancestry born and raised in the United States, regardless of socioeconomic position.<sup>7</sup>

The persistence of the Black–White gap, even after taking into account socioeconomic position and other known risk factors, has led to formulation of a new hypothesis: that racial discrimination, as a psychosocial stressor, may increase the risk of preterm and LBW deliveries.<sup>6,8–10</sup> Supporting inquiry on the impact of racial discrimination on birth outcomes are the results of 2 recent studies. One of these studies showed that self-reported experiences of racial discrimination were associated with extremely LBW deliveries in a sample of low-income Black women,<sup>11</sup> and the other produced evidence of an increased risk of pre-

**Objectives.** We examined the effects of self-reported experiences of racial discrimination on Black–White differences in preterm (less than 37 weeks gestation) and low-birthweight (less than 2500 g) deliveries.

**Methods.** Using logistic regression models, we analyzed data on 352 births among women enrolled in the Coronary Artery Risk Development in Young Adults Study.

**Results.** Among Black women, 50% of those with preterm deliveries and 61% of those with low-birthweight infants reported having experienced racial discrimination in at least 3 situations; among White women, the corresponding percentages were 5% and 0%. The unadjusted odds ratio for preterm delivery among Black versus White women was 2.54 (95% confidence interval [CI] = 1.33, 4.85), but this value decreased to 1.88 (95% CI = 0.85, 4.12) after adjustment for experiences of racial discrimination and to 1.11 (95% CI = 0.51, 2.41) after additional adjustment for alcohol and tobacco use, depression, education, and income. The corresponding odds ratios for low birthweight were 4.24 (95% CI = 1.31, 13.67), 2.11 (95% CI = 0.75, 5.93), and 2.43 (95% CI = 0.79, 7.42).

**Conclusions.** Self-reported experiences of racial discrimination were associated with preterm and low-birthweight deliveries, and such experiences may contribute to Black–White disparities in perinatal outcomes. (*Am J Public Health.* 2004;94:2125–2131)

term deliveries among women who reported high levels of racial discrimination.<sup>12</sup> Lending additional credence to this hypothesis are 2 other areas of research, one linking maternal experiences of other types of social trauma, such as violence, to risk of poor birth outcomes<sup>10,13–17</sup> and the other documenting associations between self-reported experiences of racial discrimination and other somatic health outcomes, particularly hypertension.<sup>18–21</sup>

Accordingly, in this study we addressed the following questions: Do self-reported lifetime experiences of racial discrimination contribute to Black–White differences in preterm and LBW deliveries? and if so, are such associations independent of or mediated by other physical, psychosocial, or behavioral factors hypothesized to affect the risk of these outcomes? To explore these questions, we used data from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, a longitudinal, multisite, epidemiological cohort investigation designed to examine the development of cardiovascular risk factors in a

large sample of young Black and White women and men.

## METHODS

### Sample

CARDIA's setting, sample, and data collection methods have been described elsewhere and are summarized here.<sup>22</sup> Briefly, CARDIA began in 1985 as a prospective cohort study designed to investigate factors that influence the development of coronary artery disease during young adulthood. Participants were recruited from 4 geographically diverse metropolitan areas: Birmingham, Ala; Chicago, Ill; Oakland, Calif; and Minneapolis, Minn. A stratified random sampling procedure was employed with the goal of achieving a sample that included equal numbers of Blacks and Whites, women and men, individuals aged 18 to 25 and 25 to 30 years, and individuals with less than a high school education and more than a high school education.

A total of 5115 individuals participated in the initial examination, including 1480



Black women and 1307 White women. Of the surviving baseline cohort, 91% returned at year 2 (1987–1988), 86% returned at year 5 (1990–1991), 81% returned at year 7 (1992–1993), and 79% returned at year 10 (1995–1996). Given that questions pertaining to racial discrimination were first asked in the year 7 examination, this study included only the 367 births to women who attended examinations during years 7 and 10 and gave birth after year 7 (i.e., between 1992 and 1995).

From these 367 deliveries, we excluded deliveries that resulted in multiple infants ( $n=12$ ) or stillbirths ( $n=1$ ) and those in which the gestational age was less than 20 weeks ( $n=2$ ); as a result of these exclusions, our total sample size was 352. Twenty-nine women had given birth to a live infant more than once during the interval between year 7 and year 10, and all such births were included. Self-reported data on birth outcomes were collected at year 10. Predictors of LBW and potential modifiers were measured at year 7 unless otherwise noted.

### Birth Outcomes

Participants reported their baby's birthweight in pounds and ounces; these data were converted to grams. LBW was defined as less than 2500 g. Participants reported their baby's gestational age at birth in weeks. Preterm deliveries were defined as those involving a gestational age below 37 weeks. Given the possibility of recall error or bias in reporting of birthweight and gestational age, we included a covariate for elapsed time between the birth and the year 10 (1995) interview.

### Self-Reported Experiences of Racial Discrimination

During the year 7 (1992) examination, participants completed a discrimination questionnaire<sup>18,19</sup> asking them whether they had "ever experienced discrimination, been prevented from doing something or been hassled or made to feel inferior . . . because of their race or color" in any of 7 situations: "at school, getting a job, at work, getting housing, getting medical care, on the street or in a public setting, and from the police or in the courts." Responses were combined to form a 3-level categorical variable pertaining to reports of racial discrimination in 0, 1 or 2, or 3 or more of the specified situations.<sup>18,19</sup>

### Potential Modifiers and Covariates

*Response to unfair treatment.* On the discrimination questionnaire, participants were asked "If you feel you have been treated unfairly, do you usually: accept it as a fact of life or try to do something about it?"

*Depression.* The 20-item Center for Epidemiological Studies Depression Scale<sup>23</sup> was administered during the year 5 (1990) examination. Scores on this scale can range from 0 to 60, with higher scores indicating more depression symptoms. Although this variable was measured 2 years before the year 7 examination, it represented the most recently available measurement and, therefore, the best approximation of depressive symptoms we could obtain.

*Substance use.* Although measures focusing on tobacco, alcohol, and drug use during pregnancy were not available, participants had been assessed during the year 7 (1992) examination in regard to previous use. Smoking status was categorized as never, former, or current. Alcohol use was classified as use in the past year or no use in the past year. History of drug use was included in preliminary analyses but dropped owing to its lack of association with the birth outcomes under investigation.

*Maternal anthropometric and health factors.* Participants reported occurrences of toxemia and gestational hypertension for each of their pregnancies. Self-reported gestational weight gain was recorded in pounds and converted to kilograms. To adjust for birthweight, we subtracted infant birthweight from gestational weight gain and included the net result in our models (net gestational weight gain). Data on prepregnancy body mass index (BMI; from the year 7 examination) were limited because, in the CARDIA protocol, pregnant women are not weighed; as a result, 16% of the participants were missing these data. Analyses of the subset of participants for which these data were available indicated that prepregnancy BMI was not significantly associated with the birth outcomes assessed here; therefore, we did not include this variable in our analyses.

*Sociodemographic characteristics.* Marital status, age, self-reported race/ethnicity, and 2 measures of socioeconomic position (income and education) were included as covariates. All participants classified themselves as White, non-Hispanic or Black, non-Hispanic. Categorical data indicated that annual family

incomes ranged from less than \$5000 to more than \$75 000; however, because of the relatively small percentage of low-income women in our sample, we categorized income levels as less than \$25 000, \$25 000 to \$49 999, and \$50 000 or more. Likewise, because only 5 women had less than a high school education, we categorized education levels (i.e., highest level of education completed) as less than 4 years of college and 4 or more years of college or above.

### Analyses

In preliminary analyses, we ascertained the univariate distribution of each variable among Black and White women, as well as the distribution after stratification according to preterm and LBW deliveries. On the basis of these preliminary analyses, we conducted logistic regression analyses examining associations between the outcomes of interest and variables significant at the descriptive level. The first model, designed to quantify the magnitude of the Black–White gap in preterm and LBW deliveries in the CARDIA population, included only race/ethnicity. Subsequent models included self-reported experiences of racial discrimination along with the specified potential modifiers and covariates. Finally, we included gestational age in the LBW model in an effort to determine whether the effects of racial discrimination on LBW were mediated by gestational age.

Because several women ( $n=29$ ) gave birth to more than one infant between year 7 and year 10, we used the Huber–White sandwich estimator of variance<sup>24,25</sup> in our logistic regression models to account for violation of independent observations. Only one of these women delivered an LBW infant. All models were run on a sample that included first births only, but the results were not appreciably different. The sample included in the preterm models was made up of the 328 deliveries for which we had complete data on all covariates; 49 of these deliveries were preterm. The LBW model included the 320 deliveries for which we had complete data; 15 of these were LBW deliveries.

Because the sample size was small, we expected wide confidence intervals (CIs). Thus, we present results from Hosmer–Lemeshow goodness of fit tests<sup>26</sup> in which the data were reclassified into 8 groups of nearly equal size

**TABLE 1—Univariate and Bivariate Distributions of Study Variables Among Black and White Women in the CARDIA Study, 1992–1995**

|   | Black Women     |                  |              | White Women     |                  |             |
|---|-----------------|------------------|--------------|-----------------|------------------|-------------|
|   | Total (n = 152) | Preterm (n = 32) | LBW (n = 14) | Total (n = 200) | Preterm (n = 20) | LBW (n = 5) |
| Preterm delivery, %                       | 21.1            | 100.0            | 84.6         | 10.0            | 100.0            | 60.0        |
| LBW, %                                    | 9.0             | 39.3             | 100.0        | 2.5             | 15.0             | 100.0       |
| Mean age, y, at year 10 examination (SD)  | 33.1 (3.3)      | 33.4 (3.2)       | 34.1 (2.6)   | 34.8 (3.2)      | 36.0 (3.0)       | 35.6 (2.7)  |
| Education, %                              |                 |                  |              |                 |                  |             |
| Less than college                         | 75.3            | 81.3             | 76.9         | 29.4            | 40.0             | 25.0        |
| College or more                           | 24.7            | 18.8             | 23.1         | 70.6            | 60.0             | 75.0        |
| Income, \$, %                             |                 |                  |              |                 |                  |             |
| <24 999                                   | 44.2            | 48.4             | 41.7         | 12.2            | 15.8             | 0.0         |
| 25 000–49 999                             | 37.4            | 35.5             | 33.3         | 31.6            | 42.1             | 50.0        |
| ≥50 000                                   | 18.4            | 16.1             | 25.0         | 56.1            | 42.1             | 50.0        |
| Married, %                                | 58.3            | 50.0             | 38.5         | 81.9            | 75.0             | 100.0       |
| Mean pregnancy weight gain, kg (SD)       | 13.3 (6.6)      | 12.3 (7.0)       | 10.4 (7.2)   | 15.0 (4.8)      | 13.3 (5.1)       | 13.4 (3.6)  |
| Mean net weight gain, kg (SD)             | 10.1 (6.5)      | 10.0 (6.9)       | 8.4 (7.2)    | 11.5 (4.7)      | 10.4 (5.2)       | 11.5 (3.7)  |
| Racial discrimination experiences, %      |                 |                  |              |                 |                  |             |
| ≥3  | 41.9            | 50.0             | 61.5         | 5.0             | 5.0              | 0.0         |
| 1–2                                       | 33.1            | 37.5             | 30.8         | 23.5            | 35.0             | 40.0        |
| 0   | 25.0            | 12.5             | 7.7          | 71.5            | 60.0             | 60.0        |
| Does something about unfair treatment, %  | 79.1            | 78.1             | 84.6         | 86.5            | 100.0            | 80.0        |
| Mean depressive symptomatology score (SD) | 13.0 (8.5)      | 16.9 (11.2)      | 15.0 (8.3)   | 9.9 (7.4)       | 9.8 (10.3)       | 9.3 (8.2)   |
| Prepregnancy smoking status, %            |                 |                  |              |                 |                  |             |
| Never smoked                              | 68.4            | 56.3             | 38.5         | 57.0            | 35.0             | 40.0        |
| Former smoker                             | 7.9             | 9.4              | 15.4         | 27.5            | 35.0             | 40.0        |
| Current smoker                            | 23.7            | 34.4             | 46.2         | 15.5            | 30.0             | 20.0        |
| Prepregnancy alcohol consumption, %       | 67.6            | 50.0             | 61.5         | 90.0            | 85.0             | 100.0       |
| Toxemia, %                                | 15.3            | 19.4             | 23.1         | 6.0             | 5.0              | 0.0         |
| Gestational high blood pressure, %        | 8.6             | 9.7              | 23.1         | 2.0             | 0.0              | 0.0         |
| Parity, mean (SD)                         | 2.6 (1.5)       | 2.4 (1.2)        | 2.6 (1.3)    | 2.0 (1.0)       | 2.2 (1.3)        | 2.6 (1.5)   |
| Mean gestational age, wk (SD)             | 38.7 (3.0)      | 34.2 (2.3)       | 34.7 (3.3)   | 39.3 (1.9)      | 35.3 (1.7)       | 35.6 (4.2)  |
| Mean birthweight, kg (SD)                 | 3.3 (0.6)       | 2.7 (0.7)        | 2.1 (0.5)    | 3.5 (0.5)       | 2.9 (0.7)        | 1.9 (0.5)   |

Note. LBW = low birthweight. Significant Black–White differences ( $P < .05$ ; 2-tailed  $t$  test or  $\chi^2$  test) were found for all variables other than response to unfair treatment. Numbers of participants missing data varied according to characteristic and were small (between 1% and 5% of the cohort) except in the case of pregnancy weight gain, in which 10% of the cohort was missing data.

via ordering in terms of predicted probabilities. The Hosmer–Lemeshow statistic has an approximate  $\chi^2$  distribution, and a nonsignificant  $P$  value indicates good model fit. We ran several tests to assess multicollinearity (e.g., tolerance and  $R^2$  analyses), and all values were within acceptable limits.

## RESULTS

As can be seen in Table 1, Black and White women differed significantly in regard to all characteristics other than response to unfair treatment. In comparison with White women, Black women had substantially

higher rates of preterm LBW deliveries, reported substantially more racial discrimination, had fewer socioeconomic resources (i.e., they had lower annual family incomes and less likely to have completed college), and were more likely to be unmarried, to report higher levels of depressive symptoms, to be nondrinkers, and to be current smokers. Also, they were more likely to have high rates of toxemia and gestational hypertension, to have had more births, and to show lower net gestational weight gain.

Distributions of covariates among the Black and White women with LBW or preterm deliveries were compared separately with distri-

butions among women without these conditions and were found to differ only for drinking and depressive symptomatology (Table 1). Accordingly, we included these variables in our analytic model, along with education, income, smoking status, and net weight gain, given the established associations of these variables with outcomes previously reported in the literature. Variables pertaining to self-reported responses to unfair treatment, maternal health factors, elapsed time between birth and examination, and age did not differ significantly according to birth outcome among either Black or White women, and thus they were not included in the analytic model.

**TABLE 2—Logistic Regression Analysis of Preterm Deliveries Among 328 Black and White Women in the CARDIA Study, 1992–1995**

|  | Odds Ratio (95% Confidence Interval) |                   |                   |                   |
|--|--------------------------------------|-------------------|-------------------|-------------------|
|  | Model 1                              | Model 2           | Model 3           | Model 4           |
| Race/ethnicity: Black vs White                     | 2.54 (1.33, 4.85)                    | 1.71 (0.84, 3.48) | 1.88 (0.85, 4.12) | 1.11 (0.51, 2.41) |
| Self-reported racial discrimination                |                                      |                   |                   |                   |
| 1 or 2 vs 0 experiences                            |                                      | 1.97 (0.89, 4.38) |                   | 2.05 (0.93, 4.50) |
| ≥ 3 vs 0 experiences                               |                                      | 2.42 (1.03, 5.69) |                   | 3.05 (1.29, 7.24) |
| Smoking status                                     |                                      |                   |                   |                   |
| Former vs never smoker                             |                                      |                   | 2.22 (0.89, 5.53) | 2.00 (0.79, 5.05) |
| Current vs never smoker                            |                                      |                   | 2.59 (1.16, 5.82) | 2.51 (1.13, 5.58) |
| Alcohol use: current vs not current                |                                      |                   | 0.38 (0.18, 0.79) | 0.30 (0.14, 0.66) |
| Depressive symptomatology: increase per unit score |                                      |                   | 1.03 (0.99, 1.07) | 1.02 (0.98, 1.06) |
| Education: less than college vs college or more    |                                      |                   | 0.83 (0.34, 2.04) | 0.87 (0.19, 1.33) |
| Income, \$   |                                      |                   |                   |                   |
| 25 000–49 999 vs <25 000                           |                                      |                   | 1.09 (0.50, 2.38) | 1.08 (0.49, 2.38) |
| ≥50 000 vs <25 000                                 |                                      |                   | 0.90 (0.32, 2.54) | 0.97 (0.36, 2.59) |

Note. The Hosmer–Lemeshow goodness of fit test statistic was 8.22 ( $P = .22$ ).

### Discrimination and Preterm Delivery

Overall, Black women were 2.5 times as likely to have a preterm delivery as White women (Table 2, model 1). Adding racial discrimination alone (model 2) and the other co-

variates alone (model 3) to the model each reduced the race/ethnicity odds ratio (OR). In the full model (model 4), racial discrimination and the other covariates substantially reduced the race/ethnicity odds ratio from 2.54 to

1.11. Those reporting racial discrimination in 3 or more situations were at 3.1 times the risk of preterm delivery. Depressive symptomatology was not significantly associated with risk of preterm delivery, nor did it mediate the relationship between discrimination and preterm delivery. Smoking and alcohol consumption were associated with preterm delivery but, again, did not appear to mediate the relationship. The Hosmer–Lemeshow goodness of fit test statistic was not significant, indicating a good model fit.

### Discrimination and Low Birthweight

As can be seen in Table 3, Black women were 4.2 times more likely to have an LBW delivery than White women (model 5). When self-reported experiences of racial discrimination were added to the model alone (model 6), the odds ratio for race/ethnicity was reduced. When the other covariates were added to the model alone (model 7), the race/ethnicity odds ratio increased. In the full model (model 8), women reporting high levels of racial discrimination were almost 5 times more likely than women reporting no racial discrimination to deliver LBW infants. Depressive symptoms, net pregnancy weight gain, and alcohol and tobacco consumption were not significantly asso-

**TABLE 3—Logistic Regression Analysis of Low-Birthweight Deliveries Among 320 Black and White Women in the CARDIA Study, 1992–1995**

|  | Odds Ratio (95% Confidence Interval) |                    |                    |                    |                    |
|--|--------------------------------------|--------------------|--------------------|--------------------|--------------------|
|  | Model 5                              | Model 6            | Model 7            | Model 8            | Model 9            |
| Race/ethnicity: Black vs White                     | 4.24 (1.31, 13.67)                   | 2.11 (0.75, 5.93)  | 5.90 (1.48, 23.52) | 2.43 (0.79, 7.42)  | 3.97 (0.87, 18.14) |
| Self-reported racial discrimination                |                                      |                    |                    |                    |                    |
| 1 or 2 vs 0 experiences                            |                                      | 2.04 (0.50, 8.31)  |                    | 1.96 (0.51, 7.56)  | 1.06 (0.29, 3.84)  |
| ≥ 3 vs 0 experiences                               |                                      | 4.81 (1.50, 15.40) |                    | 4.98 (1.43, 17.39) | 1.56 (0.32, 7.76)  |
| Smoking status                                     |                                      |                    |                    |                    |                    |
| Former vs never smoker                             |                                      |                    | 3.51 (0.82, 15.13) | 2.96 (0.77, 11.49) | 3.73 (0.74, 18.93) |
| Current vs never smoker                            |                                      |                    | 1.99 (0.52, 7.69)  | 2.09 (0.56, 7.66)  | 2.42 (0.44, 13.40) |
| Alcohol use: current vs not current                |                                      |                    | 0.76 (0.23, 2.46)  | 0.59 (0.18, 1.99)  | 1.15 (0.28, 4.68)  |
| Depressive symptomatology: increase per unit score |                                      |                    | 1.02 (0.96, 1.07)  | 1.01 (0.96, 1.06)  | 0.96 (0.90, 1.02)  |
| Education: less than college vs college or more    |                                      |                    | 1.11 (0.31, 4.05)  | 1.07 (0.30, 3.83)  | 0.88 (0.24, 3.25)  |
| Income, \$   |                                      |                    |                    |                    |                    |
| 25 000–49 999 vs <25 000                           |                                      |                    | 1.52 (0.46, 4.99)  | 1.43 (0.41, 4.97)  | 1.36 (0.27, 6.85)  |
| ≥50 000 vs <25 000                                 |                                      |                    | 1.71 (0.33, 8.99)  | 1.59 (0.29, 8.86)  | 1.67 (0.33, 8.55)  |
| Pregnancy net weight gain: risk per kg             |                                      |                    | 0.96 (0.87, 1.06)  | 0.96 (0.87, 1.04)  | 0.95 (0.84, 1.07)  |
| Gestational age: risk per additional week          |                                      |                    |                    |                    | 0.54 (0.42, 0.68)  |

Note. The Hosmer–Lemeshow goodness of fit test statistic was 4.91 ( $P = .56$ ).

ciated with LBW and did not mediate the relationship between discrimination and LBW. Finally, adding gestational age to the model (model 9) substantially reduced the parameter estimates for racial discrimination. The Hosmer–Lemeshow statistic was not significant, indicating a good model fit.

## DISCUSSION

Racial discrimination may affect health outcomes in a variety of different ways through its influence on factors ranging from access to health care to exposure to noxious agents.<sup>27</sup> In this study, we tested the hypothesis that racial discrimination, as a psychosocial stressor, is associated with negative health outcomes. We found that high levels of self-reported experiences of racial discrimination were associated with both preterm and LBW deliveries and might contribute to Black–White disparities in these adverse birth outcomes. Smoking, alcohol use, and depressive symptoms did not appear to mediate the relationships between self-reported discrimination and adverse outcomes, although these relationships should be tested in a larger sample, with measurements taken during the pregnancy. In addition, our findings suggest that the association between racial discrimination and LBW may be mediated by gestational age.

Our findings are unlikely to be caused by biases in the measurement of race/ethnicity, marital status, education, or depressive symptoms. Problems pertaining to misclassification and bias, however, could have affected our data in the case of self-reports of racial discrimination, pregnancy weight gain, preterm delivery, and LBW.<sup>28</sup> These potential problems, however, were unlikely to have seriously affected our results for several reasons. First, regarding racial discrimination, the participants in this study reported levels of discrimination similar to those reported by the CARDIA sample as a whole. Moreover, these exposure levels were similar to those detected in the handful of other contemporary epidemiological studies and surveys that have quantified self-reports of racial discrimination.<sup>29</sup>

Second, although mothers reported birth-related data without clinical verification, previous research indicates that maternal recall of data on birthweight and gestational age are

sufficiently accurate and unbiased by race/ethnicity to permit valid usage in epidemiological studies when data from birth records are unavailable.<sup>30–33</sup> In addition, there was no effect of elapsed time in our analyses. Nevertheless, access to clinically verified records would have strengthened our study.

Other limitations of the present study include the small sample size, the timing of measurements, and the lack of data on potentially relevant confounders. Our data were limited to births occurring after the year 7 CARDIA examination, the year in which the discrimination questionnaire was first administered. The resulting small sample size precluded analysis of models stratified according to race/ethnicity as well as testing of interactions. Similarly, our measures of socioeconomic position were limited to income and educational level; thus, our analyses may have been affected by residual confounding owing to unmeasured socioeconomic factors.

A related limitation of this study was the lack of data on several potentially important covariates for the time period under study, including onset of prenatal care, frequency of prenatal medical visits, prenatal alcohol and tobacco consumption, drug use, bacterial vaginosis,<sup>34</sup> and maternal experiences of violence during pregnancy.<sup>10,16,17</sup> Such variables may mediate the effects not only of race/ethnicity, but also of self-reported experiences of racial discrimination, on preterm and LBW deliveries. Although prepregnancy BMI was not significant when tested in a logistic model, our sample may have been too small to detect a relationship. Given the documented associations between prepregnancy BMI and birthweight<sup>35–37</sup> and between prepregnancy BMI, gestational weight gain, and birthweight,<sup>38,39</sup> future studies should examine the relationships among racial discrimination, prepregnancy BMI, gestational weight gain, and LBW.

Bias also could have been introduced by differential attrition rates, affecting estimates of outcomes as well as covariates. Notably, women not included in the present analyses were less educated, less likely to be married, and more likely to be Black than the study participants, and they had more depressive symptoms. Thus, our findings may have underestimated the effects of education, depressive symptoms, and marital status on the risk

of LBW and preterm deliveries. Given that depressive symptoms may mediate the relationship between self-reported discrimination and perinatal outcomes, we may have missed a potential relationship. It is unlikely that differential attrition according to education or marital status affected the relationships between self-reported discrimination and the outcomes under study.

Another limitation was the older age range of the individuals who took part in this study. The mean age of mothers was 34 years, whereas the majority of births in America occur among women in their 20s.<sup>1</sup> Similarly, given that CARDIA's sampling design was stratified according to race/ethnicity and education, our findings are not representative of the general population. Finally, we did not include data on preexisting chronic medical conditions or previous preterm deliveries, which could have affected the likelihood of poor perinatal outcomes.

Despite these limitations, two strands of evidence lend plausibility to our findings. First, as noted earlier, 2 recently published studies, one focusing on extremely LBW deliveries (less than 1500 g) and the other focusing on preterm deliveries, both showed that increased risks were associated with self-reported racial discrimination. The first study, conducted by Collins et al.,<sup>11</sup> was a small case–control investigation (25 case patients and 60 controls) restricted to a population of poor Black women with no private health insurance. Its central finding was that self-reported episodes of racial discrimination among low-income African American mothers were associated with deliveries of extremely LBW infants.<sup>27,40</sup>

The second study, conducted by Dole et al., involved data derived from a large, prospective cohort study of risk factors for preterm births that included 2073 White women and 1604 Black women.<sup>12</sup> Using the same discrimination measures used in this study, these authors found that high levels of self-reported racial discrimination were associated with somewhat lower but still increased risks of preterm delivery (adjusted OR=1.4, 95% CI=1.0, 2.0). Possibly contributing to their lower estimates were differences in the racial/ethnic distributions of the recruitment areas and differences in the ages of the mothers. Women in the CARDIA sample were from urban areas with



substantially higher percentages of Whites than the women in the Dole et al. study, who were from a predominantly Black region of central North Carolina and, thus, potentially had a lower likelihood of interacting with White residents. The younger mothers in the Dole et al. study may have accumulated less exposure to discrimination, and, of note, the “weathering hypothesis” indicates that the effects of social inequality on health increase with age.<sup>41,42</sup> However, notwithstanding such differences, the Dole et al. findings were similar to the findings of this study.

By contrast, a study focusing on racial discrimination and preterm deliveries conducted by Rosenberg et al.<sup>43</sup> showed little association between perceived racism and risk of preterm delivery. These authors, however, used different measures of self-reported discrimination and analyzed each item separately rather than assessing summed items. Use of noncomparable measures complicates comparisons of findings, further underscoring the importance of developing short, validated measures that can be used and compared across diverse epidemiological studies.<sup>27,44,45</sup>

A second strand of support for our findings stems from research in which the hypothesis that chronic stress can increase the risk of both preterm and LBW deliveries has been evaluated with data on biological parameters that we did not have available. Specifically, evidence indicates that psychological stress may trigger corticotropin-releasing hormone, which has been linked to preterm deliveries (see review by Rich-Edwards et al.<sup>10</sup>). Both animal and human studies suggest that stress can lead to immunosuppression, susceptibility to infection, and preterm birth.<sup>46–48</sup> Immuno-compromise has been linked to bacterial vaginosis,<sup>34,49</sup> which in turn has been associated with preterm births.<sup>50</sup> Neuroendocrine or immunological responses to the chronic stress generated by racial discrimination may in part explain the association between self-reported racial discrimination and risk of preterm and LBW deliveries we observed and should be investigated in a study focusing explicitly on pregnancy outcomes.

In addition, evidence on links between gestational hypertension and adverse birth outcomes<sup>51,52</sup> suggests an alternative pathway whereby racial discrimination elevates the risk

of gestational hypertension, thus affecting birth outcomes. In our sample, gestational hypertension was reported more frequently by Black than by White women, and the risk of LBW deliveries was elevated among Black women reporting gestational hypertension. Although our sample was too small to investigate a causal path leading from perceived discrimination to elevated blood pressure and adverse birth outcomes, future research should address this issue.

Despite the limitations noted, this study provides important evidence that a relationship exists between self-reported experiences of racial discrimination and preterm and LBW deliveries. In doing so, it adds to the small but growing body of literature<sup>18–21</sup> suggesting that racial discrimination, rather than “race” construed as “innate biology,”<sup>40</sup> underlies racial/ethnic disparities in health and places Black women and children—and potentially women and children who are members of other racial/ethnic groups—at risk for serious health consequences. ■

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#### Contributors

S. Mustillo conceived and designed the study, analyzed the data, and wrote the initial draft of the article. N. Krieger supervised and contributed to the conception and design of the study, the analysis and interpretation of the data, and the drafting of the article. E. P. Gunderson, H. McCreath, and C. I. Kiefe contributed to the analysis and interpretation of the data. S. Sidney, H. McCreath, and C. I. Kiefe contributed to acquisition of the data. All of the authors were involved in revisions of the article.

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#### Human Participant Protection

All examinations were approved by the institutional review boards at the participating institutions, and informed consent was obtained from each study participant.

#### References

1. Martin JA, Hamilton BE, Ventura SJ, Menacker F, Park MM. Births: final data for 2000. *Natl Vital Stat Rep*. February 12, 2002;50(5).
2. National Center for Health Statistics. Births: final data for 2001. Available at: [http://www.cdc.gov/nchs/data/nvsr/nvsr51/nvsr51\\_02.pdf](http://www.cdc.gov/nchs/data/nvsr/nvsr51/nvsr51_02.pdf). Accessed October 9, 2003
3. Hummer R. Black-white differences in health and mortality: a review and conceptual model. *Sociol Q*. 1996;37:105–125.
4. Schoendorf K, Hogue C, Kleinman J, Rowley D. Mortality among infants of Blacks as compared with White college-educated parents. *N Engl J Med*. 1992; 326:1522–1526.
5. Berg CJ, Wilcox LS, d'Almada PJ. The prevalence of socioeconomic and behavioral characteristics and their impact on very low birth weight in black and white infants in Georgia. *Maternal Child Health J*. 2001;5:75–84.
6. Krieger N, Rowley DL, Herman AA, Avery B, Phillips MT. Racism, sexism, and social class: implications for studies of health, disease, and well-being. *Am J Prev Med*. 1993;9:82–122.
7. David R, Collins J. Differing birth weight among infants of US-born blacks, African-born blacks, and US-born whites. *N Engl J Med*. 1997;337:1209–1214.
8. Rowley D, Hogue C, Blackmore C, et al. Preterm delivery among African-American women: a research strategy. *Am J Prev Med*. 1993;9:1–6.
9. McDonald KB. The psychosocial dimension of black maternal health: an intersection of race, gender, and class. In: Herring C, ed. *African Americans and the Public Agenda*. Thousand Oaks, Calif: Sage Publications; 1997:64–84.
10. Rich-Edwards J, Krieger N, Majzoub J, Zierler S, Lieberman E, Gillman M. Maternal experiences of racism and violence as predictors of preterm birth: rationale and study design. *Paediatr Perinat Epidemiol*. 2001; 15:124–135.
11. Collins JW Jr, David RJ, Symons R, Handler A, Wall SN, Dwyer L. Low-income African-American mothers' perception of exposure to racial discrimination and infant birth weight. *Epidemiology*. 2000;11:337–339.
12. Dole N, Savitz D, Hertz-Picciotto I, Siega-Riz A, McMahon M, Buekens P. Maternal stress and preterm births. *Am J Epidemiol*. 2003;157:14–24.
13. Parker B, McFarlane J, Soeken K. Abuse during pregnancy: effects on maternal complications and birth weight in adult and teenage women. *Obstet Gynecol*. 1994;84:323–328.
14. Bullock LF, McFarlane J. Violence during preg-

nancy does more than harm a woman physically and psychologically: battered women are also two to four times likelier to give birth to LBW infants. *Am J Nurs*. 1989;89:1153–1155.

15. Amaro H, Fried LE, Cabral H, Zuckerman B. Violence during pregnancy and substance abuse. *Am J Public Health*. 1990;80:575–579.

16. Fernandez FM, Krueger PM. Domestic violence: effect on pregnancy outcome. *J Am Osteopath Assoc*. 1999;99:254–256.

17. Shumway J, O'Campo P, Gielen A, Witter FR, Khouzami AN, Blakemore KJ. Preterm labor, placental abruption, and premature rupture of membranes in relation to maternal violence or verbal abuse. *J Maternal-Fetal Med*. 1999;8:76–80.

18. Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med*. 1990;30:1273–1281.

19. Krieger N, Sidney S. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health*. 1996;86:1370–1378.

20. Williams D, Chung A. Racism and health. In: Gibson RC, Williams D, eds. *Health in Black America*. Newbury Park, Calif: Sage Publications; 1997.

21. Williams DR, Neighbors H. Racism, discrimination, and hypertension: evidence and needed research. *Ethn Dis*. 2001;11:800–816.

22. Friedman G, Cutter G, Donahue R, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. *J Clin Epidemiol*. 1988;41:1105–1116.

23. Radloff L. The CES-D Scale: a self-report depression scale for research in the general population. *Appl Psychol Meas*. 1977;3:385–401.

24. Huber PJ. The behavior of maximum likelihood estimates under nonstandard conditions. In: *Proceedings of the Fifth Berkeley Symposium on Mathematical Statistics and Probability*. Vol. 1. Berkeley, Calif: University of California Press; 1967:221–223.

25. White H. Maximum likelihood estimation of misspecified models. *Econometrica*. 1982;50:1–25.

26. Hosmer DW, Lemeshow S. *Applied Logistic Regression*. 2nd ed. New York, NY: John Wiley & Sons Inc; 2000.

27. Krieger N. Discrimination and health. In: Berkman L, Kawachi I, eds. *Social Epidemiology*. New York, NY: Oxford University Press Inc; 2000:36–75.

28. Grimes DA, Schulz KF. Bias and causal associations in observational research. *Lancet*. 2002;359:248–252.

29. *Race and Ethnicity in 2001: Attitudes, Perceptions, and Experiences* [survey]. Washington, DC: Washington Post; 2001.

30. Ekouevi K, Morgan S. Note on the reliability and validity of mothers' retrospective reports of their children's birthweights. *Soc Biol*. 1991;38:140–145.

31. Troy L, Michels K, Hunter D, et al. Self-reported birthweight and history of having been breastfed among younger women: an assessment of validity. *Int J Epidemiol*. 1996;25:122–127.

32. Tomeo C, Rich-Edwards J, Michels K, et al. Reproducibility and validity of maternal recall of pregnancy-related events. *Epidemiology*. 1999;10:774–777.

33. Walton K, Murray L, Gallagher A, Cran G, Savage M, Boreham C. Parental recall of birthweight: a good proxy for recorded birthweight? *Eur J Epidemiol*. 2000;16:793–796.

34. Culhane JF, Rauh V, McCollum KF, Hogan VK, Agnew K, Wadhwa PD. Maternal stress is associated with bacterial vaginosis in human pregnancy. *Maternal Child Health J*. 2001;5:127–134.

35. Wong W, Tang NLS, Lau TK, Wong TW. A new recommendation for maternal weight gain in Chinese women. *J Am Diet Assoc*. 2000;100:791–796.

36. Chang S, O'Brien K, Nathanson M, Mancini J, Witter F. Characteristics and risk factors for adverse birth outcomes in pregnant black adolescents. *J Pediatr*. 2003;143:250–257.

37. Bergmann RL, Richter R, Bergmann KE, Plagemann A, Brauer M, Dudenhausen JW. Secular trends in neonatal macrosomia in Berlin: influences of potential determinants. *Paediatr Perinat Epidemiol*. 2003;17:244–249.

38. Schieve L, Cogswell M, Scanlon K, et al. Prepregnancy body mass index and pregnancy weight gain: association with preterm delivery. *Obstet Gynecol*. 2000;96:194–200.

39. Schieve L, Cogswell M, Scanlon K. Maternal weight gain and preterm delivery: differential effects by body mass index. *Epidemiology*. 1999;10:141–147.

40. Krieger N. Epidemiology, racism, and health: the case of low birth weight. *Epidemiology*. 2000;11:237–239.

41. Geronimus AT. Black/white differences in the relationship of maternal age to birthweight: a population-based test of the weathering hypothesis. *Soc Sci Med*. 1996;42:589–597.

42. Geronimus AT. Commentary: weathering Chicago. *Int J Epidemiol*. 2003;32:90–91.

43. Rosenberg L, Palmer JR, Wise LA, Horton NJ, Corwin MJ. Perceptions of racial discrimination and risk of preterm birth. *Epidemiology*. 2002;13:646–653.

44. Krieger N. Does racism harm health? Did child abuse exist before 1962? On explicit questions, critical science, and current controversies: an ecosocial perspective. *Am J Public Health*. 2003;93:194–199.

45. Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. *Am J Public Health*. 2003;93:200–208.

46. DeSouza E. Corticotropin-releasing factor and interleukin-1 receptors in the brain-endocrine-immune axis: role in stress response and infection. *Ann N Y Acad Sci*. 1993;697:9–27.

47. Goepfert A, Goldenberg R. Prediction of prematurity. *Curr Opin Obstet Gynecol*. 1996;8:417–427.

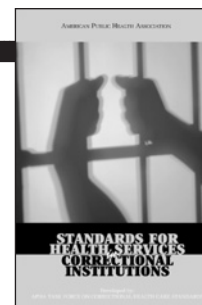
48. Culhane J, Rauh V, McCollum K, Elo I, Hogan V. Exposure to chronic stress and ethnic differences in rates of bacterial vaginosis among pregnant women. *Am J Obstet Gynecol*. 2002;187:1272–1276.

49. Jamieson D, Duerr A, Klein R, et al. Longitudinal analysis of bacterial vaginosis: findings from the HIV epidemiology research study. *Obstet Gynecol*. 2001;98:656–663.

50. Flynn CA, Helwig AL, Meurer LN. Bacterial vaginosis in pregnancy and the risk of prematurity: a meta-analysis. *J Fam Pract*. 1999;48:885–892.

51. Hauth J, Ewell M, Levine R, et al. Pregnancy outcomes in healthy nulliparas who developed hypertension. *Obstet Gynecol*. 2000;95:24–28.

52. Sibai BM, Caritis SN, Hauth JC, et al. Preterm delivery in women with pregestational diabetes mellitus or chronic hypertension relative to women with uncomplicated pregnancies. *Am J Obstet Gynecol*. 2000;183:1520–1524.



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# Very Low Birthweight in African American Infants: The Role of Maternal Exposure to Interpersonal Racial Discrimination

James W. Collins Jr, MD, MPH, Richard J. David, MD, Arden Handler, DrPH, Stephen Wall, MD, and Steven Andes, PhD

It has long been recognized that African American infants are more than twice as likely as White infants to die in their first year of life.<sup>1,2</sup> Reflecting the public health relevance of this phenomenon, *Healthy People 2010* calls for the elimination of the racial disparity in infant mortality rates.<sup>3</sup> Infant birthweight is a primary determinant of infant mortality risk. The approximately 1% of births occurring at very low birthweight (VLBW; <1500 g), pathological in all populations,<sup>1,4,5</sup> accounts for more than half of the neonatal deaths and 63% of the Black–White gap in infant mortality in the United States.<sup>4</sup> An extensive literature has treated pregnancy as a condition influenced by proximal events and has been unable to delineate the mechanisms underlying African American infants' threefold greater rate of VLBW.<sup>6–14</sup> A seminal study by Kleinman and Kessel<sup>6</sup> found not only a persistent but a widening racial gap in the incidence of VLBW infants as sociodemographic risk declines (i.e., VLBW risk declines as socioeconomic status increases). Another study found that in a prepaid health plan, the racial disparity in the rates of VLBW persisted among college-educated mothers who received adequate prenatal care.<sup>7</sup> Behavioral risk factors during pregnancy—cigarette smoking and alcohol and illicit drug usage—also have a negligible impact on the racial gap.<sup>14</sup> Numerous epidemiological studies have found that the racial differential in the rate of VLBW infants exists among women who reside in nonimpoverished neighborhoods.<sup>10–13</sup>

New conceptual models have been proposed to elucidate the contribution of chronic stress to preterm (<37 weeks) delivery and consequent VLBW risk.<sup>15–17</sup> Rich-Edwards et al.<sup>16</sup> speculated that chronic stress from maternal lifetime exposure to interpersonal racism is a risk factor for infant VLBW. Misra et al.<sup>17</sup> proposed that social

**Objectives.** We determined whether African American women's lifetime exposure to interpersonal racial discrimination is associated with pregnancy outcomes.

**Methods.** We performed a case-control study among 104 African American women who delivered very low birthweight (<1500 g) preterm (<37 weeks) infants and 208 African American women who delivered non-low-birthweight (>2500g) term infants in Chicago, Ill.

**Results.** The unadjusted and adjusted odds ratio of very low birthweight infants for maternal lifetime exposure to interpersonal racism in 3 or more domains equaled 3.2 (95% confidence intervals=1.5, 6.6) and 2.6 (1.2, 5.3), respectively. This association tended to persist across maternal sociodemographic, biomedical, and behavioral characteristics.

**Conclusions.** The lifelong accumulated experiences of racial discrimination by African American women constitute an independent risk factor for preterm delivery. (*Am J Public Health.* 2004;94:2132–2138)

(i.e., socioeconomic status) factors are antecedent to both psychosocial (i.e., stress, social support) factors and biomedical (i.e., health behaviors, preexisting diseases) factors; the latter are in turn risk factors for infant VLBW. Hogue et al.<sup>15</sup> proposed the classic host (i.e., pregnant women), environment (i.e., chronic social stressors), and agent (i.e., immediate emotional or physical stressors) triangle of epidemiological causality.<sup>5</sup>

Chronic stress is a more prominent feature in the daily lives of African American women than in the daily lives of White women.<sup>18</sup> Although there have been several studies on the relation between chronic stress and infant birthweight,<sup>19–21</sup> few studies have specifically focused on the relation between women's regular (ranging from a few times per year to nearly every day) exposure to racial discrimination—a nonrandom and race-related source of stress—and infant VLBW.<sup>18</sup> To the extent that population differences in chronic stress from lifetime exposure to interpersonal racial discrimination underlie the observed racial differential in the rate of VLBW infants, one would expect an association between this exposure and VLBW among African Americans.

A causal association between African American women's exposure to chronic stress from interpersonal racism and infant VLBW is biologically plausible. Wadhwa et al.<sup>22</sup> showed that chronic maternal exposure to stress—through maternal cardiovascular, immune/inflammatory, and neuroendocrine processes—is detrimental to infants' birthweight. Moreover, psychophysiological stress is likely to accelerate the release of corticotropin-releasing hormone, which initiates a cascade of events leading to preterm delivery.<sup>16,22</sup> Consistent with the larger literature on stress, clinical studies show that exposure to racial stressors leads to physiological reactivity.<sup>23–27</sup> African American women who were exposed to what they perceived as racial bias and internalized their responses to unfair treatment had a fourfold greater risk of hypertension.<sup>23</sup> In another study, the viewing of racist situations was associated with a significant rise in blood pressure that correlated with the African American subjects' responses on the Framington Anger Scale.<sup>24</sup> Jones et al.<sup>25</sup> also reported significant changes in heart rate, digital blood flow, and facial muscle activity in African American women who en-



countered social situations that included blatant and more subtle forms of racism.

We therefore performed a case-control study among a sample of urban African Americans to determine the extent to which women's reported lifetime and pregnancy exposure to interpersonal racial discrimination is associated with VLBW births.

## METHODS

### Study Sample

African American mothers delivering at Cook County Hospital and University of Chicago Hospital in Chicago, Ill, between November 1, 1997, and October 31, 2000, were recruited for this study. These hospitals serve critically ill and healthy infants across a broad range of socioeconomic status. Nevertheless, approximately two thirds of the participants in the study were Medicaid recipients.

The medical record was abstracted to determine infants' birthweight as defined by nursing measurement, gestational age based on physicians' physical assessment of the neonate, and maternal race as self-defined. Case subjects were restricted to mothers of singleton VLBW (<1500 g) preterm (<37 weeks) infants. Control subjects were restricted to mothers of (1) critically ill singleton non-low-birthweight (NLBW; >2500 g), term infants admitted to the neonatal intensive care unit for ventilator management; and (2) healthy singleton NLBW infants admitted to the normal newborn nursery. We approached the mothers of all eligible VLBW and critically ill NLBW infants. To ensure a 1:2 case-to-control ratio, we approached mothers of healthy NLBW infants who most approximated case infants with respect to time and day of admission within each participating hospital. We offered a \$10 participation reward to all eligible subjects. Study personnel approached African American mothers within 72 hours of their infants' admission to the neonatal intensive care unit or normal newborn nursery. We obtained informed consent from the women before study enrollment. Mothers of infants who expired within 72 hours of birth were not requested to complete the study questionnaire.

During the accrual period, 117 case subjects and 234 control subjects were potentially eligi-

ble. Of these, 3 case subjects and 5 control subjects refused interviews; 4 case subjects and 5 control subjects consented but failed to arrive at 3 scheduled appointments; we were unable to schedule interviews for 2 case subjects and 16 control subjects. The infants of 4 case subjects expired within 72 hours of birth. Thus, we obtained interview data for 104 case subjects and 208 control subjects.

### Study Questionnaire

Trained African American interviewers administered a structured questionnaire in the hospital. They collected data on mothers' age, education, marital status, parity, prenatal care initiation, cigarette smoking, and alcohol use. Using previously validated instruments, they asked about lifetime and pregnancy exposure to interpersonal racial discrimination.<sup>23,28</sup> All participants were asked their lifetime and pregnancy exposure to interpersonal racial discrimination in 5 domains: at work, getting a job, at school, getting medical care, and getting service at a restaurant or store.<sup>23</sup> The questions were formatted for yes or no answers.<sup>23</sup> We determined the distribution of reported interpersonal racial discrimination in each domain, 1 or more domains, and 3 or more domains. Current or recently employed participants were asked an additional 20 questions about their lifetime and past year's experiences with interpersonal racial discrimination at their primary place of employment.<sup>28</sup> We empirically dichotomized responses after data collection into none (none or less than once per year) and regularly (few times per year, few times per month, at least once a week, and nearly every day).

### Statistics

We calculated the odds ratio and 95% confidence intervals of exposure to measured risk factors.<sup>29</sup> Confidence intervals were estimated by the Taylor series method.<sup>29</sup> We used multivariable logistic regression (PROC LOGISTIC<sup>30</sup>) to estimate the independent association of maternal lifetime exposure to racism and VLBW.

## RESULTS

There were minimal differences between case subjects and control subjects (critically ill and healthy) with respect to marital status, in-

come, Medicaid status, prenatal care usage, parity, and alcohol consumption (Table 1). A slightly higher percentage of case subjects were found among the older, more educated women, and cigarette smokers (Table 1). When women aged older than 30 years or those having more than 12 years of education were compared with all others, a significantly increased association with VLBW was found ( $\chi^2=4.8$ ,  $P=.03$  for age,  $\chi^2=5.4$ ,  $P=.02$  for education). The distribution of sociodemographic, biomedical, and behavioral characteristics did not vary between critically ill and healthy control subjects (data available from authors by request).

Table 2 examines the relation between maternal exposure to interpersonal racism and VLBW in 5 domains. With the exception of the "getting medical care" domain, the odds ratio of VLBW for maternal lifetime exposure to interpersonal racial discrimination exceeded unity. The magnitude of the association between racial discrimination and VLBW was strongest in the "finding a job" and "at work" domains. The odds ratio of VLBW for maternal lifetime exposure to interpersonal racial discrimination in 1 or more domains was 1.9 (95% CI=1.2, 3.1). The odds ratio of VLBW for maternal lifetime exposure to interpersonal racial discrimination in 3 or more domains was 3.2 (95% CI=1.5, 6.6), suggesting a dose-response relation. In contrast, there was no consistent association of VLBW with incidents of perceived discrimination during the pregnancy.

When case subjects were compared only with critically ill control subjects, the odds ratio for exposure to racial discrimination in 1 or more and 3 or more domains equaled 1.9 (95% CI=1.1, 3.2) and 3.4 (95% CI=1.4, 8.3), respectively. When case subjects were compared only with healthy control subjects, the odds ratio for exposure to racial discrimination in 1 or more and 3 or more domains equaled 1.9 (95% CI=1.1, 3.4) and 3.0 (95% CI=1.3, 7.3), respectively. We further tested for the presence of recall bias by comparing the frequency of reported exposure to interpersonal racial discrimination in the 2 control groups of African American women with NLBW infants. The odds ratio for exposure to racial discrimination in 1 or more and 3 or more domains for critically ill (compared with well) control



**TABLE 1—Sociodemographic, Biomedical, and Behavioral Characteristics of the Study Sample: Chicago, Ill, November 1, 1997–October 31, 2000**

|                                  | Percentage (No.) of<br>VLBW Cases (n = 104) | Percentage (No.) Of<br>NLBW Controls (n = 208) | Odds Ratio (95%<br>Confidence Interval) |
|----------------------------------|---|--|---|
| Maternal age, y                  |   |  |   |
| <20                              | 27 (28)                                     | 31 (62)  | 1.1 (0.6, 2.1)                          |
| 20–24                            | 25 (26)                                     | 31 (63)  | 1.0                                     |
| 25–29                            | 19 (20)                                     | 21 (42)  | 1.2 (0.6, 2.3)                          |
| ≥ 30                             | 28 (29)                                     | 17 (35)  | 2.0 (1.0, 3.9)                          |
| Education, y                     |   |  |   |
| < 12                             | 31 (31)                                     | 39 (77)  | 0.5 (0.3, 0.9)                          |
| 12                               | 34 (34)                                     | 39 (77)  | 0.6 (0.3, 1.0)                          |
| > 12                             | 36 (36)                                     | 23 (46)  | 1.0                                     |
| Living arrangements              |   |  |   |
| Married                          | 23 (22)                                     | 15 (31)  | 1.0                                     |
| Unmarried, living together       | 12 (11)                                     | 18 (36)  | 0.4 (0.2, 1.0)                          |
| Unmarried, not together          | 65 (61)                                     | 67 (134)                                       | 0.6 (0.3, 1.2)                          |
| Income quartile, \$ <sup>a</sup> |   |  |   |
| 1: <5000                         | 28 (20)                                     | 29 (31)  | 0.8 (0.3, 1.9)                          |
| 2: 5000–15 999                   | 23 (16)                                     | 29 (31)  | 0.7 (0.3, 1.6)                          |
| 3: 16 000–30 999                 | 27 (19)                                     | 23 (25)  | 1.0 (0.4, 2.3)                          |
| 4: ≥ 31 000                      | 23 (16)                                     | 19 (20)  | 1.0                                     |
| Payment method                   |   |  |   |
| Medicaid                         | 62 (58)                                     | 68 (138)                                       | 0.8 (0.5, 1.2)                          |
| Other payments                   | 38 (35)                                     | 32 (63)  | 1.0                                     |
| Prenatal care                    |   |  |   |
| Early <sup>b</sup>               | 69 (71)                                     | 61 (127)                                       | 1.0                                     |
| Late or none                     | 31 (32)                                     | 39 (81)  | 0.7 (0.4, 1.2)                          |
| Pregnancies, No.                 |   |  |   |
| 1–3                              | 86 (89)                                     | 85 (171)                                       | 1.0                                     |
| ≥ 4                              | 14 (14)                                     | 15 (31)  | 0.9 (0.4, 1.7)                          |
| Cigarette smoking                |   |  |   |
| Smoker                           | 30 (31)                                     | 21 (43)  | 1.6 (1.0, 2.8)                          |
| Nonsmoker                        | 70 (72)                                     | 79 (163)                                       | 1.0                                     |
| Alcohol consumption              |   |  |   |
| Yes                              | 18 (19)                                     | 15 (32)  | 1.2 (0.7, 2.3)                          |
| No                               | 82 (84)                                     | 85 (176)                                       | 1.0                                     |

Note. VLBW = very low birthweight; NLBW = non-low-birthweight.

<sup>a</sup>For household income, 43% are missing data.

<sup>b</sup>Defined as initiation in the first trimester.

3 or more domains were 2.8 (95% CI=1.1, 7.1) and 7.3 (95% CI=1.9, 28.9), respectively. By contrast, for alcohol use and prenatal care categories, the racism effect was consistently stronger among women in the traditional high-risk sociodemographic, biomedical, and behavioral categories. Most important, 43 of the 48 odds ratios of VLBW for maternal lifetime exposure to interpersonal racial discrimination across the measured traditional risk factors were above unity; 95% confidence intervals often included 1.

Seventy-six percent (n=238) of women in the study sample had worked outside the home during their lifetime. Two thirds (n=163) of them were employed during their pregnancy. They worked an average of 35 hours per week. The leading employment categories were cashiers (23%), clerks (13%), teachers (10%), laborers (10%), and health care workers (8%). These 163 women answered additional questions about specific scenarios with racial discrimination at their primary place of employment, either anytime during their lifetime (10 questions) or during the past year (10 questions). For each of the questions in which there were sufficient responses for reasonably stable rate calculations, the point estimates for the association between regular (defined as “few times/year,” “few times/month,” “at least once a week,” or “nearly everyday”) exposure and VLBW exceeded unity (Table 4). The scenarios that had the strongest association with VLBW were “Because you are African American, you feel as if you have to work twice as hard” and “Whites often assume that you work in a lower status job than you do and treat you as such.” The odds ratios were between 1.1 and 2.6, although few were statistically significant.

Lastly, we performed multivariate logistic regression analyses to further explore the independent association of maternal reported lifetime exposure to interpersonal racial discrimination and pregnancy outcome. When maternal age, education, and cigarette smoking were included in logistic models, the adjusted odds ratio of infant VLBW for maternal reported exposure to interpersonal racial discrimination in 1 or more domains was 1.7 (95% CI = 1.0, 9.2); the adjusted odds ratio of infant VLBW for maternal reported expo-

subjects were 1.0 (95% CI=0.6, 1.7) and 1.1 (95% CI=0.4, 3.1), respectively.

Table 3 shows that the association between maternal lifetime exposure to interpersonal racism and infant VLBW persisted across traditional sociodemographic, biomedical, and behavioral risk categories; however, there was some evidence of effect modification. The adverse effect of perceived discrimination was strongest among women aged 20 to 29 years,

generally considered the optimal childbearing decade, whereas it was reduced or absent among teenaged women and women aged older than 30 years. Similarly, the association between maternal exposure to interpersonal racial discrimination and VLBW was strongest among women with more than 12 years of formal education. The odds ratios of infant VLBW for college-educated women who reported racial discrimination in 1 or more and

**TABLE 2—Maternal Exposure to Interpersonal Racial Discrimination and Infant Very Low Birthweight**

|                      | Reported Racial Discrimination Incidents |                                  |     |          |                                 |                                   |     |          |
|----------------------|--|----------------------------------|-----|----------|---------------------------------|-----------------------------------|-----|----------|
|                      | Lifetime                                 |                                  |     |          | This Pregnancy                  |                                   |     |          |
|                      | Percentage (No.)<br>VLBW n = 104         | Percentage (No.)<br>NLBW n = 208 | OR  | 95% CI   | Percentage (No.)<br>VLBW n = 10 | Percentage (No.)<br>NLBW n = 2088 | OR  | 95% CI   |
| Finding a job        | 29 (30)                                  | 13 (25)                          | 3.0 | 1.6, 5.4 | 2 (2)                           | 1 (3)                             | 1.3 | 0.2, 8.1 |
| At work              | 24 (25)                                  | 14 (29)                          | 2.0 | 1.1, 3.5 | 4 (4)                           | 5 (10)                            | 0.8 | 0.2, 2.6 |
| At school            | 18 (19)                                  | 11 (22)                          | 1.9 | 1.0, 3.7 | 2 (2)                           | 2 (4)                             | 1.0 | 0.2, 5.6 |
| In public settings   | 37 (38)                                  | 29 (61)                          | 1.4 | 0.8, 2.3 | 13 (14)                         | 15 (31)                           | 0.9 | 0.5, 1.8 |
| Getting medical care | 5 (5)                                    | 5 (11)                           | 0.9 | 0.3, 2.7 | 4 (4)                           | 2 (4)                             | 1.6 | 0.4, 6.2 |
| ≥ 1 domains          | 56 (58)                                  | 40 (83)                          | 1.9 | 1.2, 3.1 | 19 (20)                         | 20 (42)                           | 0.9 | 0.5, 1.7 |
| ≥ 2 domains          | 41 (32)                                  | 25 (41)                          | 2.1 | 1.2, 3.8 | 6 (6)                           | 4 (8)                             | 1.5 | 0.5, 4.4 |
| ≥ 3 domains          | 30 (20)                                  | 12 (17)                          | 3.2 | 1.5, 6.6 | 0 (0)                           | 1 (2)                             | ... | ...      |

Note. VLBW = very low birthweight; NLBW = non-low-birthweight; OR = odds ratio; CI = confidence interval.

sure to interpersonal racial discrimination in 3 or more domains was 2.6 (95% CI = 1.2, 5.3).

## DISCUSSION

Our study adds to the small but growing evidence of a relation between African American women's exposure to interpersonal racial discrimination and pregnancy outcomes. We found that African American mothers who delivered VLBW preterm infants were more likely to report experiencing interpersonal racial discrimination during their lifetime than African American mothers who delivered NLBW infants at term. Stratified analyses showed that this association persisted across the common risk categories for reproductive health. In multivariate logistic regression models, the adjusted odds ratio of VLBW for African American mothers who experienced interpersonal racial discrimination in 1 or more and 3 or more (compared with none) domains equaled 1.7 and 2.6, respectively. Interestingly, among African American women who worked outside the home, those who gave birth to VLBW infants were more likely to report racial discrimination in the workplace than were the working mothers of NLBW infants. These findings provide evidence that greater lifetime exposure to racial discrimination among African American women contributes to the racial disparity in VLBW infants.

The conventional investigative approach to the racial disparity in the rates of VLBW births has been based on the implicit assumption

that there is a set of risk factors that differ in quantity between the races but exert similar effects on African American and White women. An extensive literature has shown that established risk factors have minimal impact on the rate of VLBW for African Americans.<sup>6,7</sup> Moreover, this conceptualization does not take into account the nonrandom, pervasive, and multifaceted inequality that is bound up in the historical context of race, nor does it capture its effect on human beings over time.<sup>18,31,32</sup> Because African American women are regularly exposed to unique societal risk factors closely related to race,<sup>18,31–33</sup> restricting the search for such factors to a sample of African American women seems reasonable. We used an interviewer-administered closed-ended questionnaire to capture the variability of lifetime exposure to incidents perceived as racial discrimination and describe its association with infant birthweight. The frequency of lifetime reported incidents of interpersonal racial discrimination in at least 1 domain was 40% among our control subjects. If we take this frequency as an accurate estimate for the general population of urban African American women, then exposure to perceived racial discrimination is a common risk factor. This estimate is consistent with published prevalence rates.<sup>34</sup>

Our data show that the magnitude of the association between maternal reported lifetime exposure to racial discrimination and infant VLBW was strongest in the “finding a job” and “at place of employment” domains.

Concordant with this phenomenon, working-class African American mothers of VLBW preterm infants in our sample were more likely to regularly experience specific episodes of interpersonal racism at their primary place of employment than working-class African American mothers of NLBW term infants. These findings are consistent with the limited literature showing a negative association between pregnant African American women's psychosocial job strain and infant birthweight.<sup>35</sup> A recent study found that African American women with high job strain had infants with birthweights 273 grams less than those with low-strain jobs or those who did not work outside the home.<sup>35</sup>

Few published studies have explicitly examined the relation between maternal exposure to racial discrimination and infant birthweight.<sup>36,37</sup> Using mailed questionnaire data from the Black Women's Health Study, Rosenberg et al.<sup>37</sup> recently reported a small increase in preterm delivery among women who reported lifetime experiences of racism, particularly women with low levels of education.<sup>7</sup> In contrast, our study shows that the association between maternal reported lifetime exposure to interpersonal racism and infant VLBW is strongest among college-educated women. Because reporting discrimination may adversely affect self-esteem and perceptions of control,<sup>38</sup> differences in the methodology (i.e., mailed survey vs face-to-face interviews) used to assess lifetime incidents may contribute to the dissimilar findings. Further

**TABLE 3—Maternal Lifetime Exposure to Interpersonal Racial Discrimination and Infant Birthweight by Selected Characteristics**

|                                  | Reported Racial Discrimination Incidents in<br>≥ 1 Domains (vs No Reported Discrimination) |                                  |     |           | Reported Racial Discrimination Incidents in<br>≥ 3 Domains (vs No Reported Discrimination) |                                  |      |           |
|----------------------------------|--|----------------------------------|-----|-----------|--|----------------------------------|------|-----------|
|                                  | Percentage (No.)<br>VLBW n = 104   | Percentage (No.)<br>NLBW n = 208 | OR  | 95% CI    | Percentage (No.)<br>VLBW n = 104   | Percentage (No.)<br>NLBW n = 208 | OR   | 95% CI    |
| Maternal age, y                  |  |                                  |     |           |  |                                  |      |           |
| <20                              | 50 (14)  | 44 (27)                          | 1.3 | 0.5, 3.2  | 13 (2)   | 15 (6)                           | 0.8  | 0.2, 4.6  |
| 20–24                            | 62 (16)  | 32 (20)                          | 3.4 | 1.3, 8.9  | 33 (5)   | 4 (2)                            | 10.8 | 1.8, 63.6 |
| 25–29                            | 60 (12)  | 40 (17)                          | 2.2 | 0.7, 6.5  | 43 (6)   | 14 (4)                           | 4.7  | 1.1, 20.9 |
| ≥ 30                             | 52 (15)  | 49 (17)                          | 1.1 | 0.4, 3.0  | 33 (7)   | 18 (4)                           | 2.3  | 0.5, 9.2  |
| Education, y                     |  |                                  |     |           |  |                                  |      |           |
| < 12                             | 39 (12)  | 34 (26)                          | 1.2 | 0.5, 2.9  | 14 (3)   | 7 (4)                            | 2.0  | 0.4, 9.8  |
| 12                               | 53 (18)  | 39 (30)                          | 1.8 | 0.8, 4.0  | 24 (5)   | 15 (8)                           | 1.8  | 0.5, 6.4  |
| > 12                             | 75 (27)  | 52 (24)                          | 2.8 | 1.1, 7.1  | 57 (12)  | 15 (4)                           | 7.3  | 1.9, 28.9 |
| Married                          | 64 (14)  | 55 (17)                          | 1.4 | 0.5, 4.4  | 56 (10)  | 22 (4)                           | 4.4  | 1.0, 18.6 |
| Living together                  | 73 (8)   | 31 (11)                          | 6.1 | 1.3, 27.3 | 25 (1)   | 11 (3)                           | 2.8  | 0.2, 36.0 |
| Not together                     | 48 (29)  | 39 (52)                          | 1.4 | 0.8, 2.6  | 16 (6)   | 10 (9)                           | 1.7  | 0.6, 5.2  |
| Income quartile, \$ <sup>a</sup> |  |                                  |     |           |  |                                  |      |           |
| 1: < 5000                        | 40 (8)   | 29 (9)                           | 1.6 | 0.5, 5.3  | 8 (1)  | 4 (1)                            | 1.8  | 0.1, 32.0 |
| 2: 5000–15 999                   | 75 (12)  | 39 (12)                          | 4.8 | 1.2, 18.2 | 50 (4)   | 14 (3)                           | 6.3  | 1.0, 40.1 |
| 3: 16 000–30 999                 | 47 (9)   | 48 (12)                          | 1.0 | 0.3, 3.2  | 29 (4)   | 13 (2)                           | 2.6  | 0.4, 17.1 |
| 4: ≥ 31 000                      | 69 (11)  | 80 (16)                          | 0.6 | 0.1, 2.5  | 55 (6)   | 56 (5)                           | 1.0  | 0.2, 5.6  |
| Payment method                   |  |                                  |     |           |  |                                  |      |           |
| Medicaid                         | 50 (29)  | 37 (51)                          | 1.7 | 0.9, 3.2  | 22 (8)   | 11 (10)                          | 2.4  | 0.9, 6.7  |
| Other payment                    | 64 (23)  | 45 (29)                          | 2.1 | 0.9, 4.9  | 38 (8)   | 13 (5)                           | 4.3  | 1.2, 15.6 |
| Prenatal care                    |  |                                  |     |           |  |                                  |      |           |
| Early <sup>b</sup>               | 52 (37)  | 42 (53)                          | 1.5 | 0.8, 2.7  | 28 (13)  | 14 (12)                          | 1.7  | 1.0, 5.7  |
| Late or none                     | 63 (20)  | 37 (30)                          | 2.8 | 1.2, 6.6  | 37 (7)   | 9 (5)                            | 3.1  | 1.5, 6.2  |
| Pregnancies, No.                 |  |                                  |     |           |  |                                  |      |           |
| 1–3                              | 60 (53)  | 40 (68)                          | 2.2 | 1.3, 3.8  | 32 (17)  | 13 (15)                          | 3.2  | 1.5, 7.2  |
| ≥ 4                              | 36 (5)   | 42 (13)                          | 0.8 | 0.2, 2.8  | 25 (3)   | 5 (1)                            | 6.0  | 0.5, 66.2 |
| Cigarette smoking                |  |                                  |     |           |  |                                  |      |           |
| Smoker                           | 52 (16)  | 30 (13)                          | 2.5 | 0.9, 6.4  | 21 (4)   | 14 (5)                           | 1.6  | 0.4, 6.8  |
| Nonsmoker                        | 57 (41)  | 43 (70)                          | 1.8 | 1.0, 3.1  | 34 (16)  | 11 (12)                          | 4.0  | 1.7, 9.4  |
| Alcohol consumption              |  |                                  |     |           |  |                                  |      |           |
| Yes                              | 68 (13)  | 34 (11)                          | 4.1 | 1.2, 13.9 | 40 (4)   | 9 (2)                            | 7.0  | 1.0, 48.0 |
| No                               | 52 (44)  | 41 (72)                          | 1.6 | 0.9, 2.7  | 29 (16)  | 13 (15)                          | 2.8  | 1.3, 6.1  |

Note. VLBW = very low birthweight; NLBW = non-low-birthweight; OR = odds ratio; CI = confidence interval.

<sup>a</sup>Forty-three percent are missing data for household income.

<sup>b</sup>Defined as initiation in the first trimester.

research is needed to determine whether the inconsistencies reflect differences in unmeasured contextual variables.<sup>10,12,13,39–41</sup>

Our study provides empirical evidence supporting the conceptual model proposed by Rich-Edwards et al.<sup>16</sup> in which African American women's lifetime exposure to interpersonal racism is explicitly included as a chronic stressor.<sup>16</sup> Interestingly, we found no association between maternal self-reported exposure

to interpersonal racial discrimination during pregnancy and infant VLBW. However, the prevalence of 1 or more reported incidents during pregnancy among case subjects and control subjects was low; moreover, the prevalence of 3 or more reported incidents during pregnancy among subjects was essentially nonexistent. Given the suspected strong association between reported incidents of interpersonal racial discrimination during pregnancy

and VLBW among the subgroup of low-income African American mothers with high-risk behavioral characteristics,<sup>36</sup> our study did not have sufficient power to address the role of reported incidents during pregnancy.

Our study had a number of important limitations. First, because the experience of racial discrimination is a complex and multidimensional phenomenon, a more sensitive questionnaire may have led to better ascertain-

**TABLE 4—Maternal Exposure to Interpersonal Racial Discrimination in the Workplace and Infant Very Low Birthweight**

| Specific Perceptions  | Lifetime               |                         |     |          | Past Year              |                         |     |          |
|---|------------------------|-------------------------|-----|----------|------------------------|-------------------------|-----|----------|
|   | Percentage VLBW n = 53 | Percentage NLBW n = 110 | OR  | CI       | Percentage VLBW n = 53 | Percentage NLBW n = 110 | OR  | CI       |
| Because you are African American, you are assigned the jobs no one else will do.  | 19                     | 12                      | 1.7 | 0.7, 4.3 | 12                     | 8                       | 1.4 | 0.5, 4.2 |
| You are treated with less dignity and respect than you would be if you were White.  | 21                     | 12                      | 2.0 | 0.8, 4.7 | 23                     | 11                      | 2.3 | 1.0, 5.5 |
| You are watched more closely than other workers because of your race.   | 17                     | 8                       | 2.3 | 0.8, 6.1 | 10                     | 8                       | 1.3 | 0.4, 4.1 |
| Racial jokes or harassment are directed at you.   | ...                    | ...                     | ... | ...      | ...                    | ...                     | ... | ...      |
| Because you are African American, you feel as if you have to work twice as hard.  | 28                     | 17                      | 1.9 | 0.9, 4.1 | 25                     | 18                      | 1.6 | 0.7, 3.5 |
| Tasks that require intelligence are generally given to Whites, while African-Americans get those that don't require much thought. | 20                     | 12                      | 1.8 | 0.7, 4.6 | 14                     | 11                      | 1.3 | 0.5, 3.6 |
| You are often ignored or not taken seriously by your boss because of your race.   | ...                    | 6                       | ... | ...      | ...                    | 5                       | ... | ...      |
| Whites often assume that you work in a lower status job than you do and treat you as such.  | 29                     | 15                      | 2.3 | 1.0, 5.1 | 32                     | 15                      | 2.6 | 1.2, 5.8 |
| A White coworker with less experience and qualifications got promoted before you did.   | ...                    | 10                      | ... | ...      | ...                    | 9                       | ... | ...      |
| When different opinions would be helpful, your opinion is not asked for because of your race.                                     | 10                     | 9                       | 1.1 | 0.3, 3.3 | ...                    | 5                       | ... | ...      |
| Total positive responses  |                        |                         |     |          |                        |                         |     |          |
| ≥ 1   | 47                     | 34                      | 1.7 | 0.8, 3.5 | 49                     | 32                      | 2.0 | 1.0, 4.3 |
| ≥ 3 or more   | 26                     | 16                      | 1.7 | 0.7, 4.0 | 27                     | 16                      | 1.8 | 0.8, 4.4 |

Note. VLBW = very low birthweight; NLBW = non-low-birthweight; OR = odds ratio; CI = confidence interval; ... = undefined (<5 subjects).

ment of the exposure of chronic interpersonal racism. However, the assessment of discrimination in multiple domains and the characterization of regular exposure to discrimination in the workplace are strengths of the instruments used in our study.<sup>23,28,38</sup> In addition, the consistency of the elevated point estimates derived from 2 independently constructed instruments suggests that we accurately assessed exposure to interpersonal racial discrimination.<sup>23,28</sup> Second, our findings may have stemmed from a recall bias associated with the maternal anxiety associated with the admission of her infant to a neonatal intensive care unit. However, we found no difference in the prevalence of reported racism among control mothers of critically ill NLBW infants (a cohort with anxieties similar to those of case subjects) and the control mothers of healthy NLBW infants. Third, interviewer bias could have also influenced

our results. However, the interviewers were trained to collect data using a structured questionnaire in an identical fashion for case subjects and control subjects. They were also blinded to the study hypotheses. Fourth, sample size considerations limited our ability to fully address the association of racism and infant VLBW across the full range of maternal sociodemographic, biomedical, and behavioral characteristics. Lastly, the results of our study may be limited by the possible confounding of unmeasured variables closely related to interpersonal racial discrimination.<sup>41</sup> Lifelong exposure to interpersonal racism is unlikely to operate as a risk factor for pregnant women solely at the individual level, but it also expresses the cumulative impact of societal-level (i.e., institutional) racism exposures on birth outcome.<sup>32,39</sup> Our study suggests that a mechanism by which institutional racism affects female reproductive

health is likely to be found in the reported incidents of racial discrimination in the workplace. As such, interventions that target both the reported incidents of racial discrimination in the workplace and the structural issues of race inequality that place a large percentage of African American women in conditions of severe income insecurity are needed to narrow the racial disparity in infant VLBW.<sup>41</sup>

In conclusion, the reported lifelong accumulated experiences of interpersonal racial discrimination by African American women constitute an independent risk factor for infant VLBW. ■

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## Contributors

J. Collins originated the study, led the writing, and supervised all aspects of its implementation. R. David led the analyses and supervised subject recruitment at Cook County Hospital. A. Handler assisted with the study and questionnaire development. S. Wall assisted with the study and supervised subject recruitment at the University of Chicago. S. Andes synthesized the analyses and supervised data entry. All authors helped to conceptualize ideas, interpret findings, and review drafts of the article.

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## Human Participant Protection

Institutional review board approval was obtained at each hospital and participants provided written informed consent.

## References

- MacDorman M, Minino A, Strobino D, Guyer B. Annual summary of vital statistics—2001. *Pediatrics*. 2002;110:1037–1052.
- Centers for Disease Control and Prevention. Racial and ethnic differences in infant mortality rates—60 largest US cities, 1995–1998. *MMWR Morb Mortal Wkly Rep*. 2000;51:329–343.
- Healthy People 2010*. Washington, DC: US Department of Health and Human Services; 2001. Available at: <http://www.healthypeople.gov/document/>. Accessed September 1, 2004.
- Iyasu S, Becerra J, Rowley D, Hogue C. Impact of very low birth weight on the black-white infant mortality gap. *Am J Prev Med*. 1992;8:1333–1347.
- Matthews T, Menacker F, MacDorman M. Infant mortality statistics from the 2000 period linked birth/infant death data set. *Natl Vital Stat Rep*. 2002;50(12).
- Kleinman J, Kessel S. Racial differences in low birth weight. *N Engl J Med*. 1987;317:744–753.
- Murray J, Bernfield M. The differential effect of prenatal care on the incidence of low birth weight among blacks and whites in a prepaid health plan. *N Engl J Med*. 1988;74:1003–1008.
- Barfield W, Wise P, Rust F, et al. Racial disparities in outcomes of military and civilian births in California. *Arch Pediatr Adolesc Med*. 1996;150:1062–1067.
- David R, Collins J. Differing birth weight among infants of US-born blacks, African-born blacks, and US-born whites. *N Engl J Med*. 1997;337:1209–1219.
- Collins J, Herman A, David R. Very low birth-weight infants and income-incongruity among African-American and white parents in Chicago. *Am J Public Health*. 1997;87:414–417.
- Collins J, Schulte N, Drolet A. Differential effect of ecologic risk factors on the low birthweight components of African-American, Mexican-American, and non-Latino white infants in Chicago. *J Natl Med Assoc*. 1998;90:223–232.
- Pearl M, Braveman P, Abrams B. The relationship of neighborhood socioeconomic characteristics to birth weight among 5 ethnic groups in California. *Am J Public Health*. 2001;91:1808–1814.
- Rauh V, Andrews H, Garfinkel R. The contribution of maternal age to racial disparities in birthweight: a multilevel perspective. *Am J Public Health*. 2001;91:1815–1824.
- Berg C, Wilcox L, Almanda P. The prevalence of socioeconomic and behavioral characteristics and their impact on very low birth weight in black and white infants in Georgia. *Maternal Child Health J*. 2001;5:75–84.
- Hogue C, Hoffman S, Hatch M. Stress and preterm delivery: a conceptual model. *Paediatr Perinat Epidemiol*. 2001;15(suppl 2):30–40.
- Rich-Edwards J, Krieger N, Majzoub J, Zierler S, Liberman E, Gillman M. Maternal experiences of racism and violence as predictors of preterm birth: rationale and study design. *Paediatr Perinat Epidemiol*. 2001;15(suppl 2):122–135.
- Misra D, O'Campo P, Strobino D. Testing a sociomedical model for preterm delivery. *Paediatr Perinat Epidemiol*. 2001;15(suppl 2):110–122.
- Stancil T, Hertz-Picciotto I, Schramm M, Watt-Morse M. Stress and pregnancy among African-American women. *Paediatr Perinat Epidemiol*. 2000;14:127–135.
- Collins J, David R, Symons R, Handler A, Wall S, Andes J. African-American mothers' perception of their residential environment, stressful life events, and very low birth weight. *Epidemiology*. 1998;9:286–289.
- Lobel M, Dunkel-Schetter C, Scrimshaw S. Prenatal stress and prematurity: prospective study of socioeconomically disadvantaged women. *Health Psychol*. 1992;11:32–40.
- Orr S, James S, Miller C, Barakat B. Psychosocial stressors and low birth weight in an urban population. *Am J Prev Med*. 1996;12:459–466.
- Wadhwa P, Culhane J, Rauh V, Barve S. Stress and preterm birth: neuroendocrine, immune/inflammatory, and vascular mechanisms. *Maternal Child Health J*. 2001;5:119–125.
- Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc Sci Med*. 1990;30:1273–1281.
- Armstead C, Lawler K, Gorden G, Cross J, Gibbons J. Relationship of racial stressors to blood pressure responses and anger expression in college students. *Health Psychol*. 1980;8:541–556.
- Kreiger N, Sidney S. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health*. 1996;86:1370–1378.
- Jones D, Morris-Parther C, Thomas J, Omowale N. Affective and physiological responses to socially stressful stimuli. *Ethnicity Dis*. 1996;6:123–131.
- Butler C, Tull E, Chambers E, Taylor J. Internalized racism, body fat distribution, and abnormal fasting blood glucose among African-American Caribbean women in Dominica West Indies. *J Natl Med Assoc*. 2002;94:143–148.
- McNeilly M, Anderson N, Armstead C, et al. The perceived racism scale: a multidimensional assessment of the experiences of white racism among African-Americans. *Ethn Dis*. 1996;6:154–156.
- Schlesselman J. *Case-Control Studies: Design, Conduct, and Analysis*. New York, NY: Oxford University Press; 1982.
- SAS User's Guide, Version 6*, 4th ed, vol 1. Cary, NC: SAS Institute Inc; 1989.
- David R, Collins J. Bad outcomes in black babies: race or racism? *Ethnicity Dis*. 1991;1:236–244.
- Jones C. Levels of racism: a theoretic framework and a gardener's tale. *Am J Public Health*. 2000;90:1212–1215.
- Hogan V, Richardson J, Ferre C, Durant T, Boisseau M. A public health framework for addressing black and white disparities in preterm delivery. *J Am Med Womens Assoc*. 2001;56:177–180.
- Landrine H, Klonoff EA. The Schedule of Racist Events: a measure of racial discrimination and a study of its negative physical and mental health consequences. *J Black Psychol*. 1996;22:144–168.
- Oths K, Dunn L, Palmer N. A prospective study of psychosocial job strain and birth outcomes. *Epidemiology*. 2001;12:744–746.
- Collins J, David R, Symons R, Handler A, Wall S, Dwyer L. Low-income African-American mothers' perception of exposure to racial discrimination and infant birth weight. *Epidemiology*. 2000;11:337–339.
- Rosenberg L, Palmer J, Wise L, Horton N, Corwin M. Perceptions of racial discrimination and the risk of preterm birth. *Epidemiology*. 2002;13:646–652.
- Williams D, Neighbors H. Racism, discrimination and hypertension: evidence and needed research. *Ethn Dis*. 2001;11:800–816.
- Geronimus A. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis*. 1992;2:207–221.
- Buka S, Brennan R, Rich-Edwards J, Raudenbust S, Earls F. Neighborhood support and the birth weight of urban infants. *Am J Epidemiol*. 2003;157:1–8.
- Mullings L, Wall A. *Stress and Resilience: the Social Context of Reproduction in Central Harlem*. New York, NY: Kluwer Academic Publishers; 2001.

# An Approach to Studying Social Disparities in Health and Health Care

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With this article, we propose an approach to studying and monitoring social disparities in health and health care, using prenatal care as an example. We use the term “social disparities in health” broadly here to refer to differences in health—or likely determinants of health—that are systematically<sup>1,2</sup> associated with different levels of underlying social advantage or position in a social hierarchy.<sup>3</sup> Social advantage or position is reflected by economic resources, occupation, education, racial/ethnic group, gender, sexual orientation, and other characteristics associated with greater resources, influence, prestige, and social inclusion.<sup>3–7</sup>

Social disparities in health place people already disadvantaged by belonging to particular social groups at further disadvantage with respect to their health<sup>3,8,9</sup>; good health in turn is essential to escape from social disadvantage.<sup>9–11</sup> Efforts to reduce social disparities in health and equalize opportunities for optimal health reflect social and ethical values,<sup>8,12</sup> including solidarity or compassion<sup>8,13</sup> and distributive justice,<sup>13</sup> and are consonant with human rights principles.<sup>3,13,14</sup> The goals of *Healthy People 2010* include eliminating social disparities in health and health care.<sup>15</sup>

Social disparities in health, including gaps in maternal and child health and health care, are large and persistent in the United States.<sup>16–39</sup> There is widespread recognition that closing these gaps will require more effective strategies, including monitoring and research to guide and evaluate policies.<sup>5,40–48</sup> However, apart from racial/ethnic breakdowns of vital statistics, routine monitoring of social disparities in health in the United States has generally been limited.<sup>40,41,49–51</sup> This article was based on work supported by the Centers for Disease Control and Prevention and the Kaiser Family Foundation that examined socioeconomic and racial/ethnic disparities in 3 maternal and infant health indicators—unintended pregnancy, breastfeeding, and delayed or no prenatal care—in California during 1994–1995 and

**Objective.** We explored methods and potential applications of a systematic approach to studying and monitoring social disparities in health and health care.

**Methods.** Using delayed or no prenatal care as an example indicator, we (1) categorized women into groups with different levels of underlying social advantage; (2) described and graphically displayed rates of the indicator and relative group size for each social group; (3) identified and measured disparities, calculating relative risks and rate differences to compare each group with its a priori most-advantaged counterpart; (4) examined changes in rates and disparities over time; and (5) conducted multivariate analyses for the overall sample and “at-risk” groups to identify particular factors warranting attention.

**Results.** We identified at-risk groups and relevant factors and suggest ways to direct efforts for reducing prenatal care disparities.

**Conclusions.** This systematic approach should be useful for studying and monitoring disparities in other indicators of health and health care. (*Am J Public Health*. 2004;94:2139–2148)

1999–2001. A separate report<sup>52</sup> on that work, aimed at a wide nontechnical audience, highlights issues that policies should address. The focus of our article is primarily methodological, aiming to illustrate a systematic approach for studying and monitoring disparities that can be adapted for other indicators and populations. Space constraints limit us here to using 1 indicator—delayed or no prenatal care—as an example. Although the ideal content and number of prenatal visits are unknown,<sup>53,54</sup> few would contest the importance of at least 1 first-trimester visit for timely assessment and health promotion.<sup>55–57</sup> Healthy People 2010 objectives<sup>15</sup> include first-trimester care for at least 90% of childbearing women.

## METHODS

### Data Sources

We used cross-sectional data from 2 California statewide representative postpartum surveys, with approval from the University of California, San Francisco committee on human research and the California Health and Human Services Agency committee for the protection of human subjects. The 1999–2001 data (n=10 519) were obtained from the Maternal and Infant Health Assessment (MIHA). A col-

laborative effort of the California Department of Health Services Maternal and Child Health Branch and University of California, San Francisco, modeled on the Centers for Disease Control and Prevention's Pregnancy Risk Assessment Monitoring System,<sup>58</sup> MIHA is an annual population-based mail survey (with telephone follow-up of nonresponders) of mothers a few months after they give birth to live-born infants in California. Data for 1994 and 1995 were obtained from the Access to Maternity Care (ATM) survey, in which 10 132 mothers of live-born infants were interviewed during their postpartum stays in 19 randomly selected California hospitals. The ATM survey was conducted with support from the Agency for Health Care Policy Research, the California Department of Health Services, and the Robert Wood Johnson Foundation. Both surveys were linked with birth certificates and with census data from 2000 (MIHA) or 1990 (ATM). Residential addresses from birth certificates were geocoded to the census tract level (approximately 4000–8000 people per tract) using MapMarker Plus software<sup>59</sup> for MIHA and services from Geographic Data Technology, Inc. (Lebanon, NH), for ATM. Both procedures use several reliable and regularly updated sources of address files (e.g., US Postal

Service, Census TIGER files),<sup>60</sup> and geocoding was successful for 97.4% of addresses in MIHA and 83.8% (87.3% after excluding 1 hospital without linked birth certificates) in ATM. Both statistically weighted samples were similar to the statewide maternity populations during corresponding time periods. MIHA and ATM response rates were 71% and 86%, respectively. Methods for both surveys have been described elsewhere.<sup>29,61,62</sup>

## Variables

The indicator of health and health care used as an example was delayed or no care, which was defined as either beginning prenatal care after the first trimester or receiving no prenatal care during the index pregnancy. Social groups were defined according to (1) 3 socioeconomic variables (i.e., family income, maternal education, and neighborhood poverty), chosen a priori to categorize the sample into groups reflecting different dimensions of socioeconomic status or position plausibly related to delayed or no care<sup>28,56,61,63–65</sup>; and (2) race/ethnicity.

**Family income.** Family income was defined as the self-reported family income during pregnancy in 100% increments of the federal poverty level for the relevant year (e.g., \$17 650 for a family of 4 in 2001). Income of the nuclear family (the woman, her partner, and dependent children) was used instead of household income to conform with eligibility criteria for Medi-Cal and other programs that could influence prenatal care use.

**Maternal education.** Maternal education was defined as the respondent's self-reported highest completed educational level (i.e., did not complete high school, high-school graduate, some college, college graduate).

**Neighborhood poverty.** The definition of neighborhood poverty was based on women's residences at the index birth, defining a "poor" neighborhood as a census tract with at least 20% of persons below the federal poverty level<sup>66</sup> in 1990 (ATM) or 2000 (MIHA). We used census tracts rather than smaller block groups because tracts generally geocode at a higher rate and are simpler to use; previous studies have found similar results using tracts or block groups to define neighborhoods.<sup>67–69</sup> Although multiple characteristics of neighborhoods ideally should be examined,<sup>28,70–72</sup> for brevity we examined only poverty concentra-

tion, which has been widely used<sup>68,73–78</sup> and is easily understood by policymakers. Sample size constraints (e.g., few women in the highest income or education categories lived in "poor" neighborhoods) limited us to 2 poverty concentration categories; the 20% cutoff reflects the US Census Bureau definition of "poverty area"<sup>79</sup> and is supported by previous studies.<sup>73–76</sup>

**Race/ethnicity.** Self-reported racial/ethnic identification was categorized as African American, Asian/Pacific Islander, European American (including women from the Middle East), immigrant Latina, US-born Latina, or Native American/Alaska Native. Small numbers precluded separate multivariate analyses for Native Americans and categorizing non-Latina women by nativity.

Other covariates in 1999–2001 MIHA data were chosen on the basis of the literature<sup>56,63,65,80,81</sup> as being plausibly associated with delayed or no care, either as confounders or as mediators on pathways between social factors and prenatal care: paternal education, maternal first-trimester insurance coverage,<sup>81</sup> age, parity, marital status at the time of birth, primary language spoken at home, having a regular source of health care before pregnancy, whether the respondent felt her receipt of prenatal care was "very important" to others close to her, unintended pregnancy, initial unhappiness about the pregnancy, the respondent's general "sense of control" over her life ("mastery"),<sup>82</sup> and both smoking and drinking during pregnancy (as markers of general knowledge, attitudes, or beliefs that could influence use of care).

## Statistical Analyses

**Describing social disparities in prenatal care.** After categorizing women in each time period into social groups defined by family income, education, neighborhood poverty, and race/ethnicity, we estimated rates of delayed or no care in each group and calculated rate differences and relative risks for each group compared with the a priori most-advantaged corresponding group (Table 1). For example, each of the 4 lower income groups was compared with the highest income group. Because both risk levels and relative size of groups are relevant, we further examined disparities by income and education in 2 ways: (1) using bar graphs, with bar width reflecting the proportion of the popu-

lation in each group (suggested to us by work published by Wagstaff et al.<sup>83</sup>; this approach was used by Krieger and colleagues in 2002<sup>68</sup>); and (2) estimating 2 "summary (composite) measures"—the population-attributable risk and the relative index of inequality<sup>83–85</sup> (defined in Table 1 footnotes). Comparing 1994–1995 and 1999–2001, we examined changes between the 2 periods in the group-specific rates of delayed or no care, the sizes of the disparities, and the socioeconomic distributions.

**Identifying issues that warrant attention in efforts to reduce disparities.** Using logistic regression to estimate the odds ratio for delayed or no care in each disadvantaged social group relative to its counterpart a priori most-advantaged group, we assessed the potential contributions of different variables to the observed disparities by comparing the unadjusted and adjusted odds ratios from a series of models. We considered the variables used to define the social groups of a priori interest—income, maternal education, neighborhood poverty, and racial/ethnic group—together in the initial model. We next added other covariates in sequential models and in a final model including all variables, observing the effects on the odds ratios for each social variable. For simplicity, and because the results generally had similar implications, we report only the findings from the (1) unadjusted models, (2) initial multivariate model including the 4 social variables, and (3) full model; sequential models are not displayed.

Using 1999–2001 data, we identified at-risk social groups warranting particular attention because they did not meet the *Healthy People 2010* objective of 90% with early care and had elevated risks relative to their a priori most-advantaged counterparts. We conducted separate logistic regression analyses, including all covariates listed above, to explore risk factors for delayed or no care in each at-risk group. Because policy implications depend in part on numbers of affected people, we also calculated the prevalence of each covariate within each at-risk group.

All analyses were conducted with SUDAAN software<sup>86</sup> to account for effects of the clustered survey sampling designs<sup>87</sup> and to alleviate difficulties with statistical inference introduced by including both individual and family- and neighborhood-level variables in models.<sup>88,89</sup> Previous studies used a similar

**TABLE 1—Rates, Rate Differences, and Relative Risks of Delayed or No Care, by Income, Education, Race/Ethnicity, and Neighborhood Poverty, and Summary Measures of Socioeconomic Disparities: Postpartum Women Surveyed in California, 1994–1995 and 1999–2001**

|   | % of Total | % Delayed<br>or No Care | 95 % CI                   | Rate<br>Difference | Relative<br>Risk | 95 % CI      | PAR% <sup>a</sup> | RII <sup>b</sup> |
|---|------------|-------------------------|---------------------------|--------------------|------------------|--------------|-------------------|------------------|
| 1994–1995 (n = 10 132) <sup>c</sup>               |            |                         |                           |                    |                  |              |                   |                  |
| % of federal poverty level                        |            |                         |                           |                    |                  |              |                   |                  |
| Missing   | 3.2        | 25.0                    | (14.2, 35.8)              | 21.2               | 6.6              | (2.5, 17.2)* |                   |                  |
| 0–100   | 44.9       | 37.5 <sup>d</sup>       | (34.4, 40.6) <sup>d</sup> | 33.7 <sup>d</sup>  | 9.9              | (5.6, 17.5)* |                   |                  |
| 101–200   | 18.1       | 17.9                    | (13.4, 22.4)              | 14.1               | 4.7              | (2.1, 10.6)* | 77.06             | –2.03            |
| 201–300   | 11.9       | 11.8                    | (9.4, 14.2)               | 8.0                | 3.1              | (1.6, 5.9)*  |                   |                  |
| 301–400   | 8.6        | 8.1                     | (3.0, 13.2)               | 4.3                | 2.1              | (0.6, 7.4)   |                   |                  |
| ≥ 401   | 13.2       | 3.8                     | (1.8, 5.8)                |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| Maternal education <sup>e</sup> (completed level) |            |                         |                           |                    |                  |              |                   |                  |
| < High school                                     | 30.0       | 38.0 <sup>d</sup>       | (32.9, 43.1) <sup>d</sup> | 31.5 <sup>d</sup>  | 5.8              | (3.8, 8.9)*  |                   |                  |
| High school graduate/GED                          | 31.3       | 24.4                    | (20.5, 28.3)              | 17.9               | 3.7              | (2.7, 5.2)*  |                   |                  |
| Some college                                      | 23.9       | 14.6                    | (10.3, 18.9)              | 8.1                | 2.2              | (1.2, 4.1)*  | 72.22             | –1.69            |
| College graduate                                  | 14.8       | 6.5                     | (3.8, 9.2)                |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| Neighborhood poverty                              |            |                         |                           |                    |                  |              |                   |                  |
| Missing   | 18.2       | 22.2                    | (10.6, 25.9)              | 2.0                | 1.2              | (0.7, 2.0)   |                   |                  |
| ≥ 20% poor  | 22.7       | 31.6 <sup>d</sup>       | (27.9, 35.3) <sup>d</sup> | 11.4 <sup>d</sup>  | 1.6              | (1.4, 1.7)*  |                   |                  |
| < 20% poor  | 59.2       | 20.2                    | (17.1, 23.3)              |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| Race/ethnicity <sup>f</sup>                       |            |                         |                           |                    |                  |              |                   |                  |
| African American                                  | 6.8        | 21.6                    | (15.7, 27.5)              | 6.5                | 1.4              | (0.8, 2.6)   |                   |                  |
| Asian/Pacific Islander                            | 9.9        | 25.9                    | (17.5, 34.3)              | 10.8               | 1.7              | (0.9, 3.2)   |                   |                  |
| Foreign-born Latina                               | 35.4       | 31.5                    | (26.8, 36.2)              | 16.4               | 2.1              | (1.4, 3.1)*  |                   |                  |
| US-born Latina                                    | 12.5       | 24.0                    | (18.5, 29.5)              | 8.9                | 1.6              | (1.3, 1.9)*  |                   |                  |
| Native American/Alaska Native                     | 0.4        | 26.5                    | (3.6, 49.4)               | 11.4               | 1.8              | (0.7, 4.4)   |                   |                  |
| European American                                 | 34.9       | 15.1                    | (10.0, 20.2)              |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| 1999–2001 (n=10,519) <sup>g</sup>                 |            |                         |                           |                    |                  |              |                   |                  |
| % of federal poverty level                        |            |                         |                           |                    |                  |              |                   |                  |
| Missing   | 9.9        | 22.3                    | (19.6, 25.0)              | 18.9               | 6.6              | (5.0, 8.6)*  |                   |                  |
| 0–100   | 31.2       | 27.9 <sup>d</sup>       | (26.3, 29.5) <sup>d</sup> | 24.5 <sup>d</sup>  | 8.2              | (6.4, 10.5)* |                   |                  |
| 101–200   | 20.6       | 18.3                    | (16.5, 20.1)              | 14.9               | 5.4              | (4.2, 7.0)*  | 75.50             | –2.19            |
| 201–300   | 9.9        | 12.8                    | (10.6, 15.0)              | 9.4                | 3.8              | (2.8, 5.0)*  |                   |                  |
| 301–400   | 7.3        | 6.0                     | (4.2, 7.8)                | 2.6                | 1.8              | (1.2, 2.6)*  |                   |                  |
| ≥ 401   | 21.1       | 3.4                     | (2.6, 4.2)                |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| Maternal education <sup>e</sup> (completed level) |            |                         |                           |                    |                  |              |                   |                  |
| < High school                                     | 22.8       | 28.4 <sup>d</sup>       | (26.4, 30.4) <sup>d</sup> | 22.0 <sup>d</sup>  | 4.4              | (3.7, 5.3)*  |                   |                  |
| High school graduate/GED                          | 23.6       | 21.9                    | (20.1, 23.7)              | 15.5               | 3.4              | (2.8, 4.1)*  |                   |                  |
| Some college                                      | 30.4       | 12.7                    | (11.5, 13.9)              | 6.3                | 2.0              | (1.6, 2.4)*  | 62.38             | –1.70            |
| College graduate                                  | 23.1       | 6.4                     | (5.2, 7.6)                |                    | 1.0              |              |                   |                  |
|   | 100.0      |                         |                           |                    |                  |              |                   |                  |
| Neighborhood poverty                              |            |                         |                           |                    |                  |              |                   |                  |
| Missing   | 2.8        | 15.2                    | (10.7, 19.7)              | 0.9                | 1.1              | (0.8, 1.4)   |                   |                  |
| ≥ 20% poor  | 30.0       | 23.8 <sup>d</sup>       | (22.2, 25.4) <sup>d</sup> | 9.5 <sup>d</sup>   | 1.7              | (1.5, 1.8)*  |                   |                  |

Continued



TABLE 1—Continued

|                               |       |      |              |      |     |             |
|-------------------------------|-------|------|--------------|------|-----|-------------|
| <20% poor                     | 67.1  | 14.3 | (13.4, 15.2) |      | 1.0 |             |
|                               | 100.0 |      |              |      |     |             |
| Race/ethnicity <sup>f</sup>   |       |      |              |      |     |             |
| African American              | 6.4   | 18.8 | (16.6, 21.0) | 9.5  | 2.0 | (1.7, 2.4)* |
| Asian/Pacific Islander        | 10.2  | 17.8 | (15.3, 20.3) | 8.5  | 1.9 | (1.6, 2.3)* |
| Foreign-born Latina           | 28.6  | 25.1 | (23.3, 26.9) | 15.8 | 2.7 | (2.4, 3.1)* |
| US-born Latina                | 16.0  | 19.2 | (17.0, 21.4) | 9.9  | 2.1 | (1.8, 2.4)* |
| Native American/Alaska Native | 0.6   | 17.8 | (7.0, 28.6)  | 8.5  | 1.9 | (1.0, 3.5)  |
| European American             | 38.2  | 9.3  | (8.3, 10.3)  |      | 1.0 |             |
|                               | 100.0 |      |              |      |     |             |

Note. CI = confidence interval; GED = general equivalency diploma.

<sup>a</sup>PAR%: Population attributable risk percentage (PAR) is the percentage reduction in delayed or no care in the population overall that would occur if all groups of pregnant women were to experience the rate of the most-advantaged group.

<sup>b</sup>RII: The relative index of inequality (RII) reflects the experiences of the entire population, taking into account the relative size of each socioeconomic group. It involves calculating the mean health status of each socioeconomic group and then ranking the groups by their socioeconomic status. A summary measure (the slope index of inequality) is formed by means of weighted regression analysis. The RII is calculated above by dividing the slope index of inequality (the average decline in the standardized rate of delayed or no care moving from the most-disadvantaged to the most-advantaged socioeconomic group) by the rate of delayed or no care among women overall.

<sup>c</sup>Overall rate of delayed or no care in 1994–1995: 23.5% (95% CI = 20.3, 26.7).

<sup>d</sup>Significantly different ( $P < .05$ ) from the corresponding estimates in the other time period.

<sup>e</sup>Excludes women with unknown education: 0.3% in 1994–1995 and 0.9% in 1999–2001.

<sup>f</sup>Excludes women with unknown or other race/ethnicity: 0.8% in 1994–1995 and 2.4% in 1999–2001.

<sup>g</sup>Overall rate of delayed or no care in 1999–2001: 17.1% (95% CI = 16.3, 17.9).

\*Statistically significant difference compared with most-advantaged group ( $P < .05$ ).

approach.<sup>71,90–94</sup> Explicit multilevel linear modeling techniques were not used here because generally few women were sampled per tract (<5 in 90% of tracts in 1999–2001).<sup>95</sup>

## RESULTS

### Describing Social Disparities in Prenatal Care

Table 1 displays the income, maternal education, neighborhood poverty, and race/ethnicity distributions and corresponding delayed or no care rates during 1994–1995 and 1999–2001. In both periods, an income gradient in delayed or no care rates was suggested: the lower a woman's income, the more likely she was to lack first-trimester care. Figure 1 displays this graphically, along with the proportions of women in each income group. Compared with the highest income group, significantly higher rates (i.e., significant rate differences) and relative risks of delayed or no care were seen in both periods not only for the poorest women but for each income group up to 300% of poverty, and first-trimester care rates in all of these groups were below the 90% *Healthy People 2010* objective. Similarly, even women who had attended but not graduated from college had

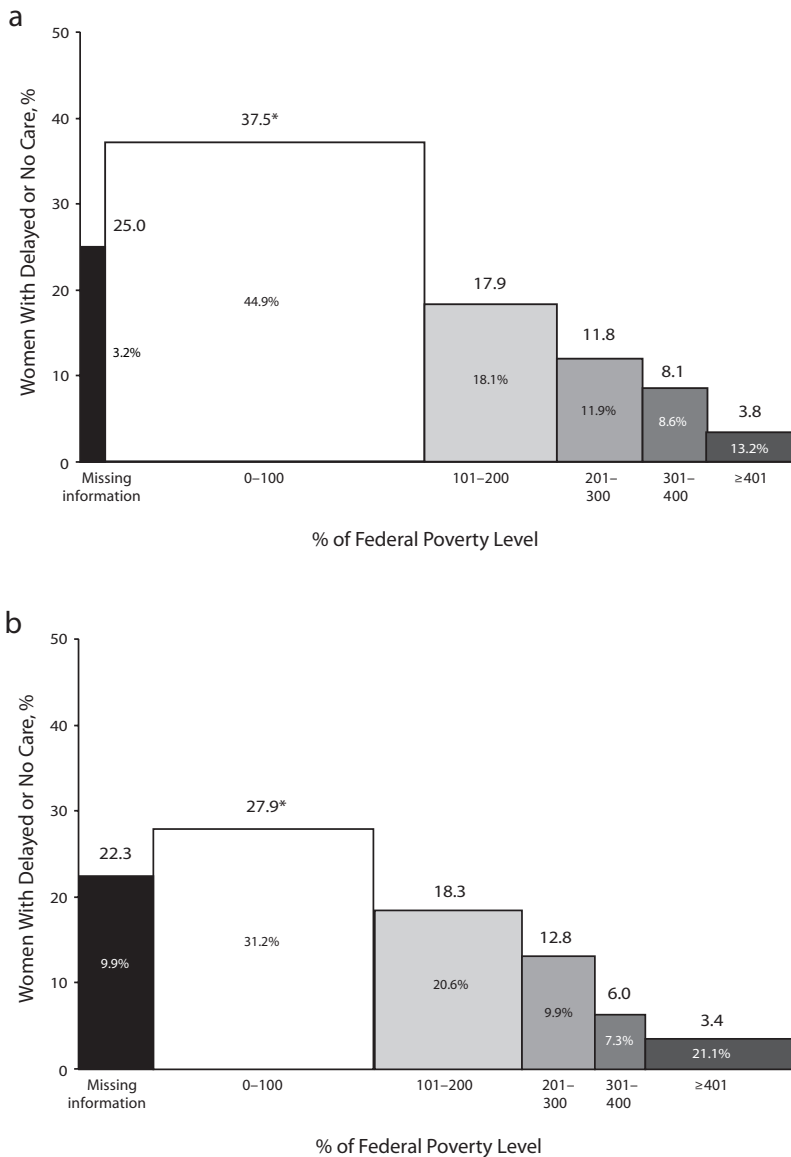
higher rates of delayed or no care than college graduates. At both times, delayed or no care rates were higher for women in poor compared with nonpoor neighborhoods; this difference was observed within most income, education, and racial/ethnic groups (not displayed). During both periods, all other racial/ethnic groups appeared to have higher rates of delayed or no care than European Americans, although these differences were not always statistically significant.

As shown in Table 1 and Figure 1, the percentage of women who were poor declined from 45% in 1994–1995 to 31% in 1999–2001. Comparing the 2 periods, we found declines in delayed or no care rates overall and within the most disadvantaged socioeconomic groups (poor, not high school graduates, residents of poor neighborhoods), with apparent declines in the corresponding rate differences for these groups. Comparing relative risks in the 2 periods, we found no significant reductions in the size of the disparities in care initiation. For example, although a smaller percentage of poor women had delayed or no care in 1999–2001 compared with 1994–1995, the relative gap between the poorest and most affluent groups was not significantly smaller. Comparing summary

measures of socioeconomic disparities for the 2 periods also suggested no significant improvement in income or education disparities.

### Identifying Issues that Warrant Attention in Efforts to Reduce Disparities

Table 2 displays prevalence rates and unadjusted odds ratios of delayed or no care in the entire 1999–2001 sample for each social group variable and covariate, along with multivariate results. Results from the initial model (model 1), including only the 4 variables defining the social groups of a priori concern, show that adjusted odds ratios for all income groups up to 300% of poverty remained significantly elevated and were not significantly lower than the unadjusted estimates; adjusted odds ratios for the education groups without any college remained elevated but were significantly reduced; and differences by neighborhood poverty were no longer significant. Compared with European Americans, other racial/ethnic groups remained at significantly higher risk of delayed or no care, but the odds ratio for each group except Asian/Pacific Islanders was significantly reduced after adjusting for the 3 socioeconomic variables. Full model (model 2) results again showed



Note. Width of bar shows percentage of population in each income group.

\*The proportion for this group in 1999-2001 was significantly different from that in 1994-1995 (at  $\alpha = .05$ ).

**FIGURE 1—Proportions of women with delayed or no prenatal care, by income: postpartum women surveyed in California in (a) 1994-1995 and (b) 1999-2001.**

marked and statistically significant income disparities; however, no other social group except Asian/Pacific Islanders appeared to be at elevated risk.

Separate models were run for the 3 groups of women—those with incomes up to 300% of poverty, lacking college degrees, or living in poor neighborhoods—identified as at-risk

(not displayed). In all 3 groups, significantly higher risks of delayed or no care were seen among women who were multiparous, lacked first-trimester insurance, reported that their prenatal care was not “very important” to others close to them, had unintended pregnancies, were initially unhappy about being pregnant, or were Asian/Pacific Islanders. Ele-

vated risks also were seen (but not in all 3 groups) among women who were young teens, unmarried, or who smoked or drank during pregnancy. Most of these risk factors were experienced by at least 10%—unintended pregnancy by over 40%—of women in each at-risk group.

## DISCUSSION

The objective of this article was to demonstrate the methods and potential applications of a systematic approach for studying and monitoring social disparities in health and health care. Using routinely collected population-based information for childbearing women in California during 1994-1995 and 1999-2001 and focusing on prenatal care as an example indicator, we (1) identified and measured disparities in delayed or no prenatal care across social groups defined by family income, maternal education, neighborhood poverty, and race/ethnicity; and (2) identified factors to consider in future efforts to reduce disparities. Results on the example indicator—delayed or no care—are discussed here to illustrate how this approach might provide useful information for other indicators, rather than to provide a comprehensive discussion of how to reduce prenatal care disparities.

Despite significant improvements in early prenatal care rates among childbearing women in California overall and within disadvantaged groups, disparities did not appear significantly smaller in 1999-2001 than in 1994-1995. In both periods, most groups of childbearing women in California had elevated delayed or no care rates, in absolute and relative terms. Only women with incomes above 300% of poverty, college graduates, and European Americans met the *Healthy People 2010* target. Although the proportion of childbearing women in poverty declined, as did rates of poverty in the general population at that time,<sup>96</sup> disparities by income persisted. In earlier work, we found marked improvements and reduced disparities in early prenatal care corresponding with federal and state initiatives during the late 1980s and early 1990s.<sup>65,97</sup> The absence of continued reductions in disparities during the later

**TABLE 2—Odds Ratios for Delayed or No Prenatal Care, by Income, Maternal Education, Race/Ethnicity, and Neighborhood Poverty: Postpartum Women (n = 10 210) Surveyed in California, 1999–2001**

|  |            | Unadjusted Odds<br>Ratio (95% CI) | Adjusted Odds Ratio (95% CI) |                   |
|--|------------|-----------------------------------|------------------------------|-------------------|
|  | % of Total |                                   | Model 1                      | Model 2           |
| Primary social variable                      |            |                                   |                              |                   |
| % of federal poverty level                   |            |                                   |                              |                   |
| Missing                                      | 10.2       | 8.28 (6.18, 11.10)                | 5.98 (4.32, 8.29)            | 3.42 (2.32, 5.04) |
| 0-100  | 31.9       | 10.91 (8.41, 14.14)               | 6.94 (5.05, 9.54)            | 2.98 (2.03, 4.39) |
| 101-200                                      | 20.4       | 6.31 (4.80, 8.30)                 | 4.55 (3.31, 6.26)            | 2.39 (1.64, 3.49) |
| 201-300                                      | 9.5        | 4.02 (2.93, 5.53)                 | 3.33 (2.35, 4.70)            | 2.62 (1.78, 3.85) |
| 301-400                                      | 7.1        | 1.65 (1.09, 2.52)                 | 1.49 (0.96, 2.30)            | 1.46 (0.89, 2.37) |
| ≥ 401  | 20.8       | Reference                         | Reference                    | Reference         |
| Maternal education (completed level)         |            |                                   |                              |                   |
| Less than high school                        | 23.8       | 5.90 (4.80, 7.25)                 | 1.95 (1.49, 2.54)            | 1.33 (0.96, 1.85) |
| High school/GED                              | 23.8       | 4.16 (3.37, 5.13)                 | 1.66 (1.28, 2.14)            | 1.30 (0.95, 1.78) |
| Some college                                 | 29.6       | 2.16 (1.74, 2.67)                 | 1.20 (0.93, 1.54)            | 1.05 (0.78, 1.40) |
| College graduate                             | 22.8       | Reference                         | Reference                    | Reference         |
| Neighborhood poverty <sup>a</sup>            |            |                                   |                              |                   |
| ≥ 20% poor                                   | 30.9       | 1.90 (1.69, 2.13)                 | 0.99 (0.87, 1.13)            | 1.01 (0.87, 1.18) |
| < 20% poor                                   | 69.1       | Reference                         | Reference                    | Reference         |
| Race/ethnicity <sup>b</sup>                  |            |                                   |                              |                   |
| African American                             | 6.5        | 2.25 (1.87, 2.70)                 | 1.25 (1.01, 1.53)            | 1.26 (1.00, 1.60) |
| Asian/Pacific Islander                       | 10.3       | 2.10 (1.69, 2.60)                 | 2.17 (1.74, 2.72)            | 2.29 (1.70, 3.09) |
| Foreign-born Latina                          | 29.8       | 3.26 (2.80, 3.78)                 | 1.38 (1.16, 1.65)            | 1.20 (0.88, 1.63) |
| US-born Latina                               | 16.0       | 2.31 (1.94, 2.76)                 | 1.23 (1.01, 1.49)            | 1.24 (0.98, 1.57) |
| European American                            | 37.5       | Reference                         | Reference                    | Reference         |
| Covariate                                    |            |                                   |                              |                   |
| Paternal education (completed level)         |            |                                   |                              |                   |
| Missing                                      | 8.0        | 6.49 (5.10, 8.26)                 |                              | 1.20 (0.85, 1.71) |
| Less than high school                        | 23.6       | 5.52 (4.49, 6.78)                 |                              | 1.15 (0.82, 1.59) |
| High school/GED                              | 28.5       | 3.44 (2.80, 4.22)                 | ...                          | 1.13 (0.83, 1.52) |
| Some college                                 | 17.0       | 1.98 (1.56, 2.52)                 |                              | 1.03 (0.76, 1.41) |
| College graduate                             | 23.0       | Reference                         |                              | Reference         |
| Unmarried                                    | 34.5       | 2.67 (2.38, 2.99)                 | ...                          | 1.26 (1.06, 1.49) |
| Maternal age, y                              |            |                                   |                              |                   |
| 15-17  | 3.9        | 5.17 (3.96, 6.74)                 |                              | 2.45 (1.65, 3.62) |
| 18-19  | 6.8        | 2.50 (1.95, 3.20)                 | ...                          | 1.38 (0.97, 1.96) |
| 20-34  | 73.5       | 1.47 (1.23, 1.75)                 |                              | 1.12 (0.88, 1.43) |
| ≥ 35   | 15.7       | Reference                         |                              | Reference         |
| Parity                                       |            |                                   |                              |                   |
| ≥ 5 births                                   | 4.3        | 2.40 (1.89, 3.05)                 |                              | 1.75 (1.23, 2.48) |
| 2-4 births                                   | 55.7       | 1.14 (1.01, 1.28)                 | ...                          | 1.37 (1.15, 1.63) |
| First birth                                  | 40.1       | Reference                         |                              | Reference         |
| No coverage in first trimester               | 17.5       | 6.60 (5.81, 7.50)                 | ...                          | 4.61 (3.93, 5.40) |
| No usual source of prepregnancy care         | 30.0       | 2.05 (1.82, 2.30)                 | ...                          | 1.10 (0.94, 1.28) |
| Non-English language spoken at home          | 40.3       | 2.18 (1.94, 2.43)                 | ...                          | 1.09 (0.86, 1.39) |
| Prenatal care not "very important" to others | 6.8        | 3.01 (2.52, 3.59)                 | ...                          | 1.92 (1.52, 2.42) |

*Continued*

1990s may reflect a lack of major new initiatives, “welfare reform,” or changes in policies affecting immigrants.<sup>98–103</sup>

The findings presented here suggest that interventions to further reduce prenatal care disparities should be more broadly targeted to reach women with incomes up to 300% of poverty (approximately three quarters of the California maternity population in 1999–2001) and those without college degrees (also approximately three quarters of childbearing women), as well as Asian/Pacific Islanders (10% of childbearing women) who are not generally considered at-risk. Our results confirmed earlier evidence that interventions to promote early prenatal care should focus on first-trimester insurance coverage,<sup>81</sup> family planning,<sup>63</sup> and general population attitudes about prenatal care.<sup>63</sup> Even with these efforts, the findings suggest that social disparities in prenatal care are unlikely to be eliminated without addressing underlying economic inequalities. Significant income disparities persisted after adjusting for education, insurance, and many other factors that may be on pathways from economic disadvantage to delayed or no prenatal care. Notably, racial/ethnic disparities were greatly reduced for most groups when income, education, and neighborhood poverty were considered.

We believe that the general approach presented here and summarized in Table 3 provides a model for monitoring social disparities and informing efforts to reduce them. Particularly relevant for states with Centers for Disease Control and Prevention Pregnancy Risk Assessment Monitoring System surveys, the approach requires routinely collected population-based data—on key social factors, potential risk or protective factors, and indicators of health (including health status, health care, health-related behaviors, and other likely health determinants)—that can be disaggregated to compare groups with different levels of underlying social advantage.<sup>5,41,104–106</sup> Socio-economic, racial/ethnic, gender, and geographic groups should always be considered when potentially relevant. Another crucial element of the approach is to examine indicators of health separately for each social group, comparing all other social groups with the a priori most-advantaged group. With that group

TABLE 2—Continued

|   |       |                   |     |                   |
|---|-------|-------------------|-----|-------------------|
| Smoked during pregnancy                       | 10.5  | 1.84 (1.57, 2.15) | ... | 1.33 (1.07, 1.64) |
| Drank during pregnancy                        | 18.7  | 1.12 (0.97, 1.29) | ... | 1.50 (1.25, 1.81) |
| Sense of control (7 = least, 28 = most), mean | 23.05 | 0.90 (0.88, 0.91) | ... | 0.99 (0.97, 1.01) |
| Unintended pregnancy                          | 46.7  | 2.84 (2.52, 3.19) | ... | 1.52 (1.29, 1.80) |
| Somewhat/very unhappy about pregnancy         | 18.7  | 2.55 (2.25, 2.89) | ... | 1.36 (1.15, 1.61) |

Note. CI = confidence interval; GED = general equivalency diploma.

<sup>a</sup>Women with missing data on neighborhood poverty were excluded.

<sup>b</sup>Native American/Alaska Native, other, or unknown race/ethnicity were excluded because of too few numbers.

TABLE 3—Overview of an Approach to Studying Social Disparities in Health and Health Care

1. Define groups with different levels of social advantage.
  - Categorize the population into socioeconomic and racial/ethnic groups (and other social groups of concern), identifying the a priori most-advantaged group in each category and viewing all other groups as relatively disadvantaged.
  - Use at least 2 different socioeconomic measures (e.g., education and income) and categorize into as many groups as sample sizes permit.
2. Describe the social disparities.
  - Examine the distributions of each social variable for each time period and rates of the health or health care indicator in each social group. Display the information as clearly as possible, in tabular and graphical form (Figure 1), using bar graphs in which (a) the height of each bar corresponds to the rate of the indicator in the social group and (b) the width corresponds to the relative size of the group (the proportion of the population included in the group).
  - Quantify the size of the gaps at each time using relative risks and rate differences, comparing each disadvantaged group with the a priori most-advantaged group. If possible, confirm socioeconomic comparisons using summary measures.
  - Compare differences across time periods in (a) group-specific rates of the health-related indicator, (b) the sizes of the disparities, and (c) the sizes of the social groups.
  - Identify at-risk social groups (a priori disadvantaged groups with elevated rates of adverse outcomes).
3. Identify important issues that should be addressed to reduce disparities.
  - Review previous research and local experience to identify likely risk and protective factors in at-risk groups; whenever possible, consult representatives of those groups.
  - When technical capabilities permit, use multivariate analyses to identify significant risk factors—including the social variables—in the population overall and in the identified at-risk social groups.
  - Calculate the prevalences of significant risk factors overall and in each at-risk group.
4. Disseminate findings to policymakers, advocates, and the public, highlighting how the results might inform further efforts to reduce disparities.

as a reference, disparities can be measured using rate differences and relative risks (and, when feasible, more complex summary measures), and at-risk social groups can be identified. Examining changes over time provides critical information for guiding policy. Although not definitive, a narrowing disparity may indicate that current policies should continue; a widening gap may suggest the need for changes. Policy responses are also informed by changes in the sizes of different socioeconomic groups (particularly in the proportions

who are poor or near-poor, of low educational levels, or living in poor neighborhoods) and in the prevalence of likely risk factors.

When informing policymakers about social disparities in health, a major challenge is to present information clearly and meaningfully without being simplistic. Descriptive findings can be presented in tables and graphically. Although summary measures (reflecting both the overall distribution of a socioeconomic variable and differences in risk across groups defined by that vari-

able<sup>83–85</sup>) are not widely used in research and may have limited intuitive meaning for policymakers, they can help to confirm conclusions based on simpler measures and to compare socioeconomic (but not racial/ethnic) disparities across states or time. A simpler alternative, illustrated in Figure 1, is to graphically display changes over time in both the observed socioeconomic disparities and the socioeconomic distribution. Policymakers are familiar with the concepts involved in these descriptive analyses, and health departments should have the necessary capabilities. When the requisite technical expertise is available, multivariate modeling can help identify risk factors to consider. Regardless of the analytic techniques used, quantitative results must be interpreted in the context of policies in all sectors that could influence health, informed by the literature and local knowledge. Although beyond the scope of this discussion, for many reasons we recommend that any study of disparities include representatives of relevant social groups to help identify issues and interpret findings.<sup>105,107,108</sup>

Work to describe and understand disparities, including selecting social groups to compare and covariates to examine, must be tailored to each health or health care indicator.<sup>109</sup> Using this approach with other indicators and in other states will require adaptations to accommodate differences in data sources, population sizes and characteristics, and technical capabilities. Several limitations we encountered are also likely to affect other efforts. No study can capture all relevant socioeconomic information, but every study should include at least 1 measure of economic resources. Income is limited as a measure of economic resources; however, at least in the US, data are more widely available on income than on accumulated assets. Education is important in itself but should not be used as a proxy for income.<sup>5,72,110,111</sup> The choice of socioeconomic and racial/ethnic variables will generally be limited in studies like this that rely on existing data. The surveys we used were restricted to women who spoke or read English or Spanish, which could have affected findings on Asian-Pacific Islanders. With data from different surveys and only 2 time periods, we could not formally assess trends over time.



Differences in neighborhood-level poverty results between time periods should be interpreted with particular caution for several reasons, including the following: the neighborhoods of surveyed women may not represent neighborhoods statewide; geocoding completeness and accuracy could differ between surveys (e.g., 16.2% of the earlier sample could not be geocoded); and effects may vary depending on the neighborhood socioeconomic characteristic being studied.<sup>28,70,71</sup> Because our primary goal was to demonstrate an overall approach, we did not explore many other area-level factors (e.g., the geographic distribution of health care facilities or providers) with potential relevance for prenatal care. Other states will also face limitations related to sample size, particularly for less prevalent indicators, requiring longer periods of data collection.

We hope that this work will generate discussion leading to more systematic and comprehensive approaches to studying and monitoring social disparities in health, particularly at the state level. Analyses must be framed and findings interpreted with the explicit goal of informing efforts to reduce disparities, systematically focusing on improvements among the socially disadvantaged.<sup>112</sup> Although health policymakers cannot dictate policies in other sectors, they can call attention to health-related disparities and advocate for action in other sectors. The economic recession and budget crises currently faced by California and other states threaten to severely cut back services that very likely contributed to earlier improvements.<sup>65,97</sup> In this environment, ongoing monitoring and analysis of state-level disparities are critical to inform policies and to ensure that scarce resources are used effectively. Monitoring and research are clearly not sufficient to eliminate disparities in health, but they are crucial.<sup>5,41,105,113,114</sup> ■

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### Contributors

All of the authors were involved in developing the conceptual framework and analytic approach presented in the article. P.A. Braveman contributed to writing the article, supervised all aspects of the project, and was the lead investigator on both postpartum surveys used in this work. S.A. Egerter contributed to writing the article and was involved in the methodological development of both surveys. C. Cubbin analyzed the data and contributed to writing the article. K.S. Marchi analyzed the data, contributed to writing the article, and was the project director for both surveys.

### Human Participant Protection

Approval for the original surveys and for the use of survey data in this work was obtained from the University of California, San Francisco committee on human research and the California Health and Human Services Agency committee for the protection of human subjects.

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### References

1. Starfield B. Improving equity in health: a research agenda. *Int J Health Serv.* 2001;31:545-566.
2. International Society for Equity in Health (ISEqH). Working definitions. Available at: <http://www.iseqh.org/en/workdef.htm>. Accessed June 6, 2003.
3. Braveman P, Gruskin S. Defining equity in health. *J Epidemiol Community Health.* 2003;57:254-258.
4. Krieger N. Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int J Health Serv.* 1999;29:295-352.
5. Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health.* 1997;18:341-378.

6. Amick BC, Levine S, Tarlov AR, Walsh DC, eds. *Society and Health.* New York: Oxford University Press; 1995.
7. Berkman LF, Kawachi I, eds. *Social Epidemiology.* New York: Oxford University Press; 2000.
8. Whitehead M. The concepts and principles of equity in health. *Int J Health Serv.* 1992;22:429-445.
9. Sen A. *Development as Freedom.* New York: Random House; 1999.
10. Anand S. The concern for equity in health. *J Epidemiol Community Health.* 2002;56:485-487.
11. *Macroeconomics and Health: Investing in Health for Economic Development.* Report of the Commission on Macroeconomics and Health. Geneva: World Health Organization; 2001.
12. Chang W-C. The meaning and goals of equity in health. *J Epidemiol Community Health.* 2002;56:488-491.
13. Mooney G. What does equity in health mean? *World Health Stat Q.* 1987;40:296-303.
14. Mann JM, Gruskin S, Grodin MA, Annas GJ, eds. *Health and Human Rights.* New York: Rutledge; 1999.
15. *Healthy People 2010: Understanding and Improving Health.* Washington, DC: US Dept of Health and Human Services; 2000.
16. Pamuk E, Makuc D, Heck K, Reuben C, Lochner K. *Socioeconomic Status and Health Chartbook: Health, United States, 1998.* Hyattsville, Md: National Center for Health Statistics; 1998.
17. Hogan VK, Richardson JL, Ferre CD, Durant T, Boisseau M. A public health framework for addressing black and white disparities in preterm delivery. *J Am Med Womens Assoc.* 2001;56:177-180.
18. Hogue CJR, Hargraves MA. Class, race, and infant mortality in the United States. *Am J Public Health.* 1993;83:9-12.
19. Kleinman JC, Kessel SS. Racial differences in low birth weight. *N Engl J Med.* 1987;317:749-753.
20. Kogan MD, Alexander GR. Social and behavioral factors in preterm birth. *Prenat Neonat Med.* 1998;6:29-31.
21. Kogan MD, Kotelchuck M, Alexander GR, Johnson WE. Racial disparities in reported prenatal care advice from health care providers. *Am J Public Health.* 1994;84:82-88.
22. Montgomery LE, Kiely JL, Pappas G. The effects of poverty, race, and family structure on US children's health: data from the NHIS, 1978 through 1980 and 1989 through 1991. *Am J Public Health.* 1996;86:1401-1405.
23. Singh GK, Yu SM. Infant mortality in the United States: trends, differentials, and projections, 1950 through 2010. *Am J Public Health.* 1995;85:957-964.
24. Singh GK, Yu SM. Adverse pregnancy outcomes: differences between US- and foreign-born women in major US racial and ethnic groups. *Am J Public Health.* 1996;86:837-843.
25. Braveman P, Oliva G, Miller MG, et al. Adverse outcomes and lack of health insurance among newborns in an eight-county area of California, 1982 to 1986. *N Engl J Med.* 1989;321:508-513.
26. Braveman PA, Egerter S, Bennett T, Showstack J. Differences in hospital resource allocation among sick

- newborns according to insurance coverage. *JAMA*. 1991;266:3300–3308.
27. Braveman P, Egerter S, Edmonston F, Verdon M. Racial/ethnic differences in the likelihood of cesarean section. *Am J Public Health*. 1995;85:625–630.
28. Pearl M, Braveman P, Abrams B. The relationship of neighborhood socioeconomic characteristics to birth-weight among 5 ethnic groups in California. *Am J Public Health*. 2001;91:1808–1814.
29. Cubbin C, Braveman P, Marchi K, et al. Socioeconomic and racial/ethnic disparities in unintended pregnancy among postpartum women in California. *Matern Child Health J*. 2002;6:237–246.
30. Gazmararian JA, Adams MM, Pamuk ER. Associations between measures of socioeconomic status and maternal health behavior. *Am J Prev Med*. 1996;12:108–115.
31. Cramer JC. Social factors and infant mortality: identifying high-risk groups and proximate causes. *Demography*. 1987;24:299–322.
32. Institute of Medicine. *Preventing Low Birthweight*. Washington, DC: National Academy Press; 1985.
33. Starfield BH. Child health and socioeconomic status. *Am J Public Health*. 1982;72:532–534.
34. Starfield B, Shapiro S, Weiss J, et al. Race, family income, and low birth weight. *Am J Epidemiol*. 1991;134:1167–1174.
35. Starfield B, Riley AW, Witt WP, Robertson J. Social class gradients in health during adolescence. *J Epidemiol Community Health*. 2002;56:354–361.
36. *Trends in the Well-Being of America's Children and Youth*. Washington, DC: US Dept of Health and Human Services; 2002.
37. Wise PH, Kotelchuck M, Wilson ML, Mills M. Racial and socioeconomic disparities in childhood mortality in Boston. *N Engl J Med*. 1985;313:360–366.
38. Newacheck PW, Hughes DC, Stoddard JJ. Children's access to primary care: differences by race, income, and insurance status. *Pediatrics*. 1996;97:26–32.
39. Krieger N, Rowley DL. Re: Race, family income, and low birth weight. *Am J Epidemiol*. 1992;136:501–502.
40. Krieger N, Fee E. Measuring socioeconomic status inequalities in health in the United States: a historical review, 1990–1950. *Int J Health Serv*. 1996;26:391–418.
41. Krieger N. The making of public health data: paradigms, politics, and policy. *J Public Health Policy*. 1992;13:412–427.
42. Krieger N, Moss NE. Accounting for the public's health: an introduction to selected papers from a US conference on "Measuring Social Inequalities in Health." *Int J Health Serv*. 1996;26:383–390.
43. Krieger N, Moss NE, Syme L. Recommendations of the conference "Measuring Social Inequalities in Health." *Int J Health Serv*. 1996;26:521–527.
44. Williams DR. Race, socioeconomic status, and health: the added effects of racism and discrimination. *Ann N Y Acad Sci*. 1999;896:173–188.
45. Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. *Ann Rev Sociol*. 1995;21:349–386.
46. Williams DR. Race and health: basic questions, emerging directions. *Ann Epidemiol*. 1997;7:322–333.
47. Rowley DL. Beyond the medical models: research on social factors and preterm delivery. *Prenat Neonat Med*. 1998;3:170–172.
48. Kramer MS. Preventing preterm birth: are we making progress? *Prenat Neonat Med*. 1998;3:10–12.
49. Krieger N. Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am J Public Health*. 1992;82:703–710.
50. Williams DR. Race/ethnicity and socioeconomic status: measurement and methodological issues. *Int J Health Serv*. 1996;26:483–505.
51. Krieger N, Fee E. Social class: the missing link in U.S. health data. *Int J Health Serv*. 1994;24:25–44.
52. Egerter S, Marchi K, Cubbin C, Braveman P. *Socioeconomic and Racial/Ethnic Disparities in Maternal and Infant Health in California*. Kaiser Family Foundation; 2004.
53. Tyson J, Guzick D, Rosenfeld CR, et al. Prenatal care evaluation and cohort analyses. *Pediatrics*. 1990;85:195–204.
54. Kogan MD, Martin JA, Alexander GR, Kotelchuck M, Ventura SJ, Frigoletto FD. The changing pattern of prenatal care utilization in the United States, 1981–1995, using different prenatal care indices. *JAMA*. 1998;279:1623–1628.
55. Brown S, ed. *Prenatal Care: Reaching Mothers, Reaching Infants*. Washington, DC: National Academy Press; 1988.
56. *Caring for Our Future: The Content of Prenatal Care*. Washington, DC: US Dept of Health and Human Services; 1989.
57. American Academy of Pediatrics, American College of Obstetrics and Gynecology. *Guidelines for Perinatal Care*. Elk Grove Village, Ill: American Academy of Pediatrics; 1997.
58. Pregnancy Risk Assessment Monitoring System (PRAMS). Available at: [http://www/cdc.gov/nccdphp/drh/srv\\_prams.htm](http://www/cdc.gov/nccdphp/drh/srv_prams.htm). Accessed December 22, 2003.
59. MapMarker Plus software (MapInfo Corp, Troy, NY).
60. Krieger N, Waterman P, Lemieux K, Zierler S, Hogan JW. On the wrong side of the tracks? Evaluating the accuracy of geocoding in public health research. *Am J Public Health*. 2001;91:1114–1116.
61. Braveman P, Egerter S, Marchi K. The prevalence of low income among childbearing women in California: implications for the private and public sectors. *Am J Public Health*. 1999;89:868–874.
62. Galbraith A, Egerter S, Marchi K, Chavez G, Braveman P. Newborn early discharge revisited: are California newborns receiving recommended postnatal services? *Pediatrics*. 2003;111:364–371.
63. Braveman P, Marchi K, Egerter S, Pearl M, Neuhaus J. Barriers to timely prenatal care among women with insurance: the importance of prepregnancy factors. *Obstet Gynecol*. 2000;95:874–880.
64. Roberts EM. Neighborhood social environments and the distribution of low birthweight in Chicago. *Am J Public Health*. 1997;87:597–603.
65. Braveman P, Marchi K, Sarnoff R, Egerter S, Rittenhouse D, Salganicoff A. *Promoting Access to Prenatal Care: Lessons from the California Experience*. Washington, DC: Kaiser Family Foundation; 2003.
66. O'Hare WP. A new look at poverty in America. *Popul Bull*. 1996;51:2–46.
67. Soobader M, LeClere F, Hadden W, Maury B. Using aggregate geographic data to proxy individual socioeconomic status: does size matter? *Am J Public Health*. 2001;91:632–636.
68. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramariam SV, Carson R. Geocoding and monitoring of US socioeconomic inequalities in mortality and cancer incidence: does the choice of area-based measure and geographic level matter? The Public Health Disparities Geocoding Project. *Am J Epidemiol*. 2002;156:471–482.
69. Krieger N, Chen JT, Waterman PD, Soobader MJ, Subramariam SV, Carson R. Choosing area based socioeconomic measures to monitor social inequalities in low birth weight and childhood lead poisoning: the Public Health Disparities Geocoding Project (US). *J Epidemiol Community Health*. 2003;57:186–199.
70. Lee RE, Cubbin C. Neighborhood context and cardiovascular behavioral risk factors in youth. *Am J Public Health*. 2002;92:428–436.
71. Cubbin C, LeClere FB, Smith GS. Socioeconomic status and fatal injuries: individual and neighborhood determinants. *J Epidemiol Community Health*. 2000;54:517–524.
72. Braveman P, Cubbin C, Marchi K, Egerter S, Chavez G. Measuring socioeconomic status in studies of racial/ethnic disparities: examples from maternal and child health. *Public Health Rep*. 2001;116:449–463.
73. *Health, United States, 1982*. Washington, DC: US Dept of Health and Human Services; 1982.
74. Cunradi CB, Caetano R, Clark C, Schafer J. Neighborhood poverty as a predictor of intimate partner violence among white, black, and Hispanic couples in the United States: a multi-level analysis. *Ann Epidemiol*. 2000;10:297–308.
75. Polednak AP. Stage at diagnosis of prostate cancer in Connecticut by poverty and race. *Ethn Dis*. 1997;7:215–220.
76. Enslinger ME, Lamkin RP, Jacobson N. School leaving: a longitudinal perspective including neighborhood effects. *Child Dev*. 1996;67:2400–2416.
77. McWhorter WP, Schatzkin AG, Horm JW, Brown CC. Contribution of socioeconomic status to black/white differences in cancer incidence. *Cancer*. 1989;63:982–987.
78. Breen N, Figueroa JB. Stage of breast and cervical cancer diagnosis in disadvantaged neighborhoods: a prevention policy perspective. *Am J Prev Med*. 1996;12:319–326.
79. *Statistical Brief: Poverty Areas*. Washington, DC: US Dept of Commerce; 1995.
80. Nothnagle M, Marchi K, Egerter S, Braveman P. Risk factors for late or no prenatal care following Medicaid expansions in California. *Matern Child Health J*. 2000;4:251–259.
81. Egerter S, Braveman P, Marchi K. The timing of insurance coverage and use of prenatal care among low-income women. *Am J Public Health*. 2002;92:423–427.

82. Pearlin LI, Lieberman MA, Menaghan EG, Mullan JT. The stress process. *J Health Soc Behav.* 1981;22: 337–356.

83. Wagstaff A, Paci P, van Doorslaer E. On the measurement of inequalities in Health. *Soc Sci Med.* 1991; 33:545–557.

84. Mackenbach JP, Kunst AE. Measuring the magnitude of socioeconomic inequalities in health: an overview of available measures illustrated with two examples from Europe. *Soc Sci Med.* 1997;44:757–771.

85. Pamuk E. Social-class inequality in infant mortality in England and Wales from 1921 to 1980. *Eur J Popul.* 1988;4:1–21.

86. SUDAAN Version 8.0 User's Manual. Research Triangle Park, NC: Research Triangle Institute; 2001.

87. Skinner CN, Holt D, Smith TMF, eds. *Analysis of Complex Surveys.* New York: John Wiley & Sons; 1989.

88. Soobader M. *Effects of Income Inequality on Morbidity* (Dissertation). Ann Arbor, Mich: University Professors Program, Boston University, UMI; 1998.

89. Goldstein H. *Multilevel Statistical Models.* London, England: Arnold Publishers; 1999.

90. LeClere FB, Rogers RG, Peters K. Neighborhood social context and racial differences in women's heart disease and mortality. *J Health Soc Behav.* 1998;39: 91–107.

91. Cubbin C, Hadden WC, Winkleby MA. Neighborhood context and cardiovascular disease risk factors: the contribution of material deprivation. *Ethn Dis.* 2001;11:687–700.

92. Soobader MJ, LeClere FB. Aggregation and the measurement of income inequality: effects on morbidity. *Soc Sci Med.* 1999;48:733–744.

93. Robert SA. Community-level socioeconomic status effects on adult health. *J Health Soc Behav.* 1998;39: 18–37.

94. Diez Roux AV. Investigating neighborhood and area effects on health. *Am J Public Health.* 2001;91: 1783–1789.

95. Duncan C, Jones K, Moon G. Context, composition and heterogeneity: using multilevel models in health research. *Soc Sci Med.* 1998;46:97–117.

96. Historical Poverty Tables, Table 21. Number of Poor and Poverty Rate, by State: 1980 to 2001. Washington, DC: US Bureau of the Census; 2002. Available at: <http://www.census.gov/hhes/poverty/histpov/hstpov21.html>. Accessed December 22, 2003.

97. Rittenhouse DR, Braveman P, Marchi K. Improvements in prenatal insurance coverage and utilization of care in California: an unsung public health victory. *Matern Child Health J.* 2003;7:75–86.

98. Chavkin W, Wise PH. The data are in: health matters in welfare policy. *Am J Public Health.* 2002;92: 1392–1395.

99. Chavkin W, Romero D, Wise PH. State welfare reform policies and declines in health insurance. *Am J Public Health.* 2000;90:900–908.

100. Baumeister L, Hearst N. Why children's health is threatened by federal immigration policies. *West J Med.* 1999;171:58–61.

101. Fenton JJ, Moss N, Khalil HG, Asch S. Effect of California's proposition 187 on the use of primary care clinics. *West J Med.* 1997;166:16–20.

102. Pati S, Romero D, Chavkin W. Changes in use of health insurance and food assistance programs in medically underserved communities in the era of welfare reform: an urban study. *Am J Public Health.* 2002;92: 1441–1445.

103. Park LS, Sarnoff R, Bender C, Korenbrot C. Impact of recent welfare and immigration reforms on use of Medicaid for prenatal care by immigrants in California. *J Immigr Health.* 2000;2:5–22.

104. Krieger N, Chen JT, Ebel G. Can we monitor socioeconomic inequalities in health? A survey of US health departments' data collection and reporting practices. *Public Health Rep.* 1997;112:481–494.

105. Braveman P. *Monitoring Equity in Health and Health Care: A Policy-Oriented Approach in Low- and Middle-Income Countries.* Geneva: World Health Organization; 1998. Equity Initiative Document No. 3, WHO document WHO/CHS/HSS98.1.

106. Braveman P, Tarimo E. Social inequalities in health within countries: not only an issue for affluent nations. *Soc Sci Med.* 2002;54:1621–1635.

107. Metzler MM, Higgins DL, Becker CG, et al. Addressing urban health in Detroit, New York City, and Seattle through community-based participatory research partnerships. *Am J Public Health.* 2003;93: 803–811.

108. Global Equity Gauge Alliance. *The Equity Gauge: Concepts, Principles, and Guidelines.* Durban, SA: Health Systems Trust; 2003. 30 pp. Available at: <http://www.gega.org.za> or <http://www.hst.org.za>. Accessed December 22, 2003.

109. Braveman P, Cubbin C. Optimal SES indicators cannot be prescribed across all outcomes. *Am J Public Health.* 2003;93:12–13.

110. Daly MC, Duncan GJ, McDonough P, Williams DR. Optimal indicators of socioeconomic status for health research. *Am J Public Health.* 2002;92: 1151–1157.

111. Backlund E, Sorlie PD, Johnson NJ. The shape of the relationship between income and mortality in the United States: evidence from the National Longitudinal Mortality Study. *Ann Epidemiol.* 1996;6:12–20.

112. Whitehead M. *Equity and Ethics in Health.* Regional Committee for Europe, 44th session, 1994: Technical discussions: Annex. Copenhagen: World Health Organization Regional Office for Europe; 1995 (EUR/ICP/RPD 419).

113. Dahlgren G, Whitehead M. *Policies and Strategies to Promote Equity in Health.* Copenhagen: World Health Organization Regional Office for Europe; 1992.

114. De Kadt E, Tasca R. *Promoting Equity: A New Approach from the Health Sector.* Washington, DC: Pan American Health Organization (PAHO/WHO); 1993.



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# Socioeconomic Position and Hormone Replacement Therapy Use: Explaining the Discrepancy in Evidence From Observational and Randomized Controlled Trials

Debbie A Lawlor, PhD, MSc, MBChB, George Davey Smith, DSc, MD, and Shah Ebrahim, DM, MSc

The disparity between findings from observational studies and randomized controlled trials of the effects of hormone replacement therapy (HRT) on coronary heart disease (CHD)<sup>1–4</sup> has created considerable debate among researchers, practitioners, and post-menopausal women.<sup>5–10</sup> Observational studies have consistently found that use of HRT is protective against CHD, with a meta-analysis of observational studies yielding a summary relative risk for ever use of HRT of 0.56 (95% confidence interval [CI]=0.50, 0.61).<sup>4</sup> By contrast, recent randomized trials among women with established CHD and healthy women have found HRT to be associated with slightly increased risk of CHD or null effects. For example, the large Women's Health Initiative randomized trial found that the hazards ratio for CHD associated with being allocated to HRT was 1.29 (95% CI=1.02, 1.63), after 5.2 years of follow-up.<sup>1</sup>

A number of explanations have been suggested for these disparities. Although some researchers have suggested that the results of the trials were biased because of contamination, and in the case of the Women's Health Initiative, early termination of the arm assessing the effect of combined HRT, the consistency across a number of trials of a null effect makes these explanations unlikely. More plausible explanations are that women who participated in the trials were importantly different from those who participated in the observational studies or that the observational study results were confounded.<sup>5,8,9,11</sup>

Women in the Women's Health Initiative trial were older than the average age at which women take HRT and were more obese than the women who have been included in the observational studies.<sup>1</sup> These women may be more likely to have established atherosclerosis than younger and leaner women and therefore may be more

prone to the prothrombotic effects of HRT.<sup>5</sup> However, there was no evidence of interactions of treatment assignment with age, prior hormone use, or body mass index for any cardiovascular outcomes in the Women's Health Initiative.<sup>1,12</sup>

Of particular interest is whether the results in the observational studies are explained by residual confounding. Despite the fact that use of HRT is strongly socially patterned<sup>13</sup> and that socioeconomic status or position (SEP) is associated with CHD,<sup>14</sup> in many observational studies, adjustment for adult SEP has failed to have a marked impact on the HRT–CHD association.<sup>15</sup> However, residual confounding by SEP across the life course may be particularly important.<sup>16,17</sup> SEP in childhood is strongly associated with CHD risk, independent of adult SEP.<sup>14,18</sup> The association between adverse SEP in early life and CHD is in part mediated by adult behavioral and physiological risk factors.<sup>14</sup> Therefore, early life SEP could be an important confounder only if it were associated with HRT use and this association were independent of adult SEP and proximal adult risk factors that in part explain the association between early life SEP and CHD risk.

**Objectives.** We assessed the association between life-course socioeconomic status or position (SEP) and hormone replacement therapy (HRT).

**Methods.** We conducted a cross-sectional analysis of 4286 women aged 60 to 79 years.

**Results.** Women experiencing adverse socioeconomic circumstances across the life course were less likely to have used HRT. The associations of childhood socioeconomic measures with HRT use were independent of adult SEP, behavioral risk factors, and physiological risk factors for heart disease.

**Conclusions.** SEP from across the life course is associated with HRT use. Because the association between early life SEP and HRT is not fully explained by adult risk factors, residual confounding (which is not captured by adjustment for adult variables only) may explain some of the disparity between observational studies and randomized controlled trials in this area. (*Am J Public Health.* 2004; 94:2149–2154)

Our hypothesis was that the protective effect of HRT against CHD found in observational studies is explained at least in part by residual confounding related to early life socioeconomic factors that are not completely captured by adult risk factors. To assess this possibility, the primary aim of this study was to determine whether SEP in early life is associated with HRT use. Furthermore, we aimed to determine whether any association between early life SEP and HRT is fully explained by adult socioeconomic, behavioral, and physiological risk factors. If this association exists, then adequate adjustment for these adult risk factors should be sufficient to capture any potential confounding effect of early life SEP.

## METHODS

Data from the British Women's Heart and Health Study were used. Full details of the selection of participants and measurements used in the study have been previously reported.<sup>19,20</sup> Women aged 60 to 79 years were randomly selected from general practitioner lists in 23 British towns. A total of 4286 women (60% of those invited) participated, and baseline data were collected between



April 1999 and March 2001. Local ethics committee approvals were obtained.

Data on socioeconomic indicators across the life course included data on the longest held occupation of the participant's father during her childhood, childhood household amenities (i.e., bathroom, hot water, bedroom sharing, and car access), age at completion of full-time education, the longest held occupation of the participant and her spouse, adult housing tenure, car access, and pension arrangements. Childhood social class of each woman was based on her father's longest held occupation and adult social class was based on her husband's longest held occupation or her own longest held occupation for single women.<sup>21</sup> Adult and childhood social class were defined according to the registrar general's classification of occupations (I, II, III nonmanual, III manual, IV, and V, with I indicating professional occupations and V indicating manual unskilled occupations). We repeated the analyses using each woman's own occupation for married women who were not permanent housewives and declared an occupation (74%). The results from these analyses were essentially unaltered from those presented here although, because of reduced numbers, they were less precise. Most of the indicators of SEP were binary variables. For the main analyses, we dichotomized adult and childhood social class into nonmanual (I, II, III nonmanual) and manual (III manual, IV, V) groups to minimize any possible misclassification bias. Pension arrangements were dichotomized as state only or state plus other and adult housing tenure as local authority or other. Age at leaving full-time education was dichotomized around the median value (15 years).

Use of HRT, socioeconomic indicators, age at menopause, history of a hysterectomy or oophorectomy, smoking history, and physical activity were obtained from the self-completed questionnaire and/or the research nurse interview, to which women were requested to bring their current medications.<sup>19,20</sup> Blood samples were taken after a minimum 6-hour fast (except for patients using insulin treatment) using evacuated tubes and were used to determine insulin resistance and lipid levels.<sup>19,20</sup> Blood pressure, weight, height, and waist and hip circumference were measured

using standard procedures.<sup>19,20</sup> Coronary heart disease was considered to be present in any woman with a medical record of myocardial infarction (verified with respect to World Health Organization criteria<sup>22</sup>), angina, angioplasty or coronary artery bypass grafting, and/or any woman with a self-report of a physician diagnosis of these.<sup>19</sup>

Of the 4286 participants, 911 (21.0%) stated that they had ever (current and past) used HRT and 368 stated (8.6%) that they were currently using HRT. Of those who had ever used HRT, 43% did not know the name or type of preparation (or gave only vague details such as "tablets" or "patches"), 32% used a combined estrogen–progestogen preparation, 18% used unopposed estrogen, and 7% were not actually using HRT (e.g., tibolone, raloxifene). Of current users, only 9% did not know the name or type, 40% were taking a combined preparation, 39% were taking unopposed estrogen, and 12% were not actually using HRT. Those who had not or were not actually using HRT were categorized as not using HRT; those who did not know the type of HRT that they had used were all assumed to have used HRT. Thus, in the main analysis, 848 women (19.8%) were categorized as ever using HRT, and 323 (7.5%) were categorized as currently using HRT. In a sensitivity analysis, all women who defined themselves as ever ( $n=911$ ) or currently taking HRT ( $n=368$ ) were defined as exposed. The results of this sensitivity analysis did not differ substantively from those presented here. All of those who were currently using unopposed estrogen had had a hysterectomy.

### Statistical Analysis

Age-adjusted prevalences and 95% confidence intervals for each indicator of SEP are presented for all women in the study and for current, past, and never users of HRT. Multiple logistic regression was used to assess the associations of each individual indicator of SEP with HRT use. For each indicator, 3 logistic regression models were undertaken. In the first, crude associations were assessed. In the second, childhood indicators were adjusted for age (entered as a continuous variable) and adult indicators of SEP (adult social class, car access, local authority housing, and pension arrangements entered as full categorical [indi-

cator] variables, except car access, which is binary). In the third model, all other adult behavioral and physiological risk factors that might capture any association between childhood SEP and HRT use were added to the age- and adult SEP-adjusted model. In this model, systolic blood pressure, high-density lipoprotein cholesterol, triglyceride levels (logged), body mass index, waist-to-hip ratio, and age of menopause were all entered as continuous variables; smoking and physical activity were entered as indicator variables. Homeostasis model assessment scores (insulin resistance) were not estimated for individuals with diabetes, and an indicator variable representing insulin resistance–diabetes was calculated as fifths of homeostasis model assessment scores for nondiabetics together with a sixth category for patients with diabetes.<sup>18</sup> Covariates were decided a priori, rather than being data driven, for example, by stepwise regression.<sup>23</sup> Uptake of HRT has increased over recent decades, and it is possible that as HRT use becomes more widespread, any associations with SEP will be weaker in younger birth cohorts. To assess this possibility, age was dichotomized as 60 to 69 years and 70 to 79 years, and likelihood ratio tests were used to determine statistical evidence for any interactions between age and socioeconomic indicators in their association with HRT use.

In addition to assessing the association of each individual life-course indicator of SEP, we assessed the cumulative effect of life-course SEP by generating a life-course SEP score from the 10 dichotomized indicators. Two scores were developed, one in which equal weight was given to each indicator and another in which the inverse of prevalence weights was used. The first score has the advantage of being easy to understand because the score gives the actual number of adverse indicators. The score ranged from 0 (most advantaged position across the life course) to 10 (most disadvantaged position across the life course). Because there were very small numbers in the 0 category ( $n=77$ ) and in the 10 category ( $n=57$ ), the 0 category was combined with the 1 category and the 10 category with the 9 category. The second score in which each indicator was weighted by the inverse of its prevalence gave the greatest weight to adverse indicators that were least

**TABLE 1—Prevalence of Life-Course SEP Indicators Among All Study Participants and By Use of Hormone Replacement Therapy: British Women's and Heart and Health Study, 1999–2001**

|   | % With Indicator (95% CI)          |                             |                            |                             |                                |
|---|------------------------------------|-----------------------------|----------------------------|-----------------------------|--------------------------------|
|   | No. With Complete Data on Variable | All Participants (n = 4286) | Nonusers of HRT (n = 3438) | Past Users of HRT (n = 525) | Current Users of HRT (n = 323) |
| Childhood SEP indicator                   |                                    |                             |                            |                             |                                |
| Manual social class                       | 4286                               | 80.0 (78.8, 81.2)           | 81.4 (80.0, 82.6)          | 76.0 (72.1, 79.6)           | 72.1 (66.9, 77.9)              |
| No bathroom in house                      | 4052                               | 38.7 (37.2, 40.2)           | 41.0 (39.3, 42.7)          | 31.1 (27.1, 35.3)           | 28.3 (23.4, 33.7)              |
| No hot water in house                     | 4022                               | 35.3 (33.8, 36.8)           | 37.3 (35.6, 38.9)          | 29.1 (25.1, 33.2)           | 24.9 (20.2, 30.1)              |
| Shared bedroom                            | 3994                               | 52.7 (51.1, 54.3)           | 54.0 (52.3, 55.8)          | 48.4 (44.0, 52.9)           | 46.3 (40.7, 52.0)              |
| No car access                             | 3936                               | 82.6 (81.4, 83.8)           | 84.1 (82.7, 85.3)          | 77.6 (73.6, 81.2)           | 76.2 (71.1, 80.8)              |
| Completed full-time education by age 15 y | 3938                               | 88.5 (87.5, 89.5)           | 89.3 (88.2, 90.4)          | 87.2 (84.0, 90.0)           | 82.5 (77.8, 86.6)              |
| Adult SEP indicator                       |                                    |                             |                            |                             |                                |
| Manual social class                       | 4286                               | 57.3 (55.8, 58.8)           | 58.7 (57.1, 60.4)          | 49.7 (45.4, 50.1)           | 54.2 (48.6, 59.7)              |
| Local authority housing                   | 4070                               | 13.6 (12.6, 14.7)           | 15.0 (13.8, 16.2)          | 9.1 (6.8, 11.9)             | 7.30 (4.7, 10.8)               |
| No car access                             | 4069                               | 28.9 (27.5, 30.3)           | 32.5 (30.9, 34.2)          | 15.1 (12.1, 18.4)           | 14.6 (10.9, 19.0)              |
| State pension only                        | 3828                               | 28.8 (27.4, 30.3)           | 30.7 (29.1, 32.4)          | 23.2 (19.5, 27.2)           | 18.4 (14.2, 23.3)              |

Note. CI = confidence interval; HRT = hormone replacement therapy; SEP = socioeconomic position.

prevalent. The resulting weighted score was highly positively skewed, with a range from 0 to 28.9. The 2 composite socioeconomic scores were strongly correlated (Spearman rank correlation coefficient=0.95) and showed identical linear trends in their association with HRT use. Results for the unweighted score only are therefore presented. Likelihood ratio tests were used to assess departure from linearity in the associations between the scores and HRT use.

## RESULTS

Most women provided data on each of the socioeconomic indicators, with details of pension arrangements being the variable with the greatest amount of missing data (3828 [89%] women provided data for this indicator). There were no significant differences in SEP indicators or risk factor distributions between women with complete data on all indicators and those with some missing data (all *P* values >.25). Table 1 shows the prevalence of each indicator of SEP for all study participants and by HRT use.

Table 2 shows the results of logistic regression analyses for the associations of each indicator of childhood SEP with HRT use. In general, childhood indicators of SEP were more strongly associated with HRT use than adult indicators, although the single strongest asso-

ciation was with adult car access. All indicators of childhood SEP were associated with reduced odds of ever and current use of HRT even with adjustment for adult SEP and a full range of adult behavioral and physiological risk factors, although the association of completing full-time education before age 15 years with ever use of HRT and of sharing a bedroom with current HRT did not reach conventional levels of 5% statistical significance. There was no statistical evidence of any interactions between age and any indicators of SEP in their associations with HRT use (all *P* values >.3). There was a cumulative effect of life-course SEP on HRT use as demonstrated by strong linear trends across the composite score (Table 3) for both ever and current use of HRT.

Among the 3496 women with complete data on all indicators of SEP, 514 (15.5%) women had CHD, and the prevalence of CHD did not differ between women with these complete data and all women in the cohort (*P*=.23). Table 4 shows the association of ever and current use of HRT with prevalent CHD and the effect on this association of adjustment for life-course SEP and all other adult risk factors, and also the effect on this association of adjustment for just adult SEP (all indicators of adult SEP) and adult risk factors. In crude analyses, both ever use and current use of HRT are associated with a protective effect.

When adjustment is made for life-course SEP (using the cumulative life-course socioeconomic score) and adult behavioral and physiological risk factors, both of these associations are reversed to slight increases in risk (although both are nonsignificant at the conventional 5% level). When adjustment is made just for all indicators of adult SEP together with adult behavioral and physiological risk factors, the results are attenuated but still suggest some benefit of HRT (although again not significant at the conventional 5% level).

## DISCUSSION

In this cohort of British women aged 60 to 79 years, adverse socioeconomic indicators from across the life course were associated with use of HRT. Indicators of socioeconomic deprivation in childhood were associated with a reduced odds of using HRT, and these associations were independent of adult SEP, behavioral risk factors, and physiological risk factors. Because childhood SEP is independently associated with CHD,<sup>14,18</sup> our findings suggest that the protective effect of HRT use found in observational studies may be attributable to residual confounding. The logic behind this argument starts from the conflicting evidence between observational studies and trials. Well-conducted trials should not be affected by confounding, and therefore residual

**TABLE 2—Associations Between Use of Hormone Replacement Therapy and Indicators of SEP Across the Life Course, With Adjustment for Potential Confounding and Mediating Variables: British Women's Heart and Health Study (n = 3496),<sup>a</sup> 1999–2001**

|   | OR (95% CI) for Ever Use of HRT Compared With Never Use |   |  | OR (95% CI) for Current Use of HRT Compared With Never Use |   |  |
|---|---|---|--|--|---|--|
|   | Crude   | Adjusted for Age and Adult/Childhood SEP <sup>b</sup> | Adjusted for Age, Adult/Childhood SEP, and Other Adult Risk Factors <sup>c</sup> | Crude  | Adjusted for Age and Adult/Childhood SEP <sup>b</sup> | Adjusted for Age, Adult/Childhood SEP, and Other Adult Risk Factors <sup>c</sup> |
| <b>Childhood SEP indicator</b>            |   |   |  |  |   |  |
| Manual social class                       | 0.63 (0.52, 0.76)                                       | 0.72 (0.59, 0.88)                                     | 0.72 (0.58, 0.89)  | 0.60 (0.46, 0.79)  | 0.67 (0.51, 0.89)                                     | 0.66 (0.49, 0.89)  |
| No bathroom in house                      | 0.68 (0.57, 0.81)                                       | 0.73 (0.61, 0.88)                                     | 0.73 (0.60, 0.88)  | 0.67 (0.52, 0.87)  | 0.66 (0.50, 0.88)                                     | 0.68 (0.51, 0.91)  |
| No hot water in house                     | 0.71 (0.59, 0.85)                                       | 0.79 (0.66, 0.96)                                     | 0.78 (0.64, 0.95)  | 0.66 (0.50, 0.87)  | 0.70 (0.53, 0.93)                                     | 0.69 (0.51, 0.93)  |
| Shared bedroom                            | 0.77 (0.66, 0.91)                                       | 0.84 (0.71, 1.00)                                     | 0.84 (0.70, 1.00)  | 0.77 (0.61, 0.97)  | 0.81 (0.63, 1.05)                                     | 0.79 (0.61, 1.03)  |
| No car access                             | 0.67 (0.55, 0.82)                                       | 0.74 (0.60, 0.91)                                     | 0.73 (0.58, 0.91)  | 0.70 (0.53, 0.93)  | 0.71 (0.53, 0.96)                                     | 0.68 (0.50, 0.92)  |
| Completed full-time education by age 15 y | 0.72 (0.56, 0.91)                                       | 0.83 (0.65, 1.07)                                     | 0.80 (0.61, 1.02)  | 0.60 (0.43, 0.82)  | 0.68 (0.48, 0.96)                                     | 0.65 (0.45, 0.93)  |
| <b>Adult SEP indicator</b>                |   |   |  |  |   |  |
| Manual social class                       | 0.71 (0.61, 0.83)                                       | 0.90 (0.76, 1.08)                                     | 0.93 (0.77, 1.12)  | 0.86 (0.68, 1.08)  | 1.16 (0.90, 1.50)                                     | 1.30 (0.99, 1.71)  |
| Local authority housing                   | 0.61 (0.46, 0.80)                                       | 0.70 (0.52, 0.95)                                     | 0.76 (0.55, 1.05)  | 0.56 (0.36, 0.88)  | 0.71 (0.45, 1.13)                                     | 0.81 (0.49, 1.32)  |
| No car access                             | 0.51 (0.41, 0.63)                                       | 0.55 (0.44, 0.69)                                     | 0.58 (0.45, 0.74)  | 0.57 (0.41, 0.79)  | 0.67 (0.48, 0.95)                                     | 0.74 (0.51, 1.06)  |
| State pension only                        | 0.67 (0.55, 0.81)                                       | 0.78 (0.64, 0.97)                                     | 0.81 (0.65, 1.01)  | 0.59 (0.44, 0.81)  | 0.74 (0.54, 1.02)                                     | 0.76 (0.55, 1.07)  |

Note. OR = odds ratio; CI = confidence interval; HRT = hormone replacement therapy; SEP = socioeconomic position.

<sup>a</sup>Number for whom complete data were available on all SEP indicators and all covariates included in the final model.

<sup>b</sup>Childhood SEP indicators are adjusted for adult SEP indicators (social class, housing tenure, car access, pension arrangements); adult SEP indicators are adjusted for childhood SEP indicators (social class, bathroom in house, hot water in house, bedroom sharing, car access, and age at leaving full-time education).

<sup>c</sup>Other adult risk factors: systolic blood pressure, high-density lipoprotein cholesterol, triglyceride levels, diabetes, body mass index, waist-to-hip ratio, age at menopause, hysterectomy/oophorectomy, physical activity, and smoking.

**TABLE 3—Association of Cumulative Life-Course SEP Score with Ever With Current Use of Hormone Replacement Therapy: British Women's and Heart and Health Study (n = 3496), 1999–2001**

| Cumulative Life-Course SEP Score (No. of Adverse Indicators) |     | OR (95% CI) of Ever Use HRT Compared With Never Use |   | OR (95% CI) of Current Use HRT Compared With Never Use |   |
|--|-----|---|---|--|---|
|  |     | Crude Association                                   | Fully Adjusted Association <sup>a</sup> | Crude Association                                      | Fully Adjusted Association <sup>a</sup> |
| 0-1  | 425 | 1.00  | 1.00                                    | 1.00   | 1.00                                    |
| 2  | 393 | 0.91 (0.66, 1.26)                                   | 0.93 (0.66, 1.31)                       | 0.97 (0.63, 1.50)                                      | 0.94 (0.59, 1.49)                       |
| 3  | 500 | 0.88 (0.65, 1.19)                                   | 0.91 (0.66, 1.27)                       | 0.81 (0.54, 1.24)                                      | 0.78 (0.49, 1.22)                       |
| 4  | 517 | 0.76 (0.56, 1.03)                                   | 0.73 (0.52, 1.02)                       | 0.66 (0.43, 1.02)                                      | 0.59 (0.37, 0.95)                       |
| 5  | 455 | 0.69 (0.50, 0.96)                                   | 0.72 (0.51, 1.02)                       | 0.56 (0.35, 0.89)                                      | 0.54 (0.33, 0.90)                       |
| 6  | 457 | 0.54 (0.39, 0.76)                                   | 0.52 (0.34, 0.75)                       | 0.46 (0.28, 0.76)                                      | 0.48 (0.29, 0.82)                       |
| 7  | 353 | 0.45 (0.30, 0.67)                                   | 0.41 (0.26, 0.65)                       | 0.53 (0.30, 0.94)                                      | 0.56 (0.30, 1.03)                       |
| 8  | 226 | 0.26 (0.15, 0.47)                                   | 0.23 (0.12, 0.44)                       | 0.34 (0.15, 0.77)                                      | 0.25 (0.10, 0.65)                       |
| 9-10   | 170 | 0.23 (0.11, 0.49)                                   | 0.25 (0.12, 0.55)                       | 0.23 (0.07, 0.75)                                      | 0.27 (0.08, 0.92)                       |
| P linear trend   |     | <.001   | <.001                                   | <.001  | <.001                                   |
| P nonlinearity   |     | .49   | .39                                     | .97  | 0.93                                    |

Note. OR = odds ratio; CI = confidence interval; HRT = hormone replacement therapy; SEP = socioeconomic position.

<sup>a</sup>Fully adjusted association: systolic blood pressure, high-density lipoprotein cholesterol, triglyceride levels, type 1 diabetes, body mass index, waist-to-hip ratio, age at menopause, hysterectomy/oophorectomy, physical activity, and smoking.

confounding in the observational studies is a persuasive explanation for the difference. Our belief is that observational studies did not adequately adjust for SEP from across the life

course. This belief is supported by the findings in this study in the following ways. First, the fact that childhood SEP is associated with HRT use, independent of adult SEP, behav-

ioral risk factors, and physiological risk factors, suggests that adjusting for these proximal risk factors will not take fully into account the effect of early life SEP on HRT use. Second, we have shown a cumulative effect of SEP from across the life course, indicating that not only does life-course SEP need to be accounted for but that a single measure of SEP also is unlikely to be adequate. Finally, although these cross-sectional data are not ideal for assessing HRT–CHD associations, our analysis of this association also supports our hypothesis. The crude associations were consistent with previous observational studies.<sup>4</sup> When we adjusted for 4 indicators of adult SEP and all adult risk factors, the association attenuated but still suggested some protective effect; this adjusted result was consistent with adjusted results in previous observational studies.<sup>4</sup> When we adjusted for SEP across the life course, together with adult risk factors, HRT use was associated with a slightly increased risk of CHD, consistent with evidence from randomized controlled trials.<sup>1</sup>

Our response rate (60%) is moderate but consistent with other baseline data collected in large epidemiological surveys.<sup>24</sup> Respon-

**TABLE 4—Association of Ever and Current Use of Hormone Replacement Therapy with Coronary Heart Disease, Adjustment for Life-Course SEP, and Other Adult Risk Factors and Adjustment Just for Adult Indicators of SEP and Other Adult Risk Factors: British Women's and Heart and Health Study (n = 3496), 1999–2001**

|                                     | OR (95% CI) of CHD |  |   |
|-------------------------------------|--------------------|--|---|
|                                     | Crude              | Adjusted for Life-Course Cumulative SEP Score and Adult Behavioral and Physiological Risk Factors <sup>a</sup> | Adjusted for Adult Indicators of SEP and Adult Behavioral and Physiological Risk Factors <sup>b</sup> |
| Ever vs never use of HRT            | 0.66 (0.53, 0.83)  | 1.09 (0.81, 1.45)  | 0.87 (0.67, 1.13)   |
| Current vs past or never use of HRT | 0.74 (0.54, 1.02)  | 1.15 (0.78, 1.70)  | 0.84 (0.59, 1.15)   |

Note. OR = odds ratio; CI = confidence interval; HRT = hormone replacement therapy; SEP = socioeconomic position; CHD = coronary heart disease.

<sup>a</sup>Adjusted for life-course cumulative SEP score, systolic blood pressure, high-density lipoprotein cholesterol, triglyceride levels, type 1 diabetes, body mass index, waist-to-hip ratio, age at menopause, hysterectomy/oophorectomy, physical activity, smoking, and low-fat diet.

<sup>b</sup>Adjusted for adult social class, car access as an adult, housing tenure as an adult, pension arrangements, systolic blood pressure, high-density lipoprotein cholesterol, triglyceride levels, type 1 diabetes, body mass index, waist-to-hip ratio, age at menopause, hysterectomy/oophorectomy, physical activity, and smoking.

ders were younger and less likely to have had a stroke than nonresponders, although CHD prevalence was similar among responders and nonresponders.<sup>19</sup> The social class distribution of the British Women's Heart and Health Study is similar to that found for the 1991 census for England and Wales (57% manual social class in British Women's Heart and Health Study vs 55% of women aged 65 and older in the 1991 census), which provides some evidence to suggest that our sample is not affected by selection bias based on SEP.

Our study is cross-sectional and so may be affected by reverse causality and survivor bias. In the association of early life SEP with HRT use, reverse causality is not an issue, and for adult SEP it is difficult to imagine HRT use having an effect on socioeconomic circumstances. Our results for the association between HRT and CHD are consistent with those from prospective cohort studies.<sup>4</sup> Survivor bias would be important for the association between childhood SEP and HRT use if the association between these 2 among women who died prematurely was either null or in the opposite direction to that presented here (i.e., women from poor SEP were more likely to use HRT). Although this cannot be ruled out, it seems unlikely.

We have no information on how women who were prescribed HRT were screened by

their physicians, and it is likely that confounding by indication also will have biased previous observational studies.<sup>11</sup> That is, doctors may have been less likely to prescribe HRT to women who were at greater risk of CHD because of obesity, high blood pressure, or other CHD risk factors. To some extent, this may be controlled for by adjustment for these adult risk factors, but adjustment for life-course SEP may capture this effect to a greater extent by reflecting these exposures over the life course. However, our study is not suitable for fully examining the importance of confounding by indication in the HRT–CHD associations.

Our study cohort consisted of women who were born in Great Britain between 1919 and 1940, and the results may not be generalizable to women from other countries and those from different birth cohorts. For example, a study of women born in 1946 in Great Britain found no association between childhood SEP and HRT use.<sup>25</sup> Because observational studies of the protective effect of HRT were largely conducted on cohorts born before the 1940s,<sup>4</sup> our results have relevance for the current debate about the disparities between observational and trial results but do not necessarily mean that for all populations childhood SEP will be associated with HRT use.

Data on HRT use were confirmed by review of medication among current users and by self-report for past users, which may have led to some misclassification for the ever use category. Over two fifths (43%) of women who stated that they had ever used HRT were unable to name the preparation, and 4% who named their preparation were using a related but nonhormonal preparation such as raloxifene. However, the results of this study were consistent for current use of HRT (where actual preparations were checked at the interview) and ever use (where some misclassification is likely). Furthermore, most other observational studies have relied on self-report of HRT use only and are likely to have included some women who were using nonhormonal preparations, as in this study. Finally, our results for the association between HRT use and CHD are consistent with previous prospective studies that have used either self-report or medical record data.<sup>4</sup>

We have not assessed all factors that may affect HRT use and CHD risk and may thus have confounded the associations presented in earlier observational studies. For example, ethnicity may determine HRT use and is associated with CHD risk. Over 99% of women in this study were White; we were therefore unable to determine the effect of ethnicity on HRT use in this study.

Childhood SEP may affect future use of HRT by means of a number of mechanisms, including the individual's attitudes toward health, preventive treatment, and natural physiological processes such as menopause and aging, gained from their parent's attitudes toward these; the ability to access health care; and discrimination based on patient characteristics. Although the actual mechanisms are not discernible from our data, it is plausible that adult attitudes toward the use of HRT and access to HRT are formed by SEP in earlier life.

The importance of our results is in the contribution that they make to the debate concerning disparities in observational and trial evidence. We believe that these results support the trial evidence of no protective effect. Our results also have general implications for observational epidemiological studies. Future observational studies, in this and other areas, should aim to collect (even retrospectively) in-



formation on socioeconomic circumstances from across the life course to be able to adjust as fully as possible for potential confounding factors. Sensitivity analyses to assess the possibility of residual confounding should also become routine practice in observational epidemiology.<sup>26,27</sup> In addition, specificity of association should be considered.<sup>26,28</sup> As long ago as 1986, Diana Petitti pointed out in observational studies that HRT was apparently equally protective against accidental and violent deaths as it was against death resulting from cardiovascular disease.<sup>29</sup> She pointed out that given the lack of any biologically plausible link between HRT and these external causes of death, both associations should be considered to be attributable to residual confounding.<sup>29</sup> We have discussed approaches to strengthening inferences from observational studies in detail elsewhere.<sup>30,31</sup> ■

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### Contributors

All authors developed the study aim. S. Ebrahim and D.A. Lawlor managed data collection, storage, and cleaning for the British Women's Heart and Health Study. D.A. Lawlor undertook the analysis and coordinated writing of the article. All authors contributed to writing the article. D.A. Lawlor acted as guarantor.

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### Human Participant Protection

The British Women's Heart and Health Study has ethics approval from UK local ethics committees in each town in which the study participants reside.

### References

- Writing committee for the Women's Health Initiative randomized controlled trial. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results from the Women's Health Initiative randomized controlled trial. *JAMA*. 2002;288:321–333.
- Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women: Heart and Estrogen/progestin Replacement Study (HERS) Research Group. *JAMA*. 1998;280:605–613.
- Grady D, Rubin SM, Petitti DB, et al. Hormone therapy to prevent disease and prolong life in postmenopausal women. *Ann Intern Med*. 1992;117:1016–1037.
- Stampfer MJ, Colditz GA. Estrogen replacement therapy and coronary heart disease: a quantitative assessment of the epidemiologic evidence. *Prev Med*. 1991;20:47–63.
- Michels KB. Hormone replacement therapy in epidemiologic studies and randomized clinical trials: are we checkmate? *Epidemiology*. 2003;14:3–5.
- Dentzer S. Science, public health, and public awareness: lessons from the Women's Health Initiative. *Ann Intern Med*. 2003;138:352–353.
- Kirschstein R. Menopausal hormone therapy: summary of a scientific workshop. *Ann Intern Med*. 2003;138:361–364.
- Piantadosi S. Larger lessons from the Women's Health Initiative. *Epidemiology*. 2003;14:6–7.
- Whittemore AS, McGuire V. Observational studies and randomized trials of hormone replacement therapy: what can we learn from them? *Epidemiology*. 2003;14:8–10.
- Rayner C. Why it's worth taking the risk. *Guardian*. Thursday, July 11, 2002:7–9.
- Vandenbroucke JP. When are observational studies as credible as randomised controlled trials? *Lancet*. 2004;363:1728–1731.
- The WHI Steering Committee and Writing Group. Risks of postmenopausal hormone replacement therapy. *JAMA*. 2002;288:2823–2824.
- Barrett-Connor E. Hormone replacement therapy. *BMJ*. 1998;317:457–461.
- Davey Smith G, Ben-Shlomo Y, Lynch J. Life course approaches to inequalities in coronary heart disease risk. In: Stansfeld S, Marmot M, eds. *Stress and the Heart*. London, UK: BMJ Books; 2002:20–49.
- Grodstein F, Manson JE. Relationship between hormone replacement therapy, socioeconomic status, and coronary heart disease. *JAMA*. 2003;289:44–45.
- Davey Smith G. Reflections on the limitations to epidemiology. *J Clin Epidemiol*. 2001;54:325–331.
- Krieger N. Postmenopausal hormone therapy. *N Engl J Med*. 2003;348:2363–2364.
- Claussen B, Davey Smith G, Thelle D. Impact of childhood and adulthood socioeconomic position on cause specific mortality: the Oslo Mortality Study. *J Epidemiol Community Health*. 2003;57:40–45.
- Lawlor DA, Bedford C, Taylor M, Ebrahim S. Geographic variation in cardiovascular disease, risk factors and their control in older women: British Women's Heart and Health Study. *J Epidemiol Community Health*. 2003;57:134–140.
- Lawlor DA, Ebrahim S, Davey Smith G. Socioeconomic position in childhood and adulthood and insulin resistance: cross sectional survey using data from the British women's heart and health study. *BMJ*. 2002;325:805–807.
- Marmot M, Brunner E. CHD risk among women: Whitehall II and other studies. In: Sharp I, ed. *Coronary Heart Disease: Are Women Special?* London, UK: National Forum for Coronary Heart Disease Prevention; 1994:57–70.
- Report of the joint international society and federation of cardiology/World Health Organisation Task force on standardisation of clinical nomenclature. Nomenclature and criteria for diagnosis of ischaemic heart disease. *Circulation*. 1979;59:607–609.
- Hernan MA, Hernandez-Diaz S, Werler MM, Mitchell AA. Causal knowledge as a prerequisite for confounding evaluation: an application to birth defects epidemiology. *Am J Epidemiol*. 2002;155:176–184.
- Erens B, Primatesta P. *Health Survey for England 1998: Cardiovascular Disease*. London, UK: The Stationery Office; 1999.
- Kuh D, Hardy R, Wadsworth M. Social and behavioural influences on the uptake of hormone replacement therapy among younger women. *Br J Obstet Gynaecol*. 2000;107:731–739.
- Davey Smith G, Ebrahim S. Data dredging, bias, or confounding. *BMJ*. 2002;325:1437–1438.
- Greenland S. Basic methods for sensitivity analysis of biases. *Int J Epidemiol*. 1996;25:1107–1116.
- Weiss NS. Can the "specificity" of an association be rehabilitated as a basis for supporting a causal hypothesis? *Epidemiology*. 2002;13:6–8.
- Petitti DB, Perlman JA, Sidney S. Postmenopausal estrogen use and heart disease. *N Engl J Med*. 1986;315:131–132.
- Davey Smith G, Ebrahim S. "Mendelian randomization": can genetic epidemiology contribute to understanding environmental determinants of disease? *Int J Epidemiol*. 2003;32:1–22.
- Lawlor DA, Davey Smith G, Bruckdorfer KR, Kundu D, Ebrahim S. Those confounded vitamins: what can we learn from the differences between observational versus randomized trial evidence? *Lancet*. 2004;363:1724–1727.

# Unemployment and Early Cause-Specific Mortality: A Study Based on the Swedish Twin Registry

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Loss of a job has pronounced negative effects on an individual's life situation.<sup>1–10</sup> Several studies have shown an increased risk of early mortality among the unemployed, but the nature of this association is not clear.<sup>1,2,6–8,11–17</sup> To better understand the relationship between unemployment and mortality, one should consider the meaning of employment beyond earning a living, including the impact on lifestyle, self-image, social integration, and psychological well-being.<sup>3,18</sup> Unemployment entails an increase in general distress, anxiety, and depression and a decrease in activity,<sup>4,7,8,17</sup> which in the long run may increase the risk of early death.

The excess risk of mortality associated with unemployment has been attributed mainly to external causes of death, including suicide and undetermined causes<sup>1,2,4,5,7–9,12,13,16,19,20</sup>; diseases of the circulatory system<sup>1,21–23</sup>; and lung cancer.<sup>2,10,12,21,24</sup>

Mortality rates seem to increase with the duration of unemployment<sup>1,12,17</sup> and are higher for the unemployed than the employed in all social classes.<sup>6,20</sup> Several studies have suggested that unemployment has a direct effect on health over and above the effects of socioeconomic status, poverty, risk behaviors, or prior ill health.<sup>1–3,6,16,20,21,25</sup> Unemployment has also been suggested as more important than other socioeconomic variables as a risk factor for premature death.<sup>20</sup>

It has been suggested that when unemployment rates are low, those with impaired health status are more likely than healthy people to become unemployed.<sup>1,26,27</sup> In Sweden, unemployment was by all international standards low (2%–4%) between 1950 and 1990 but increased to about 8% during the 1990s.<sup>18</sup>

Individuals actively employed may have better health status on average than those outside the workforce because healthy individuals are more likely to enter the workforce. Employees with impaired health or certain risk indicators, such as high alcohol consumption, may also be

more likely to lose their jobs. The effect of unemployment on mortality could therefore also be attributable to confounding from other risk factors.<sup>11,12,21,24,25,28</sup> In earlier studies, analyses took into account demographic and social characteristics to some extent,<sup>2,11,16,21,29</sup> while adjustment for potential confounding from other factors was limited.

We have previously shown an increased risk of overall early mortality (i.e., before 70 years of age) among individuals who experienced unemployment.<sup>29</sup> The results were similar for women and men, which could reflect that losing or holding a job is equally important among both Swedish men and women. In Denmark, which has a similar labor market, relative risks of about the same magnitude among unemployed men and women were reported.<sup>9</sup>

In our study, we extended the analyses to specific causes of death, accounting for potential confounding from social, behavioral, health, and personality characteristics. We also studied the relation between unemployment and total mortality using pairs of twins, one of whom had experienced employment while the other had not, thus controlling for genetic factors and for social and environmental conditions during childhood and youth. An additional aim was to analyze to what extent the effect of unemployment was modified by the presence of other

**Objectives.** We investigated the association between unemployment and early cause-specific mortality to determine whether the relationship was modified by other risk indicators.

**Methods.** Female and male twins (n = 20 632) were followed with regard to mortality from 1973 through 1996. Questionnaire data from 1973 were used to obtain information on experience of unemployment and on social, behavioral, health, and personality characteristics.

**Results.** Unemployment was associated with an increased risk of suicide and death from undetermined causes. Low education, personality characteristics, use of sleeping pills or tranquilizers, and serious or long-lasting illness tended to strengthen the association between unemployment and early mortality.

**Conclusions.** An increased risk of death from external causes implies a need for support for those experiencing unemployment, particularly susceptible individuals. (*Am J Public Health.* 2004;94:2155–2161)

risk indicators of early mortality. The study was performed with information from the Swedish twin registry.<sup>30</sup>

## METHODS

### Study Population

The study population comprised 18 516 women and 18 020 men, constituting in principle all same-sex twins born in Sweden between 1926 and 1958.<sup>30</sup> Data were based on a 1973 mailed questionnaire, which was answered by 15 683 women and 14 287 men (response rate = 85% for women and 79% for men), and on information from the Swedish Causes of Death Registry. All responders with a job title indicating gainful employment were included in the study (i.e., 9500 women and 11 132 men). Housewives, students, retired individuals, persons on disablement pension, and persons liable for military service at baseline were excluded from the analyses.

### Unemployment

A short occupational history, including history of unemployment, was recorded in the 1973 questionnaire that included unemployment. Information about unemployment was based on answers to the following questions: "Are you employed at the present time?"; "Are

you now or have you ever been unemployed?"; "For how long have you been unemployed?"

In our main analyses, we compared ever unemployed (875 women and 1309 men) with never unemployed according to the 1973 data. In some analyses, we distinguished between "short-term" and "long-term" unemployment. Short-term unemployment was defined as being unemployed in 1973 and experiencing less than 1 year of lifetime unemployment (144 women and 185 men). Long-term unemployment was defined as being unemployed for 1 year or more altogether during the life course (260 women and 311 men). Small numbers precluded separate analyses of mortality among the short- and long-term unemployed.

### Social, Behavioral, Health, and Personality Factors

In the analyses, the following social, behavioral, health, and personality factors were considered: marital status, children, education, smoking and alcohol habits, use of sleeping pills and tranquilizers, stress, shift work, personality factors, long-lasting/serious illness, and socioeconomic status. The selection of potential confounding factors originates from our previous study.<sup>29</sup> The variables were dichotomized; "exposed" categories are listed in Table 1 (reference groups were those "not exposed.") The reference category for marital status was married and cohabitant; smokers and former smokers were compared with never smokers. Alcohol consumption was analyzed by consumed grams of absolute alcohol per month.<sup>30</sup> No alcohol consumption and consumption of more than 250 g of alcohol per month were compared with consumption of 1 to 250 g of alcohol per month.

"Stressful life" was defined by the question, "Do you experience your everyday life as being very stressful?" The personality factors "instability" and "introversion-extraversion" were based on 9 items each selected from the "neuroticism" and "extraversion" dimensions of the Eysenck personality inventory; these short scales were developed to be used in comprehensive questionnaire investigations.<sup>31,32</sup> Unstable personality (>4 points) was compared with stable personality (<5 points), and extravert personality (>4 points) with introvert personality (<5 points). The question "Have you ever had any long-term or serious illness?" was used

as an indicator of ill health. Unskilled/skilled workers and low-level white-collar workers were compared with medium- and high-level white-collar workers.

### Mortality

All individuals of the study population were followed regarding mortality from January 1, 1973, to December 31, 1996. For deaths that occurred between 1973 and 1986, specific causes of death were taken from the *International Classification of Diseases, Eighth Revision (ICD-8)*<sup>33</sup>; for deaths between 1987 and 1996, the *International Classification of Diseases, Ninth Revision (ICD-9)*<sup>34</sup> was used. The 2 revisions were then harmonized. The following underlying causes of death were analyzed: malignant neoplasms (ICD-8 codes 140–209); malignant neoplasms of trachea, bronchus, and lung (162); diseases of the circulatory system (390–458); ischemic heart diseases (410–414); injuries, poisoning, and other external causes (E800–E999); suicide (E950–E959); injury, undetermined whether accidentally or purposely inflicted (external undetermined cause; E980–E989); other diseases (001–139, 210–389, 460–799); and alcohol-related diseases (303, 571, 577).

### Statistical Analysis

Differences in social, behavioral, health, and personality characteristics between individuals with and without experience of unemployment were analyzed with adjustment for age (5-year intervals), and the statistical precision was indicated by 95% confidence intervals.

We estimated the mortality rate ratio, referred to here as the relative risk, together with 95% confidence intervals by Cox proportional hazards regression,<sup>35</sup> using PHREG software (SAS 6.12; SAS Institute Inc, Cary, NC). The analyses included a full 24-year follow-up as well as a follow-up restricted to the first 10 years.

In these analyses, both twins in a pair were included and treated as independent individuals. To ensure that confidence intervals were not erroneously narrow owing to similarities within pairs, we performed proportional hazards regression analyses with variance estimates adjusted for correlated outcomes.<sup>36–38</sup> We accomplished this through the use of a SAS macro that stems from the same theoretical

background and yields the same results as the published Fortran program of Lin.<sup>39</sup>

Synergistic effects were analyzed on the basis of odds ratios from logistic regression models, according to methods suggested by Rothman.<sup>40</sup> The factors showing a significant ( $P < .05$ ) prevalence difference between ever- and never-unemployed individuals were considered to be of interest for an assessment of potential interaction with unemployment. Individuals unexposed to both unemployment and the other factor under study constituted the reference group. A synergy index was computed that indicated to what extent the presence of the factor influenced the effect of unemployment on mortality. A synergy index of 1 means no interaction, and a synergy index of 2 means an effect among those with combined exposures that is twice what would be expected from an additive effect of the 2 exposures. To calculate confidence intervals, we used methods of Hosmer and Lemeshow<sup>41</sup> and computer programs described by Lundberg et al.<sup>42</sup>

To control also for genetic and early childhood factors measured by the 1973 questionnaire, we analyzed mortality from all causes among 1067 twin pairs, 1 twin of whom had experienced unemployment while the other had not. We based the risk estimates (odds ratios) on conditional logistic regression for matched data using PHREG.

## RESULTS

Among women subjects, elementary school, smoking, use of alcohol, use of tranquilizers, shift work, personality factors, illness, and low socioeconomic status showed at least a 10% higher prevalence among those who had experienced short-term unemployment or long-term unemployment compared with those who had no experience of unemployment (Table 1). Being divorced and use of sleeping pills was also more prevalent among those experiencing unemployment.

Unemployed men more often were unmarried, were childless, smoked, used tranquilizers, had introvert personalities, had long-lasting illnesses, and had low socioeconomic status compared with those without experience of unemployment. Furthermore, being divorced, using sleeping pills, and doing shift

**TABLE 1—Prevalence (%) of Social, Behavioral, Health, and Personality Characteristics Among Women and Men, by Unemployment History: Sweden, 1973**

| Characteristic                        | Women                   |                         |                        |   |              | Men                     |                         |                        |   |              |
|---------------------------------------|-------------------------|-------------------------|------------------------|---|--------------|-------------------------|-------------------------|------------------------|---|--------------|
|                                       | History of Unemployment |                         |                        | Age-Adjusted Difference <sup>a</sup> (95% CI) |              | History of Unemployment |                         |                        | Age-Adjusted Difference <sup>a</sup> (95% CI) |              |
|                                       | Never                   | Short-Term <sup>b</sup> | Long-Term <sup>c</sup> | Short-Term                                    | Long-Term    | Never                   | Short-Term <sup>b</sup> | Long-Term <sup>c</sup> | Short-Term                                    | Long-Term    |
| Marital status                        |                         |                         |                        |   |              |                         |                         |                        |   |              |
| Unmarried                             | 27                      | 41                      | 38                     | -4 (-10, 1)                                   | 5 (-0.6, 10) | 32                      | 67                      | 53                     | 14 (6, 22)                                    | 15 (10, 21)  |
| Divorced                              | 5                       | 6                       | 10                     | ... <sup>d</sup>                              | 7 (3, 11)    | 3                       | 3                       | 6                      | ... <sup>d</sup>                              | 5 (1, 8)     |
| Widow/widower                         | 1                       | 1                       | 0                      | ...   | ...          | 0                       | 0                       | 0                      | ...   | ...          |
| No children                           | 38                      | 56                      | 40                     | -1 (-10, 8)                                   | -6 (-11, 1)  | 41                      | 71                      | 62                     | 11 (2, 19)                                    | 15 (10, 21)  |
| Education: elementary school          | 41                      | 35                      | 54                     | -0.6 (-11, 9)                                 | 16 (9, 22)   | 45                      | 41                      | 41                     | 2 (-7, 10)                                    | -2 (0, -4)   |
| Smoking status                        |                         |                         |                        |   |              |                         |                         |                        |   |              |
| Current smoker                        | 40                      | 61                      | 63                     | 21 (11, 31)                                   | 22 (16, 29)  | 52                      | 61                      | 66                     | 8 (-0.7, 17)                                  | 12 (6, 18)   |
| Former smoker                         | 13                      | 14                      | 12                     | -2 (-7, 3)                                    | -0.4 (-5, 4) | 14                      | 10                      | 13                     | 0.1 (-7, 7)                                   | -0.2 (-4, 4) |
| Alcohol consumption                   |                         |                         |                        |   |              |                         |                         |                        |   |              |
| None                                  | 43                      | 43                      | 46                     | 4 (-6, 13)                                    | 4 (-3, 12)   | 22                      | 25                      | 26                     | 5 (-5, 14)                                    | 4 (-2, 10)   |
| >250 g/mo                             | 15                      | 28                      | 18                     | 10 (0.5, 18)                                  | 2 (-4, 7)    | 46                      | 52                      | 50                     | 4 (-7, 15)                                    | 4 (-3, 11)   |
| Use of sedatives                      |                         |                         |                        |   |              |                         |                         |                        |   |              |
| Sleeping pills occasionally/regularly | 6                       | 9                       | 14                     | 6 (-1, 14)                                    | 9 (4, 14)    | 3                       | 6                       | 10                     | 5 (-1, 10)                                    | 9 (4, 13)    |
| Sleeping pills regularly              | 1                       | 3                       | 7                      | 2 (-2, 6)                                     | 6 (3, 10)    | 1                       | 3                       | 4                      | ...   | 4 (1, 7)     |
| Tranquilizers occasionally/regularly  | 14                      | 16                      | 27                     | 7 (-2, 16)                                    | 17 (11, 23)  | 7                       | 13                      | 16                     | 9 (2, 16)                                     | 11 (7, 16)   |
| Tranquilizers regularly               | 4                       | 8                       | 10                     | 7 (-0.3, 14)                                  | 8 (4, 13)    | 2                       | 7                       | 6                      | 5 (1, 10)                                     | 6 (3, 10)    |
| Stressful life situation              | 15                      | 13                      | 18                     | -1 (-8, 6)                                    | 5 (-0.2, 10) | 19                      | 17                      | 20                     | 3 (-5, 10)                                    | 2 (-3, 6)    |
| Shift work                            | 11                      | 20                      | 25                     | 9 (0.4, 17)                                   | 14 (9, 20)   | 23                      | 27                      | 29                     | 4 (-3, 11)                                    | 7 (1, 12)    |
| Personality                           |                         |                         |                        |   |              |                         |                         |                        |   |              |
| Extrovert                             | 51                      | 57                      | 47                     | 13 (5, 21)                                    | -4 (-10, 3)  | 62                      | 55                      | 58                     | -16 (-24, -8)                                 | -5 (-11, 1)  |
| Unstable                              | 26                      | 47                      | 48                     | 19 (9, 29)                                    | 24 (17, 30)  | 15                      | 30                      | 28                     | 14 (7, 22)                                    | 14 (9, 19)   |
| Long-lasting/serious illness          | 15                      | 19                      | 19                     | 12 (3, 21)                                    | 8 (2, 13)    | 15                      | 22                      | 20                     | 11 (3, 19)                                    | 7 (2, 12)    |
| Low socioeconomic status              | 77                      | 87                      | 90                     | 9 (2, 16)                                     | 14 (10, 18)  | 70                      | 85                      | 82                     | 11 (3, 18)                                    | 11 (7, 16)   |

Note. CI = confidence interval.

<sup>a</sup>Difference in exposure prevalence between short-term or long-term unemployed and never unemployed.

<sup>b</sup>Unemployed in 1973, lifetime unemployment less than 1 year.

<sup>c</sup>Ever unemployed, lifetime unemployment at least 1 year.

<sup>d</sup>Too few cases for age-adjusted analyses.

work were more prevalent among the unemployed. The latter results were statistically significant, but the difference in prevalence was less than 10%.

Among women, external causes of death—suicide in particular—showed a strong association with unemployment, with an almost threefold relative risk for the 24-year follow-up period and a sixfold increase for the first 10 years of follow-up (Table 2). The increased rates among the unemployed remained after adjustment for possible confounding factors, although at a somewhat lower level. Disregarding specific causes, the ever unemployed showed an increased mortality rate based on

the 24-year follow-up, with identical but less precise estimates for the first 10 years.

For men, a strong association between unemployment and death from external undetermined cause was found, even after adjustment for potential confounding factors (Table 2). An association on a lower level was also found for all external causes. There tended to be an association between malignant neoplasms and unemployment, which was weakened and still imprecise when potential confounders were controlled for. Total mortality over the 2 follow-up periods was higher among the ever unemployed; adjusting for potential confounding factors somewhat lowered the estimates.

In analyses in which the confidence intervals were adjusted for correlated outcomes, the confidence intervals of the mortality rate ratios were in general identical to those of the unadjusted analyses, showing little influence from correlation within twin pairs (data not shown).

For women, a synergy index of 7.0 was found for the joint presence of unemployment and use of sleeping pills (Table 3), indicating a mortality effect 7 times higher than expected from additivity. For women in the exposed group, use of tranquilizers, extravert personality, and unstable personality combined with unemployment showed a synergistic relation to mortality, although the statistical precision



**TABLE 2—Relative Risks for Specific Causes of Death and Total Mortality Among Women and Men, by Exposure to Unemployment: Sweden, 1973**

| Cause of Death (ICD-8 Codes)                                 | RR (95% CI) at 24-y Follow-Up |   |                   |                         | RR (95% CI) at 10-y Follow-Up |   |                   |                         |
|--|-------------------------------|---|-------------------|-------------------------|-------------------------------|---|-------------------|-------------------------|
|  | Total No. of Deaths           | No. of Deaths Among Those Exposed to Unemployment | Age-Adjusted      | Full Model <sup>a</sup> | Total No. of Deaths           | No. of Deaths Among Those Exposed to Unemployment | Age-Adjusted      | Full Model <sup>a</sup> |
| <b>Women</b>   |                               |   |                   |                         |                               |   |                   |                         |
| Total mortality  | 399                           | 42  | 1.7 (1.2, 2.4)    | 1.4 (1.0, 1.9)          | 73                            | 8   | 1.7 (0.8, 3.7)    | 1.4 (0.7, 3.0)          |
| Malignant neoplasms (140–209)                                | 208                           | 16  | 1.3 (0.8, 2.1)    | 1.1 (0.7, 1.9)          | 37                            | 1   | 0.4 (0.1, 3.2)    | 0.4 (0.1, 2.8)          |
| Malignant neoplasm of trachea, bronchus, or lung (162)       | 22                            | 1   | 0.9 (0.1, 6.8)    | 0.6 (0.1, 4.2)          | 2                             | 0   | ...               | ...                     |
| Diseases of the circulatory system (390–458)                 | 68                            | 6   | 1.6 (0.7, 3.7)    | 1.3 (0.5, 3.0)          | 11                            | 0   | ...               | ...                     |
| Ischemic heart diseases (410–414)                            | 31                            | 3   | 1.8 (0.5, 5.9)    | 1.3 (0.4, 4.5)          | 3                             | 3   | ...               | ...                     |
| Injuries, poisoning, other external causes (E800–E999)       | 50                            | 11  | 2.8 (1.4, 5.7)    | 2.0 (1.0, 4.1)          | 18                            | 6   | 6.2 (2.2, 17.5)   | 4.2 (1.5, 12.3)         |
| Suicide (E950–E959)  | 30                            | 8   | 4.1 (1.7, 9.5)    | 2.7 (1.2, 6.5)          | 13                            | 4   | 5.3 (1.6, 18.0)   | 3.7 (1.0, 13.0)         |
| External undetermined cause (E980–E989) <sup>b</sup>         | 4                             | 2   | 15.8 (1.3, 196.4) | 10.7 (0.9, 133.0)       | 2                             | 1   | ...               | ...                     |
| Other diagnoses  | 73                            | 9   | 2.0 (1.0, 4.0)    | 1.3 (0.6, 2.7)          | 7                             | 1   | 1.8 (0.2, 16.1)   | 1.6 (0.2, 13.8)         |
| Alcoholism, cirrhosis of liver, pancreatitis (303, 571, 577) | 10                            | 1   | 1.3 (0.2, 11.0)   | 1.0 (0.1, 8.1)          | 2                             | 1   | 14.7 (0.9, 252.9) | 10.4 (0.6, 185.2)       |
| <b>Men</b>   |                               |   |                   |                         |                               |   |                   |                         |
| Total mortality  | 792                           | 97  | 1.6 (1.3, 1.9)    | 1.3 (1.0, 1.6)          | 208                           | 32  | 1.9 (1.3, 2.8)    | 1.5 (1.0, 2.2)          |
| Malignant neoplasms (140–209)                                | 224                           | 26  | 1.6 (1.0, 2.4)    | 1.4 (0.9, 2.1)          | 47                            | 8   | 2.5 (1.1, 5.3)    | 2.1 (0.9, 4.6)          |
| Malignant neoplasm of trachea, bronchus, or lung (162)       | 38                            | 5   | 1.7 (0.7, 4.5)    | 1.5 (0.6, 4.1)          | 5                             | 1   | 3.1 (0.3, 29.0)   | 2.9 (0.3, 28.0)         |
| Diseases of the circulatory system (390–458)                 | 237                           | 23  | 1.4 (0.9, 2.2)    | 1.2 (0.8, 1.8)          | 40                            | 5   | 1.9 (0.7, 5.0)    | 1.4 (0.5, 3.8)          |
| Ischemic heart diseases (410–414)                            | 161                           | 15  | 1.4 (0.8, 2.4)    | 1.2 (0.7, 2.1)          | 27                            | 2   | 1.1 (0.3, 4.7)    | 0.8 (0.2, 3.6)          |
| Injuries, poisoning, other external causes (E800–E999)       | 200                           | 35  | 1.8 (1.2, 2.6)    | 1.5 (1.0, 2.2)          | 91                            | 16  | 1.8 (1.0, 3.1)    | 1.5 (0.8, 2.6)          |
| Suicide (E950–E959)  | 91                            | 12  | 1.3 (0.7, 2.4)    | 1.0 (0.6, 2.0)          | 38                            | 4   | 1.1 (0.4, 3.2)    | 0.9 (0.3, 2.6)          |
| Undetermined cause of death (E980–E989) <sup>b</sup>         | 23                            | 10  | 7.7 (3.3, 17.9)   | 5.8 (2.4, 14.0)         | 8                             | 4   | 8.6 (2.1, 36.2)   | 5.8 (1.3, 25.4)         |
| Other diagnoses  | 131                           | 13  | 1.2 (0.7, 2.2)    | 0.9 (0.5, 1.5)          | 30                            | 3   | 1.4 (0.4, 4.7)    | 0.9 (0.3, 3.2)          |
| Alcoholism, cirrhosis of liver, pancreatitis (303, 571, 577) | 40                            | 6   | 2.0 (0.8, 4.8)    | 1.3 (0.5, 3.3)          | 14                            | 2   | 2.2 (0.5, 10.2)   | 1.6 (0.3, 7.5)          |

Note. RR = relative risk; CI = confidence interval; ICD-8 = *International Classification of Diseases, Eighth Revision*.<sup>33</sup>

<sup>a</sup>For women, relative risk is adjusted for age, marital status, smoking status, alcohol consumption, use of tranquilizers, extroverted personality, and long-lasting/serious illness. For men, relative risk is adjusted for age, marital status, smoking status, alcohol consumption, use of sleeping pills, unstable personality, and long-lasting/serious illness.

<sup>b</sup>Uncertainty whether injury is accidental or intentional.

was weak owing to small numbers. For men, elementary school education, use of sleeping pills, and long-lasting or serious illness combined with unemployment were associated with a mortality rate ratio that exceeded expectancy based on additivity.

In the mortality analysis within twin pairs, where 1 twin had experienced unemployment while the other had not, the estimated relative risk of death for unemployed was 1.5 (95% confidence interval [CI]=0.7, 3.1) among women. A corresponding estimate of 1.4 (95% CI=1.0, 2.0) was found for men. Controlling for social, behavioral, health, and personality factors from the 1973 questionnaire lowered the relative risks marginally, to 1.4 (CI=0.6, 3.4) and 1.3 (CI=0.9, 1.9) for women and men, respectively.

## DISCUSSION

The results from this study suggest that unemployment is associated with an increased risk of early death even after adjustment for several potential confounding factors, including socioeconomic status, lifestyle factors, and genetic and early childhood factors. In particular, unemployment was associated with increased mortality from suicide and external undetermined cause. Among unemployed men, an increased risk of death from malignant neoplasms was also suggested. Furthermore, the results indicate that the risk of early mortality following unemployment may be strengthened by social, health, and personality factors.

One way to assess unemployment's public health impact is to estimate the attributable frac-

tion (the proportion of deaths that would be eliminated if mortality among the unemployed were reduced to that among the employed). The estimate depends on the strength of the association between exposure and outcome and also on the prevalence of the exposure. On the basis of our study results (24-year follow-up), and keeping the statistical imprecision in mind, the attributable fraction for suicides was 20% among women. For men, the attributable fraction for external undetermined cause was 38%.

## Unemployment

Information about current or previous unemployment was collected at baseline in 1973 only. The number of unemployment episodes and the duration of each episode was not recorded. "Ever unemployed" may therefore

**TABLE 3—Interactions Between Unemployment and Risk Indicators of Mortality for Women and Men: Sweden, 1973**

| Risk Indicator                                  | n <sup>a</sup> | Ever Unemployed,<br>Without Risk Indicator, <sup>b</sup><br>OR (95% CI) | Never Unemployed,<br>With Risk Indicator, <sup>b</sup><br>OR (95% CI) | Ever Unemployed,<br>With Risk Indicator, <sup>b</sup><br>OR (95% CI) | Synergy Index <sup>c</sup><br>(95% CI) |
|---|----------------|---|---|--|--|
| <b>Women</b>                                    |                |   |   |  |  |
| Unmarried, divorced, widow                      | 477            | 1.6 (1.0, 2.5)  | 1.4 (1.1, 1.7)  | 2.5 (1.6, 3.9)   | 1.5 (0.5, 4.4)                         |
| No children                                     | 526            | 2.1 (1.4, 3.1)  | 1.2 (0.9, 1.5)  | 1.6 (0.9, 2.7)   | 0.5 (0.1, 2.3)                         |
| Elementary school                               | 381            | 1.9 (1.2, 3.0)  | 1.1 (0.9, 1.4)  | 1.8 (1.1, 2.9)   | 0.8 (0.2, 2.7)                         |
| Smoker  | 492            | 1.6 (0.9, 2.7)  | 1.7 (1.4, 2.1)  | 2.9 (1.9, 4.3)   | 1.5 (0.6, 3.7)                         |
| Alcohol consumption >250 g                      | 123            | 2.0 (1.4, 2.8)  | 1.4 (1.1, 1.9)  | 1.2 (0.4, 3.4)   | 0.2 (0.0, 40.1)                        |
| Use of sleeping pills                           | 70             | 1.4 (1.0, 2.1)  | 1.4 (1.0, 2.0)  | 6.6 (1.1, 12.5)  | 7.0 (2.2, 22.5)                        |
| Use of tranquilizers                            | 175            | 1.4 (0.9, 2.1)  | 1.5 (1.2, 2.0)  | 3.5 (2.1, 5.7)   | 2.7 (1.0, 7.6)                         |
| Shift work                                      | 176            | 1.9 (1.3, 2.7)  | 1.3 (0.9, 1.7)  | 1.5 (0.7, 3.2)   | 0.4 (0.0, 4.8)                         |
| Extravert personality                           | 445            | 1.2 (0.7, 2.1)  | 1.3 (1.0, 1.5)  | 2.8 (1.8, 4.1)   | 3.9 (0.7, 22.7)                        |
| Unstable personality                            | 405            | 1.1 (0.7, 2.0)  | 1.4 (1.1, 1.7)  | 3.0 (2.0, 4.5)   | 3.8 (0.9, 15.6)                        |
| Long-lasting or serious illness                 | 154            | 1.6 (1.1, 2.4)  | 1.8 (1.5, 2.4)  | 3.4 (2.0, 5.9)   | 1.6 (0.7, 4.0)                         |
| Blue-collar worker or lower white-collar worker | 779            | 1.5 (0.5, 4.3)  | 1.1 (0.9, 1.5)  | 2.0 (1.4, 3.0)   | 1.5 (0.1, 16.5)                        |
| <b>Men</b>                                      |                |   |   |  |  |
| Unmarried, divorced, widower                    | 704            | 1.8 (1.3, 2.4)  | 1.9 (1.6, 2.3)  | 2.1 (1.5, 2.9)   | 0.6 (0.3, 1.3)                         |
| No children                                     | 788            | 1.9 (1.4, 2.6)  | 1.5 (1.2, 1.7)  | 1.6 (1.1, 2.3)   | 0.4 (0.2, 1.1)                         |
| Elementary school                               | 543            | 1.1 (0.8, 1.6)  | 1.2 (1.0, 1.4)  | 2.3 (1.8, 3.1)   | 4.1 (1.0, 16.7)                        |
| Smoker  | 800            | 1.5 (1.0, 2.3)  | 1.9 (1.6, 2.2)  | 2.7 (2.0, 3.6)   | 1.2 (0.7, 2.2)                         |
| Alcohol consumption >250g                       | 530            | 1.4 (1.0, 1.9)  | 1.3 (1.2, 1.6)  | 2.3 (1.7, 3.1)   | 1.7 (0.8, 3.9)                         |
| Use of sleeping pills                           | 60             | 1.5 (1.1, 1.8)  | 1.8 (1.2, 2.5)  | 3.5 (1.9, 6.8)   | 2.1 (0.7, 6.1)                         |
| Use of tranquilizers                            | 130            | 1.5 (1.1, 1.9)  | 1.5 (1.1, 1.9)  | 2.6 (1.6, 4.2)   | 1.7 (0.6, 4.5)                         |
| Shift work                                      | 395            | 1.5 (1.1, 1.9)  | 1.1 (0.9, 1.3)  | 1.8 (1.3, 2.6)   | 1.4 (0.5, 4.1)                         |
| Extravert personality                           | 783            | 1.8 (1.3, 2.5)  | 1.1 (0.9, 1.3)  | 1.5 (1.1, 2.0)   | 0.5 (0.2, 1.6)                         |
| Unstable personality                            | 350            | 1.3 (1.0, 1.8)  | 1.5 (1.3, 1.9)  | 2.6 (1.8, 3.6)   | 1.8 (0.8, 3.8)                         |
| Long-lasting or serious illness                 | 233            | 1.4 (1.0, 1.8)  | 1.4 (1.2, 1.7)  | 2.7 (1.9, 4.0)   | 2.2 (1.0, 5.0)                         |
| Blue-collar worker or lower white-collar worker | 1085           | 1.8 (1.1, 3.0)  | 1.3 (1.1, 1.5)  | 1.8 (1.4, 2.3)   | 0.7 (0.3, 1.9)                         |

<sup>a</sup>Number of unemployed also exposed to the risk indicator.<sup>b</sup>Odds ratio (OR) and 95% confidence interval (95% CI) are adjusted for age. The reference group (OR = 1) refers to cases and referents among the individuals unexposed to both unemployment and the risk indicator under study.<sup>c</sup>Synergy index: 1.0 = no interaction, 2.0 = an effect among those with combined exposure twice what would be expected from an additive effect of the 2 exposures, etc.

apply to 1 or several occasions of different length, remote from or close in time to the start of follow-up. In addition, we had no information on unemployment occurring after 1973. Exposed individuals could repeatedly be unemployed during follow-up. This does not lead to misclassification of the exposure because we did not take different levels of exposure into account—the exposure we analyzed was “ever unemployed.” Individuals classified as never unemployed as of 1973 could encounter unemployment later on and therefore be misclassified. This limitation would lead to underestimated risk estimates if those who became unemployed after 1973 had the same mortality pattern as those reported as ever unemployed

as of 1973. If the individuals who became unemployed after 1973 had a lower death rate than those earlier classified as unemployed, then the reported estimates could be biased upward; however, we have no reason to believe this to be the case. On the other hand, it is uncertain to what extent the results obtained are valid for more recent time periods, when worker groups other than those of the present study are facing unemployment.

We used a 24-year follow-up as well as a shorter period: the first 10 years of the follow-up (1973–1982). For several of the cause-specific diagnoses, higher mortality rate ratios were found for the 10-year period than for the longer follow-up. This finding could

owe partly to increased misclassification of unexposed individuals over time. It could also be that unemployment entails an increased risk of death (during a limited time period) that gradually fades away. The results suggest that unemployment may have an impact on mortality, not only in a short-term but also in a long-term perspective.

### Selection and Pathway

In accordance with other studies,<sup>12,21,25</sup> several factors related to mortality were significantly more prevalent among the unemployed than among the never unemployed (Table 1). These characteristics could either lead to or be a consequence of unemployment. The associa-

tion between unemployment and mortality may therefore partly be a result of selection mechanisms, in that individuals with certain risk indicators are more likely to become unemployed than those without the risk indicators in question. However, unemployment may also contribute to the development of these risk indicators and, in turn, to poor health. The cross-sectional data did not permit a clear determination of the timing between risk indicators and unemployment in this study.

Adjustment for risk indicators that constitute links in the causal chain between an exposure and an outcome may inaccurately reduce an association and mask an actual effect or part of the effect attributable to the exposure. Low education, personality factors, and low socioeconomic status are comparatively stable over time and may in general precede unemployment. Shift work should also mainly (but not exclusively) precede unemployment, assuming that occupational mobility is comparatively low in terms of this occupational characteristic. Furthermore, we see no reason why unemployment should increase the probability of holding a job with shift work. Serious or long-lasting illness could also be a precursor, since subjects with poor health may be more likely to lose their job. Divorce may to some extent be a consequence of unemployment, and smoking and use of sleeping pills and tranquilizers could be a coping behavior owing to psychological stress caused by unemployment. In the extended multivariate analyses, the relative risks were often attenuated compared with the age-adjusted relative risks, and this attenuation may be partly attributable to unwarranted adjustment for factors in the causal chain.

A major advantage of our study was that it controlled for confounding owing to genetic and early social and environmental conditions by analyzing unemployment among discordant twin pairs. In general, twins have early social and environmental conditions in common; in addition, monozygotic twins are genetically identical and dizygotic twins have half of their genes in common. The twin analyses showed an increased risk of death for the exposed twin compared with his or her twin sibling. The confidence intervals were wide owing to a rather small number of deaths. Nevertheless, it is notable that this analysis, which controlled for many predisposing life conditions, yielded risk

estimates comparable to the analyses based on the full cohort.

### Specific Causes of Death

We found an association between unemployment and external causes of death for both women and men. This finding is consistent with those of other studies,<sup>9,12,13</sup> although few of these included women.<sup>9</sup> Suicide was clearly associated with unemployment among women, but not among men. On the other hand, men who experienced unemployment had an increased risk of death by external undetermined cause. This finding raises the possibility that suicide is less likely to be identified among men than among women. In some studies reporting an association between suicide and unemployment among men, deaths by external undetermined cause and suicides were combined.<sup>19,20</sup>

The results indicate that unemployment has an important impact on mental health. Unemployment may cause a deterioration of economic situation, downgrading of social status, broken social relations, changed risk behaviors, impaired psychological well-being, and depression, consequences that may develop into severe illness.<sup>1-4,6-8,17,18,25</sup>

Several studies have reported an increased risk of mortality from cardiovascular disease with unemployment.<sup>1,21-23</sup> Our study showed no such increased risk, which is in accordance with another recent Swedish study.<sup>16</sup> It has been suggested that inability to control for behavioral and medical parameters before and after unemployment, and a too short follow-up period, contribute to difficulties in showing a possible relationship between unemployment and cardiovascular diseases.<sup>15</sup> Our results do not support this view but rather indicate that an association between unemployment and death from cardiovascular diseases observed in some studies may be confounded by other risk factors.

Other studies have pointed out that the excess cancer mortality among the unemployed owes mainly to an increased risk of lung cancer.<sup>2,11,12,21,24</sup> Our results for men are consistent with this observation, although they lack precision owing to small numbers. As in our study, other studies have reported a higher prevalence of smokers among the unemployed, and they have shown that smoking habits stay quite stable during unemployment.<sup>11,21</sup> In our study,

the prevalence of smoking among short-term and long-term unemployed women was similar, and there was only a slight difference among men, suggesting that, to some extent, smoking may be a precursor of unemployment. It is possible that smokers have a greater risk of losing their job either because of smoking or because of other factors related to smoking.

An increased mortality from alcohol-related diseases among the unemployed was reported by Martikainen.<sup>12</sup> Our data were consistent with such an association among men, although the results were based on few deaths and the risk estimate decreased in the full model. Morris et al. reported a higher prevalence of alcohol use among the unemployed.<sup>21</sup> In our study, increased alcohol use among the unemployed was seen only among women who experienced short-term unemployment in 1973.

### Interaction

As far as we know, no other study has focused on the question of interaction between unemployment and the characteristics of the individual relative to early death. It has been suggested previously that buffering effects from social support, for example, could reduce the negative effects of the stress of losing a job.<sup>4,7,8,10</sup> Our results indicate that modifying the effects of individual characteristics could strengthen the association between unemployment and mortality. For men, but not for women, low education seemed to enhance the association between unemployment and mortality. It is possible that unemployment means greater strain in economic and social terms, particularly for men with low education.

Furthermore, our results suggest that use of sleeping pills or tranquilizers may enhance the risk of early death among individuals who experience unemployment, particularly women. Use of these drugs may be caused by unemployment, and this type of coping may reflect a particularly strong reaction. Apart from the availability of drugs, which may be the direct cause of death, the interaction could also signify that women using these drugs are more vulnerable owing to different psychosocial problems or mental diseases, and that unemployment may augment a prevailing difficult situation. In addition, the synergistic effects indicated for unstable personality traits and for serious or long-lasting illness may indicate that

unemployment among individuals already burdened by psychological disorders, somatic illness, or both may overwhelm the individual.

## CONCLUSIONS

Unemployment is associated with an increased risk of early death, especially from suicide and external undetermined cause. Our results suggest that characteristics of the individual prior to unemployment cannot explain this increased risk. They further indicate that the association between unemployment and mortality may be strengthened by social factors, personality characteristics, and health-related factors. An increased risk of early mortality related to unemployment should be recognized. ■

## About the Authors

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## Contributors

M. Voss and L. Nylén contributed substantially in the conceptualization and design of the study and had main responsibility for the analyses and the reporting. B. Floderus initiated the study and supervised all aspects of its implementation. F. Diderichsen contributed to conceptualization of ideas. P.D. Terry assisted with the analyses. All authors helped to interpret the findings and reviewed drafts of the article.

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## Human Participant Protection

Participation in the study was optional for all members of the Swedish Twin Registry, and all subjects received general information about the general purpose of the registry. The Ethical Committee at Karolinska Institutet approved the principles for use of the Twin Registry and also this particular study.

## References

1. Jin RL, Shah CP, Svoboda TJ. The impact of unemployment on health: a review of the evidence. *CMAJ*. 1995;153(5):529–540.
2. Wilson SH, Walker GM. Unemployment and health: a review. *Public Health*. 1993;107:153–162.
3. Jahoda M. The impact of unemployment in the 1930s and the 1970s. *Bull Br Psychol Soc*. 1979;32:309–314.
4. Hammarström A. Health consequences of youth unemployment—review from a gender perspective. *Soc Sci Med*. 1994;38(5):699–709.
5. Platt S. Unemployment and suicidal behaviour: a review of the literature. *Soc Sci Med*. 1984;19(2):93–115.
6. Mathers CD, Schofield DJ. The health consequences of unemployment: the evidence. *Med J Aust*. 1998;168:178–182.
7. Dooley D, Fielding J, Levi L. Health and unemployment. *Annu Rev Public Health*. 1996;17:449–465.
8. Kasl SV, Jones BA. The impact of job loss and retirement on health. In: Berkman LF, Kawachi I, eds. *Social Epidemiology*. New York, NY: Oxford University Press; 2000:118–136.
9. Iversen L, Andersen O, Andersen PK, Christoffersen K, Keiding N. Unemployment and mortality in Denmark, 1970–80. *BMJ*. 1987;295:879–884.
10. Levi L. Psychosocial environmental factors and psychosocial mediated effects of physical environmental factors. *Scand J Work Environ Health*. 1997;23(suppl 3):47–52.
11. Lynge E. Unemployment and cancer: a literature review. *IARC Sci Publ*. 1997;138:343–351.
12. Martikainen PT. Unemployment and mortality among Finnish men, 1981–5. *BMJ*. 1990;301:407–411.
13. Moser KA, Fox AJ, Jones DR. Unemployment and mortality in the OPCS longitudinal study. *Lancet*. 1984;2:1324–1329.
14. Stefansson C-G. Long-term unemployment and mortality in Sweden, 1980–1986. *Soc Sci Med*. 1991;32(4):419–423.
15. Weber A, Lehnert G. Unemployment and cardiovascular diseases: a causal relationship? *Int Arch Occup Environ Health*. 1997;70(3):153–160.
16. Gerdtham U-G, Johannesson M. A note on the effect of unemployment on mortality. *J Health Econ*. 2002;22:505–518.
17. Björklund A, Eriksson T. Unemployment and mental health: evidence from research in the Nordic countries. *Scand J Soc Welfare*. 1998;7:219–235.
18. Nordenmark M. *Unemployment, Employment Commitment and Well-Being: The Psychosocial Meaning of (Un)Employment Among Women and Men* [dissertation]. Umeå, Sweden: Department of Sociology, Umeå University; 1999.
19. Kposowa AJ. Unemployment and suicide: a cohort analysis of social factors predicting suicide in the US National Longitudinal Mortality Study. *Psychol Med*. 2001;31(1):127–138.
20. Lewis G, Sloggett A. Suicide, deprivation and unemployment: record linkage study. *BMJ*. 1998;317:1283–1286.
21. Morris JK, Cook DG, Shaper AG. Loss of employment and mortality. *BMJ*. 1994;308:1135–1139.
22. Brenner MH. Heart disease mortality and economic changes, including unemployment, in Western Germany 1951–1989. *Acta Physiol Scand Suppl*. 1997;640:149–152.
23. Forbes JF, McGregor A. Male unemployment and cause-specific mortality in postwar Scotland. *Int J Health Serv*. 1987;17(2):233–240.
24. Lynge E, Andersen O. Unemployment and cancer in Denmark, 1970–1975 and 1986–1990. *IARC Sci Publ*. 1997;138:353–359.
25. Janlert U. Unemployment as a disease and diseases of the unemployed. *Scand J Work Environ Health*. 1997;23(suppl 3):79–83.
26. Martikainen PT, Valkonen T. Excess mortality of unemployed men and women during a period of rapidly increasing unemployment. *Lancet*. 1996;348:909–912.
27. Bartley M, Owen C. Relation between socioeconomic status, employment, and health during economic change, 1973–93. *BMJ*. 1996;313:445–449.
28. Martikainen PT, Valkonen T. The effects of differential unemployment rate increases of occupation groups on changes in mortality. *Am J Public Health*. 1998;88(12):1859–1861.
29. Nylén L, Voss M, Floderus B. Mortality among women and men relative to unemployment, part time work, overtime work, and extra work: a study based on data from the Swedish twin registry. *Occup Environ Med*. 2001;58(1):52–57.
30. Medlund P, Cederlöf R, Floderus B, Friberg L, Sörensen S. *A New Swedish Twin Registry*. Stockholm, Sweden: Karolinska Institutet; 1976.
31. Floderus B. *Psycho-Social Factors in Relation to Coronary Heart Disease and Associated Risk Factors* [dissertation]. Stockholm, Sweden: Department of Environmental Hygiene, Karolinska Institutet. *Nordisk Hygienisk Tidskrift*. 1974(suppl 6):1–148.
32. Floderus-Myrhed B, Pedersen N, Rasmuson I. Assessment of heritability for personality, based on a short-form of the Eysenck Personality Inventory: a study of 12,898 twin pairs. *Behav Genetics*. 1980;10(2):153–162.
33. *International Classification of Diseases, Eighth Revision*. Geneva, Switzerland: World Health Organization; 1965.
34. *International Classification of Diseases, Ninth Revision*. Geneva, Switzerland: World Health Organization; 1980.
35. Clayton D, Hills M. *Statistical Models in Epidemiology*. New York, NY: Oxford University Press; 1993.
36. Lin DY. Cox regression analysis of multivariate failure time data: the marginal approach. *Stat Med*. 1994;13(21):2233–2247.
37. Wei LJ, Lin DY, Weissfeld L. Regression analysis of multivariate incomplete failure time data by modeling marginal distributions. *J Am Stat Assoc*. 1989;84(408):1065–1073.
38. White H. Maximum likelihood estimation of misspecified models. *Econometrica*. 1982;50(1):1–25.
39. Lin DY. MULCOX2: a general computer program for the Cox regression analysis of multivariate failure time data. *Comput Methods Programs Biomed*. 1993;40(4):279–293.
40. Rothman KJ. *Modern Epidemiology*. Boston, Mass: Little, Brown and Company; 1986.
41. Hosmer DW, Lemeshow S. Confidence interval estimation of interaction. *Epidemiology*. 1992;3(5):452–456.
42. Lundberg M, Fredlund P, Hallqvist J, Diderichsen F. A SAS program calculating three measures of interaction with confidence intervals. *Epidemiology*. 1996;7(6):655–656.



# Hurling Alone? How Social Capital Failed to Save the Irish From Cardiovascular Disease in the United States

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Increasing evidence indicates that a full etiological explanation for major adult chronic diseases must include consideration of influences across the life course.<sup>1–4</sup> Current rates of coronary heart disease in Ireland and parts of Scotland with high rates of Irish immigration rank among the highest in the developed world,<sup>5</sup> and rates are twice the European Union average in the Republic of Ireland.<sup>6</sup> In addition, Irish immigrants to the United Kingdom retain an overall increased risk of ill health for at least 2 subsequent generations,<sup>7,8</sup> which can be partly accounted for by lifestyle and social conditions.<sup>9</sup>

In this article we examine how the early- and later-life conditions of the Irish, one of the major ethnic groups to immigrate to the United States in the 19th and early 20th centuries, contributed to their overall patterns of cardiovascular mortality. Some 4.5 million Irish immigrated to the United States over a period of 80 years, particularly after the great Irish famine of 1847.<sup>10</sup> This famine was the most devastating example in modern European history of the acute effects of a crop failure, resulting directly and indirectly in a halving of Ireland's population. The cultural story of these Irish immigrants has been documented in remarkable detail.<sup>11–15</sup> The Irish settled throughout the United States, and particularly in large East Coast cities. When a general ancestry question was reintroduced into the United States Census in 1980, 40.2 million people, or 20.64% of the White/European population, declared themselves to be of Irish ancestry.<sup>10</sup> Despite criticisms of the reliability of this measure,<sup>16</sup> demographic analysis indicates that this number is likely to be reasonably accurate.

## METHODS

### Ethnic Origin and Mortality in US Census Vital Statistics Records

In a 1933 report, considerable and unexplained variation in infant mortality rates across

**Objectives.** We performed a historical review of cardiovascular risk profiles of Irish immigrants to the United States, 1850–1970, in regard to lifestyle, socioeconomic circumstances, and social capital.

**Methods.** We analyzed US Census data from 1850–1970, area-based social and epidemiological data from Boston, data from Ireland's National Nutrition Surveillance Centre, and literature on Irish migration.

**Results.** The Irish were consistently at increased risk of cardiovascular diseases, a risk that related initially to material deprivation, across the life course of at least 2 generations.

**Conclusions.** The principal difference between the Irish and other disadvantaged immigrant groups, such as the Italians, was dietary habits influenced by experiences during the Irish famine. Although there was a psychosocial component to the disadvantage and discrimination they experienced as an ethnic group, the Irish also exhibited strong community networks and support structures that might have been expected to counteract discrimination's negative effects. However, the Irish's high levels of social capital were not protective for cardiovascular disease. (*Am J Public Health.* 2004;94:2162–2169)

Boston's census tracts was found.<sup>17</sup> In 1985, findings from the Ireland–Boston Diet Heart Study<sup>18</sup> were published. These 2 reports constitute the tip of the iceberg of what is a largely neglected story. Both used a unique and extensive US vital statistics database to examine ethnic variations in disease risk.<sup>19–31</sup> For this analysis we reviewed all hard-copy census reports and undertook a literature search for related publications, with a particular focus on the City of Boston. From 1850, country of nativity was recorded routinely as part of the US Census, and from 1870 to 1970, nativity of parents was recorded as well.<sup>20</sup> (The exact terminology varies from census to census, as we will present, but respondents may be categorized according to [a] whether they were native born vs foreign born, [b] whether they were of native-born vs foreign-born parentage, or [c] their country of origin.) Furthermore, both all-cause and disease-specific mortality were recorded, first retrospectively through census enumeration and then through state-level registration processes that achieved national coverage by the 1930s. It is possible, therefore, to document the variation in disease patterns related to country of origin for immigrants and their first-generation American

children. Because country-of-nativity questions deal specifically with the experiences of respondents or their parents, they are more precise than the recently employed general ancestry question.<sup>16,31</sup> An examination of each of these original census records revealed that the Irish had excess mortality throughout the 1850–1970 period, particularly from diseases of the heart and circulatory system. Readers should note that processes of classification of circulatory diseases were not standardized at the end of the 19th and the beginning of the 20th centuries—diagnostic criteria developed over this period.<sup>19</sup> Thus, it is unavoidable that we refer to several classifications of circulatory diseases (“circulatory disease,” “cerebrovascular disease,” etc.) as they were used in the different historical reports.

## RESULTS

The extensive US vital statistics database is summarized in Table 1. The first census report, in 1850, clearly documented that the Irish were at increased risk.<sup>21</sup> The 1860 census report concerned itself only with the health differences between Blacks and Whites.<sup>22</sup> How-

**TABLE 1—The Irish Transition in the United States: Summary of US Census-Related Data on Socioeconomic Circumstances and Cardiovascular Disease, 1850–1980**

| Census Data Year | Observations and Analysis of the Health of Ethnic Irish Groups in the US and Comparisons With Other Immigrant and Nonimmigrant Groups  |
|------------------|--|
| 1850             | Irish born constituted 4.81% of the total population in the US but made up 6.23% of deaths from circulatory disease, 7.12% of deaths from respiratory disease, and 6.33% of deaths from tuberculosis. All-cause death rates were 16.41 per 1000 deaths, comparable with the US average (16.16 per 1000 deaths).  |
| 1870             | Crude death rates from circulatory disease for Irish-born Americans were 85.9 per 100 000 deaths, comparable with rates among Americans born in England and Wales (90.1 per 100 000) but higher than for all foreign-born immigrants to the US (55.7 per 100 000) and for US-born White Americans (41.4 per 100 000).  |
| 1880             | Health status of Americans of Irish and German extraction is contrasted. Deaths from heart disease and dropsy stood at 62.3 per 1000 deaths among Americans of Irish-born parentage, 60.9 per 1000 deaths among Americans of German-born parentage, 64.5 per 1000 deaths among Black Americans, and 56.1 per 1000 deaths among White Americans generally. Tenement conditions were implicated in high death rates of poor White Americans. Rates of tuberculosis infection were excessive among immigrants to the US of Irish-born parentage.  |
| 1890             | Heart disease and dropsy death rates were higher overall among foreign-born Americans than among US-born Americans. Irish rates of heart disease and dropsy are the highest of any immigrant group in the United States—Americans with mothers born in Ireland at 15–45 years of age: 96.86 per 100 000 (compared to 66.32 per 100 000 for native-born White Americans); at 45–65 years: 401.94 per 100 000 (compared to 305.68 per 100 000 for native-born White Americans); and at 65 years and older: 1 199.33 per 100 000 (compared to 1129.01 per 100 000 for native-born White Americans).   |
| 1900             | Irish-born immigrants represented 2.12% of the US population, but Americans of Irish-born parentage number 4 981 047, or 6.53% of the total population. However, this Irish group (both Irish-born and of Irish-born parentage) contributes 10.54% of total deaths from circulatory disease in the US.   |
| 1910             | Death rates from organic heart disease were 5.63 per 1000 among Americans born in Ireland compared to 0.82 per 1000 of Americans born in Italy. Death rates for other circulatory disease were 1.90 per 1000 for Americans born in Ireland and 0.23 per 1000 in Americans born in Italy. Americans of Irish origin accounted for 10.11% of organic heart disease cases and 11.24% of circulatory disease cases in the US, though immigrants born in Ireland made up only 1.47% of the total US population. Dublin and Baker <sup>32</sup> reviewed in more detail the available data for Pennsylvania and New York, confirming excessive mortality for those of foreign and mixed (one parent not from US) parentage and those who were foreign born—this excessive mortality was especially true for the Irish of all categories, being about double the rate for second-generation Americans. Heart disease for males born in the US aged 45–64 years was 232.5 per 100 000 in Pennsylvania and 316.3 per 100 000 in New York, compared to 529.3 per 100 000 for Irish-born males in Pennsylvania and 580.2 per 100 000 in New York. Rates of heart disease were also much higher among similar groups of Irish women when compared with overall rates for women in Pennsylvania and New York. |
| 1920             | Age-adjusted death rates from cerebral hemorrhage per 100 000 population were reported according mother's country of birth—New York City: 99.9 Ireland vs 81.9 US, Pennsylvania: 95.7 Ireland vs 86.1 US, New York State: 85.6 Ireland vs 67.2 US, and Chicago: 82.4 Ireland vs 60.4 US. Americans with mothers born in Ireland had higher rates of cerebral hemorrhage than any other group, including Americans with US-born mothers. The highest rate of heart disease was in New York (389.3 per 100 000) for those with mothers born in Ireland and the lowest, in Pennsylvania, for those with mothers born in Italy. In a detailed monograph, Carpenter <sup>33</sup> reported the health status of immigrants and their children, including socioeconomic circumstances; he singled out the Irish for special mention as being at particularly excessive risk of poor health.  |
| 1930             | The total foreign-born population from Ireland was 1 037 234 in the US. National mortality data were not published, but a Boston-based census tract-level study found that the highest rates of infant mortality were in Charlestown and South Boston (neighborhoods in Boston, Mass), <sup>33,34</sup> though the Irish were integrated across the city. A strong inverse relationship was also found between 1930s socioeconomic indicators and present-day health status. The correlation between infant mortality rates during the 1930s and the coronary heart disease rate in the 2001 Health of Boston report <sup>35</sup> was 0.564 ( $P < .05$ ), with the highest rates being in Charlestown and South Boston.  |
| 1950             | Two area-based studies showed excess mortality among those of Irish extraction in the US. Trulson et al. <sup>36</sup> showed that first-generation Irish have higher death rates than US-born Bostonians with US-born parents and Stamlar et al. <sup>37</sup> showed considerably excessive risk at 45–64 years for the Irish in America compared with other immigrant groups, US-born Americans, and the Irish in Ireland.  |
| 1980             | Rosenwaike and Hempstead, <sup>38</sup> analyzing data through the 1980 US census, concluded that the SMR (Standardized Mortality Ratio) for heart disease is 0.95 for those male immigrants to the US born in Ireland compared with US-born males, but with a ratio of 1.16 to the rate for Italian American males and that an excess of cerebrovascular disease existed, with SMR 1.13 in men and 1.56 in women compared with the US-born population.  |

*Note.* We refer to several classifications of circulatory diseases ("circulatory disease," "cerebrovascular disease," etc.) as they were used in the different historical reports. The terminology regarding race/ethnicity also varies from census to census—respondents may be categorized according to (a) whether they were native born vs foreign born, (b) whether they were of native-born vs foreign-born parentage, or (c) their country of origin. All data were taken from US Census Bureau reports<sup>21–30</sup> unless otherwise cited.

ever, the subsequent 4 census reports across the latter half of the 19th century continued to indicate excess cardiovascular mortality risk among citizens of Irish extraction.<sup>23–26</sup> In 1910 and 1920, rates were elevated among various

foreign-born groups, but particularly among the Irish.<sup>27,28</sup> For instance, in all the urban areas with the highest age-specific death rates for stroke (another condition that may have early-life origins<sup>39,40</sup>) in 1920, men and

women of Irish parentage were at demonstrably increased risk, whether US-born or not.

From the period of the 1910 census onward, a number of monographs and reports examined the effect of migration on health with

careful, age-standardized approaches.<sup>32–38,41–44</sup> During this period, the overwhelming majority of immigrants were Whites of European origin. In examining the documents, we found a general consensus among them that immigrants, and indeed their first-generation children, were at excess risk of circulatory diseases compared with US Whites of native parents, and that the Irish were consistently at higher risk than other immigrant groups. This phenomenon of Irish immigrants being at particularly high risk for cardiovascular disease persisted over a period of 150 years.<sup>36,38–44</sup> The important question is, *why*?

Although early demographers considered the effects of ethnicity and adverse social conditions on longevity and health,<sup>41,42</sup> newer generations of epidemiologists were more inclined to attribute these effects to a so-called process of Americanization mainly related to individual-level adult lifestyle.<sup>36–38,44</sup> However, no one adequately explained why the Irish were consistently at higher risk. Was their excess risk related to constitutional or genetic factors, adverse lifestyle practices, processes of material disadvantage, or psychosocial processes operating at the individual or community level? As suggested in the title of this article, one way of restating this question is to paraphrase it in terms of Robert D. Putnam's most influential work, *Bowling Alone: The Collapse and Revival of American Community*, in which he describes—beginning with the example of the rise in popularity of bowling but the decline of bowling leagues—Americans' increasing disconnectedness with each other.<sup>45</sup> Putnam maintains that this “bowling alone”—a marker of the decline in social capital—is partly responsible for the apparent collapse of community in America and it may have far-reaching health impacts.<sup>45–47</sup> The Irish immigrants were not bowlers (at least not initially) but they did have their own ancient and unique community team sport called “hurling” in their country of origin, which also serves as a symbol of social capital. So was the high risk of cardiovascular disease in the Irish in the US somehow caused by the fact that they were *hurling alone*?

### Community Networks and Health

This brings us to the question of social disadvantage among the Irish and the degree to which its origins are material or psychosocial.

The Boston Health League in the early 1930s<sup>17</sup> investigated the predisposition of certain areas to higher infant mortality with 2 detailed reports that incorporated social and health statistics.<sup>33,34</sup> There were then 14 census tract areas in Boston (Table 2). For each of these areas, the following data were collected: (1) ethnicity (percentage of all foreign-born, US-born of foreign parents, US-born of native parents, Negro [*sic*], and foreign-born from several countries, notably Ireland, Italy, and Canada), (2) citizenship status (percentage naturalized citizens, aliens, and those with “first papers” [those in the process of naturalization]), (3) health indicators (infant mortality, tuberculosis incidence, and adult mortality), and (4) economic status (unemployment; criminal delinquency; numbers receiving unemployment aid, dependent aid, mother's aid, and old-age assistance; and housing type and median monthly rental [\$]).

A variation in infant mortality was found; the highest proportion of Irish-born was found for the 2 areas with highest mortality, Charlestown and South Boston (Table 2). When the interrelationship between variables is explored using Pearson's correlation method, these data present a convincing pattern of each ethnic group in social transition. The Irish, as the longest-established immigrant group, were distributed across the city and were likely to live in areas with high numbers of US-born people of foreign-born parentage ( $r=0.719$ ,  $P=.004$ ) and high numbers of naturalized citizens ( $r=0.716$ ,  $P=.004$ ) but were unlikely to be living in areas with high numbers of aliens ( $r=-0.759$ ,  $P=.002$ ). The Canadians, by contrast, were more affluent and were most likely to be living in communities with high numbers of US-born people of native-born parentage ( $r=0.902$ ,  $P<.0001$ ) and were highly unlikely to live in communities with high proportions of foreign-born people ( $r=-0.950$ ,  $P<.0001$ ), unemployment ( $r=-0.622$ ,  $P=.018$ ), or criminal delinquency ( $r=-0.741$ ,  $P<.560$ ). The Italians, more recently arrived, were unlikely to live in areas with high numbers of either Irish or Canadians or high numbers of native-born people of native-born parentage ( $r=-0.620$ ,  $P<.018$ ), and they were also more likely to reside in areas with high rates of dependent support, various forms of relief, and juvenile criminal delinquency. Table 3 indicates strong, consistent interrelationships be-

tween health and social indicators, with median rental income inversely associated with infant mortality rate and with incidence and mortality rates of tuberculosis.

In line with the hypothesis of early-life influences on adult health,<sup>1–4</sup> the question arises as to whether these previous patterns of association between social and health indicators can be related to present-day health profiles. The net effect of social mobility over time and between areas of any large city is complex—so interpretation of such long-term, complex changes must be done cautiously. However, 13 of the 14 original census areas still exist, though subdivisions and changes make them only indirectly comparable. For instance, the West End is now part of Back Bay and the Beacon Hill neighborhood, and Mattapan and Roslindale are now considered separately.

In a special study of these changing community profiles, Gamm used sociodemographic data by census tract (ethnicity continued to be recorded to some degree between 1940 and 1970) to examine patterns of migration of Jewish and Catholic groups in Boston—he complemented this census data with church and synagogue records.<sup>48</sup> He also took account of major policy initiatives around affordable housing, including the Boston Banks Urban Renewal Group scheme. Gamm found that there was surprisingly little shift in the Catholic populations, largely owing to strong affiliation to religious parishes. Because these populations are predominantly of Irish extraction, we can therefore be somewhat confident of a continuing pattern of people remaining in their areas of birth, particularly among the older generations. Table 2 shows present-day rates of age-standardized coronary heart disease and stroke, which are still reported by neighborhood in Boston.<sup>35</sup> Overall, there is a significant correlation between infant mortality rates in 1930–1934 and coronary heart disease rates averaged for 1994–1998 ( $r=0.564$ ,  $P=.04$ ). Present-day rates of coronary heart disease are clearly highest in Charlestown and South Boston (Table 2). Although the relationship between overall infant mortality rate in the 30s and present-day Irish-born percentage is not quite as strong ( $r=0.46$ ,  $P=.09$ ), both coronary heart disease rates for the 1994–1998 ( $r=0.608$ ,  $P=.027$ ) and stroke rates for 1994–1998 ( $r=0.591$ ,  $P=.033$ ) are signifi-

**TABLE 2—Characteristics of 14 Census Tract Areas in 1930s Boston (Boston Neighborhood Study<sup>33,34</sup>) and Corresponding 1990s Stroke and Coronary Heart Disease Rates (Health of Boston 2001<sup>35</sup>)**

|                                     | Back Bay | Brighton | Charlestown | Dorchester North | Dorchester South | East Boston | Hyde Park | Jamaica Plain | North End | Roxbury | South Boston | South End | West End | West Roxbury |
|-------------------------------------|----------|----------|-------------|------------------|------------------|-------------|-----------|---------------|-----------|---------|--------------|-----------|----------|--------------|
| <b>Boston Neighborhood Study</b>    |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Nativity, %                         |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| All Foreign-born                    | 26.00    | 35.8     | 44.9        | 52.9             | 55.5             | 68.7        | 47.10     | 43.00         | 87.40     | 48.00   | 55.6         | 50.80     | 62.5     | 40.3         |
| US-born to foreign parents          | 24.00    | 31.00    | 34.00       | 30.00            | 26.00            | 22.00       | 30.00     | 34.00         | 10.00     | 24.00   | 30.00        | 16.00     | 22.00    | 35.00        |
| US-born to US-born parents          | 48.00    | 32.00    | 20.00       | 17.00            | 18.00            | 9.00        | 23.00     | 23.00         | 3.00      | 14.00   | 14.00        | 17.00     | 13.00    | 24.00        |
| Irish-born                          | 13.20    | 23.80    | 49.30       | 21.10            | 13.70            | 5.20        | 13.20     | 28.20         | 0.00      | 22.70   | 35.20        | 14.00     | 0.00     | 18.90        |
| Italian-born                        | 0.00     | 0.00     | 8.40        | 9.20             | 0.00             | 63.70       | 22.80     | 0.00          | 94.80     | 0.00    | 10.50        | 15.80     | 26.70    | 11.30        |
| Canadian-born                       | 37.00    | 26.40    | 18.80       | 16.90            | 12.30            | 14.00       | 19.00     | 20.40         | 0.70      | 17.60   | 10.10        | 17.70     | 0.00     | 19.60        |
| Citizenship status, %               |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Naturalized citizen                 | 49.20    | 61.80    | 63.00       | 61.50            | 60.90            | 41.00       | 54.30     | 66.90         | 30.90     | 60.00   | 49.00        | 45.20     | 39.80    | 65.20        |
| With first papers                   | 13.20    | 9.60     | 11.20       | 9.90             | 9.20             | 11.00       | 10.30     | 10.10         | 10.00     | 10.00   | 12.80        | 12.40     | 13.20    | 9.00         |
| Aliens                              | 37.60    | 28.50    | 25.80       | 28.60            | 39.90            | 48.00       | 35.40     | 23.00         | 59.10     | 30.00   | 38.20        | 42.40     | 47.00    | 25.80        |
| Unemployed, %                       | 3.70     | 4.40     | 8.30        | 7.00             | 6.40             | 9.90        | 7.50      | 5.20          | 17.70     | 8.00    | 9.40         | 14.10     | 6.95     | 5.40         |
| Criminal delinquency, %             |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| 7–16 y                              | 19.40    | 13.50    | 31.00       | 14.00            | 9.30             | 34.10       | 11.70     | 12.80         | 30.80     | 18.40   | 25.90        | 27.40     | 38.40    | 18.80        |
| 17–20 y                             | 70.10    | 66.20    | 118.40      | 77.00            | 75.40            | 87.10       | 73.10     | 68.80         | 102.00    | 94.00   | 130.00       | 113.90    | 129.80   | 61.40        |
| Public assistance, %                |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Unemployment                        | 1.50     | 2.50     | 12.10       | 6.30             | 3.80             | 17.20       | 14.30     | 5.80          | 20.40     | 10.30   | 13.30        | 23.40     | 12.30    | 4.70         |
| Dependant aid                       | 1.80     | 2.00     | 6.30        | 3.10             | 1.80             | 6.00        | 8.80      | 2.10          | 11.20     | 6.60    | 5.60         | 16.70     | 8.70     | 1.20         |
| Mothers' aid                        | 0.03     | 0.28     | 1.20        | 0.70             | 0.50             | 1.00        | 0.60      | 0.50          | 1.70      | 0.70    | 1.00         | 0.30      | 1.00     | 0.30         |
| Old-age assistance                  | 1.30     | 1.10     | 2.90        | 1.60             | 1.50             | 1.30        | 1.50      | 1.50          | 0.90      | 2.40    | 1.80         | 5.80      | 1.70     | 0.90         |
| Housing type, %                     |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Own home                            | 10.10    | 21.30    | 26.30       | 28.00            | 35.00            | 28.80       | 46.90     | 34.20         | 7.10      | 17.80   | 26.90        | 16.10     | 12.30    | 52.70        |
| Rent                                | 89.90    | 78.70    | 73.70       | 72.00            | 65.00            | 71.20       | 53.10     | 65.80         | 92.90     | 82.20   | 73.10        | 83.90     | 87.70    | 47.30        |
| In lodgings <sup>a</sup>            | 9.20     | 0.30     | 0.30        | 0.30             | 0.30             | 0.30        | 0.30      | 0.30          | 5.90      | 0.30    | 0.30         | 18.30     | 7.40     | 0.30         |
| Median monthly income, \$           | 54.60    | 52.90    | 21.70       | 39.80            | 42.30            | 25.50       | 35.90     | 40.90         | 24.10     | 33.30   | 24.20        | 27.60     | 29.00    | 46.00        |
| Mortality                           |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Infant mortality rate <sup>b</sup>  | 64.50    | 50.50    | 85.70       | 59.60            | 49.00            | 66.80       | 47.50     | 48.20         | 62.70     | 64.40   | 74.40        | 73.10     | 48.80    | 46.20        |
| Tuberculosis incidence <sup>c</sup> | 125.00   | 104.60   | 172.00      | 112.60           | 87.60            | 133.80      | 90.60     | 101.80        | 160.10    | 171.50  | 168.60       | 359.40    | 149.00   | 113.80       |
| Tuberculosis mortality <sup>c</sup> | 34.50    | 35.90    | 76.90       | 43.40            | 31.60            | 45.60       | 33.00     | 47.70         | 55.50     | 73.30   | 85.40        | 154.60    | 52.20    | 48.50        |
| <b>Health of Boston</b>             |          |          |             |                  |                  |             |           |               |           |         |              |           |          |              |
| Stroke <sup>d</sup>                 | 12.80    | 25.10    | 28.90       | 29.00            | 20.20            | 16.00       | 19.90     | 14.10         | 16.30     | 28.10   | 23.30        | 19.10     | ...      | 22.10        |
| Coronary heart disease <sup>d</sup> | 79.80    | 112.50   | 151.20      | 136.90           | 109.70           | 125.80      | 108.90    | 92.30         | 76.30     | 149.10  | 172.80       | 133.80    | ...      | 108.00       |

Note. The terminology used is that of the historical reports.

<sup>a</sup>"In lodging" refers to renting a room within the landlord's home, whereas "renting" indicates separate accommodations.

<sup>b</sup>Average infant mortality rates 1930–1933 per 1000 live births.

<sup>c</sup>Average tuberculosis incidence and mortality rates 1930–34 per 100 000 population.

<sup>d</sup>Age-standardized stroke and coronary heart disease rates per 100 000 1994–1998.

cantly related to proportion Irish born in Charlestown and South Boston at the 1930 census. No relationship with present-day rates is seen for Italians, although Italians were more concentrated in fewer census tract areas.

Taken together, these findings indicate that socioeconomic circumstances in early life are likely to have played a role in the etiology of cardiovascular disease regardless of ethnic ori-

gin, in keeping with previous findings.<sup>1–4</sup> However, a contrast between the health and socioeconomic circumstances of the Irish and Italians indicates some residual factors as well. This detailed social portrait in 1 city corroborates findings at the national level mentioned previously (Table 1)—that some ethnic groups are more at risk of cardiovascular diseases than others. To the extent that the Irish were disad-

vantaged, a relationship between childhood material deprivation and later health outcomes existed. However, this association between being a member of an Irish American community and cardiovascular disease within a single city echoes the pattern of high rates seen in other regions with significant Irish populations.<sup>7,8</sup> In Ireland itself,<sup>49</sup> infant mortality rates during the 1930s were only weakly related to



**TABLE 3—Pearson Correlations and P Values for Association Between (a) Measures of Socioeconomic Status and Rates of Infant Mortality in 1930–1933 and Tuberculosis Incidence and Tuberculosis Mortality in 1930–1934<sup>33,34</sup> and (b) Rates of Stroke and Coronary Heart Disease in 1994–1998<sup>35</sup>: Boston**

| Measure of Socioeconomic Status   | (a) 1930–1934    |      |                        |      |                        |      | (b) 1994–1998 |      |                        |      |
|-----------------------------------|------------------|------|------------------------|------|------------------------|------|---------------|------|------------------------|------|
|                                   | Infant Mortality | P    | Tuberculosis Incidence | P    | Tuberculosis Mortality | P    | Stroke        | P    | Coronary Heart Disease | P    |
| Own home                          | -.486            | .078 | -.604*                 | .022 | -.393                  | .164 | .093          | .762 | -.016                  | 9.57 |
| Rent home                         | -.486            | .078 | -.604*                 | .022 | -.393                  | .164 | .093          | .762 | -.016                  | 9.57 |
| In lodgings <sup>a</sup>          | .204             | .485 | .413                   | .142 | .248                   | .393 | -.524         | .066 | -.375                  | .206 |
| Median monthly rent               | -.587*           | .027 | -.692**                | .006 | -.701**                | .005 | -.176         | .566 | -.467                  | .108 |
| Unemployed                        | .552*            | .041 | .648*                  | .012 | .626*                  | .017 | .044          | .887 | .352                   | .239 |
| Criminal delinquency, 7–16 y      | .538*            | .047 | .758**                 | .002 | .618*                  | .019 | -.104         | .734 | .231                   | .448 |
| Criminal delinquency, 17–20 y     | .644*            | .013 | .736**                 | .003 | .688**                 | .007 | .236          | .437 | .626*                  | .022 |
| Receiving unemployment assistance | .341             | .233 | .538*                  | .047 | .560*                  | .037 | -.082         | .789 | .247                   | .415 |
| Receiving old-age assistance      | .498             | .070 | .544*                  | .044 | .549*                  | .042 | .393          | .184 | .756**                 | .003 |
| Receiving dependant aid           | .295             | .306 | .557*                  | .039 | .517                   | .058 | .017          | .957 | .259                   | .394 |
| Receiving mothers' aid            | .337             | .239 | .421                   | .134 | .436                   | .119 | .260          | .392 | .359                   | .228 |

Note. The terminology used is that of the historical reports.

<sup>a</sup>"In lodging" refers to renting a room within the landlord's home, whereas "renting" indicates separate accommodations.

\* $P < 0.01$ ; \*\* $P < 0.001$ .

present-day adult coronary heart disease rates ( $r=0.26$  for men and  $0.29$  for women). Nevertheless, when infant mortality rates for selected urban and rural areas of Ireland<sup>50</sup> and countries to which Irish people migrated are ranked, a strong influence of urban deprivation on these patterns is clearly apparent (Table 4). In the 1930s, infant mortality rates were lowest in rural Ireland and highest in urban Dublin, with intermediate rates in the American cities to which the Irish immigrated in large numbers; Boston, as discussed in the section on community networks and health, presents a wide variation. Two processes must be understood before interpreting the relationship between (a) infant mortality rate, ethnicity, and urban deprivation and (b) later-life health: the effect of disadvantage on the health of Irish immigrants and also the possibility that something particular about the Irish as an ethnic group causes them to continue to incur excessive risk even as they become more affluent.

## DISCUSSION

### Social Capital and the Irish

The recent focus on social capital as a potentially important explanatory pathway between

**TABLE 4—Estimates of Infant Mortality per 1000 Live Births, by Area: Boston Neighborhood Study<sup>33,34</sup> and Irish Free State Vital Statistics Report,<sup>50</sup> 1930–1933**

|  | Year      | Estimated Infant Mortality |
|--|-----------|----------------------------|
| Irish Free State (urban) <sup>a</sup>  | 1931      | 90                         |
| Liverpool, England <sup>a</sup>        | 1930      | 82                         |
| Edinburgh, Scotland <sup>a</sup>       | 1931      | 82                         |
| Charlestown, Boston <sup>b</sup>       | 1930–1933 | 86                         |
| Belfast, Northern Ireland <sup>a</sup> | 1930      | 70                         |
| All Boston <sup>b</sup>                | 1930–1933 | 61                         |
| London, England <sup>a</sup>           | 1930–1933 | 59                         |
| Irish Free State (rural) <sup>a</sup>  | 1931      | 56                         |
| West Roxbury, Boston <sup>b</sup>      | 1930–33   | 46                         |

<sup>a</sup>Irish Free State report.

<sup>b</sup>Boston Neighbourhood study.

relative disadvantage and ill health is particularly apposite in this situation, because the cardiovascular health experiences of the Irish and the Italians contrasts so sharply during the period of their assimilation into the American way of life. Concepts of trust, reciprocity, net-

works, and social support are all inherent to the social capital paradigm. Putnam has focused on the importance of civic participation for community well-being and cohesion. In *Bowling Alone*, he elaborated on this concept in the context of the US by positing 2 patterns of civic participation: "Machers," who build up and take part in community organizations, and "Schmoozers," who socialize and contribute positively to community networks.<sup>42</sup> Similarly, Wilkinson has cited both the British nation during World War II and the Roseto, Pennsylvania, community during the height of the coronary heart disease epidemic in the United States as examples of how social cohesion and an egalitarian community structure not only enhanced well-being and cohesion but protected health.<sup>51</sup>

The Irish who immigrated to America during the 19th and 20th centuries were extremely materially deprived, and they had a tough, socially equivocal, and politically controversial history. Handlin,<sup>11</sup> in a landmark text, described their assimilation over 2 centuries, and, in particular, documented the prejudice they encountered in this country. The Irish were caricatured as feckless, drunken, and fatalistic for a variety of reasons including their adherence to the Roman Catholic religion in a society dominated by nonconformist Protestants. In reality, these immigrants were prepared to work under conditions so appalling that even Black slaves were not permitted to labor under them (being judged by their cynical owners to be too economically valuable to be risked).<sup>12</sup> Large numbers of Irish women found their independence as housemaids and supported families at home in Ireland.<sup>14,15</sup> What these people particularly wished to avoid was the grinding labor of subsistence farming that they had left behind, and for this, too, they were criticized by demographers for not taking up farming.<sup>41</sup>

Many social factors influenced the rate of assimilation of various ethnic groups into the United States. We know, for instance, that patterns of education differed for the Irish, Jews, Italians, and Blacks.<sup>52</sup> Irish immigrants to the United States were also accused of not valuing education as much as other immigrant groups did, but this accusation stemmed from a singular failure to acknowledge the context of Irish sociopolitical history. In 1981, Sowell bizarrely asserted that the apparent lack of interest in education he observed in Irish immigrants was

a vestige of an ancient Celtic culture that was “hostile to literacy”<sup>53</sup> and that Ireland was the only Western country that did not build a university during the Middle Ages. In fact, the historical record clearly shows that the manuscripts of Irish monastic scholars almost certainly saved the remnants of Greco-Roman culture for posterity.<sup>54</sup> The Irish preserved their cultural identity through religious belief and the Gaelic language. The strongly religious Irish immigrants in early-twentieth-century US cities, therefore, favored denominational schools but were not necessarily as interested in leaving blue-collar work situations and communities as other immigrants were,<sup>52</sup> in part perhaps because of their strong social and community identity.

Many of the values prominent among Irish people are highly consistent with notions of social support and social capital. The Irish fleeing the famine came from a country in which the first mass movement of modern history, an almost classic example of social capital in practice, originated—the Catholic Emancipation movement of Daniel O’Connell,<sup>12,14</sup> which helped achieve the right to full social and political participation by Catholics in Ireland in 1829. This emancipation movement exemplifies a phenomenon of cross-class support for centrist, charismatic leaders that still continues today but that also has concealed serious economic inequality. Emancipation itself perpetuated a class distinction among rural tenant farmers by raising the land-value threshold of those entitled to vote.<sup>14</sup> Nor could this mass populism stem the horror of the famine itself, which in very large measure was directly attributable to British economic policy at the time. Contemporary interpretations by Putnam and others<sup>45–47,51</sup> of the importance of social networks and support in promoting and maintaining health therefore present the case of the Irish as a paradox.

Although initially despised as an ethnic group, the Irish became one of the most highly successful social networking groups in the United States,<sup>15</sup> contributing constructively to the political and cultural life of their adopted country from the period of the American Revolution onward.<sup>13</sup> In cities such as Boston, Chicago, and New York, the Irish have formed the backbone of local politics and municipal services. They were joiners of societies, particularly ones associated with Catholicism such as the

Knights of Columbus, and, as Gamm pointed out, their parish networks were so strong in many areas that they were more reluctant than other immigrant groups to join the urban exodus of the 1950s and later.<sup>48</sup> Coogan represents just one of many commentators and social historians to have chronicled these developments, and, as he noted, “in South Boston the Irish look after their own.”<sup>15</sup> It is instructive that John F. Kennedy’s Pulitzer prize-winning book was calculatedly devoted to aspects of heroic citizenship.<sup>55</sup> However, as is well documented, this community solidarity possessed a dark side. More recently, Ignatiev<sup>12</sup> described numerous examples of how the Irish, in the course of their social ascent, ruthlessly forged an identity separate from African Americans (who were also in extremely adverse social circumstances)—often, Ignatiev asserted, this resulted in racial prejudice and hostility. Also, political influence can be open to corruption on occasion.<sup>15</sup>

Nonetheless, the Irish are characterized by strong family and community support, church-going, and extensive civic participation. However, the Irish do not appear to have benefited from these stocks of social capital in health status terms. A present-day analysis of the relationships among deprivation, lifestyle, and voting patterns in Ireland shows the continuing importance of material indicators of deprivation.<sup>56</sup> The immigrant group with whom the Irish are most often compared in the United States, the Italians, has qualitatively similar families and networks. The Italians do indeed experience much less coronary heart disease,<sup>37,38,43,44</sup> but the assumption that this is a consequence of community social capital<sup>47,51</sup> is confounded by a number of other important factors.<sup>57</sup> For instance, it is quite clear from the historical data we review here that the community of Roseto, Pa—given such focus in the social capital literature as an apparent exception to the epidemic patterns of coronary heart disease at the time—was just one of many predominantly Italian communities with lower risks of heart disease compared to surrounding communities.<sup>57</sup> Must we therefore look to more traditional risk factors than social capital to explain the differences?

### The Ireland–Boston Diet Heart Study

The objective of the prospective Ireland–Boston Diet Heart Study was to recruit siblings

in Ireland and in the Boston area<sup>18,37</sup> to study diet and lifestyle in relation to cardiovascular disease. Initially, as a report using 1950 US census data on Boston ethnicities described, both Irish-born immigrants to the US and first-generation Irish Americans had much higher rates of cardiovascular disease and all-cause mortality than either US-born Bostonians or their counterparts in Ireland.<sup>37</sup> However, in 1985 there was no significant difference in cardiovascular disease events between recruited groups of Irish-born brothers, who either immigrated to Boston or stayed in Ireland, and US-born men of Irish parentage; but, the sample numbers were small.<sup>18</sup> The Ireland-dwelling brothers had higher calorie and carbohydrate intakes than did the US-dwelling brothers, who, nevertheless, were heavier, less physically active, and more likely to be smokers and drinkers. Saturated fat intake was not different between the 2 groups. It seems that secular factors may have confounded the original investigators’ intentions. Recruitment to the study occurred at the peak of the cardiovascular disease epidemic in the United States, but in the interval between recruitment and follow-up, rates had begun to fall dramatically. Conversely, rates began to rise in Ireland, so that by 1985, a crossover had occurred,<sup>58</sup> and rates of cardiovascular disease in Ireland have continued to be considerably higher among middle-aged people.<sup>5,6</sup> This crossover was observed in other contemporary cohort studies of Northern European immigrants as well, illustrating the critical importance of accounting for conditions in both country of origin and country of destination.<sup>59</sup>

### Lifestyle Influences on Cardiovascular Disease

The National Nutrition Surveillance Centre in Ireland has examined dietary patterns that emerged over the period since the Irish Famine.<sup>60–63</sup> The contemporary Irish diet now shows major social variation, reflected in both nutrient and food intake, consistent with emerging inequalities in rates of chronic disease.<sup>62</sup> The estimates of diet composition from a series of studies of dietary intake from 1863 to 1998 are summarized in Table 5. Fat intake rose consistently, in keeping with the upward trend in cardiovascular disease rates, from a strikingly low baseline. Unlike the Italians, the Irish were not consumers of monounsaturated

**TABLE 5—Estimates of Dietary Composition Among Irish People Since 1863: National Nutrition Surveillance Centre<sup>60,61,62</sup>**

| Year and Source of Data  | % Protein | % Fat | % Carbohydrate |
|--|-----------|-------|----------------|
| 1863 UK Dietary Survey   | 11        | 9     | 79             |
| 1905 UK survey on consumption and cost of food in workmen's families | 11        | 24    | 66             |
| 1936 Food Consumption Survey   | 12        | 29    | 59             |
| 1948 National Nutrition Survey                                       | 13        | 30    | 57             |
| 1961 Irish Statistical Bulletin                                      | 17        | 29    | 54             |
| 1971 Irish Statistical Bulletin                                      | 19        | 34    | 47             |
| 1990 National Nutrition Survey                                       | 15        | 36    | 47             |
| 1998 Survey of Lifestyles, Attitudes and Nutrition National Survey   | 17        | 34.5  | 46.5           |

Note. Data cited to other organizations within the table was collated in the National Nutrition Surveillance Centre reports.

Dublin, Republic of Ireland. At the time of the study, Joseph Tay was with the Department of Health Promotion, National University of Ireland, Galway. John Lynch and Sam Harper are with the Center for Social Epidemiology and Population Health, University of Michigan, Ann Arbor.

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## Contributors

C. Kelleher contributed to the collection, analysis, and interpretation of data. J. Lynch contributed to data analysis and interpretation of findings. S. Harper contributed to the collection of US Census archival documents and to their interpretation and analysis. J. Tay contributed to the analysis and interpretation of the Boston-area data. G. Nolan contributed to the interpretation of historical dietary data.

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No protocol approval was needed for this study.

## References

1. Lynch JW, Davey Smith G. A life course approach to chronic disease epidemiology. *Annu Rev Public Health*. In press.
2. Kuh D, Ben-Shlomo Y, eds. *A Life Course Approach to Chronic Disease Epidemiology*. 2nd edition, Oxford, England: Oxford University Press; 2004.
3. Kuh D, Hardy R. *A Life-Course Approach to Women's Health*. Oxford, England: Oxford University Press; 2002.
4. Robinson R. The foetal origins of adult disease. *BMJ*. 2001;322:375–376.
5. Department of Health and Children. *Building Healthier Hearts. The Report of the Cardiovascular Strategy Group*. Dublin, Ireland: Government Publications Office; 1999.
6. Department of Health and Children. *Health Statistics Report*. Dublin, Ireland: Government Publications Office; 2000.
7. Harding S, Balarajan R. Patterns of mortality in second-generation Irish living in England and Wales: longitudinal study. *BMJ*. 1996;312:1389–1392.
8. Harding S, Balarajan R. Mortality of third generation Irish people living in England and Wales: longitudinal study. *BMJ*. 2001;322:466–467.
9. Harrison L, Carrhill R, Sutton M. Consumption and harm-drinking patterns of the Irish, the English and the Scots in England. *Alcohol Alcoholism*. 1993;28:715–723.

fats, fruits, and other vegetables. The Irish population thrived on a peculiarly (by European standards) high-carbohydrate diet primarily because of their dependence on the potato (Table 5). It has been documented by Diner,<sup>64</sup> in an authoritative historical review of the eating patterns of Italian, Irish, and Jewish immigrants to the United States, that the Irish were more likely than other immigrant groups to adopt the prevailing diet and to adopt it more rapidly and completely. Immigrant groups for whom cuisine was culturally central, such as the Italians and the Jews, did eat differently from the Anglo-German mainstream, with its heavy reliance on meat and a relatively high salt and fat intake. Diner singled out isolated communities like Roseto, Pa, that consumed more cardioprotective products such as olive oil. In some instances, the Irish (for whom the memories of the famine were vivid) even established dining clubs at which to eat anything but their traditional fare. Conceivably, they may have been especially unprepared, in genetic terms, for the high-saturated fat diet they encountered and embraced so enthusiastically in the United States. Celiac disease is extraordinarily common in Ireland, and, arguably, gluten intolerance would have persisted in a population with relatively low exposure to grains and cereals, especially if the predisposing human leukocyte antigen phenotype carried other selective advantages.<sup>65</sup> A major selection effect therefore may have occurred in famine survivors on this high-carbohydrate diet, both before and after the famine in Ireland. These lines of evidence

related to diet are consistent with a particular genetic predisposition to heart disease persisting across generations.

## CONCLUSIONS

This study has synthesized information from the historical record and across several past and current epidemiological studies. There is convincing evidence that Irish immigrants to the United States had inordinate risk of cardiovascular disease for at least 2 generations. This risk appears to have been mainly related to material deprivation in both early and later life and aggravated by an adverse diet encountered on arrival to the United States. Additionally, the social deprivation of the Irish had an important psychosocial component, characterized by the often intense hostility, prejudice, and discrimination toward them. Nevertheless, the Irish had the support of strong religious ties, community networks, and families. Contrasting the different cardiovascular health profiles of two immigrant groups—the similar social circumstances (high material deprivation and high social capital) but the different dietary patterns of Irish and Italian Americans—suggests that in the face of powerful behavioral factors, enhanced social capital may be relatively less important to population health than previously proposed. ■

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10. Hout M, Goldstein JR. How 4.5 million Irish immigrants became 40 million Irish-Americans: demographic and subjective aspects of the ethnic composition of white Americans. *Am Sociol Rev.* 1994;59:64–82.
11. Handlin O. *Boston Immigrants (1790–1880): A Study in Acculturation.* Cambridge, Mass: Belknap Press; 1959.
12. Ignatiev N. *How the Irish Became White.* New York, NY: Routledge; 1995.
13. Cahill RE. *The Old Irish of New England.* Peabody, Mass: Chandler-Smith; 1985.
14. Keneally T. *The Great Shame. A Story of the Irish in the Old World and the New.* London, England: Chatto & Windus; 1998.
15. Coogan TP. *Wherever Green Is Worn. The Story of the Irish Diaspora.* London, England: Hutchinson; 2000.
16. Farley R. The new census question about ancestry—what did it tell us? *Demography.* 1991;28:411–429.
17. The Boston Health League. A study of infant mortality in Boston for 1932 by health areas and census tracts. *N Engl J Med.* 1933;209:858–860.
18. Kushi LH, Lew RA, Stare FJ, et al. Diet and 20-year mortality from coronary heart disease. The Ireland-Boston Diet Heart Study. *N Engl J Med.* 1985;312:811–818.
19. Condran GA, Crimmins E. A description and evaluation of mortality data in the federal census: 1850–1900. *Historical Methods.* 1979;12(1):1–23.
20. Gibson CJ, Lennon E. *Historical Census Statistics on the Foreign-Born Population of the United States: 1850–1990.* Washington, DC: US Bureau of the Census; 1999. Population division working paper No. 29.
21. US Census Office. *Mortality Statistics of the Seventh Census of the United States, 1850.* Washington, DC: US Government Printing Office; 1855.
22. US Census Office. *Statistics of the United States (Including Mortality, Property etc.) in 1860: Compiled From the Original Returns and Being the Final Exhibit of the Eighth Census.* Washington, DC: US Government Printing Office; 1866.
23. *Ninth Census—Volume II. The Vital Statistics of the United States, Embracing the Tables of Deaths, Births, Sex and Age, to Which Are Added the Statistics of the Blind, Deaf and Dumb, the Insane and the Idiotic. From the Original Returns of the Ninth Census (June 1, 1870).* Washington, DC: US Dept of the Interior, US Census Office; 1872.
24. *Report on the Mortality and Vital Statistics of the United States as Returned at the Tenth Census (June 1, 1880).* Washington, DC: US Dept of the Interior, US Census Office; 1885.
25. *Report on Vital and Social Statistics in the United States at the Eleventh Census: 1890. Part 1: Analysis and Rate Tables.* Washington, DC: US Dept of the Interior, US Census Office; 1896.
26. Census Reports. Volume 111. *Twelfth Census of the United States, Taken in the Year 1900. Vital Statistics. Part 1: Analysis and Rate Tables.* Washington, DC: US Dept of the Interior: US Census Office; 1902.
27. *Mortality Statistics 1910. Eleventh Annual Report.* Washington, DC: US Dept of Commerce, US Bureau of the Census; 1913.
28. *Mortality Rates 1910–1920: With Population of the Federal Censuses of 1910 and 1920 and Inter-Censal Estimates of Population.* Washington, DC: US Dept of Commerce, US Bureau of the Census; 1923.
29. *Fifteenth Census of the United States: 1930. Population. Volume II. General Report. Statistics by Subjects.* Washington, DC: US Dept of Commerce, US Bureau of the Census; 1933.
30. *Sixteenth Census of the United States: 1940. Population. Nativity and Parentage of the White Population. Country of Origin of the Foreign Stock.* Washington, DC: US Dept of Commerce, US Bureau of the Census; 1943.
31. *1990 Census of Population. Ancestry of the Population in the United States.* Washington, DC: US Dept of Commerce, Economics and Statistics Administration, US Bureau of the Census; 1993.
32. Dublin LI, Baker GW. Mortality of race stocks in Pennsylvania and New York, 1910. *Q Publication of Am Stat Assoc.* 1920;17:13–44.
33. Boston Council of Social Agencies. *Social Statistics by Census Tracts in Boston. A Method of Neighborhood Study.* Boston, Mass: Bureau of Research and Studies; 1933.
34. Boston Council of Social Agencies. *Social Statistics by Census Tracts in Boston.* Vol. 2. Boston, Mass: Bureau of Research and Studies; 1935.
35. Health of Boston 2001. Boston, Mass: Boston Public Health Commission Research and Technology Services; 2001. Also available at <http://www.bphc.org/orhads/hob2001>. Accessed November 2001.
36. Trulson MF, Clancy RE, Jessop WJ, Childers RW, Stare FJ. Comparisons of siblings in Boston and Ireland. *J Am Diet Assoc.* 1964;45:225–229.
37. Stamler J, Kjelsberg M, Hall Y. Epidemiologic studies on cardiovascular–renal diseases, III: analysis of mortality by age–sex–nationality. *J Chron Dis.* 1960;12:464–475.
38. Rosenwaike I, Hempstead K. Differential mortality by ethnicity: foreign-born Irish, Italians and Jews in New York City, 1979–1981. *Soc Sci Med.* 1989;29:885–889.
39. Leon D, Davey Smith G. Infant mortality, stomach cancer, stroke, and coronary heart disease: ecological analysis. *BMJ.* 2000;320:1705–1706.
40. Lawlor DA, Davey Smith G, Leon D, Sterne J, Ebrahim S. Secular trends in mortality by stroke subtype over the twentieth century: resolution of the stroke-coronary heart disease paradox? *Lancet.* 2002;360:1818–1823.
41. Carpenter N. *Immigrants and Their Children. A Study Based on Census Statistics Relative to the Foreign Born and the Native White of Foreign or Mixed Parentage.* Washington, DC: US Dept of Commerce, US Bureau of the Census; 1927.
42. Winslow EA, Wang PL. The relation between changes in nationality stock and increasing death rates in adult life. *Am J Hygiene.* 1931;14:79–88.
43. Calabresi M. The relation of country of origin to mortality for various causes in New York State. *Hum Biol.* 1945;17:340–367.
44. Kitagawa EM, Hauser PM. Differential mortality by race and other factors. In: *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology.* Cambridge, Mass: Harvard University Press; 1973:104–107.
45. Putnam RD. *Bowling Alone. The Collapse and Revival of American Community.* New York, NY: Touchstone; 2000.
46. Kawachi I, Kennedy BP. *The Health of Nations. Why Inequality Is Harmful to Your Health.* New York, NY: New Press; 2002.
47. Kawachi I, Kennedy B, Wilkinson RG, eds. *The Society and Health Population Reader. Income Inequality and Health.* New York, NY: The New Press; 1999.
48. Gamm G. *Urban Exodus: Why the Jews Left Boston and the Catholics Stayed.* Cambridge, Mass: Harvard University Press; 1999.
49. Pringle DG. Hypothesized foetal and early life influences on adult heart disease mortality: an ecological analysis of data for the Republic of Ireland. *Soc Sci Med.* 1998;46:683–693.
50. Irish Free State. *Annual Report of the Registrar General for the Year 1931.* Dublin, Ireland: Dept of Local Government and Public Health; 1932.
51. Wilkinson RG. *Health and Society: The Afflictions of Inequality.* London, England: Routledge; 1996.
52. Perlmann J. *Ethnic Differences. Schooling and Social Structure Among the Irish, Italians, Jews and Blacks in an American City, 1880–1935.* New York, NY: Cambridge University Press; 1988.
53. Sowell T. *Ethnic America: A History.* New York, NY: Basic Books; 1981.
54. Cahill T. *How the Irish Saved Civilization. The Untold Story of Ireland's Heroic Role From the Fall of Rome to the Rise of Medieval Europe.* New York, NY: Doubleday; 1995.
55. Kennedy JF. *Profiles in Courage.* London, England: Hamish Hamilton; 1964.
56. Kelleher CC, Timoney A, Friel S, McKeown D. Indicators of deprivation, voting patterns, and health status at area level in the Republic of Ireland. *J Epidemiol Community Health.* 2002;56:36–45.
57. Lynch JW, Davey Smith G. Rates and states: reflections on the health of nations. *Int J Epidemiol.* 2003;32:663–670.
58. Shelley E, O'Reilly O, Mulcahy R, Graham I. Trends in mortality from cardio-vascular diseases in Ireland. *Ir J Med Sci.* 1991;160:5–9.
59. Feinleib M, Lambert PM, Zeiner-Henriksen T, Rogot E, Hunt BM, Ingster-Moore L. The British-Norwegian migrant study—analysis of parameters of mortality differentials associated with angina. *Biometrics.* 1982;38(suppl):55–74.
60. Newell J, Nolan G, Kelleher C. *Nutrition Surveillance in Ireland 1993.* Galway, Ireland: National Nutrition Surveillance Centre, University College Galway; 1993.
61. Friel S, Nolan N. *Changes in the Food Chain Since the Time of the Great Irish Famine.* Galway, Ireland: National Nutrition Surveillance Centre, University College Galway; 1995.
62. Kelleher C, Friel S, Nolan G, Forbes B. Effect of social variation on the Irish diet. *Proc Nutr Soc.* 2002;61:527–536.
63. Friel S, NicGabhainn S, Kelleher CC. *Main Results of the National Health and Lifestyle Surveys, SLAN and HBSC.* Dublin, Ireland: Centre for Health Promotion Studies and Dept of Health and Children; 1999.
64. Diner HR. *Hungering for America. Italian, Irish and Jewish Foodways in the Age of Migration.* Cambridge, Mass: Harvard University Press; 2001.
65. Cronin CC, Shanahan F. Why is celiac disease so common in Ireland? *Perspect Biol Med.* 2001;44:342–352.



# Socioeconomic Disadvantage, Parenting Responsibility, and Women's Smoking in the United States

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Smoking has been identified as one of the major preventable causes of morbidity and premature mortality.<sup>1</sup> The increasing proportion of women in the smoking population has heightened the importance of understanding smoking behavior within this group. Smoking among women of reproductive age has been of special interest, because such smoking influences not only the health of the women themselves but also that of their children. In part as a result of knowledge about the harmful effects of maternal smoking on the fetus and young child and also as a result of intensive prevention and cessation interventions for pregnant women, smoking prevalence among this population is lower than that in the general population.<sup>2</sup> However, substantial differences remain in smoking prevalence by educational attainment and by race/ethnicity. Among pregnant women, the prevalence of smoking is 2.8% for those with a college degree, compared with 27.0% for those who did not graduate from high school.<sup>3</sup> Several smoking-cessation intervention studies have found lower rates of smoking-cessation and higher rates of relapse among less-educated and lower-income women.<sup>4–6</sup> Despite overall lower rates of smoking among pregnant women and mothers of young children, the material and social environments of mothers may also affect their smoking behavior.

Parenting can provide a great sense of accomplishment but may also be stressful—caring for children, especially children of preschool age, requires considerable time and energy.<sup>7,8</sup> Although daily chores related to child rearing may not be burdensome, their cumulative effect can, over a period of time, be experienced as a chronic strain, especially when they are combined with low levels of resources and other stressors such as poverty. Several epidemiological studies report that mental health is worse among mothers of young children than among mothers without

young children.<sup>7,9–13</sup> Naerde et al.<sup>7,14</sup> found that problems with child care arrangements combined with stressful work are associated with poor mental health among mothers of young children.

Parenting stresses associated with rearing young children might, however, be modified by the resources available to parents. Mothers with higher family incomes have been shown to exhibit better parenting behavior.<sup>15</sup> A longitudinal study of mothers of children aged 0–5 years found that child care responsibilities, such as making day-care arrangements and combining work and child care, were associated with maternal mental distress.<sup>7</sup> The same study also showed that social support with child care arrangements appeared to be associated with maternal mental well-being.<sup>7</sup> Mothers who received social support from public health nurses or from experienced volunteer mothers showed better family dynamics, parenting skills, and maternal self-esteem.<sup>16–18</sup> Mothers with greater support were significantly more positive in their attitudes and behavior, and social support moderated the adverse effects of stress on mothers' life satisfaction and behavior.<sup>19,20</sup> The prevalence of depressive episodes was higher among mothers without social support than among those with support.<sup>21</sup>

**Objectives.** We carried out analyses of smoking in relation to poverty and child care responsibility among women aged 18–54 years residing in the United States.

**Methods.** With data from the Behavioral Risk Factor Surveillance System, we assessed the interaction effects of poverty and living with young children on maternal smoking behavior among 61 700 women aged 18–54 years in 4 different racial/ethnic groups.

**Results.** For non-White racial/ethnic groups, the prevalence of smoking among women with small children in the household was lower than that among women without small children. However, White women were more likely to smoke if they were poor and living with small children (odds ratio = 1.14, 95% confidence interval = 1.03, 1.26).

**Conclusions.** These results suggest that child care responsibility confers an increased risk of smoking among low-income White women. (*Am J Public Health.* 2004;94:2170–2176)

Smoking has been reported to be a means of reducing stress, especially among women.<sup>22–24</sup> Some qualitative studies of smoking among low-income women suggested that smoking is a way of coping with daily hassles and stress.<sup>25–29</sup> Graham reported that child care responsibilities, which are disproportionately borne by women, were associated with cigarette smoking in a study of White working-class women in the United Kingdom. Mothers living in conditions of material hardship often identified the time spent smoking cigarettes as the only time they had for themselves and cigarette smoking as the only activity they did for themselves. According to Graham, smoking might help these mothers cope with the stress and monotony of daily life.<sup>25,26</sup> Greaves suggests that, when mothers feel overwhelmed by too many demands, they may turn to a cigarette as a way of temporarily distancing themselves from their children, a strategy that helps them to manage their anger and avoid acting upon it (e.g., in the form of physical abuse).<sup>27</sup> Stewart et al.<sup>28,29</sup> also reported that disadvantaged women in Canada considered their smoking to be linked with their daily lives in poverty, isolation, and care giving and used smoking as a mechanism for coping with stress and associated negative emotions.

Our study explored the combined effects of parenting responsibilities and limited material circumstances on smoking among women. Previous research on parenting and smoking has been limited to qualitative studies from the United Kingdom and Canada and to studies with small sample sizes. Because of cumulative evidence and widespread knowledge of the harmful effects on children of maternal smoking during pregnancy and childhood, overall smoking prevalence is low during pregnancy,<sup>3</sup> and raising young children also is a protective factor against tobacco use among women. However, on the basis of qualitative studies on smoking among low-income women, we hypothesized that the effect of parenting on smoking differs according to the socioeconomic status (SES) of the mother. We assume that high-SES women have more resources for coping with stress related to parenting, an advantage that enables them to avoid smoking, whereas low-SES women lack such resources. Parenting under disadvantaged circumstances is a factor that may contribute to continued tobacco use among women, despite their knowledge of its harmful effects. Therefore, by testing the interaction effect of poverty and parenting on smoking, we investigated whether parenting actually increases the risk of tobacco use among low-SES women.

Thanks to a large sample size (the Behavior Risk Factor Surveillance System [BRFSS] population), we were able to test whether previous study findings about low-income White women and smoking also apply to women of other racial/ethnic backgrounds. We performed separate analyses by racial/ethnic group, because smoking prevalence historically has varied by racial/ethnic group, and smoking behavior is considered to be influenced by cultural norms.<sup>30</sup>

## METHODS

### Sources of Data

The study was based on the 2000 BRFSS of the Centers for Disease Control and Prevention,<sup>31</sup> which consists of a representative sample of households by state in the United States. Each state selects an independent probability sample of its civilian, noninstitutionalized adult population 18 years of age or

older, using random-digit-dialing telephone survey techniques.

All 50 states, as well as the District of Columbia and Puerto Rico, participated in the BRFSS in 2000. The total sample size was 184 450. After we restricted the sample to women of reproductive age (18–54 years), the sample size was reduced to 73 457 (a 31.4% decrease by restricting to those aged 18–54 years, and an additional 42.0% decrease by excluding males). The final sample size of women who provided complete information on smoking status and other socioeconomic characteristics was 61 700. Although the income variable had many missing observations (11.3%), most other variables had only a few missing observations (<1.0).

### Outcome Measures of Tobacco Use

Self-reported smoking status was used as the measure of tobacco use. We defined smokers as those who reported current smoking. Self-report of smoking status was determined in response to the questions “Have you smoked at least 100 cigarettes in your entire life?” and “Do you smoke cigarettes every day, some days, or not at all?” “Current smokers” were defined as those who had smoked at least 100 cigarettes during their lifetime and who currently smoked cigarettes every day or some days. “Former smokers” were women who had smoked 100 cigarettes during their lifetime but who currently did not smoke cigarettes regularly, and “never smokers” were women who had not smoked at least 100 cigarettes and who did not smoke. Former and never smokers were combined into a nonsmoker category for these analyses.

### Sociodemographic Data

The BRFSS 2000 questionnaire also asked about individual characteristics. Parenting of young children was dichotomized as living with children who were younger than 5 years versus not living with young children. SES was measured by household income. To increase comparability across households of different sizes, we adjusted household income for household size by using a standard equivalization procedure (i.e., dividing the household income by the square root of the number of people in that household).<sup>32,33</sup> Household income was recalculated on the basis of midpoints of income categories, ad-

justed for family size. Income was then dichotomized as poor (<\$15 000) and nonpoor (≥\$15 000). We set the \$15 000 cut point at 150% of the 2000 US Census Bureau poverty threshold,<sup>34</sup> which is less than \$10 000 after adjustment for family size.

Age was examined as a continuous variable and was centered on the mean (36 years old). Race/ethnicity was grouped into 4 categories: non-Hispanic White, non-Hispanic Black, Hispanic, and “other.” Marital status was grouped into 4 categories: married, divorced/widowed/separated, never married, and member of an unmarried couple. Educational attainment was grouped into 4 categories: did not graduate from high school, high school graduate, some college or trade school, and college graduate. The number of children aged 5–17 years was calculated and truncated at 4 children because few households had 5 or more children.

### Statistical Analysis

Multilevel logistic regression procedures based on a logit-link function<sup>35,36</sup> were used to model the 2-level structure of 61 700 individual women (at level 1) nested within 50 US states (at level 2). Models were fitted with the MLwiN software<sup>37</sup> and second-order Penalized Quasilikelihood estimation procedures.<sup>38</sup> At the individual level, we analyzed data with and without extrabinomial variation to determine whether the binomial distributional assumption was supported.<sup>36,39,40</sup> Because the results showed that extrabinomial variation was not significantly different from 1, the level 1 variation was constrained to 1 (pure binomial variation) in all of the models reported.

To estimate the effect on smoking of the parenting of young children, we included individual predictors in the fixed part of the model while allowing for variation between states. We assessed the relationship between smoking and all of the individual predictors across all 50 US states. Models were built by sequentially adding covariates. First, the relation between raising young children and smoking was examined with control for age, number of children aged 5–17 years, educational attainment, marital status, and race/ethnicity. Next, interaction terms between poverty status (poor or nonpoor) and each

individual characteristic were added to the main effect model to determine whether the association of parenting and other characteristics differed by poverty status. When an individual characteristic was added, the likelihood ratio test for overall model fit and the Wald test for individual indicator variables were performed. Finally, we stratified the sample by racial/ethnic group and tested an interaction effect between parenting young children and poverty status within each stratum.

To account for design-based variation in probability of selection into the sample by

age, gender, and race, we weighted the data in all analyses with sampling weights provided by BRFSS 2000.

## RESULTS

Table 1 presents the characteristics of the study population. The sample was predominantly White (77.4%), the mean age was 36 years, and 35.4% of the sample had income levels that fell within the poor category (i.e., less than \$15 000). Blacks and Hispanics were much more likely than Whites to be

poor (43% of Blacks and 55% of Hispanics vs 19% of Whites). Of the 61 700 respondents in the sample, 24.9% were current smokers. Respondents living with young children (aged 0–4 years) were less likely to be current smokers than were women not living with young children (25.7% for no child vs 22.7% for 1 or more children). Rates of smoking were lower for Blacks, Hispanics, and “other” racial/ethnic groups than for Whites. Smoking rates also were lower among respondents with higher educational attainment and income.

**TABLE 1—Distribution of Sample by Individual Characteristics and Percentage of Current Smoking<sup>a</sup>: Behavioral Risk Factor Surveillance System (BRFSS) 2000**

|                                | No. (Unweighted) | Weighted Percentage <sup>b</sup> | Weighted Percentage <sup>b</sup> of current smoking |
|--------------------------------|------------------|----------------------------------|---|
| Age, y                         |                  |                                  |   |
| 18–24                          | 7148             | 11.6                             | 28.8  |
| 25–34                          | 17 036           | 27.6                             | 23.9  |
| 35–44                          | 20 395           | 33.1                             | 26.6  |
| 45–54                          | 17 121           | 27.8                             | 21.7  |
| No. of children aged 0–4 years |                  |                                  |   |
| None                           | 46 879           | 73.6                             | 25.7  |
| ≥ 1                            | 14 821           | 26.4                             | 22.7  |
| 1                              | 10 553           | 17.5                             | 23.5  |
| 2                              | 3 683            | 5.4                              | 20.6  |
| ≥ 3                            | 585              | 1.0                              | 20.7  |
| Race/ethnicity                 |                  |                                  |   |
| White, non-Hispanic            | 47 719           | 70.8                             | 27.3  |
| Black, non-Hispanic            | 5 998            | 11.2                             | 20.9  |
| Hispanic                       | 4 768            | 13.2                             | 17.7  |
| Other                          | 3 215            | 4.8                              | 19.4  |
| Marital status                 |                  |                                  |   |
| Married                        | 34 490           | 59.2                             | 19.9  |
| Divorced/separated/widowed     | 13 188           | 16.2                             | 36.4  |
| Never been married             | 12 026           | 20.5                             | 28.0  |
| Member of unmarried couple     | 1 996            | 4.1                              | 37.0  |
| Educational attainment         |                  |                                  |   |
| College graduate               | 19 861           | 30.3                             | 12.6  |
| Some college                   | 19 313           | 31.0                             | 25.4  |
| High school graduate           | 18 210           | 29.4                             | 33.4  |
| Less than high school graduate | 4 316            | 9.3                              | 36.6  |
| Income, \$                     |                  |                                  |   |
| < 15 000                       | 15 666           | 26.9                             | 32.8  |
| ≥ 15 000                       | 46 034           | 73.1                             | 22.0  |
| Total                          | 61 700           | 100.0                            | 24.9  |

<sup>a</sup>Sample was restricted to women aged 18–54 years.

<sup>b</sup>We used weighted percentages to account for differential response rates and design-based variation in probability of selection into the sample by age, gender, and race, with sampling weights provided by the BRFSS 2000.

### Non-Hispanic White Women

Table 2 shows the results of our adjusted model by poverty status as well as by racial/ethnic group. Among nonpoor White women, living with young children was inversely related to current smoking, whereas among poor White women, living with young children was positively associated with current smoking. In the nonpoor group, the odds of smoking among women living with 1 or more children of preschool age (0–4 years) was 0.9 times that of women not living with young children (95% confidence interval [CI]=0.8, 0.9). By contrast, the odds of smoking among poor White women who lived with children aged 0–4 years was 1.1 times that among women who did not live with young children (95% CI=1.0, 1.3).

### Non-Hispanic Black and Hispanic Women

Among Black and Hispanic women, living with young children uniformly decreased the odds of smoking, regardless of income level. For Black women with children, the odds of smoking were 0.7 (95% CI=0.6, 0.9) for nonpoor women and 0.9 (95% CI=0.7, 1.1) for poor women. For Hispanic women with children, the odds of smoking were 0.7 (95% CI=0.5, 0.9) for nonpoor women and 0.9 (95% CI=0.7, 1.1) for poor women. For non-White women, therefore, living with young children decreased the risk of smoking.

## DISCUSSION

Our study found a lower prevalence of smoking, for non-White racial/ethnic groups, among women with small children in the household than among women without small

**TABLE 2—Effect of Living With Young Children<sup>a</sup> on Smoking Prevalence Among Woman Aged 18–54 Years: Behavioral Risk Factor Surveillance System (BRFSS) 2000**

|                     | Nonpoor ( $\geq \$15\,000$ ) <sup>b</sup>              |                          | Poor ( $< \$15\,000$ ) <sup>b</sup>                    |                          |
|---------------------|--|--------------------------|--|--------------------------|
|                     | Weighted Percentage <sup>c</sup><br>of current smoking | OR <sup>d</sup> (95% CI) | Weighted Percentage <sup>c</sup><br>of current smoking | OR <sup>d</sup> (95% CI) |
| Total               | 22.0   | 0.85 (0.80, 0.90)        | 32.8   | 1.07 (0.99, 1.16)        |
| White, non-Hispanic | 23.4   | 0.86 (0.80, 0.92)        | 44.1   | 1.14 (1.03, 1.26)        |
| Black, non-Hispanic | 16.1   | 0.73 (0.56, 0.95)        | 27.4   | 0.89 (0.73, 1.09)        |
| Hispanic            | 18.5   | 0.70 (0.53, 0.91)        | 17.0   | 0.86 (0.69, 1.08)        |
| Other               | 15.4   | 0.87 (0.64, 1.17)        | 29.6   | 1.20 (0.88, 1.64)        |

Note. OR = odds ratio; CI = confidence interval.

<sup>a</sup>Women with 1 or more children aged 0–4 years living in the household, compared with women of the same income and race/ethnicity group with no children aged 0–4 years living in the household. Sample was restricted to women aged 18–54 years.

<sup>b</sup>Household income was equivalized to increase comparability across households of different size by division of midpoints of household income by the square root of the number of people in that household.

<sup>c</sup>We used weighted percentages to account for differential response rates and design-based variation in probability of selection into the sample by age, gender, and race/ethnicity, with sampling weights provided by the BRFSS 2000.

<sup>d</sup>The odds ratios were from a weighted logistic regression model for smoking that included individual-level age, number of children aged 5–17 years, marital status, educational attainment, and race/ethnicity. A random intercept was specified at the state level in each model.

children in the household. However, this inverse association between small children in the household and smoking was not apparent in low-income, non-Hispanic White women, who were more likely to smoke if they were living with small children.

However, we found no interaction effect of poverty status and parenting on smoking among non-White women. For Black and Hispanic women, raising young children had a protective effect on smoking, regardless of poverty status.

### Interaction Effects

For low-income White women, raising young children in the context of economic hardship amplified the risk of cigarette smoking. Our finding is consistent with qualitative findings reported by Graham et al.,<sup>26</sup> whose results were based on data from low-income White women in England. These women have multiple role demands<sup>28</sup> with fewer material and social resources than are available to more privileged women.<sup>26,29</sup>

Children influence women's smoking behavior. Parenting children has conflicting effects on smoking. On the one hand, the burdens of child care are often considered stressors associated with smoking or the relapse of smoking. On the other hand, because

of mothers' concerns about the effects of secondhand smoke, children are also a reason for women to quit smoking.<sup>41</sup>

Our study shows both positive and negative effects of children on mothers' smoking. According to Greaves, many women feel that smoking is useful for controlling emotion in a variety of situations. It helps women to quash negative feelings, dispel tension, or delay an emotional response.<sup>27</sup> In their in-depth interviews with disadvantaged women, Stewart et al. found that disadvantaged women who continued to smoke did so to cope with their immediate situation<sup>28</sup> and that coping was the women's principal explanation for their smoking behavior—they smoked to cope with the stress, chaos, and crises in their lives, including child care. These women also reported that smoking helped them cope with loneliness and isolation. Women in economically deprived circumstances suffered from loneliness and lack of social support. Cigarettes were used as a reward and for pleasure. Smoking provided a break from a monotonous, burdensome daily routine.<sup>28</sup>

Results of smoking-cessation interventions aimed at low-income women show that relapse rates are highest among low-income single White women.<sup>42</sup> Several smoking-cessation programs consisting of self-help

booklets, telephone contacts, and even systematic provision of motivational counseling improved neither prenatal cessation rates nor postpartum maintenance rates, and researchers have concluded that there is a need to develop innovative strategies to assist this group.<sup>43,44</sup> An intervention by community health centers that focused on improving low-income women's quality of life showed better smoking-cessation rates than those achieved with other intervention programs (38% vs 20%–25%).<sup>45</sup> The key goals were to integrate low-income women's social and economic circumstances into the program. When planning an intervention, one must acknowledge the association between the need of smoking as a source of relief of stress among low-income women and to recognize that lifestyle habits are influenced by personal choices, as well as by economic circumstances and social structures.<sup>46,47</sup>

### Racial/Ethnic Differences

Many researchers have pointed out the strong support networks among African American communities.<sup>48–52</sup> This extensive support system has been reported to be a Black cultural pattern.<sup>49</sup> Studies have found that racial/ethnic minorities are more likely to live in extended-family households.<sup>53–58</sup> Farley and Allen found, based on 1980 census data, that extended living arrangements were twice as common among African American households as among White households.<sup>58</sup> Black communities have used networks of intimate mutual aid and social interactions with neighbors and kin as a coping strategy against isolation from larger society.<sup>49,51,52</sup> Care of children, shopping, and counseling are among the services provided by extensive kin systems.<sup>49,50</sup> According to McAdoo's study on the extended family support network, mothers of young children, especially single mothers, benefited from the network. Mothers appeared to receive more help than they provided in this network, and the help most frequently exchanged in the network was child care.<sup>49</sup> There was evidence that mothers who received support were protected against the harmful effect of negative life circumstances.

The Hispanic population in the United States continues to increase, partly as a result



of immigration.<sup>59</sup> Many studies have suggested that income, education, and acculturation might interact in significant ways to affect the smoking behavior of Hispanic women. For example, Latina immigrants with initially lower smoking rates tend to increase their smoking rates as they become more educated and more acculturated.<sup>60</sup> Although there is a need to belong to, and assimilate into, the general mainstream of American culture, Hispanic/Latino women are also influenced by the norms of their countries of origin, where smoking tends to be relatively uncommon among women. Furthermore, immigrant Latino subgroups experience some very positive benefits from their social networks.<sup>61,62</sup> Zuniga found that, because of their linguistic, cultural, and economic isolation, immigrants were heavily dependent on the moral support and networks of their community.<sup>59</sup> Baezconde-Garbanati found relatively lower overall rates of adult smoking, psychopathology, and depression to be tied to traditional cultural values and the presence of a strong family network.<sup>62</sup> Contact with extended families from the country of origin, and even with nonfamilial kin systems, offers support and helps preserve the values of the culture among Hispanics/Latinos. These factors may play protective roles for mothers with young children and serve as a resource for coping with stress.

We hypothesize that although women in African American and Hispanic racial/ethnic minority groups generally have less easy access to material resources than do their White counterparts, they may have strong social support systems within their communities. These social support systems may help lower the stress of child rearing for mothers of young children, resulting in a lower prevalence of smoking among these women. The most disadvantaged women, such as single mothers, may also benefit the most from these strong family or community support networks.

### Limitations

This study has several limitations. First, the cross-sectional nature of the data limits our ability to make causal inferences. Thus, it is impossible to distinguish whether the evidence we observed is a result of the effect of parenting on smoking or whether it is simply a correlation between these variables.

Second, the smoking assessment was based on self-report and was not verified by objective measures. Strong emphases on the harmful effects of secondhand smoke on children's health may compromise the validity of self-report. In fact, 2 trials targeting pregnant women who received even stronger messages "not to smoke" found high percentages of deception (28% and 35%) during late pregnancy.<sup>63,64</sup> However, a meta-analysis of 26 validation studies concluded that self-reported smoking status is generally accurate.<sup>65</sup> The only exceptions are among pregnant women, adolescents, and participants of intense smoking-cessation programs.<sup>63,66,67</sup>

In addition, the estimates of smoking prevalence in the BRFSS may be lower than the true prevalence. Studies have reported that the BRFSS tends to underestimate smoking prevalence compared with the Current Population Survey (CPS), which conducts most interviews in person.<sup>68–70</sup> In 2000, about 95% of US households had telephones,<sup>71</sup> but telephone coverage is lower in many southern states.<sup>71,72</sup> Furthermore, some risk behaviors are more common among persons in households without telephones, whereas nonresponse rates are higher among smokers,<sup>73</sup> and underreporting of smoking occurs more often in telephone interviews.<sup>74</sup> The BRFSS, which uses telephone survey methods, is susceptible to these flaws. Nevertheless, studies comparing the BRFSS with the CPS and the National Health Interview Survey suggest that state estimates of smoking prevalence from the BRFSS were reasonably accurate for the purposes of ongoing state surveillance.<sup>69,75</sup>

Finally, we attempted to measure women's child care responsibilities, but what we actually measured was whether women lived with children aged 0–4 years. Although we assumed that living with children aged 0–4 years is equivalent to raising young children, the validity of this assumption may vary by how much time women spend with their children. In fact, there is a wide variation in burdens of child care responsibilities, even given the same number of children. Actual child care depends on whether a mother is working full-time, whether the child attends day care, whether the parent receives child care from professionals or relatives, and how many adults are responsible for the child. There may be systematic differences

between high- and low-SES groups or between racial/ethnic groups in the pattern of child care.

### CONCLUSIONS

Despite these limitations, our study adds to a growing body of evidence that smoking behavior is embedded in the socioeconomic circumstances of the lives of low-income women. The apparent differences between non-Hispanic White women and other racial/ethnic groups links between child care and smoking also suggest that the strong social support systems within the Black and Hispanic communities might help women in these groups to avoid smoking. These racial/ethnic differences and the potential role of social support warrant further investigation. ■

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### Contributors

H. Jun designed the study, analyzed the data, and wrote the article. S. V. Subramanian provided statistical guidance. S. Gortmaker suggested revisions to the analysis and contributed to the interpretation of the results. I. Kawachi contributed to the study design, data interpretation, and critical revisions.

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### Human Participant Protection

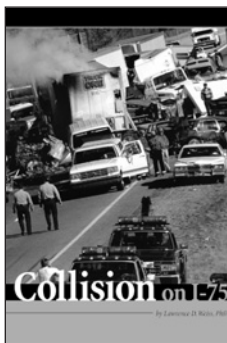
No protocol approval was needed for this study.

### References

1. US Department of Health and Human Services. *Reducing Tobacco Use. A Report of the Surgeon General*. Atlanta, Ga: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 2000.
2. US Department of Health and Human Services. *Women and Smoking: A Report of the Surgeon General*.

- Atlanta, Ga: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 2001.
3. Kendrick JS, Merritt RK. Women and smoking: an update for the 1990s. *Am J Obstet Gynecol*. 1996;175:528–535.
  4. O'Loughlin JL, Paradis G, Renaud L, Meshefedjian G, Barnett T. The "Yes, I Quit" smoking cessation course: does it help women in a low income community quit? *J Commun Health*. 1997;22:451–468.
  5. Glasgow RE, Whitlock EP, Eakin EG, Lichtenstein E. A brief smoking cessation intervention for women in low-income Planned Parenthood clinics. *Am J Public Health*. 2000;90:786–789.
  6. Lu Y, Tong S, Oldenburg B. Determinants of smoking and cessation during and after pregnancy. *Health Promotion Int*. 2001;16:355–365.
  7. Naerde A, Tambs K, Mathiesen KS. Child related strain and maternal mental health: a longitudinal study. *Acta Psychiatr Scand*. 2002;105:301–309.
  8. Umberson D. Relationships with children: explaining parents' psychological well-being. *J Marriage Family*. 1989;51:999–1012.
  9. Bebbington PE, Dunn G, Jenkins R, et al. The influence of age and sex on the prevalence of depressive conditions: report from the National Survey of Psychiatric Morbidity. *Psychol Med*. 1998;28:9–19.
  10. Brown GW, Bhrolchain MN, Harris T. Social-class and psychiatric disturbance among women in an urban population. *Sociology*. 1975;9:225–254.
  11. Brown GW, Harris T. *Social Origins of Depression: A Study of Psychiatric Disorder in Women*. New York, NY: Free Press; 1978.
  12. Richman N. Depression in mothers of preschool children. *J Child Psychol Psychiatry*. 1976;17:75–78.
  13. Moss P, Plewis I. Mental distress in mothers of pre-school children in inner London. *Psychol Med*. 1977;7:641–652.
  14. Naerde A, Tambs K, Mathiesen KS, Dalgard OS, Samuelsen SO. Symptoms of anxiety and depression among mothers of pre-school children: effect of chronic strain related to children and child care-taking. *J Affect Disord*. 2000;58:181–199.
  15. Karrass J, VanDeventer MC, Braungart-Rieker JM. Predicting shared parent-child book reading in infancy. *J Family Psychol*. 2003;17:134–146.
  16. Hakulinen T, Laippala P, Paunonen M, Pelkonen M. Relationships between family dynamics of Finnish child-rearing families, factors causing strain and received support. *J Adv Nurs*. 1999;29:407–415.
  17. Johnson Z, Molloy B, Scallan E, et al. Community Mothers Programme—seven year follow-up of a randomized controlled trial of non-professional intervention in parenting. *J Public Health Med*. 2000;22:337–342.
  18. Barnett B, Duggan AK, Devoe M, Burrell L. The effect of volunteer home visitation for adolescent mothers on parenting and mental health outcomes: a randomized trial. *Arch Pediatr Adolesc Med*. 2002;156:1216–1222.
  19. Crnic KA, Greenberg MT, Ragozin AS, Robinson NM, Basham RB. Effects of stress and social support on mothers and premature and full-term infants. *Child Dev*. 1983;54:209–217.
  20. Younger JB. A model of parenting stress. *Res Nurs Health*. 1991;14:197–204.
  21. Targosz S, Bebbington P, Lewis G, et al. Lone mothers, social exclusion and depression. *Psychol Med*. 1997;27:715–722.
  22. US Department of Health and Human Services. *The Health Consequences of Smoking: Nicotine Addiction: A Report of the Surgeon General*. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1988.
  23. Wills TA, Shiffman S. Coping and substance use: a conceptual framework. In: Shiffman S, Wills TA, eds. *Coping and Substance Use*. New York, NY: Academic Press; 1985:3–21.
  24. Kassel JD. Smoking and stress: correlation, causation, and context. *Am Psychol*. 2000;55:1155–1156.
  25. Graham H. Women's smoking and family health. *Soc Sci Med*. 1987;25:47–56.
  26. Graham H. *When Life's a Drag: Women, Smoking and Disadvantage*. London, England: Dept of Health; Her Majesty's Stationery Office; 1993.
  27. Greaves L. *Smoke Screen: Women's Smoking and Social Control*. Halifax, Nova Scotia: Scarlet Press; 1996.
  28. Stewart MJ, Gillis A, Brosky G, et al. Smoking among disadvantaged women: causes and cessation. *Can J Nurs Res*. 1996;28:41–60.
  29. Stewart MJ, Brosky G, Gillis A, et al. Disadvantaged women and smoking. *Can J Public Health*. 1996;87:257–260.
  30. Nichter M. Smoking: what does culture have to do with it? *Addiction*. 2003;98:139–145.
  31. Centers for Disease Control and Prevention. *Behavioral Risk Factor Surveillance System Survey Data 2000*. Atlanta, Ga: US Dept of Health and Human Services, Centers for Disease Control and Prevention; 2001.
  32. Organization for Economic Cooperation and Development. *The OECD List of Social Indicators*. Organization for Economic Cooperation and Development; 1982.
  33. World Institute for Development Economics Research. *World Income Inequality Database V. 1.0: User Guide and Data Sources*. United Nations University, United Nations Development Program; 2000.
  34. US Bureau of the Census. *Poverty Thresholds in 2000, by Size of Family and Number of Related Children Under 18 Years (Dollars)*. US Bureau of the Census, Current Population Survey; 2000.
  35. Goldstein H. Nonlinear multilevel models, with an application to discrete response data. *Biometrika*. 1991;78:45–51.
  36. Goldstein H. *Multilevel Statistical Models*. 3rd ed. London, England: Edward Arnold; 2003.
  37. Rasbash J, Browne W, Healy M, Cameron B, Charlton C. *MLwiN 1.10*. In. *1.10.0007 ed: Multilevel Models Project*. London, England: Institute of Education, University of London; 2001.
  38. Goldstein H, Rasbash J. Improved approximations for multilevel models with binary responses. *J R Stat Society Ser A*. 1996;159:505–513.
  39. Subramanian SV, Jones K, Duncan C. Multilevel methods for public health research. In: Kawachi I, Berkman LF, eds. *Neighborhoods and Health*. New York, NY: Oxford University Press; 2003:65–111.
  40. Collet D. *Modeling Binary Data*. London, England: Chapman & Hall; 1991.
  41. Greenberg RA, Strecher VJ, Bauman KE, et al. Evaluation of a home-based intervention program to reduce infant passive smoking and lower respiratory illness. *J Behav Med*. 1994;17:273–290.
  42. Ershoff DH, Mullen PD, Quinn VP. A randomized trial of a serialized self-help smoking cessation program for pregnant women in an HMO. *Am J Public Health*. 1989;79:182–187.
  43. Quinn VP, Mullen PD, Ershoff DH. Women who stop smoking spontaneously prior to prenatal care and predictors of relapse before delivery. *Addict Behav*. 1991;16:29–40.
  44. Ershoff DH, Quinn VP, Boyd NR, Stern J, Gregory M, Wirtschatter D. The Kaiser Permanente prenatal smoking-cessation trial: when more isn't better, what is enough? *Am J Prev Med*. 1999;17:161–168.
  45. Rafuse J. Smoking-cessation program targets low-income women. *Can Med Assoc J*. 1994;150:1683–1684.
  46. Pohl JM, Caplan D. Smoking cessation: using group intervention methods to treat low-income women. *Nurse Pract*. 1998;23:13, 17, 18, 20, 38, 39.
  47. Romano P, Bloom J, Syme S. Smoking, social support, and hassles in an urban African-American community. *Am J Public Health*. 1991;81:1415–1422.
  48. Hale J. The black woman and child rearing. In: Rodgers-Rose LF, ed. *The Black Woman*. Beverly Hills, Calif: Sage Publications; 1980:79–88.
  49. McAdoo HP. Black mothers and the extended family support network. In: Rodgers-Rose LF, ed. *The Black Woman*. Beverly Hills, Calif: Sage Publications; 1980:125–144.
  50. Rainwater L. Crucible of identity: the Negro lower-class family. *Daedalus*. 1966;95:175–216.
  51. Hill RB. *The Strengths of African American Families*. New York, NY: Emerson Hall; 1972.
  52. Billingsley A. *Black Families in White America*. Englewood Cliffs, NJ: Prentice-Hall; 1968.
  53. Tienda M, Angel RJ. Headship and household composition among blacks, Hispanics, and other whites. *Soc Forces*. 1982;61:508–531.
  54. Angel RJ, Tienda M. Determinants of extended household structure: cultural pattern or economic model? *Am J Sociol*. 1982;87:1360–1383.
  55. Taylor RJ, Tucker MB, Chatters LM, Jayakody R. Recent demographic trends in African American family structure. In: Taylor RJ, Jackson JS, Chatters LM, eds. *Family Life in Black America*. Thousand Oaks, Calif: Sage Publications; 1997:14–62.
  56. Sweet JA, Bumpass LL. *American Families and Households*. New York, NY: Russell Sage Foundation; 1987.
  57. Beck RW, Beck SH. The incidence of extended households among middle-aged black and white women: estimates from a 5-year panel study. *J Family Issues*. 1989;10:147–168.
  58. Farley R, Allen WR. *The Color Line and the Quality of Life in America*. New York, NY: Russell Sage Foundation; 1987.
  59. Zuniga ME. Latino immigrants: patterns of survival. *J Human Behav Social Environ*. 2002;5:137–155.

60. Marin G, Marin VB, Otero-Sabogal R, Sabogal F, Perez-Stable E. The role of acculturation on the attitudes, norms and expectancies of Hispanic/Latino smokers. *J Cross Cultural Psychol.* 1989;20:478-493.
61. Hayes-Bautista DE, Baezconde-Garbanati L, Schink WO, Hayes-Bautista M. Latino health in California, 1985-1990: implications for family practice. *Fam Med.* 1994;26:556-562.
62. Baezconde-Garbanati L. Do Latino social nets work? The direct and mediating effects of social resources on stressful life events and depression [doctoral dissertation]. Los Angeles: University of California, Los Angeles; 1994.
63. Kendrick J, Zahniser S, Miller N, et al. Integrating smoking cessation into routine public prenatal care: the Smoking Cessation in Pregnancy project. *Am J Public Health.* 1995;85:217-222.
64. Windsor R, Lowe J, Perkins L, et al. Health education for pregnant smokers: its behavioral impact and cost benefit. *Am J Public Health.* 1993;83:201-206.
65. Patrick D, Cheadle A, Thompson D, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health.* 1994;84:1086-1093.
66. US Department of Health and Human Services. *The Health Benefits of Smoking Cessation: A Report of the Surgeon General.* Atlanta, Ga: Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion; 1990. DHHS publication (CDC) 90-8416.
67. Velicer WF, Prochaska JO, Rossi JS, Snow MG. Assessing outcome in smoking cessation studies. *Psychol Bull.* 1992;111:23-41.
68. Anda R, Dodson D, Williamson D, Remington P. Health promotion data for state health departments: telephone versus in-person survey estimates of smoking and alcohol use. *Am J Health Promot.* 1989;4:32-36.
69. Arday DR, Tomar SL, Nelson DE, Merritt RK, Schooley MW, Mowery P. State smoking prevalence estimates: a comparison of the Behavioral Risk Factor Surveillance System and current population surveys. *Am J Public Health.* 1997;87:1665-1669.
70. Gentry E, Kalsbeek W, Hogelin G, et al. The Behavioral Risk Factor surveys, II: design, methods and estimates from combined state data. *Am J Prev Med.* 1985;1:9-14.
71. Federal Communications Commission. *Trends in Telephone Service.* Washington, DC: Industry Analysis and Technology Division, Common Carrier Bureau, Federal Communications Commission; 2000.
72. Centers for Disease Control and Prevention. *2000 Telephone Coverage: Estimated Percentages of Households With and Without Telephones by State, 2001.* Available at: [http://www.cdc.gov/brfss/technical\\_infodata/surveydata/2000/table3\\_00.htm](http://www.cdc.gov/brfss/technical_infodata/surveydata/2000/table3_00.htm). 2001.
73. Groves R, Kahn R. *Surveys by Telephone: A National Comparison With Personal Interviews.* New York, NY: Academic Press; 1979.
74. Jackson C, Jatulis D, Fortmann S. The Behavioral Risk Factor Survey and the Stanford Five-City Project Survey: a comparison of cardiovascular risk behavior estimates. *Am J Public Health.* 1992;82:412-416.
75. Nelson DE, Powell-Griner E, Town M, Kovar MG. A comparison of national estimates from the National Health Interview Survey and the Behavioral Risk Factor Surveillance System. *Am J Public Health.* 2003;93:1335-1341.



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# Prevalence of Smoking in 8 Countries of the Former Soviet Union: Results From the Living Conditions, Lifestyles and Health Study

Anna Gilmore, MSc, MFPH, Joceline Pomerleau, PhD, MSc, Martin McKee, MD, FRCP, Richard Rose, DPhil, BA, Christian W. Haerpfer, PhD, MSc, David Rotman, PhD, and Sergej Tumanov, PhD

In 1990, it was estimated that a 35-year-old man in the former Soviet Union had twice the risk of dying from tobacco-related causes before the age of 70 years as a man in the European Union (20% vs 10%).<sup>1</sup> In the former Soviet Union, 56% of male cancer deaths and 40% of all deaths are attributed to tobacco, compared with 47% and 35%, respectively, in the European Union.<sup>1</sup> Rates of circulatory disease among both men and women are approximately triple those in the European Union.<sup>2</sup> Moreover, tobacco-related mortality continues to increase in the former Soviet Union, while it has stabilized or declined in the European Union as a whole.<sup>1</sup>

Despite these deplorably high levels of tobacco-related mortality, relatively little is known about smoking prevalence rates in the region. Virtually no recent or reliable data exist for the central Asian countries (Kazakhstan, Kyrgyzstan, Tajikistan, Turkmenistan, and Uzbekistan),<sup>2,3</sup> and recent surveys conducted in Georgia have been limited to the capital, Tbilisi.<sup>4,5</sup> Data from elsewhere in the Caucasus (Armenia, Azerbaijan) are scarce,<sup>6</sup> and historical figures<sup>7</sup> are inconsistent with later findings, leading authors to rely on anecdotal reports of smoking rates.<sup>8</sup>

Historical<sup>3</sup> and more recent data, derived largely from Russia,<sup>9</sup> Ukraine,<sup>10</sup> Belarus,<sup>11</sup> and the Baltic states,<sup>12</sup> show—perhaps unsurprisingly, given the mortality figures just described—that smoking rates among men are high (45%–60%) while rates are far lower among women (1%–20%).<sup>2</sup> The higher rates previously seen among Estonian women are now being matched by rates among women in the other Baltic states<sup>2,12,13</sup> and by women in other urban areas.<sup>9,10</sup> Unfortunately, other than the Baltic states, few countries collect information using similar data collection tools,

thereby precluding accurate between-country comparisons.

These issues underlie the need in the former Soviet Union for comparable and accurate data on smoking prevalence, given that such data are widely recognized as a prerequisite for the development of effective public health policies.<sup>14–16</sup> This need is made more urgent by the profound changes occurring as a result of the former Soviet Union's recent economic transition and, more specifically, by the changes taking place in its tobacco industry.<sup>17</sup> The latter were first felt as soon as these formerly closed markets opened, with a rapid influx of cigarette imports and advertising.<sup>18–20</sup> Later, as part of the large-scale privatization of state assets, most of the newly independent states privatized their tobacco industries, and the transnational tobacco companies established a local manufacturing presence, investing more than \$2.7 billion in 10 countries of the former Soviet Union between 1991 and 2000.<sup>21</sup> Evidence from the industry's previous entry into Asia suggests that these changes are likely to have a significant upward impact on cigarette consumption.<sup>22,23</sup>

**Objectives.** We sought to provide comparative data on smoking habits in countries of the former Soviet Union.

**Methods.** We conducted cross-sectional surveys in 8 former Soviet countries with representative national samples of the population 18 years or older.

**Results.** Smoking rates varied among men, from 43.3% to 65.3% among the countries examined. Results showed that smoking among women remains uncommon in Armenia, Georgia, Kyrgyzstan, and Moldova (rates of 2.4%–6.3%). In Belarus, Ukraine, Kazakhstan, and Russia, rates were higher (9.3%–15.5%). Men start smoking at significantly younger ages than women, smoke more cigarettes per day, and are more likely to be nicotine dependent.

**Conclusions.** Smoking rates among men in these countries have been high for some time and remain among the highest in the world. Smoking rates among women have increased from previous years and appear to reflect transnational tobacco company activity. (*Am J Public Health.* 2004;94:2177–2187)

In response to these and other health and social issues facing the region, a major research project—the Living Conditions, Lifestyles and Health Study—was commissioned as part of the European Union's Copernicus program. This investigation involved surveys conducted in 8 of the 15 newly independent states: Armenia, Belarus, Georgia, Kazakhstan, Kyrgyzstan, Moldova, Russia, and Ukraine.<sup>24</sup> We present data on smoking prevalence, including age- and gender-specific smoking rates, age at initiation of smoking, and indicators of nicotine dependence.

## METHODS

### Study Population and Sampling Procedures

In autumn 2001, quantitative cross-sectional surveys were conducted in each country by organizations with expertise in survey research using standardized methods<sup>25</sup> (described in detail elsewhere<sup>26</sup>). In brief, each survey sought to include representative samples of the national adult population 18 years or older, although a few small regions had to be excluded as a result of geographic inaccess-



sibility, sociopolitical situation, or prevailing military action: Abkhazia and Ossetia in Georgia, the Transdniestrian region and the municipality of Bender in Moldova, the Chechen and Ingush republics, and autonomous districts located in the far north of the Russian Federation.

Samples were selected via multistage random sampling with stratification by region and area. Within each primary sampling unit, households were selected according to standardized random route procedures; the exception was Armenia, where household lists were used to provide a random sample. Within each household, the adult with the birthday nearest to the date of the survey was selected to be interviewed. At least 2000 respondents were included in each country; 4006 residents of the Russian Federation and 2400 residents of Ukraine were interviewed, reflecting the larger and more diverse populations of these countries.

### Questionnaire Design

The first draft of the questionnaire was created, in consultation with country representatives, from preexisting surveys conducted in other transition countries<sup>9,10,12</sup> and from New Russia Barometer surveys<sup>27</sup> adjusted to national contexts. It was developed in English, translated into national languages, back-translated to ensure consistency, and pilot tested in each country. Trained interviewers administered the questionnaire in respondents' homes.

### Statistical Analyses

Stata (Version 6; Stata Corp, College Station, Tex) was used to analyze the data. As a means of reducing the skewness of their distribution, the continuous variables of age at smoking initiation and smoking duration were transformed, via log-normal transformations, before analyses were conducted; however, they were returned to their original units in computing results.

Current smokers were defined as respondents reporting currently smoking at least 1 cigarette per day. We calculated age- and gender-specific smoking prevalence rates for each country. Given the negative health effects of early initiation, we examined age at smoking initiation among current smokers, as well as number of cigarettes smoked. We assessed level of nicotine dependence, an indi-

cation of smokers' ability or inability to quit, by identifying the percentage of current smokers who smoked more than 20 cigarettes per day and smoked within an hour of waking. This level of use is equivalent to a score of 3 or more on the abbreviated Fagerstrom dependency scale<sup>28,29</sup> and indicates moderate (score of 3 or 4) to severe (score of 5 or above) dependency.

Within each country, gender differences in smoking habits were assessed with  $\chi^2$  tests and 2-sample *t* tests; variations according to age group were estimated via logistic regression analyses in which the 18- to 29-year age group was the reference category. Logistic regression analyses with Russia as the baseline were used in making between-country comparisons in likelihood of smoking, while analyses of variance combined with Bonferroni multiple comparison tests were used in comparing geometric mean ages at smoking initiation. To allow for the large number of comparisons, we used 99% confidence intervals and set the significance level at .01.

## RESULTS

### Response Rates

A total of 18 428 individuals were surveyed. Response rates (calculated from the total number of households for which an eligible person could be identified) varied from 71% to 88% among the countries included. Rates of nonresponse for individual items were very low (e.g., 0.03% for current smoking and 0.5% for education level).

### Sample Characteristics and Representativeness

The samples clearly reflected the diversity of the region and were broadly representative of their overall populations (Table 1). Comparisons of the present data and official data are potentially limited by the failure of some of the country data to fully capture posttransition migration and other factors,<sup>30</sup> but they suggest slight underrepresentations of men in Armenia and Ukraine, of the urban population in Armenia, and of the rural population in Kyrgyzstan. Age group comparisons among the respondents 20 years or older suggested a tendency for the oldest age group to be overrepresented at the expense of the young-

est age group, particularly in Armenia, Moldova, and Ukraine.

### Smoking Prevalence

Rates of male smoking were high. In many of the countries surveyed, almost 80% of male respondents reported a history of smoking (Table 2). Rates of current smoking were lowest in Moldova (43.3%) and Kyrgyzstan (51.0%) and highest in Kazakhstan (65.3%), Armenia (61.8%), and Russia (60.4%). Smoking rates in Russia were not distinguishable from those in Kazakhstan, Armenia, or Belarus but were significantly higher than those observed in Moldova, Kyrgyzstan, Ukraine, and Georgia ( $P < .01$ ; data not shown).

Rates among women were far lower (gender comparisons were significant at the .001 level in all countries) and somewhat more variable, ranging from 2.4% to 15.5%; the lowest rates were seen in Armenia, Moldova, and Kyrgyzstan and the highest in Russia, Belarus, and Ukraine. Smoking among women in Russia was significantly more prevalent than among women in all of the other countries under study ( $P < .01$ ) although adjusting for age removed the difference between Russia and Belarus (data not shown).

The relationship between smoking and age varied by gender. Among men, with the exception of those residing in Moldova, smoking prevalence rates varied little between the ages of 18 and 59 years but then declined more markedly in men above the age of 60 years (Table 2, Figure 1). This decline with age was accounted for by increases in the older groups in terms of percentages of former smokers and never smokers. Among women, the overall trend was a decrease in reports of both current and former smoking with increasing age; very low smoking rates were observed in the oldest age group (rates of reported lifetime smoking varied from 0.8%–3.9%). However, closer inspection of the data suggested that the countries could be divided into 2 groups. In the first group (Russia, Belarus, Ukraine, and Kazakhstan), rates of current and ever smoking implied that initiation of smoking had increased rapidly between generations, especially in the youngest age group (Table 2, Figure 1). In the second group (Armenia, Georgia, Kyrgyzstan, and Moldova), the age trends were less obvious

**TABLE 1—Characteristics of Samples and Countries in the Living Conditions, Lifestyles and Health Study: 8 Countries of the Former Soviet Union, 2001**

| Characteristic   | AR    | BY    | GE    | KZ    | KG   | MD    | RU     | UA    |
|--|-------|-------|-------|-------|------|-------|--------|-------|
| <b>Sample</b>  |       |       |       |       |      |       |        |       |
| Response rate, %   | 88    | 73    | 88    | 82    | 71   | 81    | 73     | 76    |
| Gender   |       |       |       |       |      |       |        |       |
| Male, %  | 40.3  | 44.1  | 45.7  | 44.4  | 45.0 | 45.1  | 43.5   | 38.8  |
| Men aged ≥ 20 y, %   | 40.7  | 43.9  | 45.6  | 44.1  | 45.6 | 44.9  | 43.2   | 38.6  |
| No.  | 2000  | 2000  | 2022  | 2000  | 2000 | 2000  | 4006   | 2400  |
| Age group, y, %  |       |       |       |       |      |       |        |       |
| 20–29  | 15.4  | 16.9  | 13.9  | 21.9  | 26.7 | 14.5  | 16.5   | 14.6  |
| 30–39  | 21.6  | 19.2  | 20.3  | 25.8  | 26.0 | 20.1  | 19.3   | 16.4  |
| 40–49  | 24.0  | 21.6  | 21.9  | 21.5  | 21.4 | 23.1  | 20.9   | 17.9  |
| 50–59  | 11.1  | 14.5  | 16.3  | 12.0  | 10.1 | 16.4  | 15.4   | 15.5  |
| ≥ 60   | 28.0  | 27.9  | 27.6  | 18.8  | 15.9 | 26.0  | 27.9   | 35.5  |
| No. aged ≥ 20  | 1940  | 1922  | 1975  | 1890  | 1899 | 1945  | 3828   | 2324  |
| No. aged 18–19   | 60    | 78    | 47    | 110   | 101  | 55    | 178    | 76    |
| Interview location, %  |       |       |       |       |      |       |        |       |
| State/regional capital   | 44.0  | 33.9  | 41.4  | 27.0  | 27.5 | 30.4  | 35.7   | 31.5  |
| Other city/small town  | 17.0  | 34.8  | 15.6  | 25.4  | 13.5 | 11.6  | 37.1   | 36.4  |
| Village  | 39.0  | 31.4  | 43.0  | 47.6  | 59.0 | 58.1  | 27.3   | 32.1  |
| No.  | 2000  | 2000  | 2022  | 1850  | 2000 | 2000  | 4006   | 2400  |
| Reported nationality, %  |       |       |       |       |      |       |        |       |
| Nationality of country <sup>a</sup>  | 97.3  | 80.1  | 90.2  | 36.3  | 68.6 | 76.7  | 82.4   | 77.7  |
| Russian  | 0.8   | 12.1  | 1.3   | 41.5  | 18.0 | 7.7   | ...    | 16.5  |
| Other  | 1.9   | 7.8   | 8.5   | 22.1  | 13.5 | 15.7  | 17.6   | 5.8   |
| No.  | 2000  | 1979  | 2021  | 1979  | 1997 | 1980  | 3967   | 2371  |
| Education, %   |       |       |       |       |      |       |        |       |
| Secondary education or less  | 49.1  | 49.4  | 33.8  | 35.7  | 48.3 | 52.2  | 43.2   | 44.2  |
| Secondary vocational or some college   | 30.4  | 34.2  | 32.7  | 43.5  | 32.7 | 32.7  | 35.7   | 36.1  |
| College  | 20.5  | 16.4  | 33.6  | 20.8  | 19.0 | 15.2  | 21.1   | 19.7  |
| No.  | 1996  | 1984  | 1996  | 1995  | 1996 | 1984  | 4004   | 2381  |
| <b>Country data<sup>b</sup></b>  |       |       |       |       |      |       |        |       |
| Midyear population, 2001, thousands  | 3788  | 9971  | 5238  | 14821 | 4927 | 4254  | 144387 | 49111 |
| Gross national product per capita, 2001, \$  | 560   | 1190  | 620   | 1360  | 280  | 380   | 1750   | 720   |
| Men aged ≥ 20 y, 2000, %   | 47.5  | 45.4  | 46.4  | 46.6  | 47.9 | 46.3  | 45.3   | 44.8  |
| Urban population, 2001, %  | 67.3  | 69.6  | 56.5  | 55.9  | 34.4 | 41.7  | 72.9   | 68.0  |
| Age group, y, % of total ≥ 20  |       |       |       |       |      |       |        |       |
| 20–29  | 23.2  | 19.3  | 20.6  | 26.0  | 30.5 | 23.1  | 19.6   | 19.4  |
| 30–39  | 24.2  | 20.3  | 21.1  | 23.7  | 24.7 | 20.3  | 19.6   | 19.0  |
| 40–49  | 22.5  | 21.5  | 19.5  | 21.4  | 19.6 | 22.7  | 22.4   | 19.8  |
| 50–59  | 10.3  | 12.6  | 12.7  | 10.9  | 9.0  | 13.6  | 13.3   | 14.2  |
| ≥ 60   | 19.7  | 26.4  | 26.2  | 18.0  | 16.2 | 20.3  | 25.1   | 27.6  |
| Unemployment rate, % <sup>c</sup>  | 11.7  | 2.3   | 11.1  | 2.9   | 3.2  | 2.0   | 13.4   | 5.8   |
| Tobacco industry state owned (SO) or privatized (P)                                  | P     | SO    | P     | P     | P    | SO    | P      | P     |
| Foreign direct investment in tobacco industry, end of 2000, \$ millions <sup>d</sup> | 8     | 0     | 0     | 440   | ...  | 0     | 1719   | 152.9 |
| Foreign direct investment in tobacco industry per capita × 1000 <sup>d</sup>         | 0.002 | 0.000 | 0.000 | 0.030 | ...  | 0.000 | 0.012  | 0.003 |

Note. AR = Armenia; BY = Belarus; GE = Georgia; KZ = Kazakhstan; KG = Kyrgyzstan; MD = Moldova; RU = Russia; UA = Ukraine.

<sup>a</sup>Mean Armenians in Armenia, Belarussians in Belarus, Georgians in Georgia, Kazakhs in Kazakhstan, Kirghiz in Kyrgyzstan, Moldovans/Romanians in Moldova, Russians in Russia, and Ukrainians in Ukraine.

<sup>b</sup>Data sources were European Health for All Database, January 2003; Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat.

<sup>c</sup>In 1999 for Russia, 2000 for Armenia and Ukraine, and 2001 for the other countries.

<sup>d</sup>Data from Gilmore and McKee<sup>21</sup>; these are minimum investment figures.

TABLE 2—Smoking Prevalence Rates, by Country, Gender, and Age Group in 8 Countries of the Former Soviet Union, 2001

|                         | Male           |      |            |      |            |            |            |            | Female   |                |            |       |       |            |            |            | Gender Difference in Current Smoking, <i>p</i> <sup>a</sup> |            |          |
|-------------------------|----------------|------|------------|------|------------|------------|------------|------------|----------|----------------|------------|-------|-------|------------|------------|------------|---|------------|----------|
|                         | All Age Groups |      |            |      | 18-29 y, % | 30-39 y, % | 40-49 y, % | 50-59 y, % | ≥60 y, % | All Age Groups |            |       |       | 18-29 y, % | 30-39 y, % | 40-49 y, % |   | 50-59 y, % | ≥60 y, % |
|                         | No.            | %    | 99% CI     |      |            |            |            |            | No.      | %              | 99% CI     |       |       |            |            |            |   |            |          |
| Armenia                 |                |      |            |      |            |            |            |            |          |                |            |       |       |            |            |            |   |            |          |
| Current smoker          | 498            | 61.8 | 56.2, 67.4 | 62.5 | 76.8       | 68.3       | 67.1       | 44.4       | 28       | 2.4            | -5.0, 9.7  | 0.9   | 3.1   | 3.9        | 2.9        | 1.0        | <.001   |            |          |
| Former smoker           | 120            | 14.9 | 6.5, 23.3  | 8.3  | 5.5        | 14.2       | 17.1       | 25.1       | 7        | 0.6            | -6.8, 8.0  | 0.9   | 0.4   | 0.4        | 0.7        | 0.7        |   |            |          |
| Never smoker            | 188            | 23.3 | 15.4, 31.3 | 29.2 | 17.7       | 17.5       | 15.8       | 30.5       | 1159     | 97.1           | 95.8, 98.3 | 98.1  | 96.5  | 95.7       | 96.4       | 98.4       |   |            |          |
| Odds of current smoking |                |      |            | 1.00 | 1.98       | 1.29       | 1.22       | 0.48       |          |                |            | 1.00  | 3.43  | 4.3        | 3.15       | 1.05       |   |            |          |
| <i>P</i>                |                |      |            |      | .006       | .272       | .499       | .001       |          |                |            | .121  | .059  | .19        | .952       |            |   |            |          |
| Belarus                 |                |      |            |      |            |            |            |            |          |                |            |       |       |            |            |            |   |            |          |
| Current smoker          | 495            | 56.1 | 50.4, 61.9 | 58.2 | 65.3       | 59.8       | 60.2       | 40.3       | 135      | 12.1           | 4.9, 19.3  | 30.4  | 18.5  | 12.7       | 3.1        | 0.9        | <.001   |            |          |
| Former smoker           | 125            | 14.2 | 6.1, 22.2  | 9.2  | 12.1       | 12.9       | 11.0       | 23.9       | 60       | 5.4            | -2.1, 12.9 | 13.5  | 7.7   | 2.3        | 4.4        | 1.5        |   |            |          |
| Never smoker            | 262            | 29.7 | 22.4, 37.0 | 32.7 | 22.5       | 27.3       | 28.8       | 35.8       | 922      | 82.5           | 79.3, 85.8 | 56.0  | 73.9  | 85.1       | 92.5       | 97.6       |   |            |          |
| Odds of current smoking |                |      |            | 1.00 | 1.35       | 1.07       | 1.08       | 0.49       |          |                |            | 1.00  | 0.52  | 0.33       | 0.07       | 0.02       |   |            |          |
| <i>P</i>                |                |      |            |      | .159       | .743       | .726       | <.001      |          |                |            | <.001 | <.001 | <.001      | <.001      | <.001      |   |            |          |
| Georgia                 |                |      |            |      |            |            |            |            |          |                |            |       |       |            |            |            |   |            |          |
| Current smoker          | 491            | 53.3 | 47.4, 59.1 | 62.8 | 64.8       | 61.5       | 50.7       | 33.9       | 69       | 6.3            | -1.2, 13.9 | 5.8   | 11.6  | 7.8        | 3.4        | 3.4        | <.001   |            |          |
| Former smoker           | 71             | 7.7  | -0.5, 15.9 | 2.0  | 4.4        | 4.5        | 10.4       | 14.5       | 10       | 0.9            | -6.8, 8.7  | 2.3   | 1.4   | 1.3        | 0.0        | 0.0        |   |            |          |
| Never smoker            | 360            | 39.1 | 32.4, 45.7 | 35.1 | 30.8       | 34.0       | 38.9       | 51.6       | 1012     | 92.8           | 90.7, 94.9 | 91.9  | 87.0  | 90.9       | 96.6       | 96.6       |   |            |          |
| Odds of current smoking |                |      |            | 1.00 | 1.09       | 0.94       | 0.61       | 0.30       |          |                |            | 1.00  | 2.13  | 1.38       | 0.58       | 0.57       |   |            |          |
| <i>P</i>                |                |      |            |      | .707       | .799       | .037       | <.001      |          |                |            | .051  | .426  | .295       | .219       |            |   |            |          |
| Kazakhstan              |                |      |            |      |            |            |            |            |          |                |            |       |       |            |            |            |   |            |          |
| Current smoker          | 579            | 65.3 | 60.2, 70.4 | 66.0 | 72.7       | 65.9       | 64.2       | 50.0       | 103      | 9.3            | 1.9, 16.6  | 16.1  | 10.9  | 11.2       | 3.4        | 0.4        | <.001   |            |          |
| Former smoker           | 119            | 13.4 | 5.4, 21.5  | 7.6  | 9.5        | 16.2       | 18.4       | 24.2       | 48       | 4.3            | -3.2, 11.9 | 5.8   | 7.8   | 4.3        | 0.9        | 0.4        |   |            |          |
| Never smoker            | 189            | 21.3 | 13.6, 29.0 | 26.4 | 17.8       | 17.9       | 17.4       | 25.8       | 962      | 86.4           | 83.6, 89.3 | 78.1  | 81.3  | 84.6       | 95.8       | 99.1       |   |            |          |
| Odds of current smoking |                |      |            | 1.00 | 1.37       | 1.00       | 0.92       | 0.52       |          |                |            | 1.00  | 0.64  | 0.66       | 0.18       | 0.02       |   |            |          |
| <i>P</i>                |                |      |            |      | .111       | .982       | .744       | .003       |          |                |            | .087  | .113  | .002       | <.001      |            |   |            |          |
| Kyrgyzstan              |                |      |            |      |            |            |            |            |          |                |            |       |       |            |            |            |   |            |          |
| Current smoker          | 457            | 51.0 | 44.9, 57.0 | 56.2 | 60.4       | 49.8       | 50.0       | 25.0       | 49       | 4.5            | -3.1, 12.1 | 4.2   | 5.4   | 6.0        | 4.9        | 1.7        | <.001   |            |          |
| Former smoker           | 79             | 8.8  | 0.6, 17.0  | 4.9  | 5.5        | 8.3        | 6.8        | 25.8       | 22       | 2.0            | -5.7, 9.7  | 2.7   | 1.8   | 2.5        | 0.0        | 1.7        |   |            |          |
| Never smoker            | 361            | 40.3 | 33.6, 46.9 | 39.0 | 34.1       | 42.0       | 43.2       | 49.2       | 1022     | 93.5           | 91.5, 95.5 | 93.1  | 92.8  | 91.5       | 95.1       | 96.7       |   |            |          |
| Odds of current smoking |                |      |            | 1.00 | 1.19       | 0.77       | 0.78       | 0.26       |          |                |            | 1.00  | 1.31  | 1.45       | 1.18       | 0.39       |   |            |          |
| <i>P</i>                |                |      |            |      | .353       | .166       | .313       | <.001      |          |                |            | .474  | .357  | .759       | .140       |            |   |            |          |

Continued

TABLE 2—Continued

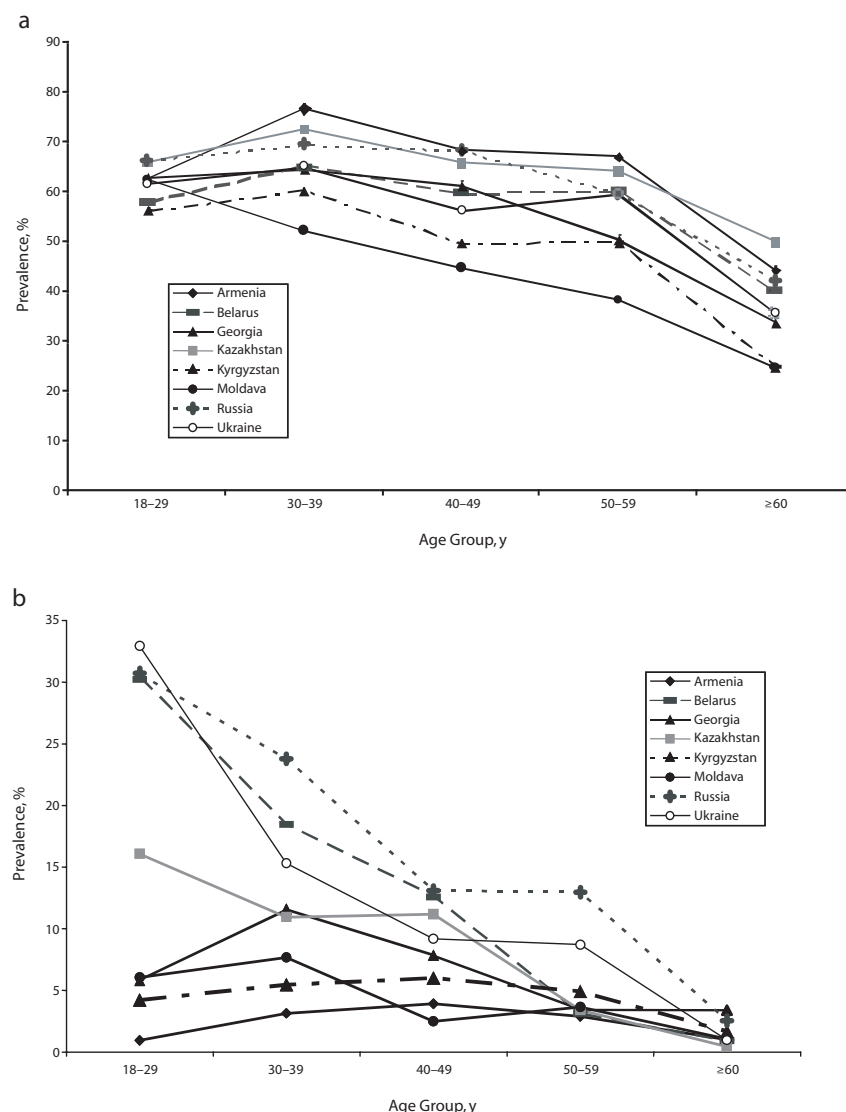
[illegible]

Note. CI = confidence interval.

<sup>a</sup>Average, assuming the same number of respondents in each country.

<sup>b</sup>Results of  $\chi^2$  test on binary variable current versus never and former smokers. Average, assuming the same number of respondents in each county.





**FIGURE 1—Current (a) male and (b) female smoking prevalence rates, by age group.**

and were nonsignificant (with the exception of the comparison of the oldest and youngest age groups in Moldova).

### Age at Initiation

The majority of male smokers reported that they began smoking before the age of 20 years, and, on average, a quarter reported that they began in childhood (Table 3). Far fewer women reported beginning in childhood, and sizable percentages began after the age of 20 years; for example, 86% of women residing in Armenia and more than 40% of women residing in Georgia, Kyrgyzstan, and

Moldova reported that they initiated smoking after this age. These gender differences were significant in all of the countries under study.

Differences also were observed between countries; in Belarus, Kazakhstan, Russia, and Ukraine, geometric mean ages at smoking initiation were younger than 18 years among men and younger than 20 years among women, compared with older ages at smoking initiation elsewhere. Overall, between-country differences were significant for both women and men ( $P < .001$ ); however, Bonferroni multiple comparisons showed that there were significant differences among women only in

comparisons involving Armenia and countries other than Georgia and Moldova ( $P < .01$ ; data not shown). Among men, significantly younger ages at initiation were observed in Russia and Ukraine versus Armenia, Georgia, Kyrgyzstan, and Moldova; in Belarus versus Armenia and Kyrgyzstan; and in Kazakhstan versus Kyrgyzstan (all  $P < .01$ ; data not shown).

### Amount Smoked and Nicotine Dependence

Men were found to smoke more cigarettes than women; the majority of men smoked 10 or more cigarettes per day, while most women smoked fewer than 10 per day. Between-gender differences in percentages of respondents smoking more than 20 cigarettes per day were significant only in the case of Belarus, Kazakhstan, Russia, and Ukraine ( $P < .001$ ).

The majority of smokers reported smoking their first cigarette within an hour of waking, although, in all countries other than Georgia, a far higher proportion of men than women did so ( $P < .01$ ). Thus, men were more likely to be moderately to severely dependent on nicotine, although gender differences were significant only for Belarus, Kazakhstan, Russia, and Ukraine.

### DISCUSSION

The surveys conducted in this study provide important new data on the prevalence of smoking in 8 countries representing more than four fifths of the population of the former Soviet Union. In the case of some of these countries, these data represent the first accurate, countrywide smoking prevalence data reported. In addition, they provide some of the first truly comparative data for countries of the former Soviet Union other than the Baltic states,<sup>31,32</sup> and, because of the focus on obtaining accurate information on sample characteristics, they offer advantages over data available in public databases. Response rates were relatively high, and the samples were broadly representative of the overall country populations.

### Study Limitations

The underrepresentation of men in Armenia and Ukraine should not have affected the gender-specific rates observed, but, as a result

**TABLE 3—Smoking Characteristics of Current Smokers in 8 Countries of the Former Soviet Union, 2001**

|   | AR, % | BY, % | GE, % | KZ, % | KG, % | MO, % | RU, % | UA, % | All, <sup>a</sup> % | Between-Country Comparison, <i>P</i> <sup>b</sup> |
|---|-------|-------|-------|-------|-------|-------|-------|-------|---------------------|---|
| Age at smoking initiation, y  |       |       |       |       |       |       |       |       |                     |   |
| Men   |       |       |       |       |       |       |       |       |                     |   |
| Mean age  | 18.5  | 17.4  | 18.2  | 17.6  | 19.1  | 18.2  | 17.0  | 17.2  | 17.9                |   |
| Geometric mean age  | 17.8  | 16.6  | 17.7  | 17.1  | 18.6  | 17.6  | 16.2  | 16.2  | 17.2                | <.001   |
| < 16  | 22.2  | 32.8  | 18.0  | 27.9  | 14.7  | 22.8  | 36.4  | 35.2  | 26.2                |   |
| 16–20   | 56.8  | 54.2  | 66.0  | 57.0  | 61.8  | 59.9  | 49.8  | 48.5  | 56.7                | <.001   |
| > 20  | 21.0  | 13.0  | 16.0  | 15.1  | 23.5  | 17.3  | 13.9  | 16.3  | 17.0                |   |
| No.   | 447   | 430   | 400   | 502   | 408   | 347   | 993   | 435   | 3962                |   |
| Women   |       |       |       |       |       |       |       |       |                     |   |
| Mean age  | 28.0  | 18.9  | 22.7  | 20.7  | 21.5  | 23.0  | 20.9  | 21.2  | 22.1                | <.001   |
| Geometric mean age  | 27.0  | 18.5  | 21.3  | 19.9  | 20.7  | 21.5  | 19.8  | 19.9  | 21.1                |   |
| < 16  | 0.0   | 20.0  | 18.5  | 15.4  | 12.5  | 22.9  | 13.1  | 15.1  | 14.7                | <.001   |
| 16–20   | 14.3  | 56.7  | 38.5  | 50.6  | 43.8  | 22.9  | 52.6  | 57.2  | 42.1                |   |
| > 20  | 85.7  | 23.3  | 43.1  | 34.1  | 43.8  | 54.3  | 34.4  | 27.6  | 43.3                |   |
| No.   | 28    | 120   | 65    | 91    | 48    | 35    | 329   | 152   | 868                 |   |
| Between-gender comparison in geometric mean age <sup>c</sup>                | <.001 | .002  | <.001 | <.001 | .002  | <.001 | <.001 | <.001 |                     |   |
| Number of cigarettes smoked daily   |       |       |       |       |       |       |       |       |                     |   |
| Men   |       |       |       |       |       |       |       |       |                     |   |
| 1–2   | 1.8   | 3.4   | 1.9   | 4.5   | 15.4  | 8.2   | 2.4   | 4.6   | 5.3                 |   |
| Up to 10  | 18.7  | 32.3  | 12.7  | 30.9  | 50.1  | 43.3  | 24.6  | 25.4  | 29.8                |   |
| 10–20   | 51.4  | 50.5  | 63.3  | 48.0  | 28.7  | 37.4  | 52.2  | 53.5  | 48.1                | <.001   |
| > 20  | 28.1  | 13.7  | 22.2  | 16.6  | 5.8   | 11.0  | 20.8  | 16.5  | 16.9                |   |
| Odds ratio for likelihood of smoking >20 cigarettes per day                 | 1.487 | 0.606 | 1.085 | 0.756 | 0.234 | 0.471 | 1.00  | 0.753 |                     |   |
| <i>P</i>  | .002  | .001  | .539  | .038  | <.001 | <.001 |       | .049  |                     |   |
| No.   | 498   | 495   | 482   | 579   | 449   | 390   | 1052  | 484   | 4429                |   |
| Women   |       |       |       |       |       |       |       |       |                     |   |
| 1–2   | 32.1  | 23.7  | 11.9  | 19.4  | 36.2  | 37.2  | 18.7  | 22.2  | 25.2                |   |
| Up to 10  | 28.6  | 48.9  | 29.9  | 53.4  | 46.8  | 41.9  | 56.6  | 45.7  | 44.0                |   |
| 10–20   | 32.1  | 25.2  | 46.3  | 23.3  | 17.0  | 18.6  | 19.8  | 26.5  | 26.1                | .065  |
| > 20  | 7.1   | 2.2   | 11.9  | 3.9   | 0.0   | 2.3   | 4.9   | 5.6   | 4.7                 |   |
| Odds ratio for likelihood of smoking > 20 cigarettes per day                | 1.50  | 0.44  | 2.64  | 0.79  | ...   | 0.46  | 1.00  | 1.15  |                     |   |
| <i>P</i>  | 0.602 | 0.199 | 0.032 | 0.672 | ...   | 0.461 |       | 0.749 |                     |   |
| No.   | 28    | 135   | 67    | 103   | 47    | 43    | 348   | 162   | 933                 |   |
| Between-gender comparison of % smoking > 20 cigarettes per day <sup>d</sup> | .015  | .000  | .053  | .001  | .090  | .073  | <.001 | <.001 |                     |   |
| Time when usually smoke first cigarette                                     |       |       |       |       |       |       |       |       |                     |   |
| Men   |       |       |       |       |       |       |       |       |                     |   |
| First 30 minutes after awakening  | 63.5  | 47.9  | 52.9  | 42.8  | 39.0  | 44.1  | 56.5  | 55.8  | 50.3                |   |
| First hour after awakening  | 24.9  | 40.4  | 34.0  | 46.6  | 39.4  | 38.2  | 34.3  | 33.3  | 36.4                | <.001   |
| Before midday meal  | 4.6   | 6.9   | 5.0   | 5.0   | 7.1   | 6.7   | 4.7   | 6.0   | 5.7                 |   |
| After midday meal or in the evening   | 7.0   | 4.9   | 8.1   | 5.5   | 14.5  | 11.0  | 4.6   | 5.0   | 7.6                 |   |
| Odds ratio for likelihood of smoking in first hour                          | 0.77  | 0.77  | 0.67  | 0.86  | 0.37  | 0.47  | 1.00  | 0.83  |                     |   |
| <i>P</i>  | .140  | .129  | .021  | .394  | <.001 | <.001 |       | .292  |                     |   |
| No.   | 498   | 495   | 480   | 579   | 449   | 390   | 1051  | 484   | 4426                |   |

Continued

TABLE 3—Continued

|   |       |       |      |       |      |       |       |       |      |      |
|---|-------|-------|------|-------|------|-------|-------|-------|------|------|
| Women   |       |       |      |       |      |       |       |       |      |      |
| First 30 minutes after awakening  | 50.0  | 31.9  | 44.6 | 35.0  | 27.7 | 14.3  | 33.7  | 27.8  | 33.1 | .278 |
| First hour after awakening  | 14.3  | 28.9  | 30.8 | 27.2  | 31.9 | 38.1  | 32.0  | 32.1  | 29.4 |      |
| Before midday meal  | 3.6   | 19.3  | 12.3 | 13.6  | 12.8 | 11.9  | 13.5  | 17.3  | 13   |      |
| After midday meal or in the evening   | 32.1  | 20.0  | 12.3 | 24.3  | 27.7 | 35.7  | 20.8  | 22.8  | 24.5 |      |
| Odds ratio for likelihood of smoking in first hour  | 0.94  | 0.81  | 1.60 | 0.86  | 0.77 | 0.57  | 1.00  | 0.78  |      |      |
| P   | .879  | .307  | .129 | .505  | .409 | .092  |       | .203  |      |      |
| No.   | 28    | 135   | 65   | 103   | 47   | 42    | 347   | 162   | 929  |      |
| Between-gender comparison in % smoking in first hour <sup>d</sup>   | <.001 | <.001 | .014 | <.001 | .004 | <.001 | <.001 | <.001 |      |      |
| Moderate to heavy nicotine dependence (>20 cigarettes per day and smoking within first hour of awakening) |       |       |      |       |      |       |       |       |      |      |
| Men   | 26.9  | 13.7  | 21.4 | 16.6  | 5.6  | 10.5  | 20.6  | 16.2  | 16.4 | .000 |
| Odds ratio for likelihood of moderate to severe dependency  | 1.42  | 0.62  | 1.05 | 0.77  | 0.23 | 0.45  | 1.00  | 0.74  | 0.8  |      |
| P   | .005  | .093  | .142 | .104  | .000 | .000  |       | .042  | .00  |      |
| No.   | 498   | 495   | 477  | 579   | 449  | 390   | 1051  | 483   | 4422 |      |
| Women   | 7.1   | 2.2   | 10.8 | 3.9   | 0.0  | 1.0   | 17.0  | 9.0   | 6.4  | .139 |
| Odds ratio for likelihood of moderate to severe dependency  | 1.49  | 0.44  | 2.34 | 0.78  | ...  | 0.47  | 1.00  | 1.14  | 1.0  |      |
| P   | .605  | .197  | .071 | .669  | ...  | .473  |       | .754  | .3   |      |
| No.   | 28    | 135   | 65   | 103   | 47   | 42    | 347   | 162   | 929  |      |
| Between-gender dependency comparison <sup>d</sup>   | .020  | <.001 | .045 | .001  | .097 | .091  | <.001 | .001  |      |      |

Note. AR = Armenia; BY = Belarus; GE = Georgia; KZ = Kazakhstan; KG = Kyrgyzstan; MD = Moldova; RU = Russia; UA = Ukraine.

<sup>a</sup>Average, assuming the same number of respondents in each country.

<sup>b</sup>Results of analyses of variance (geometric mean) and  $\chi^2$  tests (categorical variable) for mean age at smoking initiation;  $\chi^2$  test for no. of cigarettes smoked, time to first cigarette, and dependency.

<sup>c</sup>Results of *t* tests.

<sup>d</sup>Results of  $\chi^2$  tests.

of the urban/rural differences in the composition of the sample, prevalence rates in Kyrgyzstan (where urban areas were overrepresented) may have been overestimated, and prevalence rates in Armenia (where urban areas were underrepresented) may have been underestimated. However, these discrepancies were likely to affect only the data relating to female respondents.<sup>9–11</sup> The age group disparities noted were minor but would tend to lead to underestimates of smoking prevalence.

In addition, the surveys were based on self-reported smoking status; there was no independent biochemical validation, and thus the smoking rates observed may have been affected by reporting bias. Although there is concern on the part of some that self-reports of smoking status may produce underestimates of smoking levels, studies conducted in Western countries suggest that this technique is sensitive

and specific; they also suggest that more accurate responses are provided in interviewer-administered questionnaires than in self-completed questionnaires.<sup>33</sup> The only study conducted in the former Soviet Union that has addressed this issue showed that, among individuals claiming to be nonsmokers, 13% (48/368) of women and 17% (12/375) of men in rural northwestern Russia were in fact, according to blood cotinine levels, likely to be smokers, compared with only 2% of men and women in Finland.<sup>34</sup> Given the far lower prevalence of smoking among women, this had disproportionately large effects on reported rates of smoking among women. Although our questionnaires were administered by interviewers in respondents' homes, potentially making it more difficult for respondents who smoked to deny doing so, we may have underestimated smoking prevalence rates, particularly in the case of

women residing in areas where smoking remains culturally unacceptable.

A final shortfall of the present study was the failure to measure smokeless tobacco use, which is relatively common in parts of the former Soviet Union, mainly Azerbaijan, Tajikistan, and Turkmenistan. However, although chewing tobacco is used in some of the southern regions of Kyrgyzstan, cigarettes are the main form of tobacco used there as well as in all of the other countries in which surveys were conducted.<sup>8,35</sup>

## Findings

The results of our study confirm that smoking rates among men in this region are among the highest in the world and higher than the maximum rates recorded in the United States at the peak of its epidemic; rates above 50% were observed in all countries other than

Moldova and reached 60% or more in Armenia, Kazakhstan, and Russia. Elsewhere in Europe, rates above 50% are seen only in Turkey (51%) and Slovakia (56%), and worldwide fewer than 20 countries report rates of more than 60%.<sup>6</sup>

In the case of men, the lower prevalence of current smokers and higher prevalence of never and former smokers among those 60 years or older probably reflect the disproportionate number of premature deaths among current smokers relative to never and former smokers. However, a cohort effect has been shown in the former Soviet Union, with those who were teenagers between 1945 and 1953 carrying forward lower smoking rates because cigarettes, like other consumer goods, were in short supply in the period of postwar austerity under Stalin.<sup>36,37</sup> This cohort effect is also thought to account for the unexpected current decline in male lung cancer deaths,<sup>36</sup> which must be set against the overall rise in male tobacco-related mortality<sup>1</sup> and, in particular, increases in the already staggeringly high number of cardiovascular deaths.<sup>2</sup>

In comparison with male smoking patterns, smoking among women is far less common, varies more between countries, and exhibits a different age-specific pattern. Although rates of lifetime smoking are below 4% among individuals older than 60 years in all 8 countries, in the 4 countries with the highest smoking rates among women (Belarus, Kazakhstan, Russia, and Ukraine), smoking is now significantly more common among members of the younger generations; risk ratios between the youngest and oldest age groups range from 12.2 to 37.3, compared with a range of 1.0 to 5.5 in the other 4 countries.

Lopez et al.<sup>38</sup> outlined a 4-stage model of the patterns of a smoking epidemic based on observations from Western countries. In this model, such an epidemic is described as involving an initial rise in male smoking followed by a rise in female smoking 1 to 2 decades later, after which each plateaus and then falls as a result of tobacco-related mortality, finally rising to a peak decades later. Our findings suggest that the former Soviet Union's tobacco epidemic may have developed differently. Male smoking has a long history in this region. The first accounts of tobacco smoking in Russia date from the 17th century,<sup>39</sup> pa-

pirossi (a type of cigarette, popular in the former Soviet Union, characterized by a long, hollow mouthpiece that can be twisted before smoking) were first mentioned in 1844,<sup>39</sup> and cigarette factories were first constructed later in the 19th century.<sup>40,41</sup> Historical data on smoking<sup>3</sup> and high male tobacco-related mortality rates<sup>1</sup> suggest that smoking among men has been at a high level for some time and, contrary to the predictions of the 4-stage model just mentioned, has failed to exhibit a postpeak decline.

Smoking among women remains relatively uncommon, and rates have been far slower to rise than would be expected given male rates in the former Soviet Union and trends observed in the West. Indeed, it appears that female rates began to increase only in the mid-to late 1990s, when transnational tobacco companies arrived with their carefully targeted marketing strategies.<sup>18–20</sup> Therefore, although the exact stage of the epidemic varies slightly between the countries of the former Soviet Union, overall we suggest that men have remained between stages 3 and 4, with high rates of both smoking and mortality, while women in some countries are at stage 1 and others at stage 2, the latter with more rapidly rising smoking rates. Although rates of cardiovascular disease have been increasing, this can largely be explained by risk factors other than tobacco (including diet and stress), and female lung cancer rates have yet to increase.

Comparisons between our results and previous data are problematic given that much of the information that exists is fragmentary, of uncertain quality, and rarely nationally representative. This is particularly the case in the central Asian and Caucasian states, although limited data from Armenia and Moldova gathered between 1998 and 2001 suggest few changes in smoking prevalence rates<sup>2,6</sup>; data from Kazakhstan suggest small increases from the 60% male and 7% female prevalence rates recorded in 1996.<sup>2</sup> More data are available for Belarus, Russia, and Ukraine. These data suggest that smoking rates in men have changed little,<sup>2,10,11,42</sup> although in Russia they appeared to rise between the 1970s and 1980s<sup>2,3,7</sup> and into the mid-1990s, with little subsequent change. Among women, rates appear to have increased in all 3 countries,<sup>2,11</sup> and Russian data suggest that although rates

have been rising since the 1970s, increases were most notable during the 1990s.<sup>3,7,9,43</sup>

Between-gender and intercountry differences in smoking prevalence rates are reflected in other smoking indicators as well; for example, men are more likely than women to start smoking when they are young, to smoke more heavily, and to be nicotine dependent. Two separate groupings of countries appeared to emerge from the between-country comparisons: Belarus, Kazakhstan, Russia, and Ukraine, on one hand, and Armenia, Georgia, Kyrgyzstan, and Moldova, on the other. In addition to exhibiting higher smoking rates among women and more pronounced age-specific trends, the former group tended to show lower ages at smoking initiation (particularly in comparison with Armenia, Georgia, and Moldova) along with more marked gender differences in regard to number of cigarettes smoked per day and level of nicotine dependency.

The differences observed in this study suggest that smoking patterns in Armenia, Georgia, Moldova, and Kyrgyzstan are more traditional than those in Belarus, Kazakhstan, Russia, and Ukraine. This situation can be explained by the differing degree of transnational tobacco company penetration.<sup>21,44</sup> Industry in Moldova continues to be in the form of a state-owned monopoly; industry in Georgia and Armenia has been privatized, but this change was rather recent (occurring after 1997), and none of the major transnational tobacco companies have invested directly in those countries.<sup>21</sup> Kazakhstan, Russia, and Ukraine, by contrast, saw major investments from most major transnational tobacco companies beginning in the early 1990s. Belarus, which retains a state-owned monopoly system, and Kyrgyzstan, where the German cigarette manufacturer Reemtsma has invested, would therefore appear to be exceptions, with Belarus more typical of the countries with transnational tobacco company investments and Kyrgyzstan more typical of the countries without such investments. In Belarus, however, the state tobacco manufacturer has only a 40% market share, with smuggled and counterfeit brands accounting for an additional 40% of this share. The importance the transnational tobacco companies attach to the illegal market in



Belarus can be seen in the fact that, despite having little official market share,<sup>44</sup> British American Tobacco and Philip Morris have the highest outdoor advertising budgets and the 9th and 10th highest television advertising budgets of all companies operating in that country.<sup>45</sup> In Belarus, as in Ukraine and Russia, tobacco is the product most heavily advertised outdoors and the fourth most advertised product on television (there are now restrictions on television advertising in Ukraine and Russia).<sup>45,46</sup> Thus, it appears that with the continuing (if so far fruitless) discussions of possible reunification with Russia, the transnational tobacco companies treat Belarus as an important extension of the Russian market.<sup>47</sup>

Kyrgyzstan differs from the other countries in which there have been transnational tobacco company investments in that these investments occurred later (in 1998) and one company, Reemtsma, achieved a manufacturing monopoly.<sup>44</sup> However, Kyrgyzstan also differs from Belarus, Kazakhstan, Ukraine, and Russia in regard to its lower levels of development and industrialization and its larger rural and Muslim populations. Other potential explanations for the between-country differences observed cannot be excluded here, and such possibilities are explored in a separate article.<sup>48</sup> Whatever reasons emerge, the rising rates of smoking among women and the younger ages of smoking initiation are cause for concern in all of these countries.

Meanwhile, the present findings, combined with earlier data on disease burden,<sup>1,37</sup> confirm that high smoking rates among men continue unabated. Smoking among women in Armenia, Georgia, Kyrgyzstan, and Moldova remains relatively uncommon and does not appear to have increased significantly, as can be seen in rates among the younger relative to older generations and in limited comparisons with previous data. By contrast, smoking rates among women in Belarus, Ukraine, Kazakhstan, and Russia showed an increase from previous surveys, and age-specific rates suggest an ongoing increase in tobacco use among members of the younger generations. It is probably not a coincidence that these higher rates were observed in the countries with the most active transnational tobacco company presence.

## Conclusions

Concerted and urgent efforts to improve tobacco control must be made throughout the former Soviet Union to curtail current smoking and prevent further rises in smoking among women. Such efforts will require enactment and effective enforcement of comprehensive tobacco control policies, including a total ban on tobacco advertising and sponsorship, adequate taxation of both imported and domestic cigarettes, controls on smuggling, and restrictions on smoking in public places. The barriers to achieving these goals are considerable given the powerful influence of transnational tobacco companies and the limited development of democracy and civil society groups in much of the region.<sup>21</sup> The international community, cognizant of the role that international companies play in pushing the tobacco epidemic, should build on the work of the Open Society Institute (R. Bonnell, oral communication, September 2003) in strengthening the policy response to this threat. ■

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## Contributors

A. Gilmore contributed to questionnaire design and data analysis and drafted the article. J. Pomerleau and M. McKee contributed to questionnaire design, data analysis, and revisions of the article. R. Rose contributed to questionnaire design and generation of hypotheses. C. W. Haerpfer, D. Rotman, and S. Tumanov designed and supervised the conduct of the surveys. M. McKee, C. W. Haerpfer, D. Rotman, and S. Tumanov originated and supervised the overall study.

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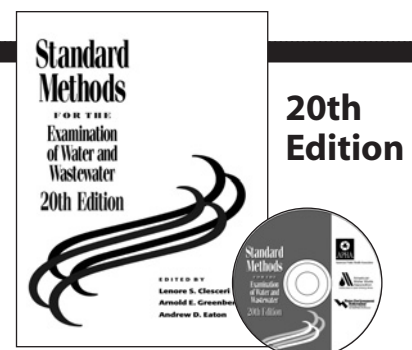
## Human Participant Protection

This study was approved by the ethics committee of the London School of Hygiene and Tropical Medicine. Verbal informed consent was obtained from all study participants at the beginning of the interviews.

## References

- Peto R, Lopez AD, Boreham J, Thun M, Heath C. *Mortality From Smoking in Developed Countries 1950–2000*. Oxford, England: Oxford University Press Inc; 1994.
- Health for All Database. Copenhagen, Denmark: World Health Organization, Regional Office for Europe; 2003.
- Forey B, Hamling J, Lee P, Wald N. *International Smoking Statistics*. 2nd ed. Oxford, England: Oxford University Press Inc; 2002.
- Grim CE, Grim CM, Petersen JR, et al. Prevalence of cardiovascular risk factors in the Republic of Georgia. *J Hum Hypertens*. 1999;13:243–247.
- Grim CE, Grim CM, Kipshidze N, Kipshidze NN, Petersen J. CVD risk factors in Eastern Europe: a rapid survey of the capital of the Republic of Georgia [abstract]. *Am J Hypertens*. 1997;10:211A.
- Corrao MA, Guindon GE, Sharma N, Shokooi DF, eds. *Tobacco Control Country Profiles*. Atlanta, Ga: American Cancer Society; 2000.
- Zaridze D, Dvornik VV, Kobljakov VA, Pisklov VP. Smoking patterns in the USSR. In: Zaridze DG, Peto R, eds. *Tobacco: A Major International Health Hazard*. Lyon, France: International Agency for Research on Cancer; 1986. IARC Scientific Publication 74.
- Tobacco or Health: A Global Status Report*. Geneva, Switzerland: World Health Organization; 1997.
- McKee M, Bobak M, Rose R, et al. Patterns of smoking in Russia. *Tob Control*. 1998;7:22–26.
- Gilmore AB, McKee M, Telishevska M, Rose R. Smoking in Ukraine: epidemiology and determinants. *Prev Med*. 2001;33:453–461.
- Gilmore AB, McKee M, Rose R. Smoking in Belarus: evidence from a household survey. *Eur J Epidemiol*. 2001;17:245–253.
- Pudule I, Grimberg D, Kadziauskiene K, et al. Patterns of smoking in the Baltic Republics. *J Epidemiol Community Health*. 1999;53:277–282.
- Raudsepp J, Rahu M. Smoking among school-teachers in Estonia 1980. *Scand J Soc Med*. 1984;12:49–53.
- Confronting the Epidemic: A Global Agenda for Tobacco Control Research*. Geneva, Switzerland: World Health Organization; 1999.
- Baris E, Waverley Brigden L, Prindiville J, Da Costa e Silva VL, Chitanondh H, Chandiwana S. Research priorities for tobacco control in developing countries: a regional approach to a global consultative process. *Tob Control*. 2000;9:217–223.

16. Lopez AD. Epidemiologic surveillance of the tobacco epidemic. *Morb Mortal Wkly Rep*. 1992; 41(suppl):157–166.
17. Connolly GN. Tobacco, trade and Eastern Europe. In: Slama K, ed. *Tobacco and Health*. London, England: Plenum Press; 1996:51–60.
18. Prokhorov AV. Getting on smokin' Route 66: tobacco promotion via Russian mass media. *Tob Control*. 1997;6:145–146.
19. Hurt RD. Smoking in Russia: what do Stalin and Western tobacco companies have in common? *Mayo Clin Proc*. 1995;70:1007–1011.
20. Krasovsky K. Abusive international marketing and promotion tactics by Philip Morris and RJR Nabisco in Ukraine. In: *Global Aggression: The Case for World Standards and Bold US Action Challenging Phillip Morris and RJR Nabisco*. New York, NY: Apex Press; 1998: 76–83.
21. Gilmore AB, McKee M. Tobacco and transition: an overview of industry investments, impact and influence in the former Soviet Union. *Tob Control*. 2004;13: 136–142.
22. Bettcher D, Subramaniam C, Guindon E, et al. *Confronting the Tobacco Epidemic in an Era of Trade Liberalisation*. Geneva, Switzerland: World Health Organization; 2001.
23. Chaloupka FJ, Laixuthai A. *US Trade Policy and Cigarette Smoking in Asia*. Cambridge, Mass: National Bureau of Economic Research; 1996. Working paper 5543.
24. EU-Copernicus Project Living Conditions: Lifestyle and Health. Vienna, Austria: Institute for Advanced Studies, 2003. Available at: <http://www.llh.at>. Accessed October 19, 2004.
25. Living Conditions, Lifestyles & Health Project Partners. Methods. Available at: [http://www.llh.at/llh\\_partners\\_start.html](http://www.llh.at/llh_partners_start.html). Accessed September 20, 2004.
26. Pomerleau J, McKee M, Rose R, Balabanova D, Gilmore A. *Living Conditions Lifestyles and Health: Comparative health report, June 2003*. London, England: London School of Hygiene and Tropical Medicine; 2003.
27. Centre for the Study of Public Policy, University of Strathclyde. New Europe Barometer Surveys. Available at: <http://www.csp.pstrath.ac.uk>. Accessed September 20, 2004.
28. Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *Br J Addict*. 1991;86:1119–1127.
29. Fagerstrom Test for Nicotine Dependence. Available at: <http://www.fpnotebook.com/PSY81.htm>. Accessed September 5, 2003.
30. Badurashvili I, McKee M, Tsuladze G, Meslé F, Vallin J, Shkolnikov V. Where there are no data: what has happened to life expectancy in Georgia since 1990? *Public Health Rep*. 2001;115:394–400.
31. Prattala R, Helasoja V, Finbalt Group. *Finbalt Health Monitor: Feasibility of a Collaborative System for Monitoring Health Behavior in Finland and the Baltic Countries*. Helsinki, Finland: National Public Health Institute; 1999.
32. Puska P, Helasoja V, Prattala R, Kasmel A, Klumbiene J. Health behaviour in Estonia, Finland and Lithuania 1994–1999. *Eur J Public Health*. 2003;13:11–17.
33. Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health*. 1994;84:1086–1093.
34. Laatikainen T, Vartiainen E, Puska P. Comparing smoking and smoking cessation processes in the Republic of Karelia, Russia and North Karelia, Finland. *J Epidemiol Community Health*. 1999;53:528–534.
35. *World Tobacco File 1997—Cigars, Smoking Tobacco and Smokeless Tobacco*. London, England: DMG Business Media; 1999.
36. Shkolnikov V, McKee M, Leon D, Chenet L. Why is the death rate from lung cancer falling in the Russian Federation? *Eur J Epidemiol*. 1999;15:203–206.
37. Ezzati M, Lopez AD. Measuring the accumulated hazards of smoking: global and regional estimates for 2000. *Tob Control*. 2003;12:79–85.
38. Lopez AD, Collishaw NE, Piha T. A descriptive model of the cigarette epidemic in developed countries. *Tob Control*. 1994;3:242–247.
39. British American Tobacco Russia. History of tobacco in Russia. Available at: [http://www.batrussia.ru/onenweb/sites/BAT\\_5FZF3V.nsf/vwPagesWebLive/DO5JVJYD?opendocument&SID=BAA08166A513AAEF3959A15BC3562EBC&DTC=20040920&TMP=1](http://www.batrussia.ru/onenweb/sites/BAT_5FZF3V.nsf/vwPagesWebLive/DO5JVJYD?opendocument&SID=BAA08166A513AAEF3959A15BC3562EBC&DTC=20040920&TMP=1). Accessed September 20, 2004.
40. British American Tobacco Russia. BAT-Yava factory history. Available at: [http://www.batrussia.ru/onenweb/sites/BAT\\_5FZF3V.nsf/vwPagesWebLive/DO5G2FWX?opendocument&SID=BAA08166A513AAEF3959A15BC3562EBC&DTC=20040920&TMP=1](http://www.batrussia.ru/onenweb/sites/BAT_5FZF3V.nsf/vwPagesWebLive/DO5G2FWX?opendocument&SID=BAA08166A513AAEF3959A15BC3562EBC&DTC=20040920&TMP=1). Accessed September 20, 2004.
41. Dragounski D. Well—this is the Russian market. *World Tob Russia Eastern Eur*. 1998;2:32–46.
42. Alcohol and Drug Information Center. Economics of tobacco control in Ukraine from the public health perspective. Available at: <http://www.adic.org.ua/adic/reports/econ>. Accessed September 20, 2004.
43. Molarius A, Parsons RW, Dobson AJ, et al. Trends in cigarette smoking in 36 populations from the early 1980s to the mid-1990s: findings from the WHO MONICA Project. *Am J Public Health*. 2001;91: 206–212.
44. *World Cigarettes 2001*. Vol. 1. Newmarket, England: ERC Group; 2001.
45. *Central and Eastern Europe Market and Mediafact*. London, England: Zenith Optimedia; 2000.
46. World Health Organization Regional Office for Europe. Tobacco control database. Available at: <http://data.euro.who.int/tobacco/>. Accessed September 20, 2004.
47. A tactical market. *Tob J Int*. 2003;1:68. Also available at: <http://www.tobacco.org/articles/country/Belarus>. Accessed September 20, 2004.
48. Pomerleau J, Gilmore A, McKee M, Rose R, Haerper CW. Determinants of smoking in eight countries of the former Soviet Union: results from the Living Conditions, Lifestyles and Health Study. *Addiction*. In press.



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# Undoing an Epidemiological Paradox: The Tobacco Industry's Targeting of US Immigrants

Dolores Acevedo-Garcia, PhD, MPA-URP, Elizabeth Barbeau, ScD, MPH, Jennifer Anne Bishop, MPH, Jocelyn Pan, PhD, MPH, and Karen M. Emmons, PhD

Tobacco use is a major health risk for groups of low socioeconomic status.<sup>1-3</sup> Among immigrants, some national-origin groups have considerably higher poverty rates and are at lower educational levels than native-born individuals.<sup>4,5</sup> However, seemingly representing a contradictory pattern—that is, an “epidemiological paradox”—rates of tobacco use are lower among certain foreign-born groups than among their US-born ethnic counterparts,<sup>6-8</sup> even when socioeconomic position is controlled.<sup>9-12</sup> For example, in 2000, only 49% of foreign-born individuals who had immigrated from Latin America to the United States were at an educational level of high school or above (as compared with 84% of foreign-born individuals from Europe and 87% of foreign-born individuals from Asia). Perez-Stable and colleagues<sup>8</sup> found that, in a sample of Latinos from 8 cities, foreign-born individuals were significantly less likely to smoke than their native-born ethnic counterparts after education had been controlled. In addition, some studies have shown that tobacco use is positively correlated with various measures of immigrant assimilation.<sup>7,8,13-23</sup>

In this article, we present preliminary evidence of the tobacco industry's efforts to market cigarettes to Asian and Hispanic immigrants residing in the United States. Because these efforts may eventually undo the protective effects of immigrant status and limited assimilation on smoking behavior, it is vital to understand them and to use this knowledge to advance tobacco control initiatives. Our interest in this issue derived from our ongoing empirical analyses of tobacco use patterns among US immigrants. Here we present the results of a pilot study investigating whether the tobacco industry has conceptualized the immigrant population as a separate market. Our findings, described subsequently, showed that the tobacco industry has a complex understanding of Asian and Hispanic immigrant

**Objectives.** We sought to ascertain whether the tobacco industry has conceptualized the US immigrant population as a separate market.

**Methods.** We conducted a content analysis of major tobacco industry documents.

**Results.** The tobacco industry has engaged in 3 distinct marketing strategies aimed at US immigrants: geographically based marketing directed toward immigrant communities, segmentation based on immigrants' assimilation status, and coordinated marketing focusing on US immigrant groups and their countries of origin.

**Conclusions.** Public health researchers should investigate further the tobacco industry's characterization of the assimilated and non-assimilated immigrant markets, and its specific strategies for targeting these groups, in order to develop informed national and international tobacco control countermarketing strategies designed to protect immigrant populations and their countries of origin. (*Am J Public Health.* 2004;94:2188-2193)

markets, has been aware of the propensity of some immigrant groups to smoke less than the general population, and has been trying to reverse such patterns since the 1970s.

## METHODS

We searched for tobacco industry documents from the 5 major US tobacco companies—American Tobacco (AT), Brown and Williamson (B&W), Lorillard, Philip Morris (PM), and RJ Reynolds (RJR)—on the University of California, San Francisco (UCSF), Legacy Tobacco Documents Library Web site (<http://legacy.library.ucsf.edu/>). Also, we searched for British American Tobacco Company (BAT) documents on the UCSF Galen Digital Library Web site (<http://www.library.ucsf.edu/tobacco/batco/>). All document searches were conducted between July and September 2003. The searches covered the period 1970 to 2003.

In the case of the 5 major US tobacco companies, we searched documents via keywords in the title field. As can be seen in Table 1, the keywords “immigrant,” “immigration,” and “assimilation” yielded a very limited number of documents. Therefore, we searched documents using the terms “Hispanic” and “Asian.” The number of documents retrieved through use of the keyword “Hispanic” was large,

ranging from 259 (Lorillard) to 3086 (RJR); in the case of PM, more than 500 documents were retrieved with the keyword “Asian.” Given the pilot nature of our study, and since our primary interest was the tobacco industry's conceptualization of these markets, we limited our searches to the keywords “Hispanic market(\*)” for Hispanics and (in the case of PM) “Asian market(\*)” for Asians; asterisks indicate wildcards that allow any form of the word “market.”

We opened and browsed all documents that were typed, original company reports; internal memos; or marketing reports produced by external consultants. We excluded duplicated documents, as well as handwritten materials and secondary documents such as press releases and scientific articles. We coded documents as “major,” “minor,” or “trivial”<sup>24</sup> in relation to their relevance to our research question, that is, whether the tobacco industry conceptualizes immigrants as a separate market. We discarded documents classified as “minor” or “trivial” (e.g., documents that did not have substantive content, such as cover letter memos in which no strategic information was conveyed, memos summarizing sales volume/amount information only, and memos summarizing distribution of promotional materials); we carefully reviewed all “major” documents.



**TABLE 1—Numbers of Documents Yielded by Tobacco Industry Document Searches**

| Title Keyword      | Tobacco Company  |                          |                      |           |               |             |
|--------------------|------------------|--------------------------|----------------------|-----------|---------------|-------------|
|                    | American Tobacco | British American Tobacco | Brown and Williamson | Lorillard | Philip Morris | RJ Reynolds |
| Immigrant          | 0                | 0                        | 0                    | 0         | 5             | 7           |
| Immigration        | 0                | 0                        | 0                    | 0         | 15            | 2           |
| Assimilation       | 0                | 0                        | 0                    | 2         | 1             | 4           |
| Hispanic           | 322              | 0                        | 767                  | 259       | 2794          | 3086        |
| Hispanic market(*) | 208              | 0                        | 102                  | 16        | 154           | 547         |
| Asian              | 35               | 39                       | 37                   | 67        | 548           | 51          |
| Asian market(*)    | N/A              | N/A                      | N/A                  | N/A       | 19            | N/A         |

*Note.* All of the documents other than those of British American Tobacco were searched at the University of California, San Francisco (UCSF), Legacy Tobacco Documents Library Web site (<http://legacy.library.ucsf.edu/>); British American Tobacco documents were searched at the UCSF Galen Digital Library Web site (<http://www.library.ucsf.edu/tobacco/batco/>). Document searches were conducted via keywords in title fields; all searches were conducted during July–September 2003. Documents involving the keyword “assimilation” were only those in which the term was used to refer to immigrant assimilation. Years covered by the searches were as follows: American Tobacco, 1970–1999; British American Tobacco, 1970–2003; Brown and Williamson, 1970–2002; Lorillard, 1970–2002; Philip Morris, 1970–2002; and RJ Reynolds, 1970–2003. Asterisks indicate wildcards that allow any form of the word “market.”

We used 2 broad initial analytical categories or themes, “immigrant market” and “assimilation,” to guide our document content analysis.<sup>25</sup> We found that the tobacco industry is aware of immigrants as potential consumers. Our subtheme analysis revealed 3 distinct marketing concepts: geographically based marketing directed at immigrant communities, segmentation based on immigrants’ assimilation status, and coordinated marketing aimed toward US immigrant groups and their countries of origin.

## RESULTS

The years covered by each of the searches are shown in Table 1. We found that the oldest documents concerning the Hispanic and Asian markets dated from the mid-1970s. The composition of the US immigrant population in terms of national origin began to change from European toward Asian and Latin American in the 1970s.<sup>26,27</sup> Therefore, these documents seem to indicate that the tobacco industry, in its market research efforts, responded quickly to changes in immigration patterns. However, tobacco companies began to address the Hispanic and Asian markets more systematically in the 1980s.<sup>28,29</sup>

The tobacco industry was keenly aware of the demographic dynamism of the immigrant population.<sup>29–33</sup> In 1985, B&W remarked

that “obviously Hispanics constitute a major market segment” because “there [are] more than 17 million Hispanics in the US, and their numbers [are] doubling every 10 years because of a high birth rate and a high level of immigration.”<sup>34</sup> B&W also noted that “Asian Americans comprised the fastest-growing population segment in the US, [providing] a steady consumer base for 555 [a brand targeted at the Asian market] to cultivate.”<sup>35</sup>

Furthermore, the industry recognized the diversity of the immigrant population and segmented the Hispanic and Asian markets along national-origin lines. For example, PM noted that Asian Americans were a “diverse population consist[ing] of at least thirteen separate ethnic groups, each with different languages and cultures.”<sup>36</sup> RJR conducted an analysis of brand preferences among Hispanic smokers according to country of origin<sup>37</sup>; similarly, PM identified that it had a larger market share among Mexicans and Central Americans than among Cubans and Puerto Ricans.<sup>38</sup>

In addition to its awareness of immigrants’ demographic importance, the tobacco industry was concerned about the low smoking rates among certain immigrant groups. According to RJR, although the Hispanic population was growing rapidly in the 1980s, smoking incidence rates among young Hispanic adults, especially young Hispanic women,

were much lower than those among the general population.<sup>29,30</sup>

## Geographic Marketing to Immigrant Communities

Tobacco companies recognized the geographic concentration patterns of various national-origin groups and used geographically focused marketing aimed at immigrant communities. RJR summarized this well: “Second only to [the] growth [of this population], the reason for targeting Hispanics lies in their geographic concentration.”<sup>29</sup>

In its Hispanic Market Development Program, B&W addressed the need to create seasonal outreach initiatives that targeted geographic areas in which there was a seasonal flow of Mexican migratory workers.<sup>34,39</sup> After monitoring the purchasing patterns of Chinese immigrants, B&W<sup>40</sup> noted that although recent Chinese immigrants residing in New York City were more geographically dispersed than members of previous immigrant waves, they still traveled to Chinatown to purchase cigarettes because of their difficulty in finding 555 cigarettes outside of that area. On the other hand, in San Francisco, “where the Chinese community is more integrated with mainstream society,” smokers were more likely to buy their cigarettes outside of Chinatown.<sup>40</sup>

## Marketing According to Assimilation Level

The tobacco industry appears to have divided immigrant markets according to their “assimilation” levels. For example, B&W recognized that many “Hispanics have not assimilated into the general population”<sup>32</sup> and that differences among Hispanic subgroups “revolve around migration history and the strength of emotional ties to the homeland.”<sup>41,42</sup> Thus, the key to reaching the Hispanic market was “striking the chord of their deep rooted heritage and language.”<sup>41</sup> Beginning in the late 1980s RJR, concerned with low smoking rates among Hispanics, suggested that this pattern was related to “Hispanic culture,” especially among female Hispanics, and that “assimilation” was a force that could promote smoking among this population.<sup>29,43</sup>

In the early 1990s, RJR and PM engaged in a more systematic effort to understand assimilation patterns among Hispanics. In



1990, National Family Opinion prepared a series of reports for RJR<sup>44–47</sup> designed “to determine the impact of assimilation for key brands in terms of purchase behavior among adult Hispanic smokers, aged 18–24.”<sup>46</sup> Specific aims included determining purchasing habits, brand and promotional awareness, and flavor preferences among Hispanics in Los Angeles, San Antonio, Houston, and Miami. On the basis of the 1989 “Hispanic tracker” (a representative sample of Hispanics in 11 local markets, as well as an additional sample of Hispanics aged 18–24 years<sup>48</sup>), these studies led to the development of an assimilation index comprising language use, birthplace, length of residence in the United States, percentage of life spent in the United States, education, and income.<sup>44</sup>

In one of these studies, it was determined that Marlboro was used primarily by “partially” to “non-assimilated Hispanics.” As Hispanics became more assimilated, awareness, trials, and purchasing levels of the Marlboro brand declined. “Fully assimilated” Hispanics preferred the Camel brand. Winston was favored less than Camel, but its brand awareness, trial, and purchasing levels increased with increasing assimilation.<sup>46</sup>

PM and its advertising agency, Leo Burnett, conducted their own set of tracking studies in the early 1990s in an effort to determine the impact of assimilation on the Hispanic market and to formulate a subsequent outreach strategy.<sup>33,49–53</sup> According to PM, understanding and tracking assimilation over time was important because Hispanics’ degree of assimilation predicted their overall values, attitudes, cultural orientations, media/product consumption patterns, and reactions to advertising.<sup>51</sup>

Some of the documents illustrated how the industry recognized variations in assimilation levels according to national origin and geographic location. For example:

Although Hispanics will probably retain their cultural differences, the degree to which they are becoming “Americanized” seems to vary from city to city. As a result of these differences, regional marketing activities, based upon both the Spanish cultural heritage and the local environment, may be more effective in reaching Hispanic smokers than a national program.<sup>30</sup>

During 1991–1992, RJR was concerned about the poor performance of Camel in heavily Hispanic markets such as Los Angeles and

attributed this problem to the poor performance of the brand among “non-assimilated” Hispanics. Consequently, the company increased its efforts to “better understand the assimilated/non-assimilated phenomenon within the Hispanic community” and “improve Camel’s performance among the non-assimilated Hispanic smoker.”<sup>54,55</sup> RJR’s definition of “non-assimilated Hispanic” included the following components: “Spanish-driven,” “less than five years of residence in the US,” “maintains cultural roots,” and “low socioeconomic level.” This definition fit 50% of the Southern California Hispanic market at the time.<sup>54,55</sup> Similarly, PM sought to understand the strong Marlboro loyalty among Mexican Americans, conducting research on the purchasing patterns of “totally assimilated,” “partially assimilated,” and “unassimilated” Mexican consumers to determine differences in brand loyalty to Marlboro among these subgroups.<sup>38</sup>

In regard to the Asian American market, RJR stressed the need to understand “the changing balance of being Asian and being American among [this] target audience.”<sup>56</sup> PM characterized the 4 largest Asian American groups according to the extent to which they were foreign born and their assimilation level. While the majority of Chinese and Korean Americans were foreign born, more than 70% of Japanese Americans were US born and consequently represented “America’s most assimilated Asian-Americans.” Filipinos were characterized as “the least ‘foreign’ of all [foreign-born] Asian-Americans” owing to their strong command of English.<sup>36</sup>

### Coordinated Marketing to Immigrants and Their Countries of Origin

The tobacco companies analyzed the domestic market performance of cigarette brands among specific immigrant groups in relation to their performance in the respective countries of origin. For instance, PM made an effort to understand why Marlboro’s market share in Mexico was only half of its share among Mexicans in the United States so that the company could increase cigarette sales among US immigrants.<sup>57</sup> RJR developed a binational border area marketing program aimed at penetrating the 5 major US–Mexico metropolitan areas in a synchronized fashion.<sup>54</sup>

Tobacco companies carefully tracked postimmigration cigarette brand switching behaviors. In a report commissioned by PM, Leo Burnett noted that “it was important to reach [new immigrants] early as they shape their brand preferences in the US.”<sup>52</sup> BAT and its subsidiary, B&W, coordinated their targeting of the Asian and Asian American markets for the 555 brand.<sup>35,40,58–60</sup> In 1992, among its advertising objectives for 555, BAT included “reach[ing] Asian Americans, primarily Chinese, with an awareness of the heritage of 555 from their immigrating countries” and “reinforce[ing] the premium, well established and quality image of 555 that is inherent in the brand from the smokers['] country of origin.”<sup>58</sup>

B&W reported that while 555 was popular in mainland China, Chinese immigrants living in the United States were reluctant to try this brand in their new homeland because they associated it with “government officials in China.”<sup>40</sup> B&W also noted that Vietnamese smokers associated this brand with “positive images and nostalgic memories of Vietnam” but that their brand loyalty might weaken if they realized that, unlike in Vietnam, 555 was not a “status symbol” in the United States.<sup>40</sup>

While 555 evoked images among both Chinese and Vietnamese smokers of their countries of origin, Marlboro was associated with “American culture.”<sup>40</sup> This brand was rejected by those individuals “who expressed negative sentiments towards the American culture and took strong pride in their own,” whereas it was appealing to individuals “who want[ed] to ‘blend in’ with American culture.”<sup>40</sup> B&W was concerned that “Marlboro’s youth-oriented image [was] likely to lure away many younger [Chinese and Vietnamese] adult smokers who were rightfully 555’s overseas,”<sup>35</sup> and noted that there was untapped potential among Indian and Pakistani smokers who had “positive exposure to 555 in their homelands.”<sup>35</sup>

## DISCUSSION

We found evidence of the tobacco industry’s efforts to achieve a sophisticated characterization of tobacco use patterns among Asian and Hispanic immigrants (e.g., brand

loyalty) according to their geographic location, assimilation level, and smoking patterns in their countries of origin. Our preliminary findings underscore the need to investigate further the tobacco industry's characterization of the assimilated and non-assimilated immigrant markets, and its specific strategies for targeting these groups, in order to develop informed national and international tobacco control countermarketing strategies designed to protect both immigrant populations and their countries of origin.

Public health researchers are currently striving to add foreign-born status to US tobacco surveillance systems and to better understand the causes of its protective effect in regard to smoking.<sup>6</sup> The present study showed that the major cigarette manufacturers in the United States are aware of the protective effect of limited assimilation on immigrants' tobacco use and have been considering ways to undo it.

As a result of its pilot nature, our study involved several limitations that can be addressed in future research. First, we were unable to mine the approximately 8000 documents generated with the keywords "Hispanic" and "Asian." It is noteworthy, though, that despite our limited search, we were able to uncover evidence of industry efforts directed toward immigrants; more comprehensive searches are likely to unearth additional rich information.

Second, we focused on the tobacco industry's conceptualization of immigrant markets. Further research is needed to understand specific marketing strategies (e.g., product creation, positioning, pricing, and promotion) directed toward increasing tobacco use among immigrants. Our searches uncovered examples of such strategies that we are pursuing further; examples are RJR's Hispanic Tracking System (which involves comprehensive surveys of the Hispanic market, including assimilation levels), established in 1988, and its Regional Initiative Program (a marketing initiative aimed at the major US Hispanic markets), established in the early 1990s.

Third, we found that, to some extent, all companies have categorized Hispanic and Asian immigrant markets, but our searches yielded more evidence of this practice on the part of the 2 largest manufacturers than on

the part of the others. PM and RJR appeared to engage in the most comprehensive and sophisticated marketing efforts aimed at these groups, as evidenced by the research studies they commissioned<sup>33,44-47,49-51</sup> with the explicit purpose of gaining an understanding of immigrant markets and the role of assimilation. Other tobacco companies recognized that RJR and PM had addressed these markets more extensively,<sup>34</sup> and B&W as well engaged in significant market research efforts in promoting the 555 brand among Asian immigrants.<sup>40,58,60</sup> Future, more comprehensive searches may reveal that American Tobacco, BAT, and Lorillard also engaged in extensive marketing efforts aimed at immigrants.

Fourth, the documents we identified, as well as the industry's characterization of its targeting of Asians in such documents,<sup>28</sup> yielded more evidence of targeting of Hispanic immigrants than of Asian immigrants, especially owing to RJR and PM's comprehensive targeting of Hispanics. However, given the size of the tobacco document collections, it would not be appropriate to draw such conclusions from this study.

Muggli et al.<sup>61</sup> analyzed 1985 to 1995 tobacco industry documents regarding the Asian/Pacific Islander population of the United States and found that this population became a priority for the industry in the 1980s. They also found strong evidence of the industry's awareness of the high population growth and purchasing power of this group, the high smoking rates in their countries of origin, and the marketing possibilities offered by their desire to assimilate to American culture. Our findings corroborated those of Muggli et al. In addition, we showed that targeting of immigrants has been a point of convergence in the industry's marketing efforts aimed at Hispanic and Asian Americans. In both markets, the tobacco industry has used geographic targeting, segmentation according to assimilation status, and coordinated targeting of immigrants and their countries of origin.

Finally, although our study uncovered evidence of targeting of immigrants and their countries of origin, a deeper understanding of such coordinated targeting efforts may help enhance global tobacco control. There is a need for studies analyzing the industry's

marketing efforts aimed at specific national-origin groups (e.g., Mexican Americans and Vietnamese Americans) as well as their countries of origin.

Tobacco use is increasingly being seen as a global health issue.<sup>62-67</sup> Of major concern are the disparities in tobacco use patterns between developed and less developed countries. In developed countries, tobacco use has declined in the past 45 years and is becoming socially unacceptable; however, in less developed countries tobacco use has increased, along with the social acceptability of smoking.<sup>65,68</sup> For example, in the United States, the smoking prevalence rate among adults decreased from 42% in 1965 to 24% in 1998.<sup>2</sup> Conversely, between the mid-1950s and mid-1990s, rates in developing countries increased from 20% to 50% among men and from virtually 0% to 8% among women.<sup>68</sup> Furthermore, the majority of recent US immigrants hail from less developed countries in Asia and Latin America, where tobacco use is becoming more widespread.

As the domestic policy environment becomes less favorable to tobacco interests, the US tobacco industry is intensifying its operations in less developed countries.<sup>69-71</sup> Our findings suggest that tobacco companies are not only aware of the interaction between assimilation and tobacco use but target US immigrant groups and their countries of origin in a coordinated fashion. Knowledge of the tobacco industry's targeting of immigrants can be used by US state health departments and community-based voluntary organizations to develop countermarketing strategies.<sup>72</sup> ■

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D. Acevedo-Garcia conceived the study, conducted the tobacco industry document searches and analyses, and wrote the article. E. Barbeau and J.A. Bishop contributed to the design and analysis of the study and edited the article. J. Pan and K.M. Emmons reviewed and edited the article.

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No protocol approval was needed for this study

## References

- Pamuk E, Makuc D, Heck K, Reuben C, Lochner K. *Socioeconomic Status and Health Chartbook*. Hyattsville, Md: National Center for Health Statistics; 1998.
- Centers for Disease Control and Prevention. Cigarette smoking among adults—United States, 1998. *MMWR Morb Mortal Wkly Rep*. 2000;49: 881–884.
- Barbeau E, Krieger N, Soobader M-J. Working class matters: socioeconomic disadvantage, race/ethnicity, gender, and smoking in the National Health Interview Survey, 2000. *Am J Public Health*. 2004;94:269–278.
- Coming to America: A Profile of the Nation's Foreign Born*. Washington, DC: US Census Bureau; 2000.
- Schmidley AD. *The Foreign-Born Population in the United States: March 2002*. Washington, DC: US Census Bureau; 2003.
- Baluja KF, Park J, Myers D. Inclusion of immigrant status in smoking prevalence statistics. *Am J Public Health*. 2003;93:642–646.
- Harris KM. The health status and risk behaviors of adolescents in immigrant families. In: Hernandez DJ, ed. *Children of Immigrants: Health, Adjustment and Public Assistance*. Washington, DC: National Academy Press; 1999:286–315.
- Perez-Stable EJ, Ramirez A, Villareal R, et al. Cigarette smoking behavior among US Latino men and women from different countries of origin. *Am J Public Health*. 2001;91:1424–1430.
- Franzini L, Ribble JC, Keddle AM. Understanding the Hispanic paradox. *Ethn Dis*. 2001;11:496–518.
- Fuentes-Afflick E, Lurie P. Low birth weight and Latino ethnicity: examining the epidemiologic paradox. *Arch Pediatr Adolesc Med*. 1997;151:665–674.
- Scribner R. Paradox as paradigm—the health outcomes of Mexican Americans. *Am J Public Health*. 1996;86:303–305.
- Palloni A, Morenoff JD. Interpreting the paradoxical in the Hispanic paradox: demographic and epidemiologic approaches. *Ann N Y Acad Sci*. 2001;954: 140–174.
- Chen X, Unger JB, Johnson CA. Is acculturation a risk factor for early smoking initiation among Chinese American minors? A comparative perspective. *Tob Control*. 1999;8:402–410.
- Acevedo MC. The role of acculturation in explaining ethnic differences in the prenatal health-risk behaviors, mental health, and parenting beliefs of Mexican American and European American at-risk women. *Child Abuse Negl*. 2000;24:111–127.
- Cobas JA, Balcazar H, Benin MB, Keith VM, Chong Y. Acculturation and low-birthweight infants among Latino women: a reanalysis of HHANES data with structural equation models. *Am J Public Health*. 1996;86:394–396.
- Amaro H, Whitaker R, Coffman G, Heeren T. Acculturation and marijuana and cocaine use: findings from HHANES 1982–84. *Am J Public Health*. 1990; 80(suppl):54–60.
- Conrod DV, Balcazar H, Brady J, Garcia S, Van Tine M. Smoking, acculturation and family cohesion in Mexican-American women. *Ethn Dis*. 1999;9: 434–440.
- Kerner JF, Breen N, Tefft MC, Silsby J. Tobacco use among multi-ethnic Latino populations. *Ethn Dis*. 1998;8:167–183.
- Tobacco Use Among US Racial/Ethnic Minority Groups—African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics: A Report of the Surgeon General*. Washington, DC: US Dept of Health and Human Services; 1998.
- Unger JB, Cruz TB, Rohrbach LA, et al. English language use as a risk factor for smoking initiation among Hispanic and Asian American adolescents: evidence for mediation by tobacco-related beliefs and social norms. *Health Psychol*. 2000;19:403–410.
- Cigarette smoking among Southeast Asian immigrants—Washington State, 1989. *MMWR Morb Mortal Wkly Rep*. 1992;41:854–855.
- Chen X, Unger JB, Cruz TB, Johnson CA. Smoking patterns of Asian-American youth in California and their relationship with acculturation. *J Adolesc Health*. 1999;24:321–328.
- Siegel D, Faigles B. Smoking and socioeconomic status in a population-based inner city sample of African-Americans, Latinos and whites. *J Cardiovasc Risk*. 1996;3:295–300.
- Balbach ED, Gasior R, Barbeau EM. Tobacco industry documents: comparing the Minnesota Depository and Internet access. *Tob Control*. 2002;11:68–72.
- Pope C, Ziebland S, Mays N. Analyzing qualitative data. *BMJ*. 2000;320:114–116.
- Rumbaut RG, Portes A. Introduction—ethnogenesis: coming of age in immigrant America. In: Rumbaut RG, Portes A, eds. *Ethnicities: Children of Immigrants in America*. Berkeley, Calif: University of California Press; 2001:1–19.
- Borjas GJ. *Heaven's Door: Immigration Policy and the American Economy*. Princeton, NJ: Princeton University Press; 1999.
- Prospectus: special markets—Asian Americans. RJ Reynolds. Bates No. 507091820–1824. Available at: <http://legacy.library.ucsf.edu/tid/rf28c00>. Accessed August 8, 2003.
- Hispanic market opportunity assessment progress update presentation. RJ Reynolds. Bates No. 505336674–6721. Available at: <http://legacy.library.ucsf.edu/tid/xli25d00>. Accessed August 8, 2003.
- Hispanic market overview. RJ Reynolds. Bates No. 504051125–1153. Available at: <http://legacy.library.ucsf.edu/tid/hbg75d00>. Accessed August 21, 2003.
- Hispanic market: 1985. RJ Reynolds. Bates No. 504667010–7019. Available at: <http://legacy.library.ucsf.edu/tid/oqa65d00>. Accessed August 21, 2003.
- The Hispanic-American market and its implications for marketers. Ogilvy & Mather. Bates No. 670616220–6427. Available at: <http://legacy.library.ucsf.edu/tid/hqu13f00>. Accessed August 26, 2003.
- Action plan for the US Hispanic market. Leo Burnett Agency, Philip Morris. Bates No. 2043022839–2946. Available at: <http://legacy.library.ucsf.edu/tid/bps03e00>. Accessed September 8, 2003.
- Kavanaugh MJ. Raleigh con Filtro/Fiesta—Hispanic market development program. Brown & Williamson. Bates No. 524102081–2082. Available at: <http://legacy.library.ucsf.edu/tid/jav23f00>. Accessed August 27, 2003.
- An ideal plan for B&W in the Asian American market. L 3 Advertising Inc, Brown & Williamson. Bates No. 502010038–0047. Available at: <http://legacy.library.ucsf.edu/tid/lqt03f00>. Accessed September 11, 2003.
- Cohen D. Asian American market study. Philip Morris. Bates No. 2045652289–2299. Available at: <http://legacy.library.ucsf.edu/tid/dsg36e00>. Accessed September 8, 2003.
- Stanley P. Consumer research reports: consumer performance in major Hispanic markets. RJ Reynolds. Bates No. 505310452–0468. Available at: <http://legacy.library.ucsf.edu/tid/rj125d00>. Accessed August 19, 2003.
- Hispanic Marlboro smokers. Philip Morris. Bates No. 2049162714–2747. Available at: <http://legacy.library.ucsf.edu/tid/txx95e00>. Accessed August 25, 2003.
- Falk D. Hispanic market in Miami, Florida. Brown & Williamson. Bates No. 555006580–6582. Available at: <http://legacy.library.ucsf.edu/tid/nyz13f00>. Accessed September 10, 2003.
- 555 performance study: 96 Asian focus groups, New York and San Francisco metro. Strategic Marketing Asia Ltd, Brown & Williamson. Bates No. 502033026–3075. Available at: <http://legacy.library.ucsf.edu/tid/apr91d00>. Accessed September 11, 2003.
- Kovatch M, Sonog F. Expanding opportunities in a growing segment profile: the Black and Hispanic market in Chicago. Brown & Williamson. Bates No. 670135322–5348. Available at: <http://legacy.library.ucsf.edu/tid/ykw79e00>. Accessed September 10, 2003.
- Yankelovich D. A study of the Hispanic market by Yankelovich, Skelly & White, Inc. Bates No. 670638118–8138. Available at: <http://legacy.library.ucsf.edu/tid/oju13f00>. Accessed September 5, 2003.
- Opportunity assessment: Hispanic market. RJ Reynolds. Bates No. 509148364–8425. Available at: <http://legacy.library.ucsf.edu/tid/rjd38c00>. Accessed August 21, 2003.
- Hispanic assimilation index. National Family Opinion, RJ Reynolds. Bates No. 507522594–2613. Available at: <http://legacy.library.ucsf.edu/tid/hdm24d00>. Accessed September 4, 2003.
- Hispanic assimilation and growth. National Family Opinion, RJ Reynolds. Bates No. 509227676–7682. Available at: <http://legacy.library.ucsf.edu/tid/szd38c00>. Accessed September 4, 2003.
- Hispanic assimilation analysis: Hispanic adult smokers aged 18–24. National Family Opinion, RJ Reynolds. Bates No. 509162818–2868. Available at:



- <http://legacy.library.ucsf.edu/tid/szz73d00>. Accessed September 4, 2003.
47. Simmons S. Relative Hispanic population sizes: 18–24 within assimilation level. National Family Opinion, RJ Reynolds. Bates No. 507521742–1745. Available at: <http://legacy.library.ucsf.edu/tid/qbm24d00>. Accessed September 4, 2003.
  48. Winston 1997: Hispanic tracker—pre-wave results. RJ Reynolds. Bates No. 517094159–4159. Available at: <http://legacy.library.ucsf.edu/tid/nkr82d00>. Accessed September 4, 2003.
  49. The U.S. Hispanic market overview for Virginia Slims. Leo Burnett Agency, Philip Morris. Bates No. 2041817689–7750. Available at: <http://legacy.library.ucsf.edu/tid/orr66e00>. Accessed September 8, 2003.
  50. Hispanic orientation: 1992. Leo Burnett Agency, Philip Morris. Bates No. 2042911541–1579. Available at: <http://legacy.library.ucsf.edu/tid/qvw95e00>. Accessed September 4, 2003.
  51. Demystifying the US Hispanic market. Leo Burnett Agency, Philip Morris. Bates No. 2060124376–4419. Available at: <http://legacy.library.ucsf.edu/tid/lsr76e00>. Accessed September 8, 2003.
  52. Marlboro: Hispanic marketing and media objectives and strategies. Leo Burnett Agency, Philip Morris. Bates No. 2045414266–4285. Available at: <http://legacy.library.ucsf.edu/tid/fhz76e00>. Accessed September 2, 2003.
  53. Rodriguez Y. Marketing to Hispanic smokers: a survey study of assimilation and cigarette brand preference. Philip Morris. Bates No. 2049162931–2980. Available at: <http://legacy.library.ucsf.edu/tid/ist83e00>. Accessed September 4, 2003.
  54. 1991 regional initiative business plan. RJ Reynolds. Bates No. 508737339–7372. Available at: <http://legacy.library.ucsf.edu/tid/puz83d00>. Accessed July 25, 2003.
  55. 1992 regional initiative program: Southern California region. RJ Reynolds. Bates No. 518049874–9911. Available at: <http://legacy.library.ucsf.edu/tid/zdb82d00>. Accessed July 28, 2003.
  56. Kang E. Asian American market. RJ Reynolds. Bates No. 522416347/6385. Available at: <http://legacy.library.ucsf.edu/tid/dmq01d00>. Accessed August 21, 2003.
  57. Marlboro Hispanic market qualitative research. Philip Morris. Bates No. 2061809833–9836. Available at: <http://legacy.library.ucsf.edu/tid/one07e00>. Accessed September 4, 2003.
  58. 555—preliminary 1992 media plan. British American Tobacco Co. Bates No. 503852954–2959. Available at: <http://www.library.ucsf.edu/tobacco/batco/html/200/297/index.html>. Accessed September 15, 2003.
  59. A P.R. proposal for State Express 555 rally program in the Asian American market. L 3 Advertising Inc, Brown & Williamson. Bates No. 502010197–0201. Available at: <http://legacy.library.ucsf.edu/tid/vsn91d00>. Accessed September 11, 2003.
  60. 555 GCS tracking study: US Asian market supplement 1994 & 1996 summary tables. Brown & Williamson. Bates No. 461219003–9021. Available at: <http://legacy.library.ucsf.edu/tid/hqj81d00>. Accessed September 11, 2003.
  61. Muggli ME, Pollay R, Lew R, Joseph A. Targeting of Asian American and Pacific Islanders by the tobacco industry: results from the Minnesota Tobacco Document Depository. *Tob Control*. 2002;11:201–209.
  62. Mackay J. The global tobacco epidemic: the next 25 years. *Public Health Rep*. 1998;113:14–21.
  63. Bartecchi CE, MacKenzie TD, Schrier RW. The global tobacco epidemic. *Scientific Am*. 1995;272(5):44–51.
  64. Rafei UM. Tobacco epidemic. *J Indian Med Assoc*. 1999;97:374.
  65. Lopez AD, Collishaw NE, Piha T. A descriptive model of the cigarette epidemic in developed countries. *Tob Control*. 1994;3:242–247.
  66. *Confronting the Epidemic: A Global Agenda for Tobacco Control Research*. Geneva, Switzerland: World Health Organization; 1999.
  67. Lopez AD. Counting the dead in China: measuring tobacco's impact in the developing world. *BMJ*. 1998;317:1399–1400.
  68. *What in the World Works?* Singapore: World Health Organization; 1999.
  69. Jenkins CN, Dai PX, Ngoc DH, et al. Tobacco use in Vietnam: prevalence, predictors, and the role of the transnational tobacco corporations. *JAMA*. 1997;277:1726–1731.
  70. Stebbins KR. Tobacco or health in the Third World: a political economy perspective with emphasis on Mexico. *Int J Health Serv*. 1987;17:521–536.
  71. Mackay J. Battling upstream against the tobacco epidemic in China. *Tob Control*. 1997;6:9–10.
  72. Ling P, Glantz S. Using tobacco-industry marketing research to design more effective tobacco-control campaigns. *JAMA*. 2002;287:2983–2989.



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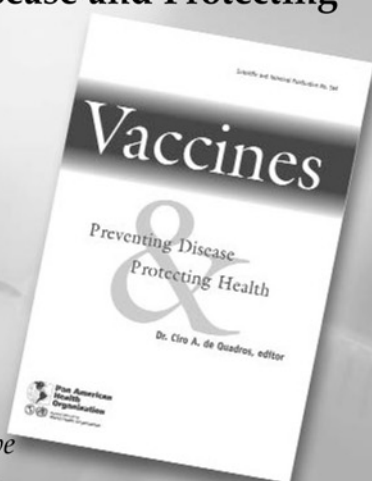
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## POST-DOCTORAL RESEARCH FELLOWSHIP IN BEHAVIORAL SCIENCES RESEARCH IN HIV INFECTION

Positions will open as of July 2005 in a NIMH-funded institutional research training program at the **HIV Center for Clinical and Behavioral Studies, Columbia University and the New York State Psychiatric Institute**. Our program is an innovative research fellowship in human sexuality as applied to HIV-related risk, health and prevention. Trainees receive up to three years of support for stipends, health insurance, travel for conferences, and research. Tuition support is available for concurrent matriculation in a Master of Science degree program in Biostatistics and other disciplines relevant to HIV and sexuality research. Applicants must be U.S. citizens or permanent residents. Information about the program is available on the internet at <http://www.hivcenternyc.org/training/trainingbigf.html>. To request an application packet call Bob Harbaum, 212-543-5751, email [harbaum@pi.cpmc.columbia.edu](mailto:harbaum@pi.cpmc.columbia.edu). **Deadline for applications is February 1, 2005. AA/EOE.**

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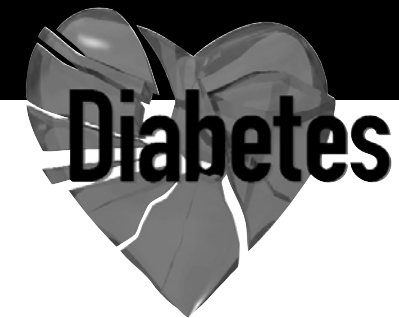
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# JOB OPPORTUNITIES

## COLORADO

**Assistant/Associate Professor in Behavioral Sciences:** The Department of Preventive Medicine and Biometrics and the Rocky Mountain Prevention Research Center (RMPRC) at the University of Colorado Health Sciences Center are recruiting to fill a new, full-time, tenure-eligible Assistant or Associate Professor position. Doctoral or comparable level training is required in public health, sociology, anthropology, social psychology or related field with expertise in areas that include nutrition, physical activity, community-based participatory research, obesity and diabetes. Salary commensurate with experience. Interested candidates please send a letter, curriculum vitae, and list of 3 references to [Debra.Becker@uchsc.edu](mailto:Debra.Becker@uchsc.edu) or Debra Becker, UCHSC-B119, 4200 East 9th Avenue, Denver CO 80262.

For details see

<http://www.uchsc.edu/pmb/epi/index.htm>. Application review begins 12-01-04 until position is filled. EOE: The University of Colorado is committed to diversity and equality in education and employment.

## KANSAS

**Dental Director:** The Kansas Department of Health and Environment in Topeka, KS is seeking qualified applicants for a new State Dental Director position (Health Officer II). Salary range is \$70,000-\$120,000. For more information about this position go to [www.accesskansas.org](http://www.accesskansas.org) and click on Kansas Government Jobs Requisition #144827. For more information about Oral Health Initiatives in Kansas go to [www.kdhe.state.ks.us/ohi](http://www.kdhe.state.ks.us/ohi). Contact person is Linda Kenney at email [lkenney@kdhe.state.ks.us](mailto:lkenney@kdhe.state.ks.us) or phone (785) 296-1310. EOE.

## NEW HAMPSHIRE

**Fellowships:** University of New Hampshire, -2004 Family Research Laboratory (FRL) has fellowships for research on family violence. These positions are open to new and experienced researchers with a Ph.D. Annual stipends run from \$35,568 to \$51,036 depending upon the number of years since receipt of doctorate. For more information visit website: [www.unh.edu/frl](http://www.unh.edu/frl). EOE.

## UMDNJ - NEW JERSEY DENTAL SCHOOL

### FACULTY OPPORTUNITIES *Endowed Professors in Community Health*

UMDNJ - New Jersey Dental School is currently seeking applicants for two Hunterdon Endowed Professors for our Department of Community Health. The successful candidates will be responsible for furthering the school's community service programs throughout the State of New Jersey and will work closely with faculty and staff from our State-Wide-Network of Oral Health Care, a network of community-based clinics offering dental care, health promotion and prevention activities to underserved communities. Candidates will also be responsible for conducting health services and behavioral science research, advancing our knowledge of factors that influence access to utilization and outcomes of health care services.

Qualified candidate must have extensive experience in dental education and research in oral health promotion with emphasis on community-based programs in health disparities in disadvantage/minority populations. Candidates should also have a successful history of continuous independent funding from NIH, HRSA, other federal, state, industry or not-for-profit agencies. A Ph.D. or equivalent terminal degree is desirable but a DMD/DDS degree will also be considered.

This position is available October 1, 2004, however the search will remain open until the position is filled by a qualified candidate. Salary and academic rank are commensurate with background and experience. Letter of interest, curriculum vitae and names of three references should be sent to: **Dr. Michael Deasy, UMDNJ-New Jersey Dental School, 110 Bergen Street, Newark, New Jersey 07103-2400.** UMDNJ is an Affirmative Action/Equal Opportunity Employer, M/F/D/V, and a member of the University Health System of New Jersey.



NEW JERSEY  
DENTAL SCHOOL

University of Medicine & Dentistry of New Jersey

## UMDNJ - NEW JERSEY DENTAL SCHOOL

### FACULTY OPPORTUNITY *Endowed Professor in Dental Public Health*

UMDNJ - New Jersey Dental School in conjunction with the UMDNJ-School of Public Health is currently seeking an individual to fill the Hunterdon Endowed Professor in Dental Public Health. The successful candidate will be responsible for the continued development of a curriculum in dental public health at the predoctoral and MPH level. The candidate is expected to conduct dental public health research, advance oral health through the design of dental public health programs, which UMDNJ offers throughout the State of New Jersey, and mentor students, graduate students and faculty interested in dental public health. A joint appointment with the UMDNJ-School of Public Health will be given.

Qualified candidate should have a successful history of continuous independent funding from NIH, HRSA, other federal, state, industry or not-for-profit agencies. Board certification in Dental Public Health is desirable along with a DMD/DDS degree and eligibility for licensure or teaching permit in the State of New Jersey. Candidates with a Ph.D. or equivalent terminal degree will also be considered.

This position is available October 1, 2004, however the search will remain open until the position is filled by a qualified candidate. Salary and academic rank are commensurate with background and experience. Letter of interest, curriculum vitae and names of three references should be sent to: **Dr. Michael Glick, UMDNJ-New Jersey Dental School, 110 Bergen Street, Newark, New Jersey 07103-2400.** UMDNJ is an Affirmative Action/Equal Opportunity Employer, M/F/D/V, and a member of the University Health System of New Jersey.



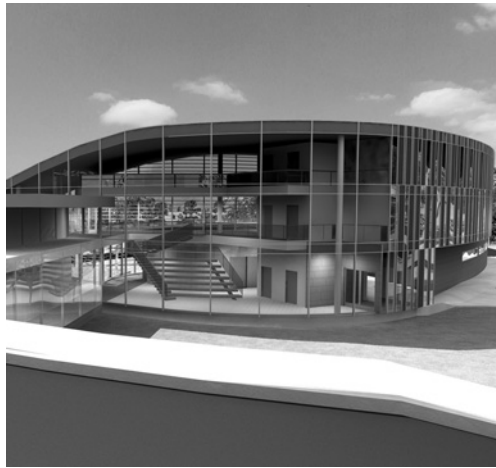
NEW JERSEY  
DENTAL SCHOOL

University of Medicine & Dentistry of New Jersey



# The Center for Health Research & Rural Advocacy

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The Center for Health Research & Rural Advocacy is uniquely positioned to conduct epidemiologic research on the broad range of conditions typically seen in primary and specialty care settings.

Geisinger's population is relatively stable geographically; most patients remain in our service area for years—frequently over several generations. This provides an ideal foundation for longitudinal studies of chronic diseases. Geisinger has a state-of-the-art, integrated electronic health record (EHR) system that has been in place since 1997. The EHR captures detailed information on patients seeking care in primary and specialty care settings and includes data on diagnosis, prescriptions and lab values, as well as imaging, structured clinical notes and supplementary patient questionnaire data. The Center is also currently building a system-wide DNA and serum repository.

**To find out more about available positions, please contact:**

Nicole Micozzi, Director of Recruitment  
Geisinger Health System  
Tel: 1-877-JOIN-GHS (564-6447)  
Fax: 570-271-7158  
e-mail: [namicozzi@geisinger.edu](mailto:namicozzi@geisinger.edu)  
100 North Academy Avenue  
Danville, Pennsylvania 17822-2428

The Center for Health Research & Rural Advocacy is seeking:

**Biostatistical Analyst II (Master's Level) and Staff Biostatistician (PhD Level):** We are seeking experienced biostatistical analysts to join our research team. Candidates will work with a multi-disciplinary team on large scale epidemiologic and health services research projects. Tools like Geisinger's comprehensive outpatient electronic health records provide unique opportunities for outcomes and effectiveness research.

**Genetic Epidemiologist:** We are seeking investigators who have interest in epidemiologic or genetics research and independent senior collaborative research in vascular diseases, diabetes, chronic lung diseases, addictions or obesity. Center investigators will have access to unique longitudinal data on clinical, pharmacy, lab, and medical claims measures and a patient population DNA and serum repository on a very stable population residing in a 38-county region of Pennsylvania. The successful candidate will have opportunities to participate in ongoing research and will be expected to have (or to establish), a collaboratively funded research program, working with multidisciplinary teams across different divisions within Geisinger. Applicants should have a doctorate in epidemiology or related discipline, training and experience in genetic epidemiology and a proven record of funded research and scholarship.

**Environmental Epidemiologist:** We are recruiting an experienced investigator who has a strong interest in environmental epidemiology. Interest in water-related disease risks is a plus. The highly stable population and unique data routinely captured by Geisinger Health System opens unique opportunities for funded research. The successful candidate will have opportunities to participate in ongoing research and will be expected to have or establish a collaboratively funded research program, working with multidisciplinary teams across different divisions within Geisinger Health System and universities in central and northeastern Pennsylvania. Applicants should have a doctorate in epidemiology, and a proven record of funded research and scholarship.

**Health Services Epidemiologist:** We are seeking investigators who have an interest in health services research with a particular focus on either electronic health record and e-patient portal technologies or effectiveness research. Research opportunities include retrospective EHR-based effectiveness studies, and prospective intervention studies applied to the evaluation of clinical guidelines, innovative use of an EHR, and new models of care delivery and finance. Experienced candidates will have the opportunity to access a large longitudinal EHR database and to work with an inter-disciplinary research and health information technology team. The successful candidates will also have opportunities to participate in ongoing research and will be expected to establish a collaboratively funded research program, working with multidisciplinary teams across different divisions within Geisinger. Applicants should have a doctorate in public health or related discipline and a proven record of funded research and scholarship.

[www.geisinger.org/research](http://www.geisinger.org/research)



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## **ASSISTANT/ASSOCIATE PROFESSOR**

The Department of Global Health, Emory University Rollins School of Public Health is recruiting for a tenure-track position at the level of Assistant or Associate Professor. The successful candidate will be appointed as a Rollins Assistant/Associate Professor, a chair that will be held until tenure is awarded.

Candidates should have a doctoral degree and demonstrated commitment to research, teaching and service. Preference will be given to candidates with a strong research record, including current funded research in any area of interest to the faculty, including: community health, environmental health, health economics, infectious diseases, nutrition, and population and reproductive health. Experience in global health policy; health communications; health service research; and/or program management, design and evaluation will be valued. The candidate will be expected to teach two courses per year, one on methods and another in his/her substantive area and to develop an externally-funded research portfolio.

The Department of Global Health consists of over 30 full-time graduate faculty or jointly appointed faculty and over 60 adjunct faculty (<http://www.sph.emory.edu/hpdih.html>). The department takes pride in its collegial environment and culture of collaborative research and teaching. About 70 MPH/MSPH students are admitted yearly to our 2-year program. Department faculty are closely involved with the Nutrition and Health Sciences PhD program.

Emory is a major, top 25 ranked, AAU Research University. Exciting opportunities for research and collaboration exist with other departments of the School, other units of Emory University, the Centers for Disease Control and Prevention, and CARE International. The department also has an extensive network of collaborating institutions and agencies abroad.

Interested persons should send a letter indicating their interest accompanied by a curriculum vitae and the name of three persons to whom we may solicit references; these should be sent to Reynaldo Martorell, Chair, Department of Global Health, 1518 Clifton Road, N.E., Atlanta, Georgia 30322 USA. Screening of applications will continue until the position is filled. Starting date is negotiable.

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## **William H. Foege Chair in Global Health**

The Department of Global Health, Emory University Rollins School of Public Health, is recruiting for the William H. Foege Chair in Global Health. The chair is established in honor of William (Bill) H. Foege, who recently retired from Emory University where he was the Presidential Distinguished Professor of Public Health. Dr. Foege is a former director of the Centers for Disease Control and Prevention, the first Executive Director of The Carter Center of Emory University, and currently the Senior Advisor for Health for the Gates Foundation.

The Department of Global Health (<http://www.sph.emory.edu/hpdih.html>) consists of over 30 full-time faculty members, many with joint appointments across the University, and over 60 adjunct faculty members. Faculty interests include infectious diseases, including HIV and AIDS, nutrition, reproductive health, global environmental health and population and community health. About 70 MPH/MSPH students are admitted annually. The department hosts several international fellows programs, including the recently-established Foege Fellowships for emerging public health leaders from developing countries.

The Rollins School of Public Health employs 120 full-time faculty members and enrolls over 800 full and part-time graduate students in its masters and doctoral programs. The School is located on the Emory University campus, adjacent to the CDC, the American Cancer Society, Emory's Schools of Nursing and Medicine and a number of laboratory and clinical facilities. The Carter Center and CARE International are nearby. The School includes six academic departments and offers joint MPH degrees with the schools of medicine, nursing, business and law. PhD programs include Nutrition and Health Sciences, Epidemiology, and Biostatistics. Emory University, a major research university with the nations' fifth largest endowment, enrolls 11,350 students in undergraduate and graduate programs taught by 2500 faculty.

Candidates should be distinguished leaders in Global Health, known internationally for seminal contributions to public health research and practice. Candidates from any discipline or area of public health will be considered. The holder of the Foege Chair will be expected to contribute to excellence in the department by furthering its mission of scholarship, research, teaching and service. The successful candidate will have an outstanding record of achievement, bring a program of excellent research, and a vision for global health.

Applicants should send a letter indicating their interest accompanied by a curriculum vitae to: Claire Sterk, Ph.D., Search Committee Chair, Rollins School of Public Health, Emory University, 1518 Clifton Road, N.E., Atlanta, GA 30322, USA. Applications will be kept confidential and references will not be contacted without the permission of applicants. Screening of applications will continue until the position is filled. Starting date is negotiable.



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# PURDUE

## UNIVERSITY

### E P I D E M I O L O G I S T

**Position:**

Tenure-track, 10 month faculty position in Epidemiology in the Department of Health & Kinesiology. The Department of Health & Kinesiology has a strong commitment to research and teaching in the areas of physical activity and health promotion throughout the life course, and is actively engaged in inter-disciplinary associations with other Departments and Schools at Purdue University. Minority and women candidates are encouraged to apply. Appointment to commence in August, 2005. The departmental webpage is: [www.sla.purdue.edu/academic/hk/](http://www.sla.purdue.edu/academic/hk/)

**Rank:**

Assistant or Associate Professor depending on the qualifications of the applicant.

**Qualifications:**

Candidates must have a PhD/MPH or equivalent with a specialty in epidemiology. There must be evidence of: 1) scholarship and extramural funding, 2) competency in research design and familiarity with public health work settings, 3) experience directing graduate students' research, and 4) teaching expertise in epidemiology, biostatistics and quantitative analysis. Proven ability as a principal investigator and as a contributor to a collaborative, multidisciplinary department is expected.

**Responsibilities:**

Independent and collaborative research that is disseminated in national and international forums is required. Extramural funding for research and the support of graduate students is expected. Teaching courses in the area of epidemiology at both the graduate and under-

graduate level is likely. Commitment to the development and implementation of an MPH degree to complement the existing Bachelor's, Master's, and Doctoral programs in the department is expected.

**Compensation:**

Purdue University has a very competitive salary structure which is commensurate with the qualifications of the applicant. The University has a comprehensive and generous benefits package with attractive retirement and medical options.

**Application Procedures:**

Send a detailed letter of application with a description of current and planned scholarly activities, complete resume, and samples of published papers from refereed professional journals. Include the names and complete contact information for three individuals who would be willing to provide letters of reference.

**Gerald C. Hyner, Professor and Chair**  
**Epidemiology Search Committee**  
**The Department of Health & Kinesiology**  
**Purdue University**  
**800 W. Stadium Ave., Lambert Building**  
**West Lafayette, Indiana 47907-2046**  
(765) 494 3151  
(765) 496 1239 facsimile  
[hyner@purdue.edu](mailto:hyner@purdue.edu)

**Timetable:**

Applications will be accepted until the position is filled. The review process will commence on November 15, 2004.

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Purdue University (1869) is the Land Grant institution in the State of Indiana and a member of the Big Ten Conference. There are 38,653 undergraduate and graduate students enrolled at its 650 acre West Lafayette campus. A full time faculty of nearly 2,200 men and women contribute to the research, teaching, and engagement missions of the University. The Health & Kinesiology Department is one of 11 departments in the School of Liberal Arts. The HK Department is actively engaged in interdisciplinary research and teaching with numerous departments in several Schools, the Center on Aging and the Life Course, the Regenstrief Center for Healthcare Engineering, and the obesity research group, among others. The opportunities for collaborative research and interdisciplinary scholarship are substantial. The University is committed to increasing diversity throughout campus and in all academic programs. Purdue is located in historic Tippecanoe County which is north of Indianapolis and south of Chicago on the banks of the Wabash River.

*PURDUE UNIVERSITY IS AN "EQUAL OPPORTUNITY/EQUAL ACCESS/AFFIRMATIVE ACTION" EMPLOYER*

**Department of Health and Human Services  
National Institutes of Health  
National Institute on Deafness and  
Other Communication Disorders**



The National Institute on Deafness and Other Communication Disorders, a major research component of the National Institutes of Health (NIH) and the Department of Health and Human Services, is recruiting for a Translational Research Branch Chief for the scientific and administrative management of a translational research program in deafness and other communication disorders. The Branch is responsible for the planning, design, implementation and administration of the NIDCD research program in translational research, epidemiology, and clinical trials (Phase I, II, and III) of pharmacological, surgical and behavioral interventions for diseases and disorders of hearing, balance, smell, taste, voice, speech and language.

The candidate should possess an M.D. or Ph.D. degree in a field relevant to the position and have clinical research experience. The ideal candidate should have a background in the mission areas of NIDCD with leadership/managerial skills and experience in the design and conduct of clinical studies and trials and/or in the evaluation of behavioral or therapeutic interventions.

Salary is commensurate with qualifications and research experience and it includes a full Federal benefits package (which include retirement, health, life and long term care insurance, Thrift Savings Plan participation, etc.). Relocation expenses will be paid. Physicians may also be eligible to receive a Physicians Comparability Allowance up to \$30k per year, depending on qualifications/experience.

For qualifications required, evaluation criteria, and application instructions, view the vacancy announcements at: <http://reports.cit.nih.gov/jobsnih/advacsearch.asp>. Announcement Number: **NIDCD-04-001** for candidates with M.D.s and **NIDCD-04-002** for candidates with Ph.Ds. For additional information on application procedures, call Ms. Felix at (301) 594-2286. Applications must be postmarked no later than **January 18, 2005**.



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***Tenure-track Assistant Professor***  
**University of Florida**

**College of Medicine**  
**Institute for Child Health Policy and the**  
**Dept. of Epidemiology & Health Policy**  
**Research**

The Institute for Child Health Policy and the Department of Epidemiology & Health Policy Research in the University of Florida College of Medicine seeks a health services researcher for a full-time tenure-track Assistant or Associate Professor position of research and teaching in an expanding child health services research program. The position will require active collaboration with other social and behavioral scientists and clinicians in the Institute, Department, and University on the design and implementation of research programs related to children and youth. Current studies underway include examination of health care delivery system factors influencing child and adolescent outcomes of care, development of health care financing and reimbursement strategies, development of the medical home in safety net settings, community intervention trials, public policy evaluations, and epidemiological studies, with a particular focus on youth. The candidate will join an active research program in a multidisciplinary environment that includes psychologists, sociologists, biostatisticians, economists, and social epidemiologists.

We are seeking a health services researcher with very strong analytical and statistical skills and a focus on children's health. Responsibilities will be mostly research, with teaching limited to masters and PhD students.

**Specific requirements** for the position include: (1) an earned doctorate in health services research, public health, sociology, psychology, or other social sciences field; and (2) a minimum of two years of research experience. Primary criteria for appointment include demonstrated expertise in child health services research, ability to author peer-reviewed publications, and grant proposals, interest and ability in collaborative multi-disciplinary research. Applicants at the Associate level must demonstrate a solid program of research and track record in attracting extramural funding.

The position is available on or about July 1, 2005. Interested applicants should email a letter of interests, curriculum vita, two recent papers, and list of three references **by January 1, 2005**, to [lmj@ichp.ufl.edu](mailto:lmj@ichp.ufl.edu), or mail to Lise Youngblade, PhD, Institute for Child Health Policy, Department of Epidemiology & Health Policy Research, University of Florida College of Medicine, 1329 SW 16th Street, room 5287, PO Box 100177, Gainesville, FL 32608. EOE



## ***Associate Director of Behavioral Research***

The National Cancer Institute, a major research component of the National Institutes of Health (NIH) and Department of Health and Human Services (DHHS), seeks a senior scientist to serve as Associate Director of the Division of Cancer Control and Population Sciences (DCCPS). The individual will lead the Behavioral Research Program (BRP), which includes the Office of the Associate Director and the following five branches: Applied Cancer Screening Research, Basic Biobehavioral Research, Health Communication and Informatics Research, Health Promotion Research, and Tobacco Control Research. The successful applicant will play a central and highly visible leadership role in the NCI's efforts in the social and behavioral sciences and their application to cancer prevention and control.

The Associate Director provides scientific and administrative leadership for the entire program, supervises the staff of the Office of the Associate Director and the five branch chiefs, and represents the NCI to a wide variety of professional, academic, and advocacy organizations. In addition, the Associate Director develops and facilitates collaborations with other social and behavioral science research funders, including NIH Institutes and Centers, the National Science Foundation, the Centers for Disease Control and Prevention, and many non-governmental organizations. The Behavioral Research Program's grants, contracts, interagency agreements and operating budgets totaled over 140 million dollars in Fiscal Year 2003. This includes over 275 grants and 19 interagency agreements.

This challenging and highly visible role requires broad scientific expertise, a passion for public service, a commitment to collaboration, and an ability to develop effective strategies for overcoming barriers to scientific progress and its application. Candidates must have a Ph.D. or equivalent degree in the social or behavioral sciences, public health, medicine, or a related discipline and a strong record of peer-reviewed publications relevant to health behavior etiology, mechanisms, and/or intervention. Experience in managing complex research projects, scientific staff, training programs, interdisciplinary collaborations, or funding programs is highly valued. The BRP of the DCCPS provides a unique and nationally visible multidisciplinary environment that participates in NCI's many internship, post-doctoral training and visiting scientist programs. The DCCPS also is committed to addressing health disparities through transdisciplinary research and its effective dissemination. This is an excepted service position (Title 42) with a salary range of \$147,476 - \$175,700. Please submit a letter of interest, including the names of at least three references and a cv to Robert T. Croyle, PhD, Director, Division of Cancer Control and Population Sciences, National Cancer Institute, 6130 Executive Blvd., Room 6138, Rockville, MD 20852. Applications will be considered until the position is filled. For more information about DCCPS/NCI, see [www.cancercontrol.cancer.gov](http://www.cancercontrol.cancer.gov).

Selection for this position will be based solely on merit, with no discrimination for non-merit reasons such as race, color, gender, national origin, age, religion, sexual orientation, or physical or mental disability.



*THE DHHS/NIH/NCI ARE EQUAL OPPORTUNITY EMPLOYERS*





## ***Branch Chief for Health Communication and Informatics Research, Behavioral Research Program***

The National Cancer Institute, a major research component of the National Institutes of Health (NIH) and Department of Health and Human Services (DHHS), seeks a senior scientist to serve as Chief of the Health Communication and Informatics Research Branch within the Behavioral Research Program (BRP) of the Division of Cancer Control and Population Sciences (DCCPS).

The successful applicant will play a central leadership role in NCI's expanding efforts to accelerate progress in the science of health communication and informatics and the application of this knowledge to cancer prevention, control, and quality of care. The Branch Chief provides scientific oversight of an extramural grants program, develops new research initiatives, represents NCI to relevant professional, academic and advocacy organizations, supervises branch staff, and participates in BRP and DCCPS planning and priority-setting to move communication research to practice in areas such as tobacco control, energy balance, and cancer screening. Effective collaborations within the NCI, including the Office of Communications and the Center for Strategic Dissemination, and with other NIH Institutes and Centers and Federal agencies, such as the Centers for Disease Control and Prevention and the Agency for Healthcare Research and Quality, are essential for the continued success of the program.

This challenging and highly visible role requires broad scientific expertise, a passion for public service, a commitment to collaboration, and an ability to develop effective strategies for overcoming barriers to scientific progress and its application. Candidates must have a Ph.D. or equivalent degree in behavioral science, public health or a related discipline, a strong record of peer-reviewed publications, and substantial experience in health communication research. Scientific expertise in health behavior change, health communication technology, consumer health informatics, and the development and evaluation of communication interventions in medical or public health settings are especially desirable. The BRP of the DCCPS provides a unique and nationally visible multidisciplinary environment that participates in NCI's many internship, postdoctoral training and visiting scientist programs. The DCCPS also is committed to addressing health disparities through transdisciplinary research and its effective dissemination. This is an excepted service (Title 42) position with a salary range of \$125,304 – 147,475. Please submit a letter of interest, including the names of at least three references and a CV to Robert T. Croyle, Ph.D., Director, Division of Cancer Control and Population Sciences, National Cancer Institute, 6130 Executive Blvd., Room 6138, Rockville, MD 20852. Applications will be considered immediately and will be accepted until the position is filled. NCI/NIH is an equal opportunity employer. For more information about DCCPS/NCI, see [www.cancercontrol.cancer.gov](http://www.cancercontrol.cancer.gov).

Selection for this position will be based solely on merit, with no discrimination for non-merit reasons such as race, color, gender, national origin, age, religion, sexual orientation, or physical or mental disability.



*THE DHHS/NIH/NCI ARE EQUAL OPPORTUNITY EMPLOYERS*



## MATERNAL & CHILD HEALTH

### ASSOCIATE OR FULL PROFESSOR FACULTY POSITION

The Department of Prevention and Community Health in the George Washington University School of Public Health and Health Services seeks an energetic, resourceful and visionary scholar to lead and administer the expansion of its Maternal and Child Health Program. Current research interests include reproductive health, adolescent health, women's health, injury prevention, CSHCN, and community-based interventions and evaluation. The wide-range of organizations in the nation's capital provides an additional avenue for collaborations.

Qualified applicants must have a doctoral degree in maternal and child health, the social and behavioral sciences, public health, or a related health science; documentation of a research portfolio; and publications in the field of MCH. Consideration will also be given to candidates who provide documentation of previous and current success in procurement of external funding. Commitment to excellence in teaching and advising of MPH and DrPH students, as well as mentoring of junior faculty is expected. This is a tenure-earning position and may be at the associate or full professor level. An academic appointment at a senior rank will be based upon experience and qualifications.

Letters of application should include a statement of interest, curriculum vitae, and contact information including the names, mailing addresses, and telephone numbers of three references, and a reprint of a recent representative publication. Application packets should be mailed to:

James Cawley, MPH, PA-C

Chair, Maternal and Child Health Search Committee

Department of Prevention and Community Health

School of Public Health and Health Services

George Washington University

2175 K Street, N.W. Suite 700

Washington, D.C. 20037

Attention: Heather Jordan, Administrator

Review of applications will begin December 15, 2004 and will continue until the position is filled. The appointment is expected by July 1, 2005.

Women and minorities are encouraged to apply. The George Washington University is an Affirmative Action/Equal Opportunity employer.



### TRAINING OPPORTUNITY

## Public Health Prevention Service

The **Centers for Disease Control and Prevention** (CDC) invites applications for the Public Health Prevention Service (PHPS), a 3-year national training and service program for masters-level health professionals. The ninth class of 25 participants is scheduled to begin in September 2005.

The PHPS program focuses on public health program management and provides Prevention Specialists with experience in program planning, implementation, and evaluation through specialized hands-on training and mentorship at CDC and state and local health agencies. Formal instruction is also provided in program management, epidemiology, surveillance, emergency response, and project evaluation. Prevention Specialists participate in a variety of activities, including seminars, evaluation projects, web-based training, temporary duty assignments, and conferences designed to provide them with essential public health management skills.

The first year of the PHPS program, Prevention Specialists will have two different assignments at CDC. The second and third years will be spent in a single assignment, with a variety of responsibilities, in a state or local health agency. These assignments prepare Prevention Specialists for program management positions in state, local, and federal health agencies, as well as voluntary, community, and managed-care organizations, upon completion of the Program.

**Eligibility:** Professionals with a strong interest in a career in public health, a master's degree related to public health, and U.S. citizenship. At least one year of public health work experience, which may include an internship or a thesis project in a community setting, as part of a master's degree, is highly desirable. Starting salary is approximately \$41,000 with annual increases. Benefits include vacation, sick leave, health insurance, and some relocation expenses. Expenses associated with the interview are the responsibility of the applicant.

**Application deadline:** January 15, 2005

**For further information or an application:** Public Health Prevention Service, Division of Applied Public Health Training, Epidemiology Program Office, Centers for Disease Control and Prevention, 1600 Clifton Road, NE, MS E-92, Atlanta, GA 30333, e-mail: [phpsepo@cdc.gov](mailto:phpsepo@cdc.gov) Phone: 404.498.6162.

<http://www.cdc.gov/epo/dapht/phps.htm>

EOE

## FACULTY POSITIONS

### Community Health Interventions, Behavioral Health Interventions, and Health Services Research/Health Policy.

The Section on Social Sciences and Health Policy in the Department of Public Health Sciences at Wake Forest University School of Medicine invites applications for tenure-track faculty positions (rank open) in any of the following areas:

- 1) **Community Health Interventions:** We are seeking applicants with research expertise in community interventions (e.g., community organizing, social marketing, design and/or analysis of community trials). Candidates' interests may cut across disease areas, behaviors, and populations. Interest in health disparities is highly desirable.
- 2) **Behavioral Health Interventions.** Applicants with expertise in behaviorally-based interventions related to health behavior change and/or health promoting behaviors are sought to develop new research initiatives and work collaboratively on multidisciplinary projects in such areas as exercise, diet, cancer screening behaviors, adherence to treatment protocols, minority health, and reducing barriers to health care and health promoting behaviors. The successful applicant must possess a broad theoretical knowledge of behavior change and social science models, and have prior experience in behaviorally-based research interventions. Candidates' interests may cut across disease areas, behaviors, and populations.
- 3) **Health Services Research/Health Policy/Health Economics.** We are seeking applicants with research expertise in the study of the institutional, legal, ethical, political, professional, and/or financial systems that affect the delivery of medical care, with a focus on how these systems interact with each other, their effects on cost, quality, effectiveness and accessibility of care, and how these systems might be improved or reformed.

Applicants must have a Ph.D. or other terminal degree in a field relevant to the position, such as public health, health behavior, sociology, communication, psychology, public policy, institutional or macro-economics, political science, or law. Experience in collaborative research in a multidisciplinary setting and excellent written and oral communication skills are required. The successful applicant should have a strong publication record. Experience in teaching at the graduate level and a history of extramural funding is highly desirable.

The Department of Public Health Sciences has over 45 faculty and 200 staff in three sections: Social Sciences and Health Policy, Epidemiology, and Biostatistics. The Department, which obtained over \$28 million in extramural research funding in 2003, ranks second in NIH funding among medical school departments of public health and preventive medicine. The Section on Social Sciences and Health Policy is comprised of over 75 staff, 15 faculty and 18 joint/cross-appointment faculty with training in psychology, sociology, economics, behavioral epidemiology, gerontology, public health, medicine, nursing, and law. Areas of excellence include: quality of life, patient satisfaction, women's health, health services research, substance abuse, cardiovascular disease, cancer, community health, violence prevention, and health policy.

Opportunities for collaboration exist with other clinical and basic science departments as well as the Wake Forest University Comprehensive Cancer Center, the Women's Health Center of Excellence, the Brenner Center for Child and Adolescent Health, the Maya Angelou Research Center on Minority Health, the Center for Health Care Research and Quality, and the Center for Community Research. For information about the Department, visit: <http://www.phs.wfubmc.edu/home.cfm>. For information about the Section of Social Sciences and Health Policy, please visit:

<http://www.phs.wfubmc.edu/sshp.home.cfm>

Applicants should send a copy of their current CV, a letter indicating area(s) of expertise and interest in the position, and a list of three references to: Michelle Naughton, Ph.D., Search Committee Chair, Section on Social Sciences and Health Policy, Department of Public Health Sciences, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 27157-1063. Applications will be accepted until February 15, 2005.

*Wake Forest is committed to equal opportunity, affirmative action and the diversity of its faculty and staff. Women and minorities are strongly encouraged to apply.*





**Director**  
**Clinical Research Unit**  
**Denver, Colorado**

Kaiser Permanente of Colorado is accepting applications for the Physician Director of Research of the Clinical Research Unit (CRU.) Kaiser Permanente of Colorado is a non-profit group model HMO, consisting of the Medical Group with over 600 physicians and the Kaiser Foundation Health Plan of Colorado (Health Plan) with 417,000 members. Kaiser Permanente of Colorado has an outstanding record of innovation, and excellence in clinical care and has had a fully automated medical record in place for six years, which complements the comprehensive administrative databases that have been in place since 1990.

The mission of the CRU is to develop, conduct and translate high-quality research into practice. The CRU, with a budget of \$9 million in 2003, 12 investigators and a staff of 70, focuses on translational research, including health services, behavioral and clinical research. CRU conducts federal, foundation, and industry funded research, including multiple research collaborations with similar units in other HMOs and academic medical centers.

Applicants must be physicians who possess leadership experience, including a successful track record of developing staff, building relationships, and developing and promoting a strategic vision for health services, behavioral and clinical research. In addition, applicants should have several years of research experience, including success in obtaining grant funding from federal agencies such as the NIH, AHRQ, and CDC, and experience supervising large and multi-institutional grants. Additional formal training in research methodologies (e.g., MPH or comparable training) is desirable. Please contact: **Chantal Papez/Physician Recruitment, 303-344-7302, E-Mail: chantal.papez@kp.org FAX 303-344-7818 EOE, M/F, V/H**



# SAN DIEGO STATE UNIVERSITY

## Director: School of Public Health, SDSU

San Diego State University (SDSU) is seeking an innovative and energetic academic leader to serve as Director of the Graduate School of Public Health (GSPH). SDSU has a highly diverse student population of over 32,000 students, including approximately 6,000 graduate students. With 72 master's programs and 15 joint doctoral programs, SDSU is a Carnegie Foundation Doctoral/Research Intensive University. During the past year, SDSU received \$123 million in grants and contracts.

The GSPH offers one undergraduate degree with about 100 majors, 6 MPH and joint MPH degrees in epidemiology, health promotion, health services and occupational and environmental health, 2 MS degrees in environmental health and two Ph.D. degrees in Epidemiology and Health Behavior. The School has approximately 350 graduate students. In 2003, the School generated more than \$22 million in extramural research and contract funds. Additional information is available at [publichealth.sdsu.edu](http://publichealth.sdsu.edu).

The Director is the chief administrator of the GSPH, reports to the Dean, College of Health and Human Services, and serves as a member of the Executive Advisory Council. The Director oversees operation of all Divisions and works closely with Division Heads who serve on an Executive Committee chaired by the Director. The Director is responsible for administering the budget, guiding the educational, professional, and research missions of the School, supervising staff, securing resources, and representing and promoting the School within governmental and public health institutions and the local, national and international communities.

**Qualifications** The successful candidate will have an earned doctorate and excellent leadership and interpersonal skills, with capabilities for promoting collegiality, building consensus, and mentoring faculty, students and staff. The candidate shall be an effective communicator, able to promote the School's goals in the University and the professional and general communities. The candidate will have extensive peer reviewed published public health research, including NIH, CDC, or equivalent extramural support. The candidate should also have generated external financial support for instructional and other academic advancement activities. Thorough knowledge of the management and operation of academic and research programs is expected, including advancing diversity, working effectively in a multicultural campus and community, and having the capacity to expand the School's academic and research programs regionally, nationally, and internationally.

**Applications/Nominations** Nominations are welcome. Candidates may apply directly by sending a letter of application, curriculum vitae, and the names, addresses, phone/fax numbers, and email of at least five referees. References will only be contacted with permission of the candidate. Application review will begin November 2004 and continue until the position is filled. Send all communications to: **Chair, Director's Search Committee, Graduate School of Public Health, San Diego State University, 5500 Campanile Drive, San Diego, CA 92182-4162.**

*SDSU is a Title IX equal opportunity employer and does not discriminate against individuals on the basis of race, religion, national origin, sexual orientation, gender, marital status, age, disability, or veteran status, including veterans of the Vietnam era.*

## **Tenure Track Assistant Professor**

Department of Mental Health

The Department of Mental Health seeks individuals for a tenure-track Assistant Professor position. Our mission is to advance understanding of the occurrence, causes, and consequences of mental health and mental and behavioral disorders, in order to improve health in the general population, and to prevent and control these disorders and their associated impairments, disabilities, and handicaps. Department faculty conduct a wide range of research in a multi-disciplinary environment. The Department is the only academic unit of its type in the world, and continues a long tradition of leadership in the field of public mental health.

The most talented candidates in any area related to research in public mental health are sought. Those with interests and experience in genetic epidemiology, mental health services research, cognitive health and aging, or quantitative and biostatistical aspects of mental health research, are particularly encouraged to apply. Faculty are expected to teach and to develop their own externally funded research.

Applicants should have a doctoral level (M.D. or Ph.D.) education. The successful candidate should have a strong publication record or an exceptional potential to publish.

Application reviews will begin in the Fall of 2004. Interested candidates should send a letter describing research and teaching interests, names and addresses of references, and a Curriculum Vita to:

William W. Eaton, Ph.D.  
Professor and Chair  
Department of Mental Health  
624 N. Broadway, Suite 850  
Baltimore, MD 21205  
[weaton@jhsp.edu](mailto:weaton@jhsp.edu)

*The Johns Hopkins University actively encourages interest from women and minorities and is an affirmative action/equal opportunity employer.*

## **BROWN MEDICAL SCHOOL**

PUBLIC HEALTH PROGRAM/  
DEPARTMENT OF COMMUNITY  
HEALTH

### **Assistant Professor of Epidemiology (tenure-track)**

The Public Health Program in the Brown Medical School seeks an epidemiologist for a new tenure track Assistant Professor position in the emerging section of Epidemiology. The position is part of a five-year expansion plan for Public Health at Brown and is available to start July, 2005. Applicants must have a doctoral degree in Epidemiology and have an independent research program preferably in areas of international health, genetic/molecular, reproductive or infectious disease epidemiology. Experience teaching epidemiological methods at the graduate level is desirable. Review of applications will begin on October 1, 2004, and will continue until the position is filled or the search is closed.

Interested applicants should send a letter of application, at least 3 letters of reference and an updated curriculum vitae to: Chair of Assistant Professor of Epidemiology Search Committee, Attention Carol Mercier, Box G-H1, Brown University Medical School, Providence, RI 02912.

*Brown University is an  
Affirmative Action/ Equal  
Opportunity Employer and  
actively solicits applications from  
women and minorities.*

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**Tobacco Control Scientist  
Faculty Position**  
**Lombardi Comprehensive Cancer Center**

The Cancer Control Program of the Lombardi Comprehensive Cancer Center is seeking a senior researcher at the Associate Professor level or higher. The individual filling this position will be an experienced tobacco control scientist with interests and expertise in one or more areas of tobacco control (e.g., epidemiology and behavior, prevention and cessation, policy and legislation). Minimum requirements include a doctoral degree in a behavioral or social science discipline with an established track record of attracting extramural funding. The successful candidate will join a highly interdisciplinary department of oncology and a cancer control program with active research in cancer screening, genetic counseling and testing, outcomes, lifespan development/aging, and community outreach. The cancer center's Cancer Control & Population Sciences division is also home to a productive cancer genetics and epidemiology program, with strong tobacco research and biomarker efforts. Georgetown University Medical Center is comprised of the Lombardi Comprehensive Cancer Center, School of Medicine, School of Nursing and Health Studies, and a biomedical research enterprise. The medical center is conveniently located in Washington, DC. Salary and recruitment package will be commensurate with qualifications and experience. Interested individuals should send a statement of interest, CV, and the names of three references to: *Tobacco Control Scientist Search Committee, Cancer Control Program, Lombardi Comprehensive Cancer Center, 2233 Wisconsin Avenue, NW, Suite 317, Washington DC 20007-4104*. Inquiries may be directed to **Kenneth Tercyak, PhD** by email at [tercyakk@georgetown.edu](mailto:tercyakk@georgetown.edu). For more information, please visit <http://lombardi.georgetown.edu>.

*Georgetown University Medical Center is an equal opportunity employer*

Health Services Management at the **University of Missouri** is recruiting for two faculty members. Both positions are 11-month tenure-track and candidates should have a Ph.D. or equivalent degree.

**An associate/full professor** with an established research record in one of the following areas: organization design and strategy, organization behavior, or operations management/process improvement. Experience needed in collaborative research and a commitment to providing leadership within a multidisciplinary research program.

**An assistant/associate professor** with some demonstrated research in the organization and management of health systems with research interests in structure, strategy, and performance of integrated health systems. Individuals with specific interest in clinical outcomes and quality with experience from academic and health services research centers are strongly encouraged to apply.

The Department of Health Management and Informatics offers graduate programs in Health Services Management (MHA) and Health and Bio Informatics (MS), both on-campus and as Executive Programs using distance-learning formats.

Interested candidates should send a cover letter describing qualifications/interests and curriculum vitae to:

**Lanis L. Hicks, Ph.D.**  
Department of Health Management and Informatics  
324 Clark Hall  
University of Missouri-Columbia  
Columbia, MO 65211  
Email: [HicksL@health.missouri.edu](mailto:HicksL@health.missouri.edu)  
Phone: (573) 882-6178  
Fax [573] 882-6158  
<http://www.hmi.missouri.edu>

*The University of Missouri-Columbia is an Equal Opportunity/Affirmative Action employer. Minorities and women are strongly encouraged to apply. To request ADA accommodations, please contact our ADA Coordinator at (573) 884-7278 (V/TTY).*

**Yale University  
School of Medicine**

**Department of Epidemiology  
and Public Health**  
**Division of Health Policy and Administration**

**Assistant/Associate Professor**

Applications are being accepted for a new faculty position at the Assistant or early Associate Professor level in the area of health policy and health services research. A doctoral degree – which may include a PhD, ScD, or a professional doctorate (e.g., MD) with advanced training in a policy-related discipline – is required.

We welcome applications from any disciplinary background (including but not limited to economics, political science, sociology, public health, and public policy). We are especially interested in individuals who apply formal, quantitative methods to policy analysis or health services research. While all areas of substantive interest will be considered, we hope to identify candidates in one of three fields of study:

- (1) **Health Services Research:** We encourage applicants whose research encompasses the individual, organizational, and societal determinants of health and health care – particularly with regard to minority health services and vulnerable populations.
- (2) **Health Policy:** Preference will be given to candidates whose research addresses both the political determinants of public policy and assessments of program performance. We are interested in individuals who are able to teach policy development and implementation at the masters and doctoral level.
- (3) **Organization, Financing, and Delivery of Health Care.** We seek individuals who are committed to research on the impact of competition, managed care, regulation, and other forms of organization on delivery, incentives, and disparities of care. Candidates should be willing to take an active role in the activities and administration of our masters program in Health Management.

The successful candidate will have a record of publications in peer-reviewed journals, a demonstrated ability to compete successfully for extramural funding, a willingness to work in a collaborative, interdisciplinary environment, and appropriate teaching experience.

Salary and rank will be commensurate with experience. Applications, nominations, and inquiries are all invited. For full consideration, applicants should submit a letter of interest, a complete curriculum vitae, three letters of reference, and a writing sample by January 1, 2005 to: Mark Schlesinger, PhD, Division of Health Policy and Administration, Department of Epidemiology and Public Health, Yale University School of Medicine, 60 College Street, Box 208034, New Haven, CT 06520-8034.

*Yale University is an affirmative action/equal opportunity employer. Men and women of diverse racial/ethnic backgrounds and cultures are encouraged to apply.*



## Faculty Positions in Cancer Control

The Comprehensive Cancer Center of Wake Forest University (CC-CWFU) and the Department of Public Health Sciences invite applications for two tenure track faculty positions (rank open) in Cancer Control.

The successful candidate will participate in an active program in cancer control as part of an NCI-funded Comprehensive Cancer Center. The CCCWFU has a large and dynamic group of faculty in numerous departments collaborating in the areas of cancer screening, quality of life, survivorship, tobacco control, environmental exposures to cancer, cancer prevention, genetic epidemiology, and complementary and alternative therapies. The CCCWFU has a Department of Defense funded Behavioral Center of Excellence, serves as a CCOP Research Base, and participates in CALGB trials. The Institution has also recently established the Genomics Center, with a major focus in prostate cancer. The Department of Public Health Sciences has over 45 faculty and 200 staff in three sections: Social Sciences and Health Policy, Epidemiology, and Biostatistics and is ranked second nationally in NIH research funding among medical school departments of public health and preventive medicine. Opportunities for collaboration exist with the Maya Angelou Research Center on Minority Health, the Women's Health Center of Excellence, and the Department of Health and Exercise Science.

*Wake Forest University Health Sciences is an equal opportunity/affirmative action employer. Applications from women and minority candidates are strongly encouraged.*

The faculty position is offered in the Department of Public Health Sciences, and/or another department, depending upon the applicant's qualifications and interests. Applicants should have a Ph.D. or M.D. with additional training/experience in epidemiology, health services research, clinical trials, behavioral medicine, or molecular/genetic epidemiology. Experience in collaborative research in a multidisciplinary setting and excellent written and oral communication skills are required. The successful applicant should have a strong publication record. Experience in teaching at the graduate level and a history of extramural funding is highly desirable. Specific area of research is open.

Wake Forest University Health Sciences is located in Winston-Salem, North Carolina, one hour east of the Great Smoky Mountains and four hours west of the Atlantic coast beaches.

Applicants should send a letter, curriculum vitae, names of 3 references, and a summary of research interests to Dr. Nancy Avis, Department of Public Health Sciences, Piedmont Plaza II, Wake Forest University Health Sciences, Winston-Salem, NC 27157. Applications will be accepted until the positions are filled. For more information about the Department of Public Health Sciences, visit: <http://www.phs.wfubmc.edu>.

The **Faculty of Health Sciences** of the **American University of Beirut** currently seeks for its **Department of Health Management and Policy** a candidate with a Ph.D. degree in Health Management and Policy or its equivalent in Public Policy. The candidate should have teaching and demonstrated experience and scholarly productivity in conducting policy-relevant research and policy analysis with emphasis on health systems and financing, disparities in access to healthcare and operations and improvement of public and private organizations.

This position is available at the ranks of assistant, associate or full professor depending on qualifications. Visiting positions at all levels may be considered.

Successful candidates are expected to actively participate in the teaching program of the Department of Health Management and Policy as well as contribute to research programs in the Department and the Faculty. For further information about AUB: URL: <http://www.aub.edu.lb> and FHS: <http://fhs.aub.edu.lb>

Interested candidates should submit a complete resume, statement of teaching and research interests and three letters of reference to: **Huda Zurayk, Dean, Faculty of Health Sciences, American University of Beirut, 3 Dag Hammarskjold Plaza, 8th floor, New York, NY 10017-2303. Fax in Beirut +961-1-744470. E-mail: [hzurayk@aub.edu.lb](mailto:hzurayk@aub.edu.lb).**

**Deadline** for receipt of applications is **January 31, 2005** for a starting date of **September 15, 2005**.

*The American University of Beirut is an Affirmative Action/  
Equal Opportunity Employer.*

## ASSISTANT OR ASSOCIATE PROFESSOR LEVEL

The Division of Health Services Research, Department of Family and Community Medicine, **Texas Tech University Health Sciences Center (TTUHSC)**, invites applications for a tenure-track faculty position at either the Assistant or Associate Professor level. The Division is a multidisciplinary team of highly productive health researchers who are seeking a PhD or MD (or equivalent) colleague with expertise in health promotion/behavior and experience in a fundable line of intervention research among multi-ethnic and underserved populations. This position requires a strong commitment to academic research and to collaborative work with physicians. Successful candidates will have published in peer-reviewed journals and will be qualified to teach graduate courses in health promotion/behavior. Salary is commensurate with experience. TTUHSC is committed to increasing representation of women and members of minority groups on its faculty and particularly encourages applications from such candidates.

**Contact:** James Rohrer, Ph.D., Search Committee Chair, attn. Jane Allison, 3601 4th Street STOP 8143, Lubbock, TX 79430-8143 or e-mail [Jane.Allison@ttuhsc.edu](mailto:Jane.Allison@ttuhsc.edu).

*TTUHSC is an EEO/AA employer and in compliance with ADA.*



# Post-doctoral Training Opportunities at The University of Illinois at Chicago School of Public Health

The Illinois Public Health Research Fellowship Program at the University of Illinois at Chicago School of Public Health (UIC SPH) announces post-doctoral interdisciplinary health protection research training opportunities in preparedness and primary prevention across the life stages, with emphasis on research to reduce health disparities and promote environmental justice.

Trainees will participate in transdisciplinary teams lead by senior faculty based in the traditional public health disciplines (epidemiology, biostatistics, health policy and administration, community health sciences, and environmental and occupational health sciences), but focused on the themes of community-based participatory research, acute/emergency response, translational research, and intervention effectiveness.

Twelve (12) post-doctoral positions are available through this CDC-funded project. Stipends are consistent with standard federal training programs and each position is funded for three years.

#### **Applicant qualifications:**

- A doctorate in any of the public health or related sciences by Summer 2005. Qualifying fields include physiology, engineering, nursing, sociology, psychology, urban planning, epidemiology, chemistry, economics, anthropology, toxicology, public policy, etc.
- Commitment to pursuing research careers in public health and a strong intellectual curiosity about and willingness to work with diverse disciplines.

*Under-represented minority scientists are strongly encouraged to apply.*

*Applicants must be U.S. citizens, non-citizen nationals or lawfully admitted permanent residents at the time of the award.*

#### **If interested, please submit:**

- A curriculum vitae
- Three letters of reference
- A 2 to 3 page statement of interest that includes primary research interests, collateral research interests, and specific pilot research application proposals as well as any proposed publications to be developed from earlier work. Applications must be submitted to Ms. Edna Rivera, University of Illinois at Chicago, School of Public Health (M/C 922), 2121 W. Taylor St, Chicago, IL 60612.

**Application deadline:** January 31, 2005. Earlier applications are encouraged.

Selected applicants will be invited to a meeting in Chicago to identify potential primary and cross-disciplinary mentors. Travel support will be provided. Meetings will take place between December 2004 and March 2005.

#### **For further information contact any of the program directors:**

Rosemary Sokas, MD, MOH, Environmental/Occupational Health Sciences, at [sokas@uic.edu](mailto:sokas@uic.edu)

Jack Zwanziger, PhD, Health Policy and Administration, at [jzwanzig@uic.edu](mailto:jzwanzig@uic.edu)

Faith Davis, PhD, Epidemiology and Biostatistics, at [fayed@uic.edu](mailto:fayed@uic.edu)

Arden Handler, DrPH, Community Health Sciences, at [handler@uic.edu](mailto:handler@uic.edu) or visit the UIC SPH website at <http://www.uic.edu/sph/>

## Assistant Professor, Department of Applied Behavioral Science, University of Kansas

Full-time, academic year, tenure-track position in health promotion. We are especially looking for applicants who can teach, conduct research, and supervise students in behavioral research and application (e.g., applied behavior analysis and interventions). The position begins on August 18, 2005, contingent on final budgetary approval.

**Required qualifications:** Ph.D. by August 18, 2005 in a discipline related to health promotion (e.g., psychology, public health), effective written and oral communication skills, and ability to work effectively with colleagues on cooperative projects. Additional qualifications include the ability to teach high-quality courses and/or practica, develop an exemplary program of research, and secure external grant funding. For a complete position announcement, see [www.ku.edu/~absc](http://www.ku.edu/~absc) or contact Ms. Amy Robbins ([amyr@ku.edu](mailto:amyr@ku.edu)). First consideration will be given to applications received by **February 1, 2005**. A complete application will include a letter of application, a curriculum vita, reprints of representative publications, and three letters of reference. Please send these to Ms. Amy Robbins, Department of Applied Behavioral Science, 4001 Dole Human Development Center, University of Kansas, 1000 Sunnyside Avenue, Lawrence, KS 66045-7555. Phone: 785-864-0503; fax: 785-864-5202; e-mail: [amyr@ku.edu](mailto:amyr@ku.edu). Search committee co-chairpersons: R. Mark Mathews ([rm-mathews@ku.edu](mailto:rm-mathews@ku.edu)) and Edward K. Morris ([ekm@ku.edu](mailto:ekm@ku.edu)). EO/AA Employer.

## Urology Outcomes Research Scientist

The Department of Urology at Beaumont Hospital in Royal Oak, Michigan, is seeking a candidate with expertise in Health Services Research with emphasis in Outcomes Analysis.

Requirements for application include possessing a MPH and/or Ph.D. title with proficiency in one or more of the following areas:

- Clinical Trials
- Survey Research
- Epidemiology
- Health Economics

Database management and previous or current urology experience is preferred.

Please send curriculum vitae to:

Ananias C. Diokno, M.D.

Beaumont Hospital

Department of Urology

3535 W. Thirteen Mile Road, Suite 438

Royal Oak, MI 48073

Email: [adiokno@beaumont-hospitals.com](mailto:adiokno@beaumont-hospitals.com)

**Beaumont**  
William Beaumont Hospital

## **Yale University School of Medicine**

Department of Epidemiology and Public Health

Division of Health Policy and Administration

### **Associate Professor/Professor with Tenure**

We seek a scholar of national prominence for a senior faculty position with tenure in the area of health policy or health services research. A doctoral degree is required.

The successful candidate should have a substantial research program including prominent publications, significant external funding, and expertise in quantitative methods as applied in health-related fields. The position duties include research, teaching, mentoring doctoral students and junior faculty, and the possibility of Division-level administrative leadership.

Salary and rank will be commensurate with experience. The closing date for applications is January 1, 2005 or until a successful candidate has been identified. For full consideration, applicants should submit a letter outlining their primary research and teaching interests, statement of professional goals, a complete curriculum vitae, and reprints of published work to:

**Jody Sindelar, Ph.D.**

**Chair, Senior Search Committee Chair**

**Health Policy and Administration**

**Department of Epidemiology and Public Health**

**Yale University School of Medicine**

**60 College Street**

**PO Box 208034**

**New Haven, CT 06520-8034**

*Yale University is an affirmative action/equal opportunity employer. Men and women of diverse racial/ethnic backgrounds and cultures are encouraged to apply.*

## **Yale University School of Medicine**

Department of Epidemiology and Public Health

Assistant/Associate Professor

### **Program on the Social and Behavioral Sciences**

Yale University School of Public Health, Division of Chronic Disease Epidemiology/ Social and Behavioral Sciences Program is recruiting for a position at the Assistant or early Associate Professor level. The position requires a doctoral level degree, evidence of the beginning of a scholarly career through publications and successful grant writing, and some teaching experience of master's and doctoral level courses.

We are developing an exciting new academic program, and looking to expand to include a focus on understanding and reducing racial/ethnic health disparities. The division has related research programs in cancer, cardiovascular disease, aging, perinatal epidemiology, genetic epidemiology, HIV/AIDS, and mental health. The selected candidate will be expected to collaborate with other faculty, teach, supervise graduate students, and establish an externally funded research program.

The closing date for applications is January 1, 2005 or until a successful candidate has been identified. Interested applicants should submit a curriculum vitae, statement of professional goals and three letters of recommendation to:

**Jeannette R. Ickovics, Ph.D.**

**Director, Social and Behavioral Sciences**

**Yale School of Public Health**

**60 College Street, Fourth Floor**

**PO Box 208034**

**New Haven, CT 06520-8034**

*Yale University is an affirmative action/equal opportunity employer. Men and women of diverse racial/ethnic backgrounds and cultures are encouraged to apply.*

## **YALE SCHOOL OF PUBLIC HEALTH**

### **Director, Office of Public Health Practice**

Applications are being accepted for the new non-ladder position of Director of Public Health Practice. Faculty rank and salary will be commensurate with training and experience. A doctoral degree and at least five years experience in public health practice are required.

The candidate should be a public health leader/practitioner who can provide leadership, guidance and linkages to practice-based research and education activities in the School and interface with public health programs in our local community.

The candidate will be expected to establish an independent, extramurally-funded practice-based research program which contributes to the overall mission of the school. Teaching responsibilities will include enhancing existing courses and developing new courses in public health practice and preparedness.

The Director will represent the school in professional activities related to public health practice, preparedness and leadership and will participate in relevant state, local, national and international initiatives.

The closing date for applications is January 1, 2005 or until a successful candidate has been identified. Interested applicants should send a full curriculum vitae and a letter describing qualifications and research interests to:

**Brian P. Leaderer, Ph.D.**

**Chair, Public Health Practice Search Committee**

**Department of Epidemiology and Public Health**

**Yale University School of Medicine**

**60 College Street**

**PO Box 208034**

**New Haven, CT 06520-8034**

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## **YALE UNIVERSITY SCHOOL OF MEDICINE**

Department of Epidemiology and Public Health

Global Health Division

### **Assistant/Associate Professor**

The Department of Epidemiology and Public Health, an accredited School of Public Health at Yale University, is accepting applications for the position of Assistant/Associate Professor. Applicants must have a doctoral degree, Ph.D., M.D., or the equivalent. Preference will be given to applicants who have an interest in global health research with a focus on prevention of major risk factors for chronic diseases such as tobacco use, unhealthy diets and physical inactivity, a record of publications in the peer reviewed literature and grant supported research. The successful candidate will be expected to teach, conduct a program of independently funded research and collaborate with other investigators.

The closing date for applications is January 1, 2005. Applicants should send letters outlining primary research and teaching interests, and statement of professional goals, curriculum vitae and reprints of published work to:

**Derek Yach, MB.ChB., M.P.H.**

**Chair, Global Health Search Committee**

**Department of Epidemiology and Public Health**

**Yale University School of Medicine**

**PO Box 208034**

**New Haven, CT 06520-8034**

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## ASSISTANT PROFESSOR OF HEALTH PROMOTION

**Coastal Carolina University**, Program in Health Promotion, Department of Health, Physical Education, and Recreation, in the College of Education, announces a full-time, tenure-track Assistant Professor position beginning in August 2005.

**QUALIFICATIONS:** Earned Doctorate in Public Health or Health Promotion, evidence of scholarly productivity and excellence in teaching. CHES certification and technology skills are preferred.

**RESPONSIBILITIES:** Undergraduate instruction in health promotion will be selected from, but not limited to, family life and sexuality, epidemiology, personal and community health, and philosophy and principles of health promotion/education. Position requires advisement of undergraduate students, mentoring student research, establishing a record of scholarship and professional service/outreach, collaboration with related academic programs, and assisting in undergraduate program approval.

Coastal Carolina University is a growing, state-supported institution with increasing emphasis placed on faculty-student research and public service. Coastal Carolina University is located approximately nine miles from Myrtle Beach, South Carolina enrolling nearly 7,000 students from 48 states and 50 countries. The University offers baccalaureate degree programs in 36 major fields of study and master's degrees in education and marine science.

Interested applicants should send a letter of application, current vita, official transcripts of all undergraduate and graduate coursework, evidence of CHES certification, and three (3) letters of recommendation to: Dr. Sharon Thompson, Department of Health, Physical Education, and Recreation, Spadoni College of Education, Coastal Carolina University, PO Box 261954, Conway, SC 29528-6054. To ensure full consideration, application materials must be received **by January 5, 2005**.

**Coastal Carolina University is an EO/AA employer.**

## Tenure-Track Epidemiologist Position Vacancy

Department of Microbiology, **Montana State University** seeks to fill tenure track position in epidemiology at rank and salary commensurate with candidate's qualifications. Position is funded for first five years through the IDeA Network of Biomedical Research Excellence (INBRE) program. Incumbent will work with cross-disciplinary teams to study human and animal disease vectors.

### Responsibilities include:

- Conducting externally funded research in environmental and/or infectious disease epidemiology involving undergraduate and graduate students.
- Developing and teaching senior/grad level course in epidemiology and/or biostatistics.
- Participating in MSU's biomedical networking program.

### Required:

- Ph.D., M.D., DVM or equivalent.
- At least 2 years of post-doctoral experience in epidemiology, public health, biostatistics or related scientific field.
- Record of publishing in peer-reviewed journals.
- Potential to develop and maintain independent research program in environmental and/or infectious disease epidemiology.
- Teaching experience in a classroom environment.
- Experience in mentoring undergraduate and graduate students engaged in independent research.

### Preferred:

- Expertise and/or interest in environmental and infectious zoonotic disease epidemiology, social epidemiology or ecology of agents of infectious disease important to the State and local communities
- Experience in preparing proposals for research.
- Prior research involving rural and/or American Indian communities

Screening begins November 3, 2004 and continues until suitable candidate is hired. Send curriculum vitae, letter addressing the required and preferred qualifications and outlining your research interests, and the names and addresses of five references to: Epidemiologist Search Committee, Department of Microbiology, P.O. Box 173520, Montana State University, Bozeman, MT 59717-3520. Electronic applications with signed hard copy letter of application accepted. Direct inquiries to Dr. Gill Geesey (phone: 406-994-3820; email: [gill\\_g@erc.montana.edu](mailto:gill_g@erc.montana.edu)).

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