LETTERS

VETERANS' HEALTH CARE

It is not enough to say thank you to the editors of the Journal for the October 2003 cover picture and article on veterans' health care, which were well received in the veterans' community.[1] Far too often veterans are yesterday's news, even when our country is engaged in a new conflict.

New York City veterans are currently involved in a struggle to keep the Veterans Affairs (VA) Hospital at First Ave and 23rd St open. We have the support of several public officials, such as Congresswoman Carolyn Maloney, New York City Council Speaker Gifford Miller, Public Advocate Betsy Gotbaum, and Council Members Margarita Lopez and Christine Quinn, but it is still an uphill fight. The VA CARES (Capital Asset Realignment for Enhanced Services) Commission has visited many VA medical facilities around the country and has slated several for major changes. The commission proposes moving all inpatient care from 23rd St to other VA hospitals.

The CARES Commission also has plans to close down the Montrose and Canandaigua VA facilities in upstate New York. These closings will be devastating to the veterans community as well as to the health care professionals who staff these facilities. The impact on teaching hospitals and funding in these areas is probably viewed as nothing more than "collateral damage," but such a change will affect many lives and probably none for the better. I am sure that New York veterans are not alone in being affected by the draconian measures recommended by the CARES Commission. Should readers want more information on this subject, I can connect them with other veterans who can offer much more knowledge than my limited grasp of this serious situation allows.

The Journal is a welcome, positive, caring voice in very uncertain times for veterans' health care. Please keep this issue alive in the public health community, as we need all the help we can get. ■

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LETTERS

ELIMINATING HEALTH DISPARITIES: FOCAL POINTS FOR ADVOCACY AND INTERVENTION

We applaud the Journal's October 2003 focus on eliminating health disparities and the University of Pittsburgh's National Minority Health Research Summit. Efforts to reduce inequalities need to be derived from multiple sources. Two recent publications about the challenges of achieving equality for American Indians, Alaska Natives, and Latino peoples show the failings of our past approaches.[1,2]

The National Institutes of Health's National Center on Minority Health and Health Disparities is paving the way for a more aggressive research agenda in this area.[3] Public health professionals need to educate policymakers about the need for support for such research, as well as for programs that translate research into practice, such as the Centers for Disease Control and Prevention's Racial and Ethnic Approaches to Community Health (REACH) 2010.[4] REACH is funded for fiscal year 2004 at only $37 million—the same amount it received in fiscal year 2003.[5]

It is clear that government intentions alone will not elevate the health status of America's marginalized and minority peoples. Voluntary professional associations such as the Sociey for Public Health Education and the American Public Health Association (APHA) must call on their members to connect with all levels of society. A central organization can plan training, promote cultural sensitivity, share information and research, and develop positions that can be used by other groups in their advocacy efforts. APHA is demonstrating its leadership in this area by designating "Eliminating Health Disparities" the theme of National Public Health Week in 2004.[6]

In 1966, Dorothy Nyswander defined an open society as "one where justice is the same for every [person]; where dissent is taken seriously as an index of something wrong or something needed; where diversity is expected; . . . where the best of health care is available to all; where poverty is a community disgrace not an individual's weakness; [and] where desires for power over [people] become satisfaction with the use of power for people."[7(p37)] In keeping with this vision, in 2000 the Society for Public Health Education (SOPHE) commissioned an Open Society Commission, which resulted in 6 resolutions aimed at eliminating health disparities and calling for widespread socioecological change. Yet stronger organizational commitment from groups such as SOPHE and APHA is crucial to helping diverse practitioners eliminate such continuing practices among health care professionals as racism, stereotyping, bias, discrimination, and cultural and professional in-

competence. Let each public health professional rise to the challenges of achieving an open society and reaching our national goal of eliminating health disparities. ■

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ERRATA

In: Roubideaux Y, Buchwald D, Beals J, Middlebrook D, Manson S, Muneta B, Rith-Najarian S, Shields R, and Acton K. Measuring the Quality of Diabetes Care for Older American Indians and Alaska Natives. *Am J Public Health.* 2004;94:60–65.

An article was inadvertently published without the following authorial disclaimer and acknowledgments:

Note [to About the Authors]. *Opinions expressed by this article are those of the authors and do not necessarily reflect the views of the Indian Health Service.*

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The authors wish to thank the many Indian Health Service (IHS), tribal, and urban Indian program providers who contributed to the IHS Diabetes Care and Outcomes Audit in 1997.

In: Ellickson PL, Orlando M, Tucker JS, and Klein DJ. From adolescence to young adulthood: racial/ethnic disparities in smoking. *Am J Public Health.* 2004;94:293–299.

Some racial/ethnic smoking rates were expressed incorrectly. On page 295 under the subhead Regular Smoking, the categories "Asians" and "African Americans" were reversed for the rates given. The text should have read:

By the age of 23 years, regular smoking rates among Hispanics and Whites (29% and 32%, respectively) were approximately twice those among Asians (16%) and 1.5 to 1.7 times those among African Americans (19%).

EDITOR'S CHOICE

The Solution Is Injury Prevention

I awoke this morning to the news that another American life was tragically lost because of a preventable injury, and I wondered why our nation has been so complacent about this problem. Then I remembered the retort I received from a policymaker the last time I sought funding for an injury program: "Injury is not a public health problem!"

In 2000, injury was responsible for 10% of health care expenditures—more than $117 billion. Injury is the leading cause of death for Americans younger than 35 years and is a leading cause of disability. In this issue of the Journal, Lynda Doll and Sue Binder of the National Center for Injury Prevention and Control lay out the case for injury as one our nation's most preventable problems. They point to the incredible toll extracted by injury in human suffering as well as the staggering fiscal costs to society.

Why, then, is it so difficult to convince policymakers that injury is a public health problem worth addressing? Maybe because they still believe in the accident paradigm. This line of thinking argues that injuries are an act of fate, and while it makes sense to be more careful, injuries will occur despite our best efforts.

The evidence against the accident paradigm is exhaustive. Automobile-related injury has been dramatically reduced by a multifaceted effort to make cars safer. By redesigning automobile brakes, steering columns, sidewall protections, seat belts, air bags, and a host of other safety features, we have made the automobile a much safer machine. The addition of programs to reduce the incidence of impaired driving and promote driver training have addressed the human aspects of automobile safety.

Pedestrian trauma from motor vehicles is on the rise and, once again, safety experts are intervening by developing novel ways to reduce the hazards of walking. On April 7, 2004, the World Health Organization is emphasizing the global scope of the problem by declaring road safety as the theme for World Health Day.

In my days as an emergency physician, I was struck by the fact that most of the injuries I treated could have been prevented if only simple precautions had been taken. My colleagues and I would give individualized advice to patients on how to prevent future injuries while we provided medical care for their current injuries. Later, when I became active in public health, I recognized the need for population-based interventions as tools to reduce injuries, such as the interventions advanced in this issue of the Journal regarding motorcycle helmet laws, speed bumps, and programs to prevent dating violence. These interventions work. They reduce injuries, save lives, and prevent disabilities. They also save money—lots of money. These are marvelous accomplishments that are largely overlooked by most policymakers.

At a time when economists predict double-digit increases in health care expenditures, I have a solution: injury prevention. At a time when we are looking for ways to address disparities in health care, I have a solution: injury prevention. At a time when we are looking to protect our children from harm, I have a solution: injury prevention. The next time someone challenges us on the propriety of addressing injury as a public health problem, point out that if something kills people or hurts people, it's our responsibility as public health leaders to find a solution. ■

Georges C. Benjamin, MD, FACP
Executive Director, APHA

Injury Prevention Research at the Centers for Disease Control and Prevention

Recognizing the critical public health burden that unintentional and violent injuries place on the United States, Congress mandated in 1992 that the Centers for Disease Control and Prevention (CDC) create the National Center for Injury Prevention and Control (NCIPC). NCIPC was established to coordinate research and programmatic responses to the problem of nonoccupational injuries.

MORBIDITY, MORTALITY, AND THE COST OF INJURIES

Violent and unintentional injuries place a severe physical, emotional, and financial burden on our communities. Injuries do not discriminate; they affect all races and ages. In fact, injuries are the leading cause of death in the first 4 decades of life.[1] In 2001, the leading causes of deaths due to injury in the United States were motor vehicle crashes, suicides, and falls.

In addition to being a major cause of death, injuries cause suffering and disability. Virtually everyone knows of someone whose life has been changed because of a motor vehicle crash or other injury-causing event. Each year, Americans make 30 to 40 million visits to emergency departments for treatment of injuries.[2] Except for teenagers and young adults, falls are the leading cause of emergency department visits; for persons aged 15 to 23 years, motor vehicle crashes and striking or being struck by objects predominate.[1]

The economic costs of injuries impose a significant burden on society as well. In 2000, the United States spent $117 billion treating injuries, accounting for 10% of all medical expenditures that year.[3] The percentage of total medical expenditures accounted for by injuries in 2000 was comparable to the percentages attributable to other leading public health issues, such as obesity (9.1%) and smoking (6.5%–14.4%).

The mission of NCIPC is to prevent or reduce injuries. To accomplish its goals, NCIPC works with numerous partners to support injury surveillance, research, and prevention programs and to disseminate information that can inform prevention programs and policies. There is strong evidence of the effectiveness of many preventive interventions, including use of seat belts[4] and bicycle helmets,[5] laws establishing 0.08 blood alcohol content as the definition for drunk driving,[6] and residential smoke alarm and fire safety education programs.[7] Effective violence prevention strategies include home visitation of new parents to prevent mistreatment of children[8] and tenant-based rental assistance programs to prevent youths from witnessing or becoming victims of crime.[9]

Widespread implementation of such interventions could save thousands of lives annually. However, despite the progress that has been made, there is still much to be learned about preventing unintentional and violent injuries and about encouraging the dissemination and adoption of strategies that have proven effective.

NCIPC RESEARCH: GOALS AND STRATEGIES

Through a participatory process, NCIPC and its partners developed an Injury Research Agenda, which was published in June 2002.[10] The agenda categorizes research needs along a continuum, beginning with risk factor identification, proceeding through intervention evaluation, and ending with dissemination research.

While the agenda recognizes the need for additional descriptive research, it emphasizes the right side of the continuum—intervention and dissemination research.

The Injury Research Agenda includes 7 broad categories: injuries occurring at home and in the community; injuries occurring during sports, recreation, or exercise; transportation injuries; intimate partner violence, sexual violence, and child maltreatment; suicide; youth violence; and acute care, disability, and rehabilitation. The first 6 emphasize prevention, while the seventh focuses on improving outcomes when prevention efforts fail (e.g., enhancing systems for emergency treatment).

The agenda includes cross-cutting themes such as alcohol use, parenting and supervision styles, economic costs, and dissemination of scientific findings. It also identifies the importance of building injury-related research infrastructure, which, for example, will provide ongoing support for young researchers. The NCIPC New Investigator

and Dissertation grant awards, as well as funds for training and pilot studies, have been established to help fill this need.

Of the research issues raised in the agenda-setting process, 48 topics were deemed highest priority, each of which will require 10 to 20 studies to address adequately. NCIPC is using its available funds to address these priority topics; researchers wishing to apply for NCIPC funds can use the research agenda to anticipate future funding announcements. In fiscal year 2003, the NCIPC research budget of $41 million funded researcher-initiated grants (37.5% of funds), research centers (29.4%), research cooperative agreements (25.5%), and research contracts (7.6%). At this level of funding, full implementation of the Injury Research Agenda may take some time. However, at CDC it is firmly believed that lives will be saved and suffering will be reduced with this funding.

INJURY RESEARCH: OPPORTUNITIES AND CHALLENGES

The context in which research is conducted is changing rapidly. Injury researchers should be aware of changing opportunities and expectations related to accountability for the expenditure of federal funds, openness during the conduct and dissemination of research findings, emphasis on research addressing linked health problems, and globalization.

Federal agencies are increasingly being held accountable not just for the appropriateness of their expenditures, but also for describing the impact of their research. Typical measures of research success (such as published findings in peer-reviewed journals), while easy to count, do not answer the fundamental question "What difference has your research made?" Answers like "It is too soon to tell" no longer satisfy policymakers faced with difficult funding decisions.

While continuing to produce publications and other products, scientists should also collect personal stories from people whose lives have been affected by the research, as well as information on specific uses of the findings. Initial research protocols should include strategies for disseminating the findings and ensuring that the next steps in the public health continuum are taken. Next steps might include conducting follow-up research, sustaining a successful demonstration program, or disseminating study results to policymakers and tracking whether the results effect policy or legislative change.

The speed and scope of the global communication infrastructure present an enormous opportunity to injury researchers. Members of research consortia have for some time used the Internet to share information. Recently, the public health community received a glimpse of its potential future when researchers around the world shared data to quickly identify the causal organism of severe acute respiratory syndrome (SARS).[11] This unprecedented collaboration provides a model for how researchers can solve complex public health problems by working together. The application of this model in nonacute settings needs to be explored.

The enhanced communication infrastructure provides opportunities to respond to the public's increasing demand for health information. Injury researchers now have the opportunity to disseminate their findings not just through academic journals, but also through Web sites, newspapers, and other venues with large audiences. Often, researchers are reluctant to reduce their findings to sound bites that fail to capture the nuances of their work. Nevertheless, by quickly disseminating their findings, researchers can take advantage of the public's interest in health and safety and can provide usable information for both the public and policymakers.

The Internet has generated interest in the sharing of government-sponsored research data. New policy requires researchers receiving federal funds above a specific amount to make their data available to others, while at the same time ensuring the confidentiality of research participants.[12] The goal of this policy is to ensure that data are used as widely as possible to inform research and program efforts. The policy may also inspire new collaborations between researchers and practitioners with shared interests.

Openness and participation also apply to the way research is conducted. This is reflected by the increasing emphasis placed on research that incorporates community partners at all stages.[13] Community participatory research not only increases the relevancy (external validity) of the work, it also enhances the adoption of scientific findings in practice settings. However, such research can be very challenging. Researchers and community partners with different research emphases may need to negotiate. Research outcomes may take longer, and questions may arise about the quality or rigor (internal validity) of the research. Lessons learned from successful, rigorous community research should be disseminated to the injury research community and be used to train students in community research methods and partnership building.

Another prevalent theme in injury research is the need to account for linkages between various health problems as they occur among individuals and in communities. This consideration often arises in the context of doing community participatory research. CDC, including NCIPC, has tended to fund research in a categorical manner, with funds provided to address specific outcomes (e.g., motor vehicle injuries) or risk factors (e.g., tobacco use). However, behaviors and other risk factors are often common to many types of injuries and public health problems. For example, risky alcohol use is a well-known factor in many types of injuries, and recent research shows impulsivity to be a potentially important risk factor for suicide,[14] unintentional injuries, deaths from motor vehicle crashes, and drownings. Understanding how to measure and improve the supervision of young children is also critical in addressing child neglect, injuries due to falls, and drownings.

NCIPC funds for community participatory research and addressing crosscutting issues have been limited. CDC, however, has received funding for extramural, peer-reviewed prevention research and has directed the funds toward these purposes,[15] thus placing a high priority on increasing its investment in such research.

While much of CDC's research, including that of NCIPC, has focused on interventions directed at individuals, etiological

studies have shown the importance of community-level factors. For example, community-level variables associated with violence include poverty, residential instability, and low neighborhood collective efficacy. These factors have an impact beyond what might be expected from the characteristics of the individuals living within the community.[16] Using these data, NCIPC hopes to stimulate research on the effectiveness of modifying community-level factors to reduce violent outcomes.

A final theme in injury prevention is the need to be an active participant in a global community that is committed to injury prevention and control. While most CDC investments in global health target infectious diseases, CDC and others recognize the growing importance of preventing and controlling noncommunicable diseases and conditions. Worldwide, injuries—whether they result from road traffic, suicide, falls, interpersonal violence, or war—take an enormous toll on lives. Motor vehicle crashes alone are anticipated to become the third leading cause of disability-adjusted life years (DALYs) by 2010, up from ninth place in 1990.[17] As we increase our ability to control infectious and nutritional causes of child death and illness, we observe that injuries make up a greater proportion of DALYs among young children. For example, in Southeast Asia, unintentional injuries are now the fifth leading cause of DALYs among children younger than 5 years.[18]

Emphasizing global efforts to reduce road traffic deaths and injuries is particularly crucial at this time. The injury burden of road traffic crashes is steadily increasing worldwide; globally, for men aged 15 to 44 years, road traffic injuries rank second only to HIV/AIDS as the leading cause of illness and premature death.[18] In recognition of this burden, the World Health Organization has dedicated World Health Day 2004 to road traffic safety.[19] On April 7, 2004, and in the weeks that follow, events around the world will draw attention to road traffic crashes and potential solutions. The United Nations will also stress the need for public health and transportation agencies to work together to address this problem.

CONCLUSION

Worldwide, injuries remain a leading cause of death and suffering. While effective interventions exist for some injury-related problems, more research is needed to better understand how successful interventions can be incorporated into practice settings. For other injury issues, much more research is needed. The CDC Injury Research Agenda lays out NCIPC research priorities for the next several years. As researchers develop and implement the ideas presented in the agenda, they should take into account the changing research scene and their role in reducing the public health burden worldwide, and they should avail themselves of opportunities to make an even greater impact. ■

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Integrating the Environment, the Economy, and Community Health: A Community Health Center's Initiative to Link Health Benefits to Smart Growth

| Peter V. McAvoy, JD, MS, Mary Beth Driscoll, BA, and Benjamin J. Gramling, BS

The Sixteenth Street Community Health Center (SSCHC) in Milwaukee, Wis, is making a difference in the livability of surrounding neighborhoods and the overall health of the families it serves. SSCHC is going beyond traditional health care provider models and working to link the environment, the economy, and community health through urban brownfield redevelopment and sustainable land-use planning.

In 1997, SSCHC recognized that restoration of local air and water quality and other environmental conditions, coupled with restoring family-supporting jobs in the neighborhood, could have a substantial impact on the overall health of families. Recent events indicate that SSCHC's pursuit of smart growth strategies has begun to pay off.

A HOLISTIC HEALTH APPROACH

OPERATING AS 1 OF 15

federally qualified, community-based health centers in Wisconsin, the Sixteenth Street Community Health Center (SSCHC) has for more than 34 years relied on a place-based mission in offering primary health care to families living in Milwaukee's Near South-Side neighborhood, which primarily comprises low-income Latinos. SSCHC's Department of Environmental Health was created in 1997 to address environmental factors that affect health, including deteriorating lead paint in housing and poor air and water quality. The department was charged with achieving a healthy environment within its service area through restoring abandoned, environmentally contaminated industrial sites; attracting high-quality investment; and creating family-supporting jobs to increase the prosperity of the low-income families it serves, thereby increasing constituents' ability to pay for quality health care, nutritious food, and suitable housing.

This approach complements the ongoing development of "smart growth" plans by Wisconsin municipalities as required by the Wisconsin State Legislature. Wisconsin's "smart growth" legislation offers financial assistance to municipalities for long-range planning that links transportation and land-use policies to quality of life in both urban and rural settings. Additional information regarding Wisconsin's "smart growth" legislation can be found at http://www.doa.state.wi.us/olis.

SSCHC's Department of Environmental Health program promotes sustainable development which will create a viable alternative to the sprawling suburban development that has come to characterize southeastern Wisconsin. Sustainable development features the reuse of existing buildings and land (including brownfields), conserving residential neighborhoods, maintaining local community character, promoting the health of the community, and protecting the environment for future generations.

THE CENTER OF IT ALL: MILWAUKEE'S MENOMONEE RIVER VALLEY

SSCHC's service area includes the Menomonee River Valley, a 1500-acre collection of properties that is adjacent to downtown Milwaukee and Lake Michigan and surrounded by the most densely populated neighborhoods in Wisconsin. This valley was the center of Wisconsin's industrial production for a century, employing more than 50 000 people at its peak. Many of the workers lived in neighborhoods bordering the valley and either walked to work or rode a trolley.

Over the last 25 years, many industrial manufacturers have either closed or relocated. With the loss of nearby jobs, many family breadwinners are forced to commute an hour or more to jobs in surrounding suburbs. Because few mass-transit alternatives are available, the few workers who own cars must join countless other commuters using the region's interstate highways. Local and regional transportation patterns, coupled with industrial and environmental factors, are associated with high rates of asthma and respiratory illness.[1] In addition, poor land stewardship, non–point-source pollution, and contaminated harbor sediments resulted in poor water quality, which contributed to

KEY FINDINGS

- A community health center can link restoration of the local environment, creation of good family-supporting jobs, and public health.
- Visioning exercises help residents visualize how a revitalized area can look and function.
- These visioning and design events have served as a catalyst for achieving high-quality, well-designed redevelopment.
- Redevelopment of industrial brownfield sites may be an alternative to suburban and exurban sprawl.

FIGURE 1—Participants in the 1999 charrette, or visioning and design workshop, were charged with designing ways to bring high-quality investors and family-supporting jobs back to the community and to reverse the Menomonee River Valley's historical environmental abuse.

beach closings (1 of every 4 days in 2003) and ongoing fish-consumption advisories.[2]

BEYOND POLICY

The success of the SSCHC-led 1999 Sustainable Development Design Charrette for Milwaukee's Menomonee River Valley (Figure 1) provided visions that fueled the need to develop site-specific land-use plans that would accommodate SSCHC's sustainability and "smart growth" objectives. The charrette, or visioning workshop, involved over 140 local design professionals from the public and private sector. In collaboration with these and other partners, SSCHC hosted the 2002 Menomonee River Valley National Design Competition: Natural Landscapes for Living Communities, which focused on a 140-acre parcel within the Menomonee River Valley (Figure 2). This property historically supported 5000 employees of the Chicago, Milwaukee, St. Paul & Pacific railroad company (the Milwaukee Road), but it employed increasingly fewer persons in the second half of the 20th century, a trend typical of many Menomonee Valley enterprises. Barriers to redeveloping this site are characteristics shared by most Menomonee Valley properties: poor access, decrepit buildings, impaired soils and groundwater, and low property values.

To obtain ideas regarding the revitalization of the Menomonee Valley, SSCHC held the aforementioned design competition sponsored in part by the National Endowment for the Arts. A field of 25 teams was narrowed to 4 finalists, each with experience in dealing with environmental contamination, landscape architecture, and natural landscaping, as well as storm-water and flood-management techniques. A group of technical advisors outlined design elements for the set of problems to which the teams responded and evaluated the final designs for technical merit before a jury of national and local experts determined the winning plan.

Held over 6 months, the competition process included opportunities for public input. The winning submission included an industrial park that will provide family-supporting jobs for surrounding neighborhoods while significantly adding to the city's property tax base. The master plan allowed for the integration of natural and open-space elements into the industrial park, including a community green, a storm-water park that will prevent water pollution, and Wisconsin's new multi-use Hank Aaron State Trail. In addition, the plan called for restoration of ecological systems within the affected segment of the Menomonee River, a step which will address the valley's and the surrounding neighborhoods' lack of recreational open space.

FINDINGS

The 1999 design charrette and the 2002 national design competition have been critical in providing residents of Milwaukee with a vision of how a revitalized Menomonee River Valley could look and function. They combine to illustrate an exercise in moving from conceptual analysis and brainstorming to real-world plan-

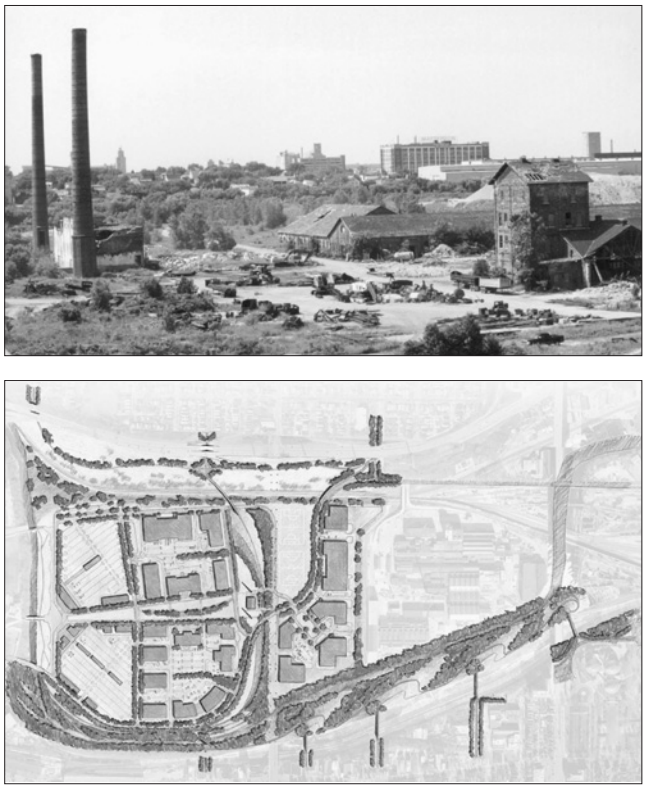

FIGURE 2—The abandoned railroad yard (top photo) was the focus of the 2002 Menomonee River Valley National Design Competition: Natural Landscapes for Living Communities, which attracted nationally recognized, award-winning teams from around the United States, the United Kingdom, Germany, and Canada. The winning master plan by Wenk Associates of Denver, Colorado (bottom photo), provides both development space for new jobs and recreational opportunities for the community in a setting where environmental damage has been repaired.

TABLE 1—The Menomonee Valley Benchmarking Initiative's Economic, Environmental, and Community Indicators for Tracking Progress in the Sixteenth Street Community Health Center's Sustainability Objectives[3]

Economy
- Business activity
- Employment
- Commercial/industrial property
- Infrastructure and access

Environment
- Water quality
- Air quality
- Land cover and habitat
- Flora and fauna

Community
- Housing
- Crime
- Arts and events
- Health

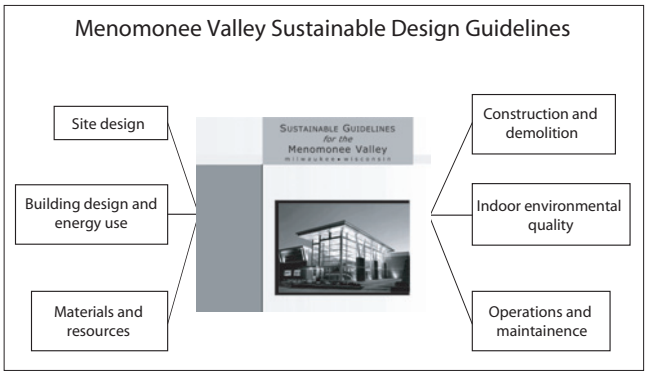

FIGURE 3—Sustainable guidelines for the Menomonee Valley, which identify sustainability objectives for developers and property owners and offer practical suggestions for achieving high performance green architecture.

ning and implementation of sustainable redevelopment practices. Their outcomes, and the widespread media coverage they received, have served as a catalyst for achieving high-quality, well-designed redevelopment that will ensure that people of the adjoining neighborhoods and surrounding communities are reconnected to the valley through new jobs and recreational opportunities.

The momentum that has built around the 1999 design charrette and the national design competition of 2002 has also spurred the successful cleanup of 3 contaminated industrial sites totaling 38 acres. In addition, the City of Milwaukee, in collaboration with the civic nonprofit organization Menomonee Valley Partners, Inc, has initiated a master planning, cleanup, and redevelopment effort for a collection of properties on the east end of the Menomonee Valley, which will add an additional 20 acres of development space. It is estimated that several thousand new jobs will be created in the valley once all properties are fully redeveloped.

To determine an employment baseline, a study conducted by SSCHC and the University of Wisconsin–Milwaukee, under the auspices of the Menomonee Valley Benchmarking Initiative (Table 1), calculated the number of jobs located in the Menomonee Valley during 2002 to be 9451 (which includes 7961 full-time positions). This study will be replicated in 2004 and in subsequent years to measure increased employment opportunities and long-term progress toward other economic, environmental, and social indicators of sustainability objectives in the valley.[3]

A community health center can make a difference in the livability of surrounding neighborhoods and the overall health of the families it serves by going beyond traditional health care provider models and working to link the environment, the economy, and community health. SSCHC and its partners are working to establish measurable standards for private-sector, sustainable development by developing sustainable design guidelines (Figure 3) and marketing the Menomonee Valley to investors committed to "smart growth" principles.

Although redevelopment of Milwaukee's Menomonee River Valley began only recently, the area is already undergoing significant change. Ultimately, success will be achieved when the valley's environment is cleaned up, new family-supporting jobs located close to housing are created and held by neighborhood residents, and the health and livability of neighborhoods surrounding the valley are substantially improved. ■

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Policies of Inclusion

Immigrants, Disease, Dependency, and American Immigration Policy at the Dawn and Dusk of the 20th Century

The racial politics of immigration have punctuated national discussions about immigration at different periods in US history, particularly when concerns about losing an American way of life or American population have coincided with concerns about infectious diseases.

Nevertheless, the main theme running through American immigration policy is one of inclusion. The United States has historically been a nation reliant on immigrant labor and, accordingly, the most consequential public policies regarding immigration have responded to disease and its economic burdens by seeking to control the behavior of immigrants within our borders rather than excluding immigrants at our borders.

Amy L. Fairchild, PhD, MPH

CARVED IN STONE IN A PILLAR adorning the National Archives in Washington, DC, is a line from Shakespeare that has captured my imagination: "What is past is prologue." Day after day I read this phrase as I entered the archives, reviewing the records of the US Public Health Service (PHS) relating to the immigrant medical inspection that was required at the nation's borders beginning in 1891. Whether boxes and boxes of records impaired my judgment, whether I was swayed by its inexorable logic, or whether it simply felt true for the ways in which we have thought about the intersection of immigration and disease, I became a firm believer: "What is past is prologue."

But the problem is that historians have provided us with multiple prologues, and this can trip us up when we try to make the past speak to the present. Despite widespread assumptions about the exclusionary nature of American immigration policy, the history of immigration at the beginning and end of the 20th century is in fact a history of inclusion.

The period 1924 to 1965 is set off by 2 landmark pieces of legislation: the Immigration Act of 1924, which made national origin the basis for admission into the United States, and the Immigration Act of 1965,[1] which eliminated the national origins systems and at the peak of the Civil Rights Movement restored what President Lyndon Johnson called "the basic principle of American democracy."[2] But in placing undue emphasis on the racially restrictive nature of policy between 1924 and 1965, it becomes too easy to view all policies—past and present—through a lens of exclusion.[3]

I had been led to explore the early history of medical inspection by a contemporary policy disaster that occurred while I was working in the Policy Unit of the New York State Department of Health's AIDS Institute in the early 1990s. At that time, the US detention of some 200 Haitian immigrants infected with HIV at the naval base at Guantanamo Bay, Cuba, reached its climax. I found it very easy, using the Immigration Act of 1924 and histories of eugenics and scientific racism as a lens, to tell a story of racial restrictions masquerading as public health policy. It was a story, I argued, emblematic of our larger immigration policy.[4] Conversely, I was prepared to see the historical origins of immigrant medical inspection as the story of public health used for racial demarcation and exclusion. This interpretation is very much in keeping with how social historians have traditionally viewed the relationship between immigration and disease.[5]

In this article, I compare the broad intentions of US policy fundamentally concerned with managing the economic burden of disease in 2 periods: the Progressive Era, in which medical inspection sought to control the consequences of disease not by turning immigrants back but by introducing them to industrial values and expectations regarding work and dependency, and the current era of immigration and welfare reform. Although the racial politics of immigration have typically framed our understanding of Progressive Era policy, in discussing that period I consider the day-to-day practice of immigrant medical inspection and the ways it was shaped by industrial demands. For the present era, in which individuals certainly live within the constraints of both federal and state policy on a day-to-

day basis, I focus on the politics and policy at the broadest level, considering the provisions of the 1996 Personal Responsibility and Work Opportunity Reconciliation Act (PRWORA, or Personal Responsibility Act), which excluded illegal and legal immigrants from many public benefits such as Medicaid.

There are, of course, important differences between the 2 eras: immigrants, particularly illegal immigrants, fuel the service industry and highly skilled immigrants fill the ranks of the information sector in the current era of globalization, whereas in the late 19th and early 20th centuries immigrants joined the unskilled industrial labor force. Likewise, the changing position and power of organized labor potentially gives different meaning to the notion of inclusion in the 2 different eras. Nonetheless, at both the dawn and dusk of the 20th century, I argue, the most consequential public policies responded to disease and its economic burdens by seeking to control the behavior of immigrants *within* our borders rather than excluding immigrants *at* our borders. That this theme of inclusion marks 2 such different eras underscores its enduring significance in American public policy.

"At both the dawn and dusk of the 20th century, the most consequential public policies responded to disease and its economic burdens by seeking to control the behavior of immigrants within *our borders rather than excluding immigrants* at *our borders. That this theme of inclusion marks 2 such different eras underscores its enduring significance in American public policy."*

INCLUSION AS BACKDROP: 1882 TO 1924

There coexisted in Progressive Era America 2 models—interconnected by questions of race and labor—of citizenship, one characterized by fitness for civic participation and the other by fitness for industrial participation. The political movement to restrict immigrants from southern and eastern Europe prioritized questions of fitness for self-government and emphasized the inherent genetic and intellectual racial inferiority of the new immigrant streams.[6] In the 1890s, Senator Henry Cabot Lodge and his Immigration Restriction League connected the literacy test with protection of American character and citizenship in the 1890s.[7] The literacy test promised to restrict the entry of "beaten men from beaten races" with "none of the ideas and aptitudes" necessary for democratic self-rule.[8]

Critically, however, expansive notions of racial restriction stemming from civic concerns did not find their way into actual immigration legislation until well after the turn of the century.[9] Even the literacy test—as it was finally passed in 1917 over President Woodrow Wilson's veto—required only that immigrants be able to read in any language, including Hebrew and Yiddish. With its entry into the control of immigration in the 1880s, Congress remained legislatively focused on the immigrant as industrial participant, aligning itself with business and against labor and the nation's proponents of scientific racism. It was a model of industrial fitness, then, that would shape US immigration policy during the Progressive Era.[10]

Between the ends of the Civil War and World War I, the United States was transformed from a society of artisans, who largely controlled the pace of production, into the world's leading industrial power. By the 1880s and 1890s, mechanization swelled the ranks of the unskilled labor force.[11] In the face of the changing nature of production and the changing composition of the work force, industry took advantage of an opportunity to assert control and authority. A new cadre of scientific and industrial managers undertook the tasks of redefining ability to work in terms of segmented tasks rather than supervision of a product from start to finish. By 1911, Fredrick Winslow Taylor's *Principles of Scientific Management*, first published in the 1890s, profoundly shaped the way the nation thought about how to organize work efficiently. Industrial leaders saw scientific management as a process for removing "the manager's brain" from "under the workman's cap."[12] While scientific racists were concerned with ensuring that the nation's inhabitants remained "well born," those concerned with the labor half of the equation insisted that this was not enough: the worker "must be trained

right as well as born right."[13] Industry, therefore, was interested in worker discipline.[14]

Dramatic changes in industrial production and management not only allowed the unprecedented expansion of American industry but also generated great economic fragility. The phenomenon of unemployment introduced a new dimension into defining and managing a necessarily fluid industrial workforce while at the same time providing a compelling rationale for disciplining those deemed destined to destitution. As workers increasingly located in urban areas and the labor supply swelled to accommodate the demands of a rapidly growing industrial power, hundreds of thousands of industrial workers became "utterly dependent upon their industrial earnings to survive."[15]

But workers could not rely on industrial earnings. Many "minor" recessions and depressions accompanied the 6 "major" economic downturns that the nation experienced from 1870 to 1921.[16] In this kind of work economy, sickness could mean the difference between survival and destitution.[17] While most of the laboring class relied primarily on the resources of family and friends rather than public or private charity or relief organizations during lean times,[18] illness, rather than the nature of the economy, was viewed as the "outstanding problem which led to dependency."[19] In this context, then, government in addition to industry had an interest in controlling the worker. Thus, when the federal government began to exercise its congressional authority over immigration in 1882,[20] it sought not to *restrict* immigration but rather to *control* it by preventing the entry of those who could not support themselves as well as "convicts, lunatics, and idiots."[21]

Control rather than restriction would characterize immigration policy for the next 4 decades. With the immigration law of 1891, the federal government created the machinery for federal officials to inspect and exclude immigrants. The law required medical officers of the PHS to issue a medical certificate to all immigrants suffering from a "loathsome or a dangerous contagious disease."[22] Loathsome and dangerous contagious diseases—also known as class A conditions—included trachoma (also known as granular conjunctivitis), an infectious eye condition that could lead to blindness; favus, a fungal infection of the scalp and nails; venereal diseases; parasitic infections; and tuberculosis.[23] A subset of class A conditions included mental conditions such as insanity, feeblemindedness, imbecility, idiocy, and epilepsy.

In 1903, the PHS created a new category of class B diseases or conditions—those rendering the immigrant "likely to become a public charge."[24] Class B conditions included hernia, valvular heart disease, pregnancy, poor physique, chronic rheumatism, nervous afflictions, malignant diseases, deformities, senility and debility, varicose veins, and poor eyesight.[25] But in the context of industrial-era America, not only class B conditions affecting ability to earn a living but also the loathsome and dangerous contagious diseases took on economic meaning in the hands of the PHS, which defined contagious immigrant diseases as "essentially chronic." Chronic, debilitating disease represented the permanent inability of an immigrant to function in society; it represented dependency.[26]

At the core of the industrial economy were the dual principles of disciplining and discarding the laboring body. It was not simply the case that the worker bound for dependency had to be barred at the nation's threshold; rather, at the nation's threshold, *all* workers had to learn the rules and expectations of industrial society. Immigrant laborers had to understand that they were expected to remain fit throughout the inevitable spells of unemployment that they would be required to weather. The message was clear to Bridget Fitzgerald, who came from Ireland in 1921 at age 18: "You know what you needed then mostly? I'll tell you. Strong and healthy, that you won't become a public charge, because then, I mean, you go right back."[27]

While federal immigration law sent roughly 79 000 immigrants home for diseases or defects, it brought *all* 25 million arriving immigrants—particularly those traveling in steerage or third class who would join the ranks of laborers—under the scrutiny of the PHS.[28] The assembly line of flesh and bone developed to defend the nation from diseased immigrants served as the inaugural event in the life of the new labor force. Immigrant medical examination centered on the "line," which became shorthand for techniques and procedures for quickly examining thousands of immigrants. In the context of immigrant medical inspection, it represented a direct and meaningful analogy to the industrial assembly line and is central to a story of inclusion.

Ellis Island, where roughly 70% of immigrants entered the United States, set the standard

for examination on the "line." After an arriving ship passed the quarantine inspection in New York Harbor,[29] Immigration Service and PHS immigrant examiners boarded and examined all first- and second-class passengers. PHS officers transferred steerage or third-class passengers to Ellis Island by barge. Proceeding one after the other and lugging heavy baggage, prospective immigrants entered the often-congested immigration station and proceeded slowly through a series of gated passageways resembling cattle pens. The winding passage leading toward the PHS officers who waited at the end ensured that each could witness the inspection of dozens of immigrants ahead. As they reached the end of the line, immigrants slowly filed past one or more PHS officers who, at a glance, quickly surveyed them for a variety of serious and minor diseases and conditions, finally turning back their eyelids with their fingers or a buttonhook to check for trachoma.[30] "Were they ready to enter? Or would they be sent back?" wondered each immigrant with faces "taut, eyes narrowed" throughout the process.[31]

Manny Steen, who immigrated from Ireland in 1925, kept the moment of entry at Ellis Island fresh in his memory for nearly 7 decades, describing it as "the worst memory I have of Ellis Island." He remembered that "doctors were seated at a long table with a basin full of potassium chloride and you had to stand in front of them, follow me, and they'd ask you and you had to reveal yourself. . . . Right there in front of everyone! I mean, it wasn't private."[32] His memory of the humiliating nature of the examination was shared by Enid Griffiths Jones, inspected at Ellis Island in 1923 at age 10: "And we went to this big, like an open room, and there were a couple of doctors there, and then they tell you, 'Strip.' And my mother had never, ever undressed in front of us. In those days nobody ever would. She was so embarrassed. And it was all these other, all nationalities, all people there."[33] Steen and Jones described not an examination but a public spectacle. Even the more intensive examination of the estimated 10% to 20% that the PHS "turned off the line" was also a public event,[34] as illustrated in photographs depicting the intensive examination of men and women at Ellis Island sometime after the turn of the century (photos this page and next).

Power, wrote Foucault, "must be spectacular, it must be seen by all almost as its triumph." The spectacle of inspection on "the line" represented a "ritual recoding" to be "repeated as often as possible."[35] The inspections represented emersion in a particular, routinized, ordered set of exercises or motions—waiting in line, moving in unison, stepping up to the medical inspector, moving forward, stepping up to the immigrant inspector, answering questions. In this fashion, they were introduced to the repetitive, monotonous habits of industrial order. For 14-year-old Bessie Kriesberg, the process impressed upon her the imperative "to obey the rules."[36] It was one of many reinforcing moments in the new immigrant's life. Ellis Island was, in the words of Michael La Sorte, part of "a seamless continuity" that began overseas "and ended somewhere in America."[37]

Public health in the Progressive Era was, of course, not

Photo courtesy fo the National Park Service, US Department of Interior.

Women undergoing secondary medical examination at Ellis Island.

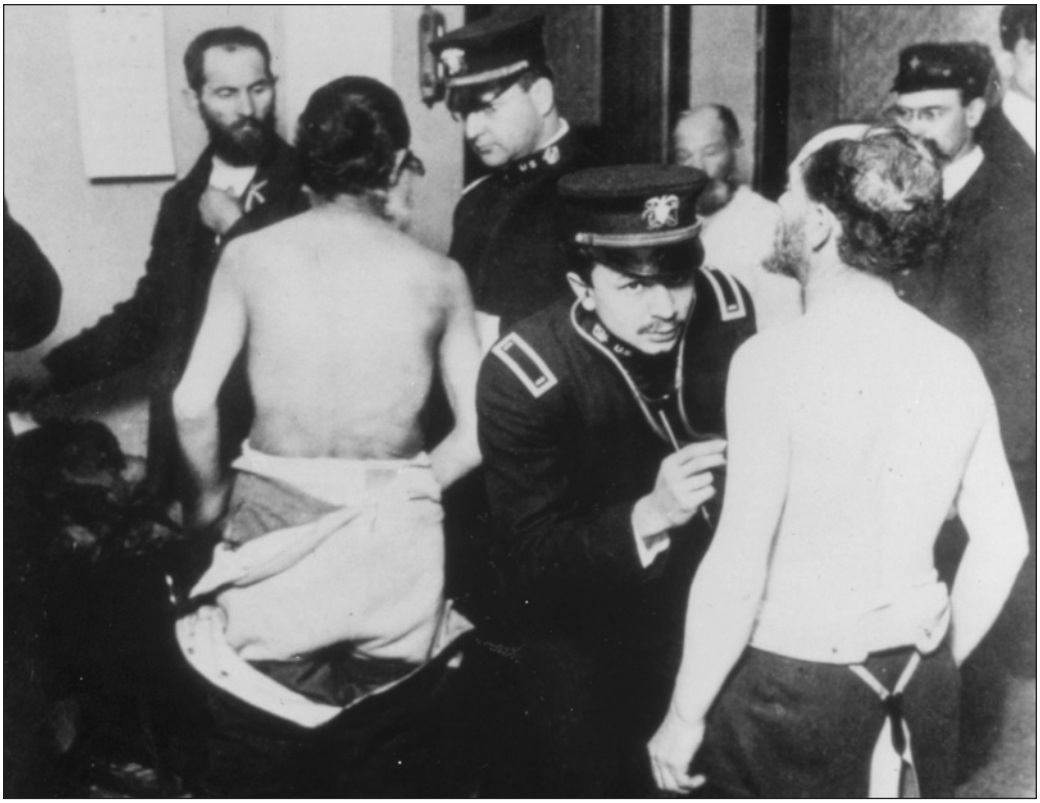


Photo courtesy fo the National Park Service, US Department of Interior.

Jewish immigrants undergoing the secondary medical examination at Ellis Island. As also reflected in the previous photograph, the secondary examination was conducted in a group setting in which immigrants witnessed the examination of others.

solely about inclusion or absorption of immigrants into the national workforce, as historians such as Howard Markel, Alan Kraut, and Nayan Shah have powerfully demonstrated.[38] In the case of the immigrant medical examination, when groups of immigrants failed to conform to societal expectations about the industrial worker, the examination worked to *exclude* those groups at the nation's borders on the understanding that they were not racially fit for industrial labor. Disease was instrumental in rationalizing these exclusions, and the medical examination served as a flexible tool to achieve higher exclusion rates in regions of the country receiving greater shares of "undesirable" immigrants. Consequently, immigrants faced considerable medical obstacles to entry and higher rates of medical certification and exclusion at the nation's Pacific Coast and Mexican border immigration stations.[39] Nonetheless, given the industrial context, the terms of inclusion must provide a backdrop to such exclusionary endeavors.

EXCLUSION AS BACKDROP: 1924 TO 1965

The backdrop does, however, change in 1924 with the national origins quota system, which was explicitly racially exclusive. The Immigration Act of 1924 capped immigration at 150 000 per year and restricted immigration to 2% of the population of each "race" recorded in the US census of 1890, representing a deliberate attempt to dramatically limit immigration from southern and eastern Europe.[40] A very vocal segment of the nation's political and intellectual elite viewed the legislation as an important means to stem a threatening tide of physically, genetically, and intellectually inferior southern and eastern European immigration.

The threat of "inferior races" and disease informed some of the most exclusionary policies from 1924 to 1965. Emily Abel, for example, describes how the fears of contagion and dependency enabled public health officials to use tuberculosis as a tool for repatriating Mexican immigrants and citizens in the West. As Abel convincingly argues, a growing consensus regarding Mexicans' lack of entitlement to US citizenship made health officials emphasize the economic consequences of tuberculosis as a chronic disease.[41] Although Herbert Hoover's policy of repatriation (ostensibly voluntary, but often viewed by immigrants as mandatory) was abandoned under Franklin Delano Roosevelt, Roger Daniels explains that "there was nothing even approaching a New Deal for immigration." Indeed, under Roosevelt, racial exclusions were extended to Filipinos in 1934.[42]

US refugee policy during World War II—or rather the absence of formal policy, epitomized in 1939 when the United States turned back the *St. Louis*, most of whose 933 passengers were Jewish refugees—stands as the greatest testament to the exclusionary practices of the United States after 1924. But even if woefully inadequate, informal presidential directives resulted in some quarter of a million refugees reaching the United States during and after the war.[43] That some effort was made to expand America's immigration policy during World War II underscores the fact that just as the Progressive Era was not characterized entirely by inclusion, nei-

ther was the period from 1924 to 1965 characterized entirely by exclusion.

Many have cited the Immigration Act of 1965 as abruptly ending the exclusionary era, fundamentally altering the face of immigration, and, indeed, causing a near-catastrophic rise in immigration.[44] No doubt the Immigration Act contributed to the rising tide of immigration and its increasing proportion of Asians and Latin Americans, but it was actually the 1924 restriction legislation that ushered in the profound, though unintended, changes in the sources of immigration.[45] As Daniels explains, the relatives of immigrants already in the United States and immigrants from Latin America and Canada were not subject to the numerical limitation under the quota law; immigration in these categories increased from about 10% of total immigration in the period before World War I to approximately 45% of immigration by 1930, considerably altering immigration patterns.[46] Figure 1 shows that the sources of immigration began to shift after the turn of the century, with the proportion of Europeans steadily declining after 1900.[47] Figure 2 further shows that while immigration fell off dramatically after 1924, it quickly began a steady increase, which was disrupted by depression in 1929 and war in 1940 (we see a similar pattern following World War I). By 1947, immigration resumed at a level we would have expected had there been no depression or war.

It was this dip in immigration during World War II that helped to create severe labor shortages and that prompted the United States to ease restrictive immigration policy, chiefly admitting Mexican and Chinese laborers. The Displaced Persons Act of 1948 created a national refugee policy, resulting in the admission of some 400 000 persons by 1952. Subsequently, the Refugee Act of 1953 allotted an additional 214 000 nonquota visas; it sought primarily to protect, in true Cold War spirit, those seeking to escape communism, but it also extended admission to Asian and Middle Eastern immigrants.[48]

America's increasingly permissive stance on immigration was not limited to refugees. 1952 also saw the passage of the McCarran–Walter Act, which, in repealing previous immigration laws, not only expanded the classes of aliens subject to exclusion and deportation and made it easier to accomplish both, but it also reduced barriers to skilled immigration and family reunification and ended the policy of Asian exclusion. The McCarran–Walter Act was, to be sure, not intended as a liberal measure to increase immigration to the United States. The backdrop to this legislation was most decidedly exclusion: Patrick McCarran sponsored the Internal Security Act of 1950 that prohibited the immigration of communists and fascists. The bill ultimately retained the national origins system, but, as Robert Divine has observed, as "an act of conservatism rather than intolerance."[49] A vast amount of immigration fell outside of the quota and nearly 3.5 million immigrants—many of them Asian—subsequently entered.

Just as the doors to the nation did not decisively slam shut in 1924, neither did they dramatically swing open in 1965. Rather, the period from 1924 through 1965 represented decades of gradual intended and unintended change in response to immigration legislation that at some moments sought even tighter restrictions on the basis of race but at others pursued more tolerant policies when the economy and humanitarianism demanded them.

> *"The inspections represented emersion in a particular, routinized, ordered set of exercises or motions—waiting in line, moving in unison, stepping up to the medical inspector, moving forward, stepping up to the immigrant inspector, answering questions."*

INCLUSION AS BACKDROP, EXCLUSION AS VEIL: WELFARE AND IMMIGRATION REFORM IN THE 1990S

It is the centrality that questions of disease and dependency would once again take in the 1990s, and how the nation would respond to them legislatively, that enables us to draw an analogy between the opening and closing decades of the 20th century. The AIDS crisis raised deep concerns that immigrants with HIV would swell the Medicaid rolls, causing a collapse of our hospital and medical systems. These were the concerns that made Guantanamo Bay possible and that fostered a broader immigration policy banning the immigration of individuals with HIV.

The new restrictions on HIV and immigration were part and parcel of a growing concern regarding the economic burden that immigrants placed on society. While in 1986 the Immigration Reform and Control Act legalized an unprecedented 2.7 million illegal immigrants living in the United States,[50] the Immigration Act of 1990 attempted to reverse the flow of immigrants who might not be self-sufficient, raising the nation's immigration ceiling by providing an unlimited number of visas to relatives of US citizens but reducing the allocation of visas for unskilled immigrants and raising the total visa quota.[51]

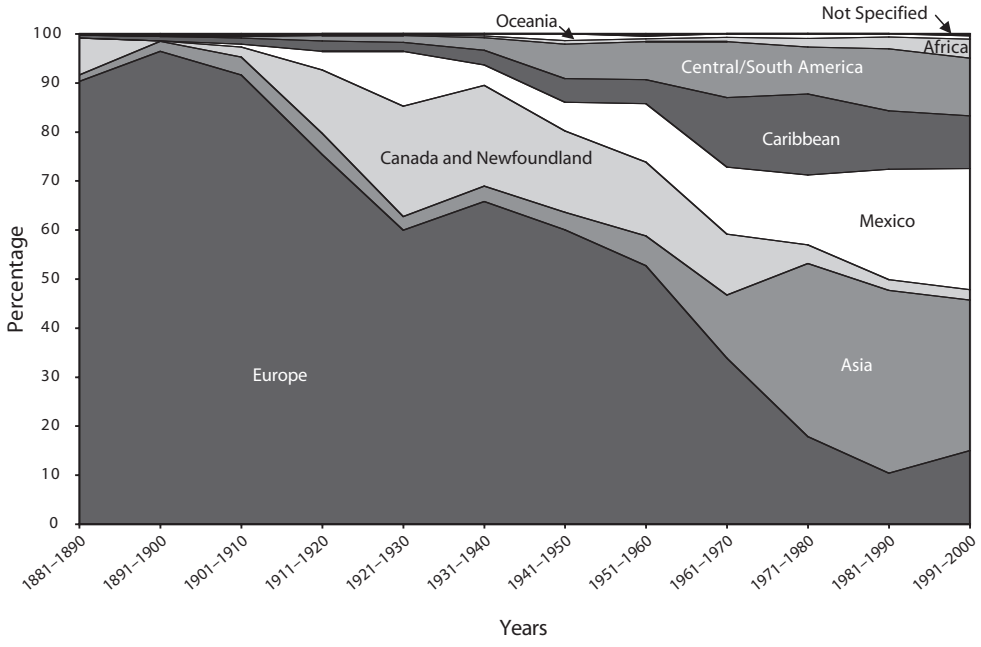
But it was not immigration policy that most clearly expressed the new concerns regarding disease and dependency; it was welfare policy. The most sweeping policy measure affecting immigrants and welfare was the Personal Responsibility Act of 1996—a policy currently undergoing reauthorization. One of the distinguishing features of the initial legislation was its withdrawal of many public benefits from legal immigrants and "undeserving" citizens. With some exceptions, the law barred immigrants from receiving Supplemental Security Income and food stamps until they became citizens.[52] Immigrants could not receive cash assistance,[53] Medicaid, Social Services Block Grant services, and other federal means-tested

programs for 5 years after arrival.[54] The law barred illegal immigrants from all but a few selected in-kind, noncash services typically involving emergency care or vaccination.[55] The income and financial resources of an immigrant's sponsor—typically a family member who had to be a citizen or lawful permanent resident—were "deemed" available to any immigrant applying for benefits.[56]

Welfare reform's provisions regarding immigrants resonated with the exclusionary leitmotif running through 20th-century immigrant policy. The 1996 Personal Responsibility Act as passed promised some $54.1 billion in savings over 6 years, with $23.8 billion (44%) to be achieved primarily at the expense of immigrants, both legal and illegal.[57] Because immigrants are more likely to engage in employment that carries no health benefits,[58] it was conceivable that welfare reform would provide a strong disincentive to legal and illegal immigration. Welfare reform's clear ties to California's Proposition 187—which passed with nearly 60% of the vote in 1994 and denied a variety of public services, including public education, to illegal immigrants—underscored the extent to which it was initially viewed as an exclusionary immigration measure.[59] Bob Dole, for instance, the 1996 Republican presidential candidate, reasoned that "if kids can't go to school, the parents will go home."[60]

In part, an enormous Hispanic voter backlash would cause Republicans gradually to alter their rhetoric and pitch welfare reform not as an immigration control measure but rather as sound social policy.[61] The rhetorical about-face, however, was not purely strategic. Welfare reform was motivated by complex and deeply rooted sentiments in the United States regarding humanitarianism and its limits, order and discipline, enforcing an "ideal" family structure, citizenship and its entitlements and obligations, and the labor market.[62] Senator Bob Bennett of Utah stated that the issue for children was to create conditions "so [they] can learn and be productive citizens." Senator Daniel Patrick Moynihan, who ultimately proved a strong opponent of the bill, also saw the *potential* for welfare reform to send a message about the expectations of citizens: "We expect of you what we expect of ourselves and our own loved ones: that you will do your share in taking responsibility for your life and the lives of the children you bring into the world."[63] The themes of citizenship, discipline, and family were not intended to resonate only for immigrants but for all of the working class.

The theme of promoting discipline within and regulation of the labor market was reflected not only in welfare policy but also in immigration policy, although exclusion remained a key contrapuntal element marking the debates. The US House of Representatives, in an amendment to the immigration reform bill that sought to increase the number of Border Patrol officers, increase workplace immigration inspections, and restrict food stamps to immigrants,[64] voted in March 1996 to deny public education benefits to illegal immigrant children.[65] Speaker of the House Newt Gingrich argued that "There is no question that offering free taxpayer goods to illegals attracts more illegals." He



Source. 2000 Statistical Yearbook of the Immigration and Naturalization Service. Available at http://uscis.gov/graphics/shared/aboutus/statistics/imm00yrbk/imm2000list.htm. Accessed February 25, 2004.

FIGURE 1—Immigration by region, expressed as a percentage of total immigration, 1891 through 2000.

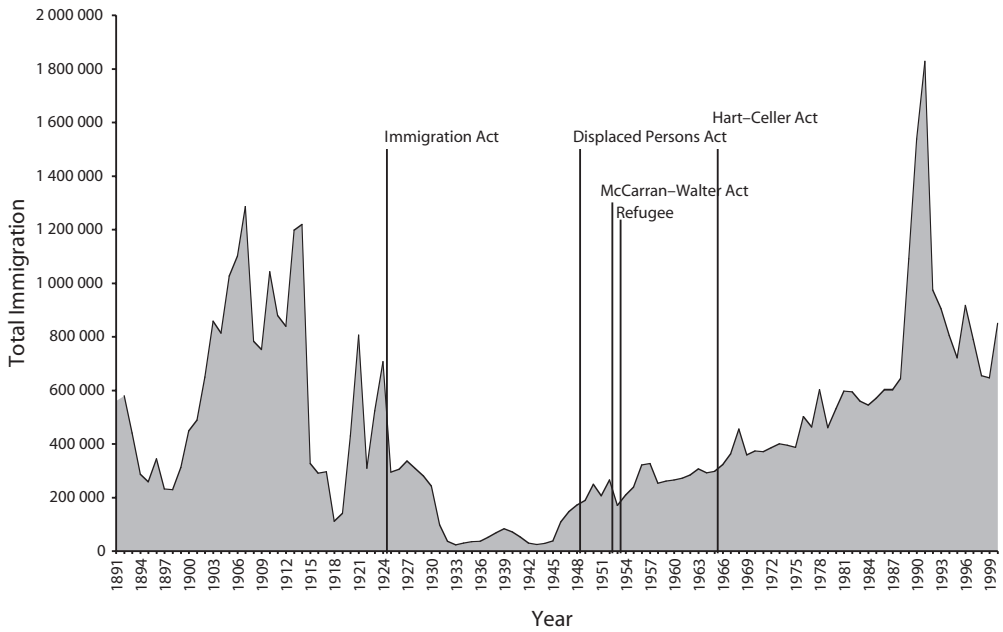
concluded: "It is wrong for us to be the welfare capital of the world."[66] Although the bill carried the strong support of Bob Dole,[67] incumbent President Bill Clinton threatened to veto the measure. Subsequently, the Senate, with the backing of conservative Texas Senators Phil Gramm and Kay Bailey Hutchinson, successfully blocked the amendment.[68]

Clinton signed the immigration reform bill on September 30, 1996, without measures denying either public education to illegal immigrants or federal treatment funds to legal immigrants infected with HIV/AIDS. Also missing were provisions to deport legal immigrants who received more than a year's worth of federal benefits within a 7-year period.[69] While the immigration reform bill remained "one of the most sweeping efforts by Congress in years to control illegal immigration,"[70] also absent from the final legislation were provisions to reduce legal immigration by 30%, as the Immigration Reform Commission had urged, and provisions to increase substantially the number of Department of Labor workplace inspectors to investigate and penalize employers for hiring illegal immigrants.[71] In refusing to limit immigration or create disincentives for businesses to hire illegal immigrants, the legislation thus made a powerful statement about the centrality of the contributions of both legal and illegal immigrants to the US economy.[72] Coming on the heels of the Personal Responsibility Act, which dramatically limited the social obligations of the nation to these immigrants, it clearly defined the terms of inclusion: immigrants entered a social contract in which they must make economic contributions but in which the nation had no reciprocal obligations.[73]

CONCLUSION: TERMS OF INCLUSION

Just as it would be a mistake to deny the exclusionary elements of public policy touching immigrants during the Progressive Era or the countervailing currents of immigration policy in the restrictive decades between 1924 and 1965, it would also be foolhardy to ignore or minimize these elements of recent policy.[74] HIV exclusion has dropped out of public discussion, but the events of September 11, 2001, have reinvigorated the metaphors and language of disease—infections, terrorist cells, eradication—and renewed interest in exclusion. The advent of Sudden Acute Respiratory Syndrome (SARS) no doubt will reinforce such interest. We find ourselves poised on the border between greater inclusion and further restriction.[75] In the period since the September 11th attacks, Congress has passed measures tightening control of the borders and intensifying the scrutiny and surveillance of immigrants.[76] In March 2002, both the House and Senate overwhelmingly passed legislation to increase the number of immigration investigators and inspectors and to establish a surveillance system for people entering with student visas. President Bush has signed "modern," "smart border" agreements with Canada and Mexico aimed at further limiting the flow of illegal immigrants, drugs, and terrorists, without slowing the flow of goods.[77]

In this context, it would be easy to draw analogies to the anti-immigration rhetoric that surfaced



Source. 2000 Statistical Yearbook of the Immigration and Naturalization Service. Available at http://uscis.gov/graphics/shared/aboutus/statistics/imm00yrbk/imm2000list.htm. Accessed February 25, 2004.

FIGURE 2—Total immigration to the United States, 1891 through 1999.

in the mid-1990s.[78] But emerging debates and even policies in the aftermath of 9/11 are not likely easily to derail the broader inclusionary impulses characterizing American immigration policy in the current period. Thus, while one interpretation of the welfare and immigration reform measures is that it sends the message "Non-Yankees Go Home,"[79] we have to look through this veil at the larger backdrop.

In March 2002, the US House of Representatives, in support of negotiations between the Bush administration and Mexico's President Vicente Fox, approved a measure to make it easier for illegal immigrants to gain legal status in the United States.[80] After encountering opposition from Democratic Senator Robert Byrd, the measure was excluded from May's reconciliation legislation, which hardened immigration enforcement laws. Despite this setback, the proposal had carried broad bipartisan support in both the House and Senate. It also enjoyed wide community and business support.[81] Indeed, Democratic Senate Majority Leader Tom Daschle reintroduced the measure on May 9, 2002.[82] Most recently, President Bush put forward a guest-worker proposal that would allow illegal immigrants to obtain renewable 3-year work permits that many critics have derided as exploitative.[83]

It is important, then, to appreciate the exact terms of inclusion as well as the extent of support behind inclusion, which includes not only Republicans, Democrats, and employers—who may or may not have a stake in improving the terms of inclusion for immigrants—but also organized labor.

The AFL-CIO, in February 2000, began to urge the legalization and unionization of illegal Mexican immigrants, representing a dramatic shift in a policy position forged during the Progressive Era.[84] At the beginning of the century, unions saw the new immigrant laborer as living outside the craftsman's ethic of collective behavior; as a contemporary labor lyric put it, "There were no men invited such as Slavs and 'Tally Annes,'/ Hungarians and Chinamen with pigtail cues and fans."[85] This "dangerous class" of unskilled labor was perceived as "inadequately fed, clothed, and housed" and, accordingly, it "threatened the health of the community."[86] This was a period, after all, in which labor struggled not only to organize but to organize against incredible odds: beginning in the 1880s, corporations gained recognition as "persons" sharing in constitutional rights in a context in which the courts increasingly reduced the rights of citizenship to "unfettered liberty of contract." The courts consistently ruled that regulation of wages and work conditions represented a fetter on this liberty. Conversely, the courts consistently prohibited labor boycotts and strikes.[87]

The change in the position of organized labor was a reaction to its stagnating membership levels and a political economy that had changed dramatically since the dawn of the 20th century.[88] Although it was decidedly weak early in the century, during and after World War II the power of unions was considerably strengthened and by century's end the national AFL-CIO membership of some 16 million had been relatively stable for decades.[89] But immigrants potentially promise increased power and position for organized labor. California's local unions, drawing on a large and largely illegal immigrant labor force, added some 132 000 members in 1999.[90] Thus, the 17.7 million immigrants in the United States[91]—perhaps 7 million of them illegal—have provided a powerful incentive for a switch in organized labor's long-held positions.[92]

Characterized by the North American Free Trade Agreement, which was strongly opposed by organized labor,[93] globalization also brought into question the terms of employment not only in this nation but also in the less-developed nations where business might locate or relocate production.[94] In this global context, while immigration policy can shape whether the nation has an information economy versus a labor or service-sector economy, as well as the distribution of wealth within it,[95] exclusion no longer represents a viable alternative for controlling labor conditions or opportunities within the nation, as it might have earlier in the century, when the United States was the leading industrial producer and American business thrived on a large, highly mobile, and responsive unskilled labor force.

But while the new position of labor offers hope for altering the terms of inclusion—a hope not possible early in the 20th century—the trajectory of welfare reform must give us pause. In the summer of 2002, the House passed a version of a reauthorization bill, still excluding immigrants,[96] that increased work requirements from 20 to 40 hours per week with no exemptions for women with children aged younger than 6 years.[97] House Republicans argued—and some Democrats agreed—that the PRWORA was a stunning success, dramatically reducing the welfare caseloads despite rising immigration and unemployment.[98]

Absent was any suggestion that increasingly strict requirements provided disincentives to immigration: legislators viewed welfare reform as a means of creating and training productive citizens. Mark Foley, a Republican representative from Florida, argued, "My grandmother came from Poland, she was a maid at the Travel Lodge Motel, she worked hard all her life. All she wanted to be is a good citizen and an honest, God-fearing person of this country." He saw the bill as "preparing our citizens" and would-be citizens "for the future of this country and its economy."[99] It was not simply that if a lifetime of menial, low-wage work was good enough for Foley's grandmother, then certainly it was good enough for today's immigrants—it was good for today's workers regardless of their immigration status. President Bush has begun touting welfare reform as ending "the culture of dependency that welfare" had created for people who should properly be thought of as "citizens of this country, with abilities and aspirations" and not "charges of the state."[100] Given that the Senate is now controlled by Republicans, the House reauthorization seems likely to pass sometime in 2004.[101]

Thus, as important as it will be in this era to remain alert to the nation's policies of exclusion, particularly when they turn on questions of race or nationality, a focus on exclusion at the borders can obscure a critical analysis of the terms of inclusion that we set not only for immigrants but for all workers. How the nation sets

those terms, and the extent to which they reflect either suspicion and distrust of immigrants as a potential burden or a recognition of mutual obligations between workers and society, will differ as the economic base of the nation changes, as the position and power of organized labor alters, and, of course, as the sources and levels of immigrant shift. As we make decisions about immigration, we must view immigration reform along with welfare reform as being fundamentally concerned with engineering the economic structure of American society, as being fundamentally about the nature of inclusion. This is a prologue that does not deny the exclusionary impulses within American immigration history and policy, but that can refocus our attention in the present. ■

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Endnotes

1. While the national origins system was eliminated, the use of quotas continued. Total hemispheric quotas capped at 290 000 per year. In 1976, hemispheric caps were abandoned and each country was allotted a quota of 20 000, and the Refugee Act of 1980 excluded refugees from the preference system. Roger Daniels, "Two Cheers for Immigration," in *Debating American Immigration, 1882–Present*, ed. Roger Daniels and Otis L. Graham (New York: Rowman & Littlefield Publishers Inc, 2001), 37, 41, 78.

2. Quoted in Daniels, "Two Cheers for Immigration," 43.

3. Rudolph J. Vecoli, "Ethnicity: A Neglected Dimension of American History," in *The State of American History, ed.* Herbert J. Bass (Chicago: Quadrangle Books, 1970), 73.

4. Amy L. Fairchild and Eileen A. Tynan, "Policies of Containment: Immigration in the Era of AIDS," *American Journal of Public Health* 84 (1994): 2011–2022.

5. Howard Markel, *Quarantine! East European Jewish Immigrants and the New York City Epidemics of 1892* (Baltimore: Johns Hopkins University Press, 1997); Matthew Frye Jacobson, *Whiteness of a Different Color: European Immigrants and the Alchemy of Race* (Cambridge, Mass: Harvard University Press); Alan M. Kraut, *Silent Travelers: Germs, Genes, and the Immigrant Menace* (New York: Basic Books, 1994); Kenneth Ludmerer, *Genetics and American Society: A Historical Appraisal* (Baltimore: Johns Hopkins University Press, 1972).

6. It was such a notion of political citizenship that in part motivated Chinese exclusion in 1882. Congress deemed the Chinese unfit for democratic self-rule and barred the Chinese not only from entry into the United States but also from naturalization. The 14th Amendment, ratified in 1868, made citizenship and equal protection under the law a constitutional birthright. The Naturalization Law of 1870 subsequently affirmed the right of persons of African descent to naturalization and their right to vote. But it also denied citizenship to first-generation Asian immigrants. Eric Foner, *The Story of American Freedom* (New York: W. W. Norton & Company, 1998), 112. The Immigration Act of 1917 and subsequent Supreme Court decisions in *Ozawa v the United States* (1922) and *The United States v Thind* (1923) reaffirmed such exclusion on the grounds that Asians were not "White." See also Ian F. Haney-Lopez, *White by Law: The Legal Construction of Race* (New York: New York University Press, 1996).

7. John Higham, *Strangers in the Land: Patterns of American Nativism, 1860–1925* (New Brunswick, NJ: Rutgers University Press, 1994), 103.

8. Francis A. Walker, "Restriction of Immigration," *Atlantic Monthly*, June 1896, p. 828.

9. Higham, *Strangers in the Land*, 43–44, 48–49, 73, 99–100, 112, 129–130, 202, 203–204, 221.

10. Likewise, the movement for Chinese exclusion was not rooted exclusively in questions of fitness to participate in American public life. Congress—in rare concordance with organized labor, which saw Chinese workers as severely depressing White wages—responded to a perceived threat that the Chinese posed to industrial civilization. Gwendolyn Mink, *Old Labor and New Immigrants in American Political Development: Union, Party, and State, 1875–1920* (Ithaca, NY: Cornell University Press, 1986), 90–91, 96; Alexander Saxton, *The Indispensable Enemy: Labor and the Anti-Chinese Movement in California* (Berkeley: University of California Press, 1971). The 1882 Exclusion Act did not bar the entry of all Chinese immigrants; it targeted laborers only.

11. David Montgomery, *The Fall of the House of Labor: The Workplace, the State, and American Labor Activism, 1865–1925* (Paris: Cambridge University Press, 1987), 24, 25.

12. William D. Haywood and Frank Bohn, Industrial Socialism (Chicago, n.d.), 25, quoted in Montgomery, *Fall of the House of Labor*, 45.

13. Fredrick Winslow Taylor, *Principles of Scientific Management* (Norcross, Ga: Engineering and Managing Press, 1911), viii–xi, 50.

14. Montgomery, *Fall of the House of Labor*, 252.

15. Alexander Keyssar, *Out of Work: The First Century of Unemployment in Massachusetts* (Cambridge, England: Cambridge University Press, 1986), 25.

16. During these years, the economy faltered 13 times, meaning that each decade brought 4 years of depression or recession. Keyssar, *Out of Work*, 47.

17. Unemployment Committee of the National Federation of Settlements, *Case Studies of Unemployment* (Philadelphia: University of Pennsylvania Press, 1931), 71.

18. Lizabeth Cohen, *Making a New Deal: Industrial Workers in Chicago, 1919–1939* (Cambridge, England: Cambridge University Press, 1990), 57.

19. *Commissioner-General's Annual Report* (Washington, DC: Government Printing Office, 1898), 2. See also memorandum abstracting information in "The Alien as Charity Seeker," Children's Bureau, US Department of Labor, Vol. IV, No. 29, October 1927; "Aliens and Charity," Immigration History Research Center, University of Minnesota, St. Paul; US Senate, *Reports of the Immigration Commission, Abstracts of Reports of the Immigration Commission, Abstract of the Report on Immigrants as Charity Seekers*, Vol. 2

20. The Supreme Court had ruled in 1849 that control of immigration, as a matter of "foreign commerce," fell within the authority of Congress, but the Immigration Act of 1882 is generally regarded as the first federal effort to assert its authority. Higham, *Strangers in the Land*, 356, note 19; Benjamin Klebaner, "State and Local Immigration Regulation in the United States Before 1882," *International Review of Social History* 3 (1958): 269–295.

21. Higham, *Strangers in the Land,* 43–44; John Higham, "Origins of Immigration Restriction, 1882–1897: A Social Analysis," *Mississippi Valley Historical Review* 39 (June 1952): 79–80.

22. The Public Health Service (PHS) was created in 1798 as the United States Marine Hospital Service under the jurisdiction of the Treasury Department, where it remained until 1939. Its initial function was to provide medical care to merchant marines. Although I refer to it consistently as the PHS here, it was renamed several times and was not known as such until 1912. Ralph Chester Williams, MD, *The United States Public Health Service, 1798–1950* (Washington, DC: Government Printing Office, 1951). Officers of the Immigration Service made the final decisions regarding whether immigrants would be deported for disease, although deportation of immigrants with class A diseases was mandatory. Although I refer to it as the Immigration Service, it, too, was renamed and reorganized several times throughout its history. Darrel H. Smith, *The Bureau of Immigration* (Baltimore: Johns Hopkins Press, 1926); Darrel H. Smith, *The Bureau of Naturalization* (Baltimore: Johns Hopkins Press, 1926).

23. Barbara Bates, *Bargaining for Life: A Social History of Tuberculosis, 1876–1938* (Philadelphia: University of Pennsylvania Press, 1992), 16–18; Sheila M. Rothman, *Living in the Shadow of Death: Tuberculosis and the Social Experience of Illness in American History* (New York: Basic Books, 1994), 13–15.

24. Bureau of Public Health and Marine-Hospital Service, *Book of Instructions for the Medical Inspection of Immigrants* (Washington, DC: Government Printing Office, 1903), 5, 10–11.

25. Amy L. Fairchild, *Science at the Borders: Immigrant Medical Inspection and the Making of the Modern Industrial Labor Force* (Baltimore: Johns Hopkins University Press, 2003), 32.

26. Fairchild, *Science at the Borders*, 14.

27. Joan Morrison and Charlotte Fox Zabusky, *American Mosaic: The Immi-*

grant Experience in the Words of Those Who Lived It (New York: E. P. Dutton, 1980), 42. As an employee, she described herself as "a 'useful girl.' "

28. On average, 4.4% of all immigrants were certified annually from 1909 to 1930, peaking at more than 8.0% in 1918 and 1919, although only about 11% were ever deported. The medical deportation rate for medical causes never exceeded 1%. Fairchild, *Science at the Borders*, 4–5. The immigrant medical inspection was designed for processing third-class or steerage passengers. Although an officer might occasionally send a first-class passenger for closer examination, he searched primarily not for physical but for social aberration: "If a passenger is seen in the first cabin, but his appearance stamps him as belonging in the steerage or second cabin, his examination usually follows." Letter from Assistant Surgeon General H.D. Geddings to Surgeon General, November 16, 1906, RG90, Central File, 1897 to 1923, Box 36, File No. 219, National Archives and Records Administration, College Park, Md.

29. A.H. Doty, "The Use of the Clinical Thermometer as an Aid in Quarantine Inspection," *Medical Record*, 1 November 1902, p. 690; A.H. Doty, "Modification of Present Port Inspection," *American Public Health Association Reports* 21 (1906): 260.

30. Letter from Assistant Surgeon General H.D. Geddings to the Surgeon General, November 16, 1923, RG90, Central File, 1897 to 1923, Box 36, File No. 219, National Archives and Records Administration, College Park, Md; E.H. Mullan, "The Medical Inspection of Immigrants at Ellis Island," *Medical Record*, 27 December 1913, p. 1168.

31. Quoted in Irving Howe, *World of Our Fathers* (New York: Galahad Books, 1976), 43.

32. Paul Sigrist, interview with Manny Steen, March 22, 1991, Ellis Island Oral History Project.

33. Janet Levine, interview with Enid Griffiths Jones, April 18, 1993, Ellis Island Oral History Project.

34. There is no precise data to support this estimate. Allan Kraut cites this figure, which appears sporadically in the PHS records. No doubt, during some of the peak immigration years before the war, a far smaller percentage was turned off the line; likewise, during the war, when immigration levels were very low and the PHS experimented with the utility of conducting a more intensive medical examination, the percentage was higher.

35. Michel Foucault, *Discipline and Punish: The Birth of the Prison* (New York: Vintage Books, 1979), 34, 111.

36. Bessie Kriesberg, Hard Soil, *Tough Roots: An Immigrant Woman's Story* (Jericho, NY: Exposition Press Inc, 1973), 138, 139.

37. Michael La Sorte, *La Merica: Images of Italian Greenhorn Experience* (Philadelphia: Temple University Press, 1985), 48.

38. Markel, *Quarantine!*; Kraut, *Silent Travelers*; Nayah Shah, Contagious Divides: Epidemics and Race in San Francisco's Chinatown (Berkeley: University of California Press, 2001); Emily Abel, "From Exclusion to Expulsion: Mexicans and Tuberculosis in Los Angeles, 1914–1940," Bulletin of the History of Medicine 77 (Winter 2003): 823–849.

39. Fairchild, *Science at the Borders,* chap 4. See also Shah, Contagious Divides, and Abel, "From Exclusion to Expulsion."

40. Robert A. Divine, *American Immigration Policy,* 1924–1952 (New York: Da Capo Press, 1957, 1972); Mae Ngai, "The Architecture of Race in American Immigration Law: A Reexamination of the Immigration Act of 1924," *Journal of American History* 86 (June 1999), available at http://www.historycooperative.org/journals/jah/86.1//ngai.html, accessed January 16, 2004.

41. Abel, "From Exclusion to Expulsion"; Ngai, "The Architecture of Race."

42. Daniels, "Two Cheers for Immigration," 26–27; Benicio Catapusan, "Filipino Immigrants and Public Relief in the United States," *Sociology and Social Research* 23 (1939): 546–554; Ronald Takaki, *Strangers From a Different Shore: A History of Asian Americans* (New York: Penguin Books, 1989).

43. Daniels, "Two Cheers for Immigration," 26–27, 30.

44. Peter Brimelow, *Alien Nation: Common Sense About America's Immigration Disaster* (New York: Random House, 1995); Roy Beck, *The Case Against Immigration: The Moral, Economic, Social, and Environmental Reasons for Reducing US Immigration Back to Traditional Levels* (New York: W.W. Norton & Company, 1996), 69–70.

45. Divine, *American Immigration Policy,* 167.

46. Daniels, "Two Cheers for Immigration," 24–29; Kitty Calavita, "US Immigration Policymaking: Contradictions, Myths, and Backlash," in *Regulation of Immigration: International Experiences,* ed. Anita Bocker, Kees Groenendijk, Tetty Havinga, and Paul Minderhood (Amsterdam: Het Sphinhuis Publishers, 1998), 141.

47. See also Christopher Jencks, "Who Should Get In? Part II," *New York Review of Books*, 20 December 2001, available at www.nybooks.com/articles/14942, accessed April 18, 2002.

48. Daniels, "Two Cheers for Immigration," 29–35, 38; E.P. Hutchinson, *Legislative History of American Immigration Policy 1798–1965* (Philadelphia: University of Pennsylvania Press, 1981), 264–265.

49. Divine, *American Immigration Policy,* 161, 164–176, 190; Hutchinson, *Legislative History*, 297–312.

50. Diane Lindquist, "In Search of Amnesty; Call to Legalize Workers Gives Mexicans Hope," *San Diego Union-Tribune*, 29 May 2000, p. 1; Robin Gerber, "Labor's Welcome Change of Course on Immigration," *Baltimore Sun*, 29 February 2000, p. 23.

51. Nancy Foner, *From Ellis Island to JFK: New York's Two Great Waves of Immigration* (New Haven, Conn: Yale University Press and Russell Sage Foundation, 2000), 11, 249 (note 73); National Research Council, *The New Americans: Economic, Demographic, and Fiscal Effects of Immigration* (Washington, DC: National Academy Press, 1997), 29. In the late 1990s, Congress authorized the Immigration and Naturalization Service to issue more temporary visas to highly skilled workers.

52. In 1997, Congress restored Supplemental Security Income eligibility to immigrants residing in the United States before welfare reform was passed; Balanced Budget Act of 1997, P.L. 105–33. In 1998 and 2000, food stamp benefits were restored to some immigrants, their children, the disabled, and the elderly; William E. Gibson, "Immigrants to US Discover Welcome Mat Is Out," *Seattle Times*, 5 September 1999, p. 9.

53. Exceptions to cash assistance included programs such as the National School Lunch Act, Head Start, and emergency medical assistance. The Temporary Assistance for Needy Families Block Grant (TANF) replaced Aid to Families With Dependent Children (AFDC). States may provide cash assistance under TANF, but they can provide vouchers or services in lieu of cash.

54. States could continue to withhold cash benefits from nonexempted immigrants even after this 5-year period; P.L. 104–193, Title I, section 402(a)(1)(B)(ii) and Title IV, sections 401–403, 411–412.

55. States largely controlled the manner in which immigrants who arrived before 1996 were covered by means-tested programs. Immigrants, therefore, remained eligible for some benefits even within 5 years of arrival in states like California and in New York City.

56. Exceptions apply to programs such as emergency medical assistance. The practice of "deeming" originated in 1980 with Supplemental Security Income benefits. Deeming, as a means of limiting access to welfare, was extended to programs such as AFDC before being incorporated into Personal Responsibility Act in 1996. George J. Borjas, *Heaven's Door: Immigration Policy and The American Economy* (Princeton, NJ: Princeton University Press, 1999), 119.

57. *Immigrant Policy News* 3 (August 1996): 1; Wendell Primus, "Immigration Provisions in the New Welfare Law," *Focus* 18 (Fall/Winter 1996–1997): 14–18; R.Y. Kim, "Welfare Reform and 'Ineligibles': Issue of Constitutionality and Recent Court Rulings," *Social Work* 46 (2001): 315.

58. Marsha Lillie-Blanton and Julie Hudman, "Untangling the Web: Race/Ethnicity, Immigration, and the Nation's Health," *American Journal of Public Health* 91 (2001): 1736–1738. Texas almost immediately implemented the PRWORA cutbacks without additional state compensation.

59. Dave Lesher, "Deadlock on Prop. 187 Has Backers, Governor Fuming," *Los Angeles Times*, 8 November 1997, p. 1; Patrick J. McDonnell, "Judge's Final Order Kills Key Points of Prop. 187," *Los Angeles Times*, 19 March 1998, p. 3; Patrick J. McDonnell and Ken Ellingwood, "Immigration—the Big Issue of '94—Disappears From '98 Debate," *Los Angeles Times*, 23 October 1998, p. 3; Patrick J. McDonnell, "Davis Won't Appeal Prop. 187 Ruling, Ending Court Battles," *Los Angeles Times*, 29 July 1999, p. 1; Evelyn Nieves, "California Class Off Effort to Carry Out Immigrant Measure," *New York Times*, 30 July 1999, p. 1.

60. Greg McDonald, "Dole Scorns 'Liberals' Over School Issue," *Houston Chronicle*, 25 September 1996, p. 13; Marc Lacey, "Immigration Debate About to Resurface," *Los Angeles Times*, 26 May 1996, p. 18.

61. McDonnell and Ellingwood, "Immigration"; McDonnell, "Davis Won't Appeal"; Elaine S. Povich, "Courting Hispanics," *Newsday*, 21 April 2002, p. 4.

62. Michael Katz, *In the Shadow of the Poorhouse: A Social History of Welfare in America* (New York: Basic Books, 1996), xi.

63. Congressional Record, Personal Responsibility and Work Act of 1995 Conference Report, Vol. 141, No. 206 (21 December 1995): S19098, S19089.

64. Eric Schmitt, "GOP Seems Ready

to Drop Political Fight," *New York Times*, 18 September 1996, p. 6.

65. Marc Lacey and Patrick J. McDonnell, "House Votes to OK Bans on Illegal Immigrant Schooling," *Los Angeles Times*, 21 March 1996, p. 1.

66. Ibid. See also Marc Lacey, "Immigration Debate About to Resurface," *Los Angeles Times*, 26 May 1996, p. 18.

67. Schmitt, "GOP Seems Ready."

68. McDonald, "Dole Scorns 'Liberals' Over School Issue."

69. Marc Lacey, "Toned Down Bill on Immigration Passes in House," *Los Angeles Times,* 29 September 1996, p. 12; "Non-Yankees Go Home: That's the Message of the Punishing New Immigration and Welfare Reform Laws," *Newsday*, 2 October 1996, p. 36.

70. The final legislation denied refugees the right to counsel, raised the income requirements of people seeking to sponsor immigrants to 25% over the poverty level, and doubled the size of the Border Patrol; McDonald, "Dole Scorns 'Liberals' Over School Issue."

71. Lacey, "Toned Down Bill"; "Non-Yankees Go Home."

72. James Flanigan, "New Bill on Immigration is Borderline at Best," *Los Angeles Times*, 29 September 1996, p. 1.

73. Even illegal immigrants, though formally excluded, have a role and function within the US economy, from the laundry industry in Chicago to agriculture in the West and Southwest. Louis Uchitelle, "INS Is Looking the Other Way as Illegal Immigrants Fill Jobs," *New York Times*, 9 March 2000, p. 1; Mark Bixler, "Immigration Deal Could Bolster Unions; Many Illegals Who Want to Join Labor Organizations Fear Deportation," *Atlanta Journal and Constitution*, 2 September 2001, p. 2; Christopher Parks and Henry Tricks, "Illicit Angels of America's Economic Miracle," *London Financial Times*, 23 February 2000, p. 10; Michael Riley, "Increase in Immigration Arrests Leaves Town's Workforce Depleted," *Denver Post*, 14 April 2002, p. 18.

74. Mae Ngai, "Legacies of Exclusion: Illegal Chinese Immigration During the Cold War Years," *Journal of American Ethnic History* 18 (Fall 1998): 3–35; Mae Ngai, "The Strange Career of the Illegal Alien: Immigration Restriction and Deportation Policy in the United States, 1921–1965," *Law and History Review* 21 (Spring 2003): 69–108; Natalia Molina, *Contested Bodies and Cultures: The Politics of Public Health and Race Within Mexican, Japanese, and Chinese Communities in Los Angeles, 1879–1939* [PhD Dissertation] (Ann Arbor: University of Michigan, 2000); Emily Abel, "From Exclusion to Expulsion."

75. Bill McAllister, "Immigration Law Unlikely to Soften; Mexican Workers' Status Can't Change for Safety Reasons, Tancredo Asserts," *Denver Post*, 26 September 2001, p. 1; Cindy Rodriguez, "In the Week of Attacks, Proposals to Toughen Laws Are Expected," *Boston Globe*, 19 September 2001, p. 26.

76. Robert Pear, "Bill on Border Security and Immigration Passes in House," *New York Times*, 9 May 2002, p. 34; Diana Jean Schemo, "Officials to Speed Start of New Student Visa Tracking System," *New York Times*, 11 May 2002, p. 12. The effects of September 11th may be further exacerbated by commentaries such as Pat Buchanan's *Death of the West: How Dying Populations and Immigrant Invasions Imperil Our Country and Civilization* (New York: St. Martin's Press, 2002).

77. Patty Reinert, "Border Fix Won't Be Quick," *Houston Chronicle*, 27 March 2002, p. 3; Elisabeth Bumiller, "White House Announces Security Pact with Mexico," *New York Times*, 22 March 2002, p. 18; Carla Baranauckas, "Bush Signs Border-Security Measure," *New York Times*, 15 May 2002, p. 20.

78. Peter Brimelow, "Time for a Change? Enough! America Is Drowning in a Sea of Immigrants," *Atlanta Journal and Constitution*, 30 April 1995, p. 1G.

79. "Non-Yankees Go Home."

80. Robert Pear, "House Passes Immigrant Bill to Aid Mexico," *New York Times*, 13 March 2002, p. 1; Robert Pear, "Immigrant Bill," *New York Times*, 17 March 2002, p. 2; Mark Bixler, "House Votes to Ease Immigration Rule," *Atlanta Journal and Constitution*, 13 March 2002, p. 3A.

81. Associated Press, "Border Security Bill Sent to Bush," *New York Times*, 9 May 2002; Pear, "Bill on Border Security"; Pear, "House Passes Immigrant Bill."

82. The bill is still in the Senate Judiciary Committee.

83. David Abraham, "American Jobs but not the American Dream," *New York Times*, 9 January 2004, p. A19.

84. "AFL-CIO Calls for New Direction in US Immigration Policy to Protect Workers, Hold Employers Accountable for Exploitative Working Conditions," February 16, 2000, available at www.aflcio.org/mediacenter/prsptm/pr02162000d.cfm, accessed January 16, 2004.

85. Michael McGovern, *Labor Lyrics, and Other Poems* (Youngstown, Ohio, 1899), 27–28, quoted in Montgomery, *Fall of the House of Labor*, 25.

86. Edith Abbott, "The Wages of Unskilled Labor in the United States," *Journal of Political Economy* 13 (1905): 324.

87. In its 1886 ruling in the Wabash case, the Supreme Court recognized corporations as "persons" protected under the 14th Amendment to the Constitution. The ruling also prohibited states from regulating interstate commerce, giving sole regulatory authority to the federal government.

88. Steven Greenhouse, "In US Unions, Mexico Finds an Unlikely Ally on Immigration," *New York Times*, 19 July 2001, p. A1.

89. Eric Brazil, "Unions Widen Their World; Ex-Foes of Undocumented Workers Now See Them as Membership Targets," *San Francisco Chronicle*, 2 September 2001, p. W1.

90. Lindquist, "In Search of Amnesty."

91. It is not clear how labor will view foreign workers who are not either legal or illegal immigrants. Two federal programs from the early 1990s helped to expand the labor force without visibly expanding the immigrant population. The H1-B visa system was established in 1990 to permit businesses to sponsor highly skilled foreign workers if domestic workers were not available. H1-B visa holders are not technically immigrants, but most are likely to remain permanently. In a declining economy, these workers are increasingly viewed as "serfs" who take American jobs and drive down wages, and measures have been introduced to reduce drastically the number of H1-B visas the Immigration and Naturalization Service may issue. Alan B. Krueger, "Work Visas Are Allowing Washington to Sidestep Immigration Reform," *New York Times*, 25 May 2000, p. C2; Tom Condon, "Vulnerable Workers Become Victims of Corporate Greed," *Hartford Courant*, 13 October 2002, p. B1; Allan Masri, "No Shortage of Trained American Engineers," *Los Angeles Times*, 8 February 2003, p. 26; Diane E. Lewis, "Congress Asked to Review IT Field; Engineers Group Upset Over H1-B Visas, Job Losses," *Boston Globe*, 23 July 2002, p. D2; Pamela R. Winnick, "Visa Versa," *Pittsburgh Post-Gazette*, 3 May 2002, p. C10. Other individuals are allowed to work in the United States if granted "temporary protected status," or TPS. US Citizenship and Immigration Services, "What Is Temporary Protected Status," available at http://uscis.gov/graphics/howdoi/tps.htm, accessed January 16, 2004.

92. Brazil, "Unions Widen Their World."

93. Lindquist, "In Search of Amnesty."

94. James B. Parks, "Recognizing Our Common Bonds," updated April 12, 2002, available at http://www.aflcio.org/aboutaflcio/magazine/commonbonds.cfm, accessed January 14, 2004.

95. Borjas, *Heaven's Door*, xiv–xvi, 5, 8–16, 62–104.

96. Congressional Record, House of Representatives, May 16, 2002, p. H2515–H2590; Robert Pear, "GOP Dispute Delays Vote on Welfare Bill," *New York Times*, 16 May 2002, A20. A Democratic substitute bill as well as planned amendments that were overruled would have restored benefits to legal immigrants along with providing an additional $5 billion for child care, given the increased work requirements; Congressional Record, p. H2559. See also Robert Pear, "House Passes a Welfare Bill With Stricter Rules on Work," *New York Times*, 17 May 2002, A1, and Hillary Rodham Clinton, Letter to the Editor, *New York Times*, 16 May 2002, A24.

97. Daniel Altman, "Welfare Bill's Tougher Love May Backfire," *New York Times*, 19 May 2002, p. 4.

98. Robert Pear, "Federal Welfare Roll Shrinks, but Drop Is Smallest Since '94," *New York Times*, 21 May 2002, p. A12.

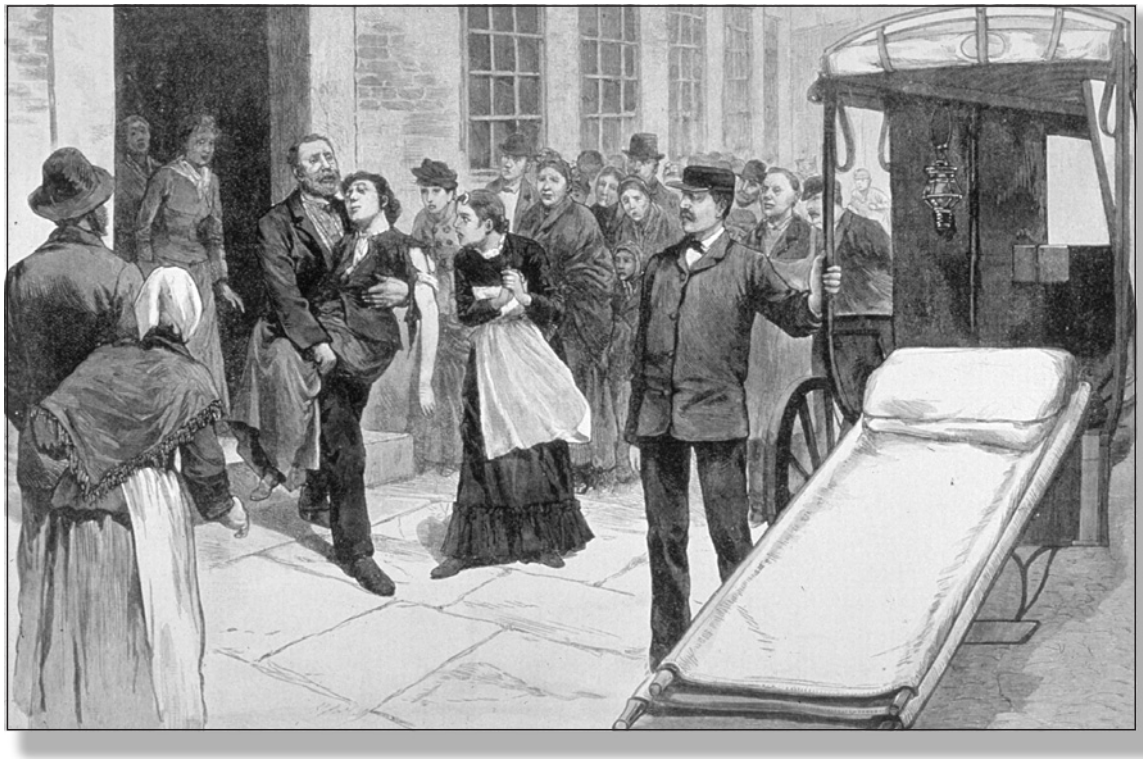
99. Congressional Record, May 16, 2002, p. H2515, and May 16, 2002, p. H2540 and H2545. The bill's detractors advanced a different vision of society and repeatedly argued that the bill failed to lift people out of poverty. They underscored that reducing the welfare rolls and combating poverty were hardly equivalent, thereby questioning the very terms of welfare reform's success. See, for example, the pointed remarks of Representative Lynn Woolsey, a Democrat from California and former welfare recipient, as well as those of Jose Serrano, a New York Democrat; Congressional Record, May 16, 2002, p. H2538. See also the editorial of Douglass McKinnon, former recipient of welfare and former press secretary to former senator Bob Dole, "The Welfare Washington Doesn't Know," *New York Times*, 21 May 2002, p. A21.

100. Ann McFeatters, "Success Stories Highlight New Welfare Push; Welfare-to-Work Law Hailed at White House," *Pittsburgh Post-Gazette*, 15 January 2003, p. A10; Editorial, "Unworkable Welfare," *Boston Globe*, 16 January 2003, p. A10.

101. Senator Max Baucus, "What's the Next Phase of Welfare Reform?" *Roll Call*, 8 December 2003 (Policy Briefings Section).

Factory Injuries and Progressive Reform

Elizabeth Fee and Theodore M. Brown



***Source*. Prints and Photographs Collection, History of Medicine Division, National Library of Medicine.**

THIS COLORED WOOD engraving, circa 1886, captures an all-too-common scene in late-19th-century America: a woman injured during the course of factory work. Scenes like this—and others far more horrific—inspired muckraking journalists and the national labor reform movement of the Progressive Era.[1] In 1907, the popular author Arthur B. Reeve wrote, "To unprecedented prosperity . . . there is a seamy side of which little is said. Thousands of wage earners, men, women, and children, [are] caught in the machinery of our record breaking production and turned out cripples. . . . Other thousands [are] killed outright. . . . How many there [are] none can say exactly for we [are] too busy making our record breaking production to count the dead."[2]

The famous socialist agitator Crystal Eastman declared, "We must pause and consider what are the essential weapons in our campaign. . . . The first thing we need is information, complete and accurate information about the accidents that are happening. It seems a tame thing to drop so suddenly from talk of revolution to talk of statistics, but I believe in statistics just as firmly as I believe in revolutions. . . . And what is more, I believe statistics are good stuff to start a revolution with."[3]

The broad labor reform movement, bringing together muckrakers, Socialists, middle-class Progressives, labor union leaders, and enlightened capitalists in an unlikely but at least temporarily powerful alliance, would lead to the creation of state bureaus of labor statistics, the passage of protective labor legislation, and the appointment of factory commissioners empowered to inspect actual working conditions and on-site adherence to the new labor laws.

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2. Reeve AB. The death roll of industry. *Charities and the Commons.* 1907; 17:791.

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Confronting the Challenges in Reconnecting Urban Planning and Public Health

Although public health and urban planning emerged with the common goal of preventing urban outbreaks of infectious disease, there is little overlap between the fields today. The separation of the fields has contributed to uncoordinated efforts to address the health of urban populations and a general failure to recognize the links between, for example, the built environment and health disparities facing low-income populations and people of color.

I review the historic connections and lack thereof between urban planning and public health, highlight some challenges facing efforts to recouple the fields, and suggest that insights from ecosocial theory and environmental justice offer a preliminary framework for reconnecting the fields around a social justice agenda. (*Am J Public Health.* 2004;94:541–546)

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DESPITE THE COMMON historical origins and interests of urban planning and public health, only minor overlaps between the 2 fields exist today. One result of this "disconnect" is an uncoordinated approach to eliminating the glaring health inequalities facing the urban poor and people of color.[1–5] While public health is increasingly concentrating on biomedical factors that might contribute to different morbidity and mortality rates between the well off and least well off, the field is just beginning to seriously investigate the role of land use decisions and how the built environment influences population health. At the same time, urban planning practice shows few signs of returning to one of its original missions of addressing the health of the least well off.[3,5] The result is that work in the 2 fields is largely disconnected, and both areas are failing to meaningfully account for the economic, social, and political factors that contribute to public health disparities.[4] However, the public health significance of the disconnect between planning and public health has not gone unnoticed.

A series of recent reports have emphasized the importance of reconnecting planning and public health. For example, a 2001 Institute of Medicine report titled *Rebuilding the Unity of Health and the Environment* emphasized that the "environment" should be understood as the interplay between ecological (biological), physical (natural and built), social, political, aesthetic, and economic environments.[6] The National Center for Environmental Health of the Centers for Disease Control and Prevention, in its 2000 report *Creating a Healthy Environment: The Impact of the Built Environment on Public Health*, argued for the reintegration of land use planning and public health, explicitly linking transportation and land use planning to public health outcomes such as increased obesity, asthma, and mental health.[7] A 1999 report published by the World Health Organization, *Healthy Cities and the City Planning Process*, emphasized the importance of developing a model of "healthy urban planning" to ensure the health of the world's increasing urban and poor populations.[8] Finally, *Healthy People 2010* lists eliminating health disparities as 1 of its 2 top priorities and acknowledges that only an interdisciplinary approach to health promotion will accomplish this goal.[9]

While these reports are important steps toward reuniting planning and public health, what is missing is an articulation of the challenges each field must confront in any reconnection effort and a theory or framework articulating why and for whom the fields should be reconnected.[10] This article highlights some of these challenges and offers a framework by drawing on insights from ecosocial epidemiology and environmental justice. I suggest that ecosocial epidemiology and environmental justice are useful paradigms because the former provides an explicit framework that attempts to explain health disparities across populations and how social relations can be pathogenic, biologically "embodied," and expressed as health inequalities,[11–13] while the latter outlines a democratic research and public decisionmaking agenda that is attentive to the distributive, procedural, and corrective justice concerns of people of color.[14–17]

THE DISCONNECT BETWEEN PLANNING AND PUBLIC HEALTH

Public health, city planning, and civil engineering in the United States evolved together as a consequence of late-19th-century efforts to reduce the harmful effects of rapid industrialization and urbanization, particularly infectious diseases.[8,18,19] Reformers recognized that poor housing conditions, inadequate sanitation and ventilation, and dangerous working conditions helped cause devastating outbreaks of cholera and typhoid.[18] Planning and public health were regularly affiliated during this era of miasma and contagion, and engineering-based sanitary reforms, largely influenced by the Chadwick report in Britain, were instituted to limit hazardous exposures through such measures as sewerage, garbage collection, and rodent control.[20–22] Planners also used the power of the state to separate out populations suspected of causing disease. Yet, both miasma and contagion failed to explain certain aspects

of population health, such as why epidemics occurred only sporadically, even with the seeming ubiquitous filth present in many urban areas, and how diseases traveled.

By the end of the 19th century, the driving ideology in public health had shifted to germ theory, and this shift continued through the first half of the 20th century. According to germ theory, there are specific agents of infectious disease, in particular microbes, and these agents relate in a one-to-one manner to specific diseases.[20] This conceptual shift was accompanied by shifts in public health and planning practice. Public health research shifted from investigating ways to improve urban infrastructure to laboratory investigations of microbes and interventions focused on specific immunization plans, with physicians, not planners, emerging as the new class of public health professionals.[12,19]

In urban planning, the German-inspired "Haussman model" of zoning began to take hold in the United States during this same period.[23] This model focused on functionality and a hierarchical ordering of land use that tended to separate residential areas from other land uses, particularly those involving industry.[24] At the core of the Haussman model was the idea of dividing up functions within the economy (e.g., zoning), isolating those functions deemed unhealthy (e.g., industry), and placing strict regulations on the kind of contact occurring between people and land use functions.[24] Zoning was aimed at "immunizing" urban populations from the undesirable externalities of the economy, such as industrial pollution.

As clinicians increasingly implemented public health measures in the mid-to-latter half of the 20th century, the field shifted toward addressing the "hosts" (e.g., individuals) of disease, because the "environment" (e.g., the world outside of microorganisms) was harder for physicians to influence.[20] During this era, public health largely ignored the social dimensions of disease and emphasized modifying individual "risk factors" reflected in one's lifestyle, such as diet, exercise, and smoking.[25] Planning, searching for an identity in postwar America, turned to promoting economic development through large infrastructure and transportation projects.[26] Planning shifted from attempting to restrain harmful "spillovers" from private market activities in urban areas to promoting suburban economic development.[27] Models of economic efficiency were used in planning new towns, regional planning authorities were established to provide inexpensive and reliable resources to these areas, and an era of urban divestment and residential segregation took hold.[26,27]

By the latter half of the 20th century, the biomedical model of disease, which attributes morbidity and mortality to molecular-level pathogens brought about by individual lifestyles, behaviors, hereditary biology, or genetics,[25] was firmly entrenched as the dominant paradigm in epidemiology. Yet, the biomedical model was oriented toward explaining molecular-level pathogenesis rather than explaining the distribution of disease among populations or disease incidence or distribution at a societal level.[20,25] Urban planning underwent an analogous shift in its orientation toward environmental health by adopting the environmental impact assessment (EIA) process.

The EIA process, institutionalized after passage of the National Environmental Policy Act of 1969, ushered in the use of the environmental impact statement (EIS) for analyzing the ecological and human health effects of plans, projects, programs, and policies.[28] The EIA process is generally accompanied by a quantitative risk assessment in which human health effects are considered.[29] Risk assessment was institutionalized as part of the EIA process in almost all site-specific analyses of human health after the 1980 Supreme Court decision supporting the use of risk assessment in the regulation of benzene.[30]

Yet, both the EIS and quantitative risk assessment have been widely criticized as methods for assessing population health because they tend to overemphasize carcinogenesis at the expense of other chronic diseases,[31,32] treat all populations as similarly susceptible while ignoring the disproportionate hazardous exposures experienced by certain populations,[33] restrict analyses to quantitative data while minimizing or ignoring other kinds of information,[34] and limit the discourse and practice to experts, which can undermine the democratic character of the process by determining who is empowered to frame analyses and who will be excluded, deemed inarticulate, irrelevant, or incompetent.[29,34,35] Thus, wholesale adoption of practices such as EIS and risk assessment leads to planning becoming disconnected from environmental health.

CHALLENGES FACING THE UNION OF PLANNING AND PUBLIC HEALTH

By the late 20th century, the fields of planning and public health were largely disconnected both from their original mission of social betterment and from working collaboratively to address the health of urban populations.[8] There were some notable exceptions in each field, such as Alice Hamilton's community health work and Paul Davidoff's "advocacy planning" movement,[28,36] both of which advocated for interventions designed to improve the lot of the least well off. However, such movements were the exception rather than the rule in their respective fields. As discussed in the sections to follow, at least 4 significant challenges for reconnecting the fields emerge from this current disconnect.

Assessing the Health of Places and "Place-Making"

The first challenge facing the recoupling of planning and public health is how to pay increased attention to the public health effects of land use and places—often referred to as the built environment—while simultaneously expanding our definition of planning to include the political processes that produce these outcomes. For instance, the fields must develop new methods to understand the effects of the physical and social environment on human health by challenging the "geographic neutrality" assumptions of most environmental laws. Geographic neutrality is assumed when environmental regulations control *activities* that cause pollution (e.g., energy production, agriculture, transportation).[28] In such instances, the regulations of the Environmental Protection Agency (EPA) involve an industry-by-industry focus or an EIS assessment of a single facility; there is little regard for whether or not multiple industries or facil-

ities are clustered in particular communities.

Geographic neutrality is also assumed when environmental controls are placed on a specific hazardous agent or pollutant (e.g., lead, asbestos, radon), the environmental medium, or, less frequently, the route of exposure (e.g., drinking water, ambient air). In the case of each of these scenarios, cumulative exposures from multiple hazardous agents that have effects on communities are rarely considered.[15,35] The EPA has recognized the importance of geography in some regulatory programs, such as the state implementation plans designated under the Clean Air Act and watershed protection programs such as those managing the Great Lakes and Chesapeake Bay regions.[28] However, the overall regulatory strategy remains firmly rooted in the geographic neutrality fallacy.

While reconnecting planning and public health will require increased attention to the health effects of plans in geographic places, it will also demand that the field recognize its role in the politics of "place-making."[37,38] Planning must increasingly be understood as a profession that manages conflicts over political power and values that arise when, for instance, state or private-sector objectives clash with those of local communities. If planning is to be reconnected with public health, planning practice must be conceptualized as a set of outcomes (e.g., housing, transportation systems, urban designs) and processes that can (1) involve the use or abuse of power, (2) respond to or resist market forces, (3) work to empower certain groups and disempower others, and (4) promote multiparty consensual decision-making discourses or simply rationalize decisions already made.[39]

In other words, planning practice involves choices regarding which information is deemed relevant, what decisionmaking processes will be used, and when, or if, various publics will be involved in making the plan.[38] Reconnecting the fields will require increased attention to the politics of planning practice (i.e., in terms of shaping public agendas and attention), available evidence and norms of inquiry, inclusive or exclusive deliberations, and responses (or lack thereof) to bias, discrimination, inequality, and recalcitrance.[39]

Addressing Health Disparities

A second challenge in reconnecting the fields is developing a coordinated, multidisciplinary approach toward eliminating health disparities. A plethora of recent evidence suggests that disparities in health between people of color and Whites have not narrowed over time, are getting worse, and are increasingly linked to the physical and social environments that fall under the traditional domain of planning, such as housing, transportation, streetscapes, and community or social capital.[40–47]

For instance, Williams and Collins[42] noted that residential segregation is a fundamental cause of differences in health status between African Americans and Whites because it shapes the socioeconomic conditions faced by Blacks not only at the individual and household levels but also at the neighborhood and community levels; it also can contribute, in residential environments, to social and physical risks that adversely affect health. While African Americans have been effectively frozen out of suburbs by racial covenants, discriminatory mortgage practices, and racial steering since the 1950s, Whites have benefited from access to low-cost suburban homes, low interest rates on government-subsidized home mortgages, and publicly funded transportation projects linking their suburban homes to employment, recreation, and commercial centers.[48,49] Such housing and transportation policies promoted segregation and continue to preclude African Americans from enjoying the accumulation of wealth associated with the improved health of populations.[42]

Developing an Urban Health Agenda

In addition to addressing health disparities, reconnecting the fields will demand a clearly articulated strategy to improve the health of urban populations. Currently, the lack of an urban health agenda has allowed each field to downplay the significance of urban–suburban–rural health disparities.[1,2] Today's absence of an urban health agenda stems in part from national and state trends of divestment in cities; this divestment has subsequently led to a deemphasis on research about, and deflected resources away from, urban issues.[26,48,49] With urban poverty rates approximately twice as high as suburban poverty rates (16.4% vs 8.0% in 1999[50]), an urban health agenda must address socioeconomic position and other social determinants of health unique to urban areas.[2] Concentrated poverty is principally an urban and racial phenomenon, and people living in poor neighborhoods often face multiple simultaneous burdens that influence their health: poor schools, unemployment, psychosocial stress, discrimination, environmental exposures, and limited access to health care.[51]

Democratizing Practice

Finally, reconnecting planning and public health will require a new conception of participatory democracy to ensure that practices are accountable to communities that have historically been excluded from decisionmaking but face the greatest burden in terms of inequalities.[52] Research and decisionmaking in both planning and public health are often criticized for relying solely on professional knowledge at the expense of democratic participation.[52–56] Such critiques also claim that professional "knowledge elites" tend to view the "public" as largely ignorant of technical and scientific issues, reflecting a professional loss of confidence in the public's capacity to make sense of complex problems and disputes.

However, increasing evidence in the natural sciences, public health, and urban planning[53–56] reveals that expert assessments can miss important contextual information and need to be tempered by the experiences and knowledge offered by lay publics. Successfully reconnecting planning and public health will require the use of expert models, but it will also demand that these same models be recognized as contingent and fallible.[57] Democratizing practice in both fields demands that professional knowledge not be compartmentalized from practical experience, that lay knowledge be considered alongside expert judgments, and that the incomplete models of the technically literate not be mistaken for the sum total of reality.[30,35,58,59]

A RECONNECTING FRAMEWORK: ECOSOCIAL EPIDEMIOLOGY AND ENVIRONMENTAL JUSTICE

Reconnecting public health with planning will require the fields to embrace their physical and social dimensions, address health disparities burdening urban populations, and democratize research and decisionmaking practices. Although the task is daunting, insights from both fields might assist in the effort. In public health, social epidemiology, particularly ecosocial epidemiology, provides an interdisciplinary, multilevel perspective for understanding the health status of, and health disparities in, populations. In planning, environmental justice provides a framework for ensuring that decisionmaking processes and outcomes are democratic and fair.

Ecosocial epidemiology makes explicit the importance of an interdisciplinary understanding of how both biology and different forms of social organization influence the well-being of individuals and populations and explicitly investigates social determinants of population distributions of health, disease, and well-being.[13] Ecosocial epidemiology stresses a multidisciplinary population perspective that requires examination of how biological, sociological, economic, and psychological phenomena influence distributions of population health while incorporating a life-course perspective that considers the role of early and multiple pathogenic exposures that contribute to cumulative disadvantage.[13] Through its population and multilevel approach to health, ecosocial epidemiology recognizes that extra-individual socioeconomic factors closely related to the physical and social infrastructure of communities affect health above and beyond a combination of individual "risk" factors.[25]

A key concept in ecosocial epidemiology is *embodiment*, or how throughout our lives we literally incorporate, biologically, the material and social world in which we live.[13] The implication for reconnecting planning and public health is that better models are needed to understand how our biology does or does not reflect the physical, social, economic, and psychosocial environments in which we live, work, and play.[12,60–62] This insight suggests that reconnecting public health and planning will do more than simply add "biology" to "social" analyses; it will provide an understanding of health as a *continual and cumulative interplay* between exposure, susceptibility, and resistance, all of which occur at multiple levels (e.g., individual, neighborhood, national) and in multiple domains (e.g., home, work, school, community).[13,63]

Insights from environmental justice help confront the decisionmaking challenges facing the recoupling of planning and public health. A basic premise of environmental justice is that all people and communities have a right to live, work, and play in places and communities that are safe, healthy, and free of life-threatening conditions.[14–17] Claims of "environmental injustice" have highlighted that people of color and poor populations bear a disproportionate burden of hazardous exposures, experience less stringent enforcement of environmental regulations, have access to fewer environmental benefits such as parks, and have been routinely excluded from environmental decisionmaking.[15,54,64] These disproportionate hazardous exposures have also been shown to contribute to adverse health outcomes.[17] Environmental justice emphasizes *corrective justice* as well, or the notion that polluters should be punished and held responsible for cleanups and should compensate or repair communities damaged by historic pollution.[15]

Reconnecting the fields could benefit from an environmental justice decisionmaking framework that evaluates the democratic character of processes on the basis of their openness, inclusiveness, and fairness.[64] A democratic process, according to the environmental justice framework, demands that those being asked to bear an environmental or health burden "speak for themselves" in the design, analysis, and implementation stages of the process.[16] The environmental justice framework also recognizes that improved democratic decisionmaking processes require planners and others to work to ensure that disadvantaged groups receive the necessary legal, financial, and technical resources to allow their meaningful participation.[14]

A redistribution of material resources must accompany efforts to enhance participatory democracy. Material redistribution is necessary because, for instance, community networks and social capital—both of which are resources viewed as central to improving democracy and population health—cannot be built without supporting economic capital.[26,48] The conundrum is that redistributing economic growth *alone* will not guarantee the development of community networks and social organizations that are viewed as integral to determining how the benefits of economic growth and development are distributed.

Ultimately, resource redistribution requires a role for the federal government,[27,49] since local governments are always constrained by interjurisdictional competition—that is, interstate and intrastate (i.e., urban–suburban–rural) competition—in formulating redistributive policies.[50] Defining a new role for the federal government in planning and public health will be an essential part of democratizing the reconnection effort. The cruel irony is that while federal policies often helped create today's urban–suburban economic, social, and health disparities, policies at this same level are necessary to revitalize urban areas, address discriminatory programs, and help reconnect planning and public health.[26,50]

TOWARD HEALTHY AND JUST URBAN PLANNING

The successful reconnection of planning and public health will require the articulation of an explicit conceptual framework, and I have suggested one such paradigm here. Efforts to achieve this reconnection must confront a host of challenges, from redefining planning to addressing health disparities and formulating an urban health agenda. This task will not be easy. However, through an interdisciplinary approach that incorporates the multilevel, life-course, population health perspective suggested by ecosocial epidemiology and the procedural, distributive, and corrective justice principles advanced by environmental justice, a reconnection framework is possible. ■

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Ranking of Cities According to Public Health Criteria: Pitfalls and Opportunities

Ranking of Cities According to Public Health Criteria: Pitfalls and Opportunities

Popular magazines often rank cities in terms of various aspects of quality of life. Such ranking studies can motivate people to visit or relocate to a particular city or increase the frequency with which they engage in healthy behaviors.

With careful consideration of study design and data limitations, these efforts also can assist policymakers in identifying local public health issues. We discuss considerations in interpreting ranking studies that use environmental measures of a city population's public health related to physical activity, nutrition, and obesity.

Ranking studies such as those commonly publicized are constrained by statistical methodology issues and a lack of a scientific basis in regard to design. (*Am J Public Health.* 2004;94:546–549)

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FOR CENTURIES, PLACES TO live have been ranked on the basis of factors that contribute to quality of life, such as friendliness, wealth, crime, and health; in a 17th-century ranking, for example, areas with more plentiful game, heavier livestock, and lower mortality from Indian attacks were promoted as more "livable."[1] Further, recent examples are numerous, such as the *Places Rated Almanac*, a book that rates and ranks 354 metropolitan areas in terms of cost of living, job outlooks, transportation, education, health care, crime, the arts, recreation, and climate.[1] Popular magazines often publish rankings as well. For instance, *Natural Health* magazine ranked "America's Healthiest Cities" in 2001 (in terms of 37 criteria in the areas of amenities, physical health, environment, and happiness),[2] and *Men's Fitness* magazine has ranked "America's Fattest Cities" annually since 1999 (in terms of 16 categories including number of fitness centers and fast-food restaurants, measures of the natural environment and climate, and number of parks and recreational areas).[3] "Best places" are also proclaimed on the Internet, examples being *Money Magazine*'s "Best Places to Live" (factors considered are climate, crime, housing, education, economy, health, arts and leisure, and transportation)[4] and *Fast Forward*'s "Sperling's Best Places" (criteria are housing, cost of living, crime, education, economy, health, and climate).[5]

Ranking studies can garner considerable press coverage, can influence local public health and environmental policies, and motivate populations to work toward healthier lifestyles. In Philadelphia, after the release of "America's Fattest Cities 2000," the mayor implemented a new public health program in which he challenged the city's population to lose 76 tons of weight in 76 days.[6] In such ways, rankings of cities can effectively raise awareness of the factors influencing quality of life. In addition, local governments may use the findings to attract new residents, businesses, or tourists. For example, the Web site of the Visitors Association of Portland, Ore, touts the city as a great place to visit and live,[7] in part as a result of the high ranking it achieved in the "America's Fattest Cities 2001" article.

Nevertheless, controversies exist about whether ratings accurately reflect the "livability" of cities and the extent to which such reports can be misleading. A city's ranking varies depending on the quality of life criteria used in a particular study. Furthermore, these criteria typically include public health prevalence data and environmental measures with multiple sources of variability that are ignored when ranking studies are done. To date, there has not, to our knowledge, been a systematic analysis of ranking studies attempting to determine the extent to which their findings are methodologically sound. Editors of studies published in popular magazines and on the Internet are not bound by criteria imposed by peer-reviewed journals such as requirements regarding complete source citations and discussion of study limitations.

We provide an analysis designed to help policymakers interpret ranking studies that appear in the popular press. We discuss considerations in developing ranking studies that use environmental measures of a city population's public health related to physical activity, nutrition, and obesity in the hopes of stimulating greater interaction between policymakers and those who publish such studies.

THEORETICAL FRAMEWORK

Ranking studies can compare cities according to disease outcomes, behavior prevalence, correlates of health measures, or a combination of these indicators. Studies limited to behavior prevalence are the simplest, because national health surveys report prevalence rates at local levels.[8] More commonly, studies compare cities primarily on the basis of environmental correlates of health measures. Ideally, the scientific community would publish a list of environmental and behavioral measures derived from multilevel ecological modeling studies, and these measures would be weighted in regard to their relative importance in determining disease outcomes. Such measures would involve the use of timely, readily available sources of comparable data for relevant geographic units, and study designers would construct indices with appropriate weights based on scientific theory and empirical evidence. However, this ideal scenario does not yet exist.

Theoretical frameworks for environment–disease relationships are still in their infancy owing to the shift in public health paradigms in the mid-1990s to encompass multilevel causes of disease.[9] For example, current frameworks for obesity-related research distinguish between physical and social environments, behaviors, and disease outcomes.[10] No clear evidence exists as yet to quantify relationships between environment, physical activity/nutrition, obesity, and disease,[10] and ecological-level studies are lacking that include a wide range of readily available indicators such as those used in city rankings. However, a recent study[11] revealed that several economic nutrition indicators (e.g., number of full-service and fast-food restaurants[12] and average cost of a meal prepared at home[13]) exhibited significant associations with obesity prevalence rates. "Walkability" (e.g., presence of sidewalks, enjoyable scenery)[14] and number of locations available for exercise (e.g., walking trails, parks, indoor gyms)[15] also have been correlated with physical activity. Future scientific research will provide additional empirical evidence on which city rankings can be based.

Meanwhile, ranking studies are popular and will continue to be published in part as a result of the plausibility of relationships between environmental factors, behavior, and health outcomes. Editors are left to do their best with limited resources, and many appropriately choose a combination of available statistics on health behaviors and environmental factors. Cities are complex systems with multiple causal pathways between environment, population dynamics, behavior, and health conditions. Ranking studies may oversimplify these complex systems. Furthermore, combining environmental, behavioral, and disease outcome measures without clarifying the differences between them may confuse and mislead readers. Public health policymakers can benefit from ranking studies while recognizing that their findings may need to be reinterpreted once more sound hierarchical linear models are developed.

DATA SOURCES

Other than lack of scientific basis, limitations posed by available data are often the greatest weakness associated with a ranking study. The primary issue is the paucity of comparable, timely data collected at the city level through the use of stable and reliable procedures and representative samples of the population of interest.

Geographical Level of Analysis

In comparisons of cities, information is required at the city level or the level of the metropolitan statistical area (MSA), which comprises the central city and the suburbs and surrounding counties economically tied to the central city. Most cities and MSAs, however, do not routinely collect all of the data required to create the desired indices for ranking studies. Until recently, state-level averages had been reported for most of the available health data collected nationally, because these data were derived from probability samples and required a minimum sample size to achieve acceptable statistical confidence.[8] Yet, state-level averages may not adequately represent the health situation in any of the state's cities, and this is particularly the case in states covering large geographic areas.

Another complication is that some environmental indicators are measured for central cities but not the entire MSA. Faced with this dilemma, editors may resort to combining city-, MSA-, and state-level data or may impute data from the mean without documenting their decisions in published reports. Policymakers are left to investigate the data sources themselves.

Timeliness

Similarly, ranking studies based on data from different years or outdated sources should be interpreted with caution. Older sources can be misleading when the phenomenon under consideration is changing at a rapid pace. For example, published summary MSA-level health statistics on obesity trends occurring in the 1990s were based on data gathered during the early part of the decade, after which obesity prevalence rates increased rapidly. Studies involving the use of older data for indicators that tend to be more stable over time, such as acres of parks per 10 000 people, do not face the same problem.

Consistency With Current Standards

Measures used to rank cities may not be calculated and defined according to currently accepted standards and should be interpreted accordingly. For example, public health definitions of "overweight," "obesity,"[16] and "physical activity"[17] have been revised in recent years, but statistics including both current and past definitions are widely used. Furthermore, beginning in 2001, household, transportation, and leisure-time physical activity were measured in national health surveys to concur with current public health recommendations; previous measures reported only leisure-time exercise, one type of all possible physical activity.[8]

Stability, Reliability, and Selection Bias

Data gathered from some of the sources used to rank cities, including federal sources (e.g., the US Bureau of the Census [http://www.census.gov], the US Environmental Protection Agency [http://www.epa.gov], and the National Oceanic and Atmospheric Association [http://www.noaa.gov]), are collected according to scientific methods and are well documented. The Centers for Disease Control and Prevention's Behavioral Risk Factor Surveillance System[8] also is considered a reliable data source that is particularly

well documented and based on a scientifically selected sample.

Other sources are more problematic and should be interpreted with caution. For example, in-house surveys are often derived from convenience samples that may systematically exclude certain groups by focusing on a nonrepresentative sample within a city or MSA. Likewise, only about half of all businesses are listed in business address databases, whereas the Census of Retail Trade[12] uses a representative sample.

CALCULATION METHODS

In ranking studies, data are often transformed to normal distributions, although statistical ranking methods were developed to analyze data that are not normally distributed. Rankings based on normal distributions identify the best and worst cities but misrepresent the relative positions of the many cities in between, the reason being that most of the scores cluster near the mean. The highest and lowest scores are easy to identify, but the remaining values could be statistically indistinguishable. Meaningful measures of statistical uncertainty regarding city scores are difficult to derive in city ranking studies, because such scores are sums or averages of point estimates for differing constructs. Therefore, the numerical scores on which these rankings are based will be more useful to policymakers.

Rankings may be based on indices in which each construct represents a combination of related measures (e.g., a climate index calculated via data on temperature, precipitation, snowfall, and sunshine) that are given numerical scores. Policymakers can group cities with similar numerical scores by, for example, assigning letter grades (A+, A, A–, etc.), a method that has intuitive appeal and commands attention in a society accustomed to being graded or evaluated. Indices and letter grades reduce the effects of imperfect data by giving individual measures low weight in the overall score and by creating categorical measures. Despite its appeal, however, the technique of creating indices via simple averaging assumes that all data sources are of equal quality and appropriateness, which may not be true.

MESSAGE COMMUNICATION

Ranking studies published in magazines and books and on Web sites often attract media and government attention and are taken seriously, regardless of their designs and limitations. When cities refer to their rankings in public health programs,[6] on the Internet, and in local chamber of commerce publications,[7] it would be useful to include a brief discussion of study limitations along with rankings and scores. As demonstrated here, all ranking studies involve limitations that affect data interpretation. Topics to be touched on include, but are not limited to, lack of a scientific basis for linking study indicators and lack of effective measures focusing on important aspects of the environment, physical activity, and nutrition; the latter issue is a consequence of the difficulty of measuring these factors, poor data quality (e.g., inconsistent geographic level of analysis, outdated statistics, poor coverage), or both.

Another limitation that should be noted is the use of multiple geographic units of analysis for statistical data; if possible, such data should be reanalyzed in hierarchical linear models as they become available. Ranking studies can be valuable tools for the public health field and for local governments if methodological limitations are assessed and taken into consideration.

CONCLUSIONS

Rankings of cities can play an important role in raising awareness of public health issues and illuminating what policymakers can do to address these issues. In addition, they provide policymakers and the public more information about the health challenges they face and allow progress to be monitored over time on a range of indicators. If their city's comparisons with higher ranked cities suggest an environmental issue that might be health related (e.g., poor air quality, higher number of fast food restaurants, lower number of recreational areas), public health policymakers can use published ranking studies to justify a local investigation into the problem. Such data also can be used to raise awareness about lifestyle choices among residents; to market "healthy cities" and "active cities" as attractive places to visit, live, and do business; and to hold government and the private sector accountable for doing what is necessary to keep residents healthy. ■

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Contributors

S.A. Ham planned and executed the study, reviewed data sources, contributed to the conceptualization and writing of the article, and revised the article. S. Levin and A.I. Zlot reviewed data sources and contributed to the conceptualization and writing of the article. R.R. Andrews reviewed data sources. R. Miles contributed to the conceptualization of the argument and to the writing of the article.

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Cost Analysis of the Built Environment: The Case of Bike and Pedestrian Trials in Lincoln, Neb

We estimated the annual cost of bike and pedestrian trails in Lincoln, Neb, using construction and maintenance costs provided by the Department of Parks and Recreation of Nebraska. We obtained the number of users of 5 trails from a 1998 census report. The annual construction cost of each trail was calculated by using 3%, 5%, and 10% discount rates for a period of useful life of 10, 30, and 50 years. The average cost per mile and per user was calculated.

Trail length averaged 3.6 miles (range = 1.6–4.6 miles). Annual cost in 2002 dollars ranged from $25 762 to $248 479 (mean = $124 927; median = $171 064). The cost per mile ranged from $5735 to $54 017 (mean = $35 355; median = $37 994). The annual cost per user was $235 (range = $83–$592), whereas per capita annual medical cost of inactivity was $622.

Construction of trails fits a wide range of budgets and may be a viable health amenity for most communities. To increase trail cost-effectiveness, efforts to decrease cost and increase the number of users should be considered. (*Am J Public Health*. 2004;94:549–553)

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ENVIRONMENTAL FACTORS affect the health of all people in both developed and developing countries. Because of industrialization and the consequent environmental pollution, environmental changes in the past several decades have led to new challenges for public health.

Many studies have documented links between the environment and human health.[1–7] For example, household amenities and other environmental exposures have been linked to children's health problems such as cancer and asthma,[1–3] and environmental pollution has been linked to high morbidity and mortality in the general population.[4–7] In recent years, the worldwide increase of obesity has prompted discussions of environmental interventions such as increasing the availability of healthy snacks and building environments that are amenable to physical activity as possible effective means to prevent and control obesity and other costly chronic diseases.[8–11]

Because of suburbanization, the transportation systems in the United States have been designed for automobile use. Although automobile-oriented transportation is a necessity for economic development and people's daily lives, the modern transportation system may pose a hazardous environment for public health. Recently, 42% of American adults expressed a great deal of concern about urban sprawl and loss of open space,[12] which can create an environment of physical inactivity, a major risk factor for several chronic diseases and obesity.[13–25] One study has demonstrated the association between the built environment and physical activity by showing the effects of urban environment on walking behavior.[22] Another study showed that environmental features such as neighborhood design appeared to affect whether residents walked to work.[24]

Pedestrian-oriented urban environments may promote physical activity,[22,23] and a combination of urban design, land use patterns, and transportation systems that promote walking and bicycling may help create more livable communities.[26–28] Lieberman recently suggested that proper design and land use patterns and policies can increase public transit use as well as walking and bicycling.[26] Efforts to increase the pedestrian-oriented environment through mixed-use development, street connectivity, and good community design can enhance both the feasibility and attractiveness of walking and bicycling.

Participation in regular physical activity depends in part on the availability and proximity to such resources as community recreation facilities and walking and bicycling trails, so building such environments holds much promise in health promotion.[29–31] Studies on the economic costs of the built environment must proceed, because they may provide critical information to policymakers regarding resource allocations. We conducted a cost analysis of building bike and pedestrian trails to provide some of this information.

DATA SOURCE

We obtained the costs of construction and maintenance of 5 bike and pedestrian trails in Lincoln, Neb, from the Department of Parks and Recreation of Nebraska, and the number of trail

TABLE 1—Number of Users and Costs of Construction and Maintenance of Trails

Trail Description	Date Built	Trail Length (mi)	Number of Users	Construction Cost (2002 $)	Maintenance Cost (2002 $)
1. Concrete, 2 bridges	1995	4.6	1638	2 366 927	26 183
2. Limestone chip, 0 bridges	1997	4.5	232	90 982	14 980
3. Concrete, 3 bridges	1996	4.1	1878	1 621 994	11 828
4. Concrete, 0 bridges	1989	3.1	238	979 600	17 196
5. Concrete, 1 bridge	1999	1.6	Not available	598 863	7 040

users from the Great Plains Trails Network (Table 1).[32] In addition to the cost and number of users, information about surface type, date built, and length was also obtained for each trail. The construction cost was a 1-time investment on building the trails. Ideally, the cost would be divided into labor cost and capital cost, but we were able to obtain only the total cost without further details. Maintenance cost was based on annual upkeep expenditures. The construction and maintenance costs were adjusted to 2002 dollars using a 5% inflation rate based on the date each trail was built.

The number of users was determined by the Lincoln Recreational Trails Census, which was conducted on Sunday, July 12, 1998 (Table 1). The census began at 7:00 AM and concluded at 9:00 PM the same day. Census volunteers, working in 2-hour shifts, counted cyclists, runners, walkers, skaters, and miscellaneous users (such as persons with skateboards, wheelchairs, and horses.) Ideally, this number would be adjusted according to weather and date of the week to determine a representative number of users, but this information was unavailable.

The census report used this information for the number of users in 1998, which is comparable to the number of users in other years. We used this number as a snapshot of the use of trails for a conservative estimate of cost-effectiveness of trails. This value is conservative because the number of users during a year should be more than that during a day. Additionally, we varied the number of trail users listed in the census report by increasing or decreasing by 50% to calculate a range for the cost per user.

DATA ANALYSIS

The construction cost is a large 1-time capital investment, so it is necessary to spread the investment over the useful life in years by determining an annual value of the capital investment. To do this, we calculated an annuity factor that takes into account time preference (r, discount rate) and length of useful life (t, number of useful years). The annuity (A) rate [$A(t,r)$] for time t years at r discount rate was derived by using $A(t,r)=1/r[1-1/(1+r)^t]$. The annual equivalent cost (AEC) of trail construction was calculated by $AEC=C\times A(t,r)$, where C is the 1-time capital expenditure.

The time preference needs to be incorporated into the cost estimate even with zero inflation because people prefer paying later and getting benefits earlier.[33] The discount rate, r, is a quantitative measure of time preference.

Different discount rates have been used in empirical studies; the normal range is 3% to 10%. We used 3%, 5%, and 10% as discount rates for cost estimation to cover a wide range of time preference. The higher the discount rate, the more people value current dollars. In the case of trail investment, a higher discount rate was associated with a higher AEC for construction. For the number of years of useful life of the trails, we used 10, 30, and 50 years to cover a wide range of situations. The longer the useful life of the trails, the lower the construction AEC.

For the case of a 5% discount rate and 30 years of useful life, we calculated annual cost per mile for construction, maintenance, and a total (construction and maintenance costs combined). In addition, for the total cost, we calculated the annual average cost per user as a measure of cost-effectiveness. We also analyzed the composition of cost (construction versus maintenance) and types of users.

RESULTS

The 5 trails were built between 1989 and 1999. Their average length was 3.6 miles (range=1.6–4.6 miles) (Table 1). Four trails had a concrete surface, and 1 had a limestone-chip surface. On the day of census, the number of users ranged from 232 persons on the limestone-chip trail (trail 2) to 1878 persons on the most heavily used concrete trail (trail 3, a concrete surface with 3 bridges). The total construction cost ranged from $90 982 ($20 218 per mile) for trail 2, the limestone-chip trail, to $2 366 927 ($514 549 per mile) for trail 1, a concrete surface with 2 bridges. The annual maintenance cost ranged from $7040 ($4400 per mile) for trail 5, a concrete surface with 1 bridge, to $26 183 ($5692 per mile) for trail 1.

The AEC for construction of the 5 trails under all the scenarios of time preference and period of useful years is useful information for those deciding on resource allocations (Table 2). Among all the scenarios, the highest cost ($542 021) was incurred for the 4.6-mile concrete trail 1 with its 2 bridges under the assumption of a 10% discount rate and 10 years of useful life. The lowest cost ($4513) was incurred for the 4.5-mile limestone-chip trail under the assumption of a 3% discount rate and 50 years of useful life.

Using a 5% discount rate and 30 years of useful life, we found that the annual average cost per mile for trail 4 (concrete with no bridges) was $45 505, and the cost for trail 3 (concrete with 3 bridges) was $37 994 per mile. The annual total cost per user for trail 4 was $592, whereas the cost per user for trail 3 was $83 (Table 3). The cost ranged from $55 to $1185 per user.

For cost composition, 85% of the total cost was construction cost (range=29%–91%) under the assumption of a 5% discount rate and 30 years of useful life (Figure 1). Because only 1 trail was made of limestone chips and it cost much less than the con-

TABLE 2—Annual Equivalent Construction Cost of Trails (2002 $)

Trail	Years of Useful Life	Annual Equivalent Construction Cost		
		3% Discount	5% Discount	10% Discount
1	Concrete, 2 bridges			
	10	390 437	458 009	542 021
	30	169 920	216 653	353 298
	50	129 442	182 434	335 912
2	Limestone chip, 0 bridges			
	10	13 613	15 969	18 898
	30	5924	7554	12 318
	50	4513	6361	11 712
3	Concrete, 3 bridges			
	10	254 816	298 916	353 746
	30	110 897	141 397	230 577
	50	84 479	119 064	219 231
4	Concrete, 0 bridges			
	10	216 524	254 023	300 619
	30	94 242	120 162	195 948
	50	71 792	101 183	186 306
5	Concrete, 1 bridge			
	10	81 271	95 337	112 824
	30	35 370	45 097	73 540
	50	26 944	37 975	69 922

crete trails, the average cost composition was very close to the cost compositions of the concrete trails. The composition was similar for all the concrete trails. The majority of users were bicyclists (73%), followed by runners/walkers (20%) (Figure 2). Because of data limitations, we did not know how the types of users varied with the type of trails.

DISCUSSION

When communities decide to build a bike or pedestrian trail, financial budgeting should be based on trail surface type, length, and other features such as bridges. Both construction and maintenance costs should be considered, because although the construction cost of the limestone-chip trail was much lower than that of the concrete surface trails, the maintenance cost was not necessarily lower.

The construction AEC varied with the discount rate and number of years of useful life. Specifically, the cost increased as the discount rate increased, and decreased as the number of years of useful life increased. The figures suggest that the cost of building a trail can vary greatly and that trails can be developed to meet a variety of budgets.

As an example of the variances, the construction AEC (at a 5% discount rate and 30 years of useful life) of building a concrete trail with 1 bridge was 6 times as expensive as building a limestone-chip trail. The total annual cost (including both maintenance and construction costs) for a concrete trail was 5 times more than that for the limestone-chip trail.

Although the cost of building and maintaining a limestone-chip trail was lower than the cost for a concrete trail, the limestone-chip trail may not be the most cost-effective strategy if the number of users is taken into account. The cost per user for the limestone-chip trail ($111) was more than for a concrete trail with 3 bridges ($83). Thus, both the total cost of trails and the number of users should be considered when decisions about trails are made. On average across all the trails, the cost per user was $235. This figure is much lower than the economic benefit of physical activity. A conservative estimate of direct medical cost savings from physical activity was $330 per person in 1987.[34] Using a 5% inflation rate, this savings is about $622 in 2002, nearly 3 times as high as the trail cost. Therefore, developing trails may be a cost-effective means to promote physical activity.

The fact that there were more users on concrete trails than on the limestone-chip trail (except trail 4, built in 1989, on which there were similar number of users) may suggest that concrete trails have more desirable features and are more convenient for cycling. Because most of these users were bicyclists (unfortunately, we did not have detailed information about user type on the limestone-chip trail versus the concrete trails), building trails to fit the needs of cyclists may substantially increase the cost-effectiveness and net health benefits of trails.

Several limitations should be noted to interpret the findings properly. (1) We cannot analyze total construction costs such as labor and material in more detail because of data limitations. This lack of information restricted our ability to examine how other major factors (e.g., material cost, land value, funding sources) influenced the total cost and how to minimize project costs. (2) The number of trails is small, and each trail was built in a different year. Technology and funding sources change over time, so the cost of each trail may not be fully comparable. Therefore, the average cost across trails may be somewhat inaccurate, although

TABLE 3—Annual Total Cost (2002 $) of Trails Using a 5% Discount Rate and 30 Years of Useful Life

Description	Trail	Construction Cost		Maintenance Cost		Total Cost		
		Total	Per Mile	Total	Per Mile	Total	Per Mile	Per User[a]
Concrete, 2 bridges	1	216 653	47 098	31 826	6919	248 479	54 017	152 (101, 303)
Limestone chip, 0 bridges	2	7554	1679	18 208	4046	25 762	5725	111 (74, 222)
Concrete, 3 bridges	3	141 397	34 487	14 377	3507	155 774	37 994	83 (55, 166)
Concrete, 0 bridges	4	120 162	38 761	20 902	6743	141 064	45 505	592 (395, 1185)
Concrete, 1 bridge	5	45 097	28 186	8557	5348	53 654	33 534	Not available
Average		106 173	30 042	18 774	5312	124 947	35 355	235 (156, 469)

[a]Figures in parentheses are the cost per user calculated by increasing or decreasing the number of users listed in the census report by 50%.

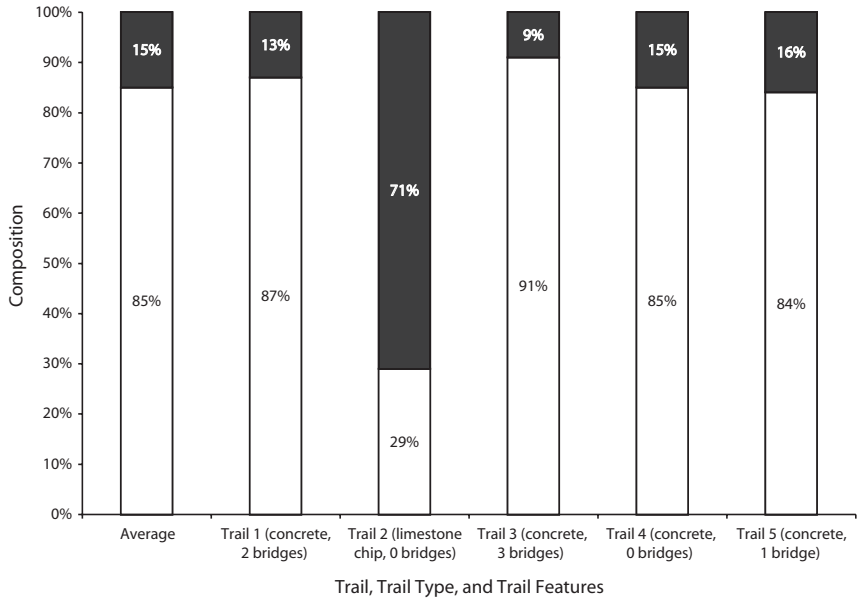

FIGURE 1—Cost composition (black = maintenance; white = construction) of 5 trails in Lincoln, Neb, using a 5% discount rate and 30 years of useful life.

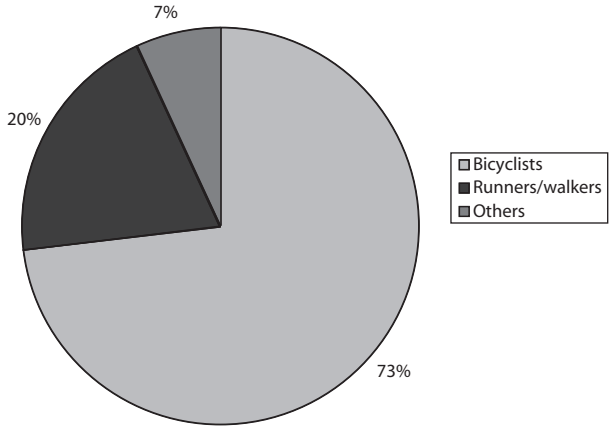

FIGURE 2—Trail user type in Lincoln, Neb.

we adjusted all the costs to 2002 dollars. (3) The census was conducted on a Sunday in summer; we cannot claim that it was representative of the number and type of users on an average day. The lack of information means we cannot adjust the number of users according to weather, day of the week, purpose of using trails, and other factors. For example, many users may commute to work. The number of users on a Sunday would not capture this. Therefore, if the majority of trail users used trails for commuting, the cost per user may be severely underestimated. (4) Information on various qualitative aspects of trails was lacking. We have information only on the surface type, length, and number of bridges for each trail. Other attributes such as safety and convenient access to trails also affect the cost of construction and maintenance. Because of these information gaps, the cost estimates according to trail length and surface type should be interpreted cautiously. (5) The trails analyzed in this study were built as a part of community design or development planning, not as a public health intervention project. Factors such as increased property value or a more attractive environment may have been major determinants of building trails rather than health promotion. These added values may have significantly biased our cost estimates because we analyzed only financial cost and did not consider the effects of other community features such as location and land value. (6) We analyzed only the cost data of trails in a local community area. The results should not be generalized to other areas because household income levels, natural characteristics, and local politics influence the development of trails.

Despite these limitations, we derived a framework of cost analysis based on the available data, and several strengths should be noted. (1) We derived the costs of construction and maintenance for each trail and adjusted all costs to 2002 dollars, which should increase the comparability of the cost across the 5 trails. (2) We incorporated different discount rates and number of useful years into the analysis, and therefore covered a wide range of possible cost values for trails. (3) We used trail length and number of users on each trail to derive the cost per mile and cost per user. The cost per mile is useful for community planners who are deciding to build trails based on financial feasibility. The cost per user is useful to demonstrate the usability of trails as a measure of cost-effectiveness.

For future economic research on the built environment, detailed cost information should be collected systematically. This information will make analysis much more useful in identifying factors influencing the cost of trails. In addition, effectiveness of trails in changing physical activity behaviors should be incorporated into the economic analysis. To do this, data such as consumer willingness to pay for trail construction and use if trails are built should be collected. When information about trail effectiveness is available, cost-effectiveness of trails on health promotion can be soundly evaluated.

CONCLUSIONS

Trails can fit a wide range of budgets depending on the needs and resources of the community. Our research demonstrates the need to increase cost-effectiveness efforts by researching ways to decrease the cost of building trails and to increase the number of users of trails. We have also outlined specific information that should be gathered to more completely explore the construction and use of trails in the future. Policymakers and community developers may use the cost information to determine their needs and the cost-effectiveness and feasibility of built environments in their community. ■

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All authors helped plan the study. G. Wang and B. Scudder-Soucie obtained the data. G. Wang also performed data analysis and wrote the article. C. A. Macera, T. Schmid, D. Buchner, G. Heath, and M. Pratt revised the article. Pratt also supervised the project.

Human Participant Protection

No protocol approval was needed for this study.

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The Epidemic of Pediatric Traffic Injuries in South Florida: A Review of the Problem and Initial Results of a Prospective Surveillance Strategy

S. Morad Hameed, MD, MPH, Charles A. Popkin BA, Stephen M. Cohn, MD, E. William Johnson, MPH, and the Miami Pediatric Traffic Injury Task Force

This study identified specific regional risk factors for the high rate of pediatric pedestrian trauma in Florida. Of the 29 cases studied prospectively, 3 (10%) occurred near ice cream trucks and 13 (45%) involved "dart-outs"; mean hospital charges were $24 478 ±$43 939. Recommendations included an engineering change for a dangerous intersection, and a population-based recommendation was to equip ice cream trucks with extending stop signs. (*Am J Public Health*. 2004;94:554–556)

Approximately 30 000 children are struck by cars each year in the United States.[1] Florida is home to 4 of the 5 most dangerous cities for pedestrians in this country, and the mortality rate after pedestrian trauma (3.9 per 100 000) is higher than the national average (2.3 per 100 000).[2] Pediatric pedestrian injuries are frequently encountered at our trauma referral center in Miami, Florida.

Efforts to reduce the rates of pedestrian injury previously centered primarily on education programs and met with little success.[3] This may be partly due to an absence of data from prospective studies. Broad demographic trends and socioeconomic and geographic risk factors identified in the literature are often either region-specific or too generalized to be useful in the creation of practical, site-specific prevention strategies.

The purpose of this study was to outline the distribution, determinants, and effects of pediatric pedestrian trauma (PPT) in our community. We hypothesized that careful data collection would uncover community-specific PPT risk factors and suggest avenues for prevention and resource allocation.

METHODS

This study, set at the Jackson Memorial Hospital/University of Miami Ryder Trauma Center (the sole trauma center for approximately 3 million Miami-Dade County residents), was performed in 2 phases.

Phase 1—Retrospective Review

Medical records of pediatric pedestrians (younger than age 16 years) who presented to our institution between January 1994 and December 1996 were reviewed. Demographic parameters were defined and analyzed to assess the impact of PPT in our communities.

Phase 2—Prospective Data Collection

Recommendations from a multidisciplinary task force (including local medical, police, and government agencies) were incorporated into a design of a 4-month prospective cohort study. Detailed information from hospital records, crash scene visits, patients, families, and police interviews was compiled on consecutive cases of PPT treated at our center (July 1 through October 31, 2000). Injury scene conditions were systematically assessed and especially emphasized in the analysis.

RESULTS

Retrospective Review

A total of 235 PPT cases were evaluated. Grade school children were most often (53%) injured, usually in the vicinity of schools. Boys predominated, and African American children accounted for 60% of the cases. High mean hospital charges ($16 553) resulted from the high incidence rates (32%) of head injuries.

Prospective Data Collection

Population, scene, environmental, and cost issues were explored in 29 consecutive cases of PPT. Many children (69%) were from single-parent homes. Although Miami is ethnically diverse, a disproportionate number of PPT events occurred in predominantly African American neighborhoods. Thirty-five percent of children came from homes where at least 1 parent had some postsecondary education.

At most sites, intervals between marked intersections were long, allowing vehicle acceleration and predisposing random pedestrian crossing patterns. Some intersections (Figure 1) were observed to be poorly regulated by misplaced traffic lights and were a source of long-standing community apprehension. Mechanisms involving obstruction of view ("dart-outs")[4] were common (46%), although most PPT incidents (64%) occurred in clear daylight conditions. Site visits provided insight into situational dynamics. For example, 3 events (10%) resulted from traffic disruption by ice cream trucks.

Hospital charges ranged from $336 to $172 283, and at the time of the site visit (25 ±13 days post-PPT), 44% of children had not returned to school.

DISCUSSION

Previous studies (Table 1) have characterized region-specific risk factors for PPT, which may not be completely generalizable to Miami, with its unique cultural and geographic milieu. As indicated by our review, South Florida is fertile ground for a comprehensive PPT prevention strategy. Groups such as the North Miami Crash Traffic Safety Team and the Safe Kids prevention programs have taken an active role in pedestrian education, but to date, prevention initiatives have not been designed with specific references to objectively measured risk factors.

Available information sources, including police reports and hospital records, lacked sufficient detail to clarify the causes of PPT. The second phase of this study was designed to provide useful information for development of directed multidisciplinary prevention policy.

All 29 cases studied during our surveillance period had implications for the design of high-risk or population-based prevention strategies. Miami's uninhibited westward growth has resulted in the creation of communities with high volumes of rapid commuter traffic and long residential streets without sidewalks. Situations such as that summarized in Figure 1 will require innova-

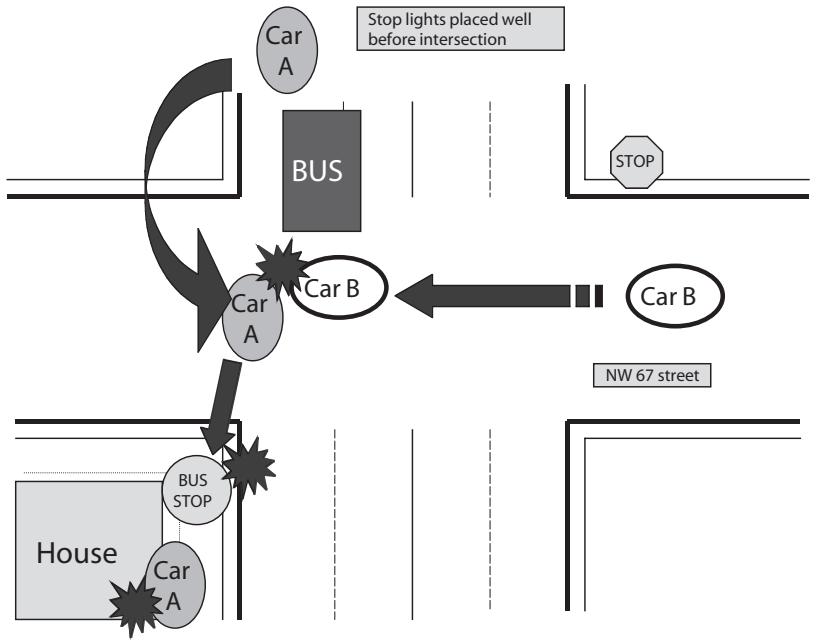

FIGURE 1—Problematic intersection at 2nd Ave and NW 67th St, Miami, Fla. During this site visit, numerous bystanders approached the investigators to find out when the city was planning to modify the control of this dangerous intersection. The visit was prompted by the injuries of a 5-year-old African American boy who had been holding his mother's hand at the bus stop on the corner. The driver of car A, after waiting behind a bus ahead of the traffic lights, swerved onto the shoulder area at a high rate of speed and entered the intersection unaware that the light had changed. The ensuing events are depicted. After the collision with car B, the driver of car A lost control, striking the boy, his sister, and their mother. The car then struck a fence at the corner and proceeded toward the wall of a nearby house with the child still trapped underneath.

tive engineering approaches to eliminate high-risk scenarios. Other high-risk situations, such as those involving ice cream trucks, will require legislation mandating the use of safety measures such as extending stop signs on these vehicles to help reduce the impact of the frequently observed dart-outs. Conscientious regulation of school bus access and pickup and drop-off practices would reduce the incidence of injuries observed during school hours.

Although this study was performed without external funding, a grant from the Florida Department of Transportation will allow us to address some of the limitations of this initial surveillance. Information will be collected over a school year along with an economic evaluation, and more objective scene measurements will be made. We hope to delineate a cost-effective surveillance-based prevention plan that reduces the incidence of children struck by motor vehicles. ■

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TABLE 1—A Summary of the Pediatric Pedestrian Traffic Injury Literature

Authors	Type of Study	No. of Injuries	Location	Main Conclusion
Rivara and Barber, 1985[5]	Retrospective	210	Memphis, Tenn	Traffic engineering modifications are practical solution
Brison et al., 1988[4]	Retrospective	71	Washington State	Prevention strategies must be age-specific
Mueller et al., 1990[6]	Case–control	98	King County, Washington	Busy streets, multifamily homes are strong risk factors
Braddock et al., 1991[7]	Retrospective	198	Hartford, Conn	High-density areas are problematic
Roberts et al., 1995[3]	Case–control	190	Auckland, New Zealand	High traffic volume in urban areas should be reduced
Agran et al., 1996[8]	Case–control	39	Orange County, California	Parked cars and reduced speed would decrease injuries
Calhoun et al., 1998[9]	Retrospective	91	Jefferson County, Alabama	Manageable environmental risk factors were identified; education should be targeted toward grade school children
Durkin et al., 1999[10]	Retrospective review of newly implemented intervention	Incidence study of all injuries (n = 981) in Harlem, New York, NY	Harlem, New York, NY	Community interventions (play areas, education) may be helpful in preventing injury
Miami Pediatric Traffic Injury Task Force, 2001[a]	Retrospective review	235	Miami–Dade County, Florida	Ongoing surveillance is required for continued development of focused prevention strategies
	Prospective surveillance	29		

[a]Unpublished data.

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Contributors

S.M. Hameed contributed to the study design, data collection, data analysis, and manuscript preparation. C.A. Popkin contributed to the data collection and manuscript preparation. S.M. Cohn contributed to the study design, data analysis, and manuscript preparation. W.M. Johnson contributed to the study design and data collection.

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The members of the Miami Pediatric Traffic Injury Task Force are Frank Pernas, BA, David Henderson, AICP, Mimi Sutherland, RN, MS, Margaret Brown, MSN, J. Esteban Varela, MD, Dimeter Hristov, MD, Kimberly Schwartz, MD, Officer Luis Taborda, BA, Julie Jackowski, RN, Tracy Byrd, BA, Gilian Hotz, PhD, Lewis Saye, BA, and Jose Guerrier, MD.

Human Participant Protection

Institutional review board approval was obtained from the University of Miami before this study was initiated.

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Florida's Motorcycle Helmet Law Repeal and Fatality Rates

Andreas Muller, PhD

On July 1 2000, the State of Florida exempted adult motorcyclist and moped riders from wearing helmets provided they have medical insurance of $10 000. Monthly time series of motorcycle occupant deaths are examined from 1/1994 to 12/2001. The interrupted time series analysis estimates a 48.6% increase in motorcycle occupant deaths the year after the law change. The impact estimate reduces to 38.2% and 21.3% when trends in travel miles and motorcycle registrations are controlled. Our findings suggest that the law's age exemption should be revoked. (*Am J Public Health*. 2004;94:556–558)

Between 1997 and 2001, nationwide motorcycle rider fatalities increased by 50% while motorcycle registrations increased by 31%.[1,2] The rise in death rates may be related to the concurrent weakening of motorcycle helmet laws in Arkansas, Texas, Kentucky, Louisiana, and Florida. In comparing rates the year before (1996) and the year after (1998) the helmet law change, Preusser et al.[3] found a 21% increase in motorcyclist deaths in Arkansas and a 30% increase in Texas. This analysis tries to determine the effect of weakening Florida's motorcycle helmet law.

Since July 1, 2000, Florida statutes have required motorcycle riders younger than 21 years of age to wear helmets. Adult motorcycle and moped riders are exempted provided they have insurance for motorcycle accident injuries with minimum medical benefit coverage of $10 000.[4] Before July 1, 2000, Florida had a helmet law that required all riders to wear safety helmets.

The State of Florida is of interest because it accounts for 9% of all motorcycle rider deaths in the United States. Coinciding with the helmet law change, the number of Florida's motorcycle registrations increased substantially. The number of motorcycle deaths in the state was high enough to permit a monthly time series analysis. The Florida motorcycle helmet law change has not been evaluated statewide.[5]

METHODS

Data

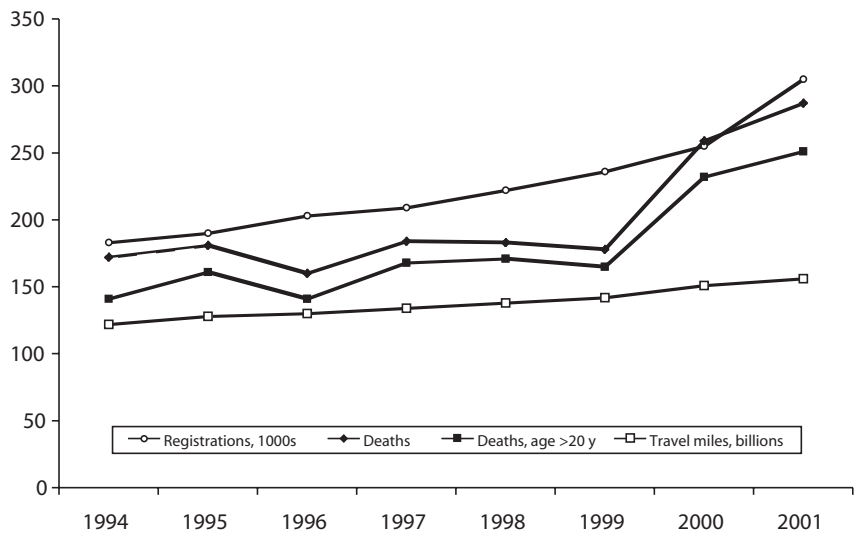
Florida's monthly motorcycle rider deaths for the period January 1994 to December 2001 were analyzed. Motorcycle rider deaths included operators or passengers of motorcycles, mopeds, minibikes, motorized three-wheelers, off-road, other, and unknown types of motorcycles. All-terrain vehicles were excluded. The definition is intentionally comprehensive to allow for comparisons over time. It matches the National Highway Traffic Safety Administration's definition of motorcycle rider death.

The time series data were obtained from the Fatal Accident Reporting System database.[1] Yearly issues of *Highway Statistics* provided motorcycle registration and travel miles for the period 1996 to 2001, and earlier years came from the 1995 summary volume.[2] To obtain a more realistic representation of the Florida motorcycle registration and travel trends, annual data were converted into a monthly series by 12-month centered moving averages. The smoothing operation removes 12 observations and reduces the sample size to 84 months, July 1994 to June 2001.

The following series were analyzed: motorcycle rider deaths, motorcycle rider deaths per billion travel miles, and motorcycle rider deaths per 10 000 registered motorcycles. Restricting the analysis to rider deaths of adult motorcyclists (>20 years) generates 3 additional series.

Time Series Models

The method of analysis was interrupted time series analysis using Box–Jenkins models.[6,7] To approximate normal distributions closely, all time series were converted into natural log units. A step function for an abrupt, permanent impact models Florida's motorcycle helmet law change beginning in July 2000. The residuals of all time series follow random process properties (data available from the author upon request).



Source. Fatality Analysis Reporting System[1] and Federal Highway Administration.[2]

FIGURE 1—Florida motorcycle registrations, motorcycle rider deaths, and travel miles for all motor vehicles, 1994 to 2001.

RESULTS

Figure 1 presents annual trends in motorcyclist deaths, motorcycle registrations, and travel miles in Florida. During the year 2000, Florida motorcyclist deaths increased by 81 (45.5%), motorcycle registrations by 19 494 (8.1%), and travel miles by 9 billion (6.3%). The upward trends in motorcycle registrations and travel miles are noteworthy.

Figure 2 presents impact estimates based on the analyses of 6 monthly time series. The estimates indicate that the change in Florida's helmet law increased motorcycle rider deaths. The impact on all motorcycle rider deaths is strongest, 48.6%. Controlling for travel miles reduces the estimate to 38.2%, and correcting for the motorcycle registration trend reduces the estimate to only 21.3%. Restricting the analysis to adults reduces the previous estimates only slightly.

DISCUSSION

The analysis suggests that exempting adult motorcyclists from wearing helmets increased the number of motorcyclist fatalities in Florida. However, the effect of the law change depends on which "exposure" measures are controlled. Since travel increased in Florida, the impact estimates based on the absolute number of deaths are probably overstated. Conversely, the estimates that correct for the motorcycle registration trend may understate the law's impact. The large increase (19.6%) in Florida motorcycle registrations in 2001 (Figure 1) suggests that changing the law may have stimulated interest in motorcycling and increased motorcycle registrations. Substantial increases in motorcycle registrations also occurred in Arkansas (47%), Louisiana (13%), and Texas (12%) the year after their helmet laws were weakened. The extent of such a law-induced effect is currently unknown. On the basis of registration and miles traveled, it is estimated that between 46 and 82 additional motorcyclists died in Florida the year after the helmet law changed.

In 2001, only 53% of Florida underage motorcyclists who died in crashes wore motorcycle helmets; for adults the figure was 39%.[1] That is, the legal age restriction is barely effective and amounts to a de facto helmet law repeal.

CONCLUSION

This study finds that the current age-restricted version of Florida's motorcycle helmet law resulted in more motorcyclist deaths

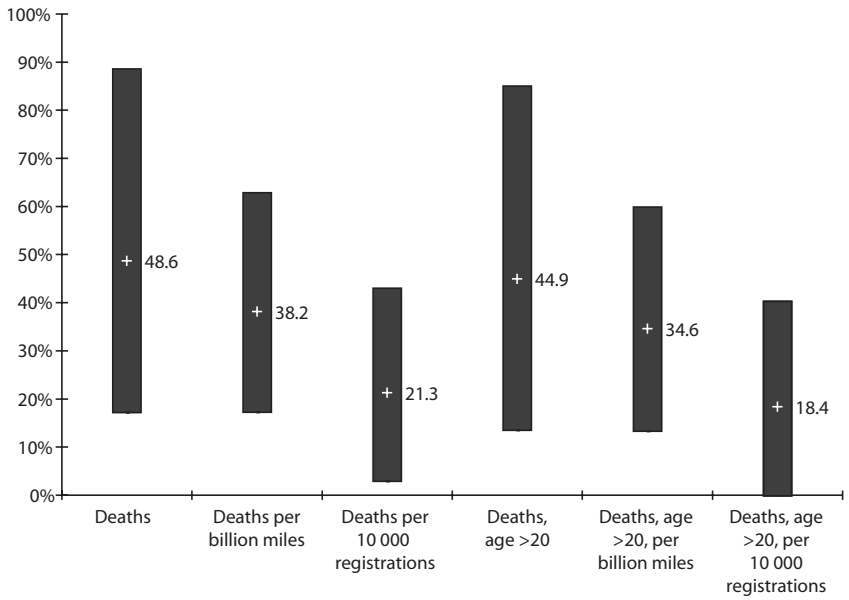


FIGURE 2—Estimated impact of the change in Florida's motorcycle helmet law by series, with 95% confidence limits.

even after adjustment for concurrent increases in motorcycle registrations or miles traveled. Exempting adult motorcycle riders from wearing motorcycle helmets is counterproductive for motorcyclists' health and unnecessarily increases insurance and medical care expenses. ■

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Human Participation Protection

No protocol approval was needed because no individuals are identified by the analysis.

Acknowledgments

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Femur Fractures in Infants and Young Children

Desmond Brown, MD, and Elliott Fisher, MD, MPH

Using an administrative database, we determined rates of femur fracture by year of age for children younger than 6 years and by month of age. The highest rate of femur fracture was in children younger than 1 year and in 2-year-olds; the greatest number of fractures occurred during the third month of life. While femur fractures in children are often due to accidental injury, the reasons for the peak in the first year and the subsequent decline are not clear. (*Am J Public Health*. 2004;94:558–560)

The incidence of femur fractures in children is believed to have 2 peaks, one at the age of 2 to 3 years and another during adolescence.[1] This view is based, however, on older studies from Scandinavia[2–4] and a more recent study from Maryland[5] and may not reflect the experience of the US population. Previous studies have also categorized children by year of age, which may be insufficiently precise for the infant or young child in whom rapid changes in size, physical ability, and behavior may affect the risk of fracture.

Although most femur fractures in children are caused by falls or other unintentional injuries, abuse is considered more likely in the child aged younger than 1 year or not yet able to walk. In this brief, we focus on this youngest group, reporting data on hospital discharges for femur fractures from a national database in which children were categorized by age in months.

METHODS

The 1997 Kids' Inpatient Database[6] contains 1.9 million records of hospital discharges for children aged 18 or younger, representing nearly a third of the estimated 6.7 million pediatric discharges during that year. Using *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*) codes for fracture of the proximal femur, femoral shaft, and distal femur (diagnosis codes 820–821.39), we identified 3308 records of children under the age of 6 discharged from a hospital with a diagnosis of femur fracture. Fractures occurring during childbirth were excluded.

Using population weights provided with the database, we calculated national estimates for the number of femur fractures in each 1-year age group. We determined fracture incidence rates by dividing the number of fractures by the estimated number of children in each age group, using population estimates for 1997 from the US Bureau of the Census. To examine the relationship between age and femur fractures more closely, we identified 2753 records for which the age in months was available. Because we lacked the population denominator to determine rates of fracture, we report the counts for this subset of patients.

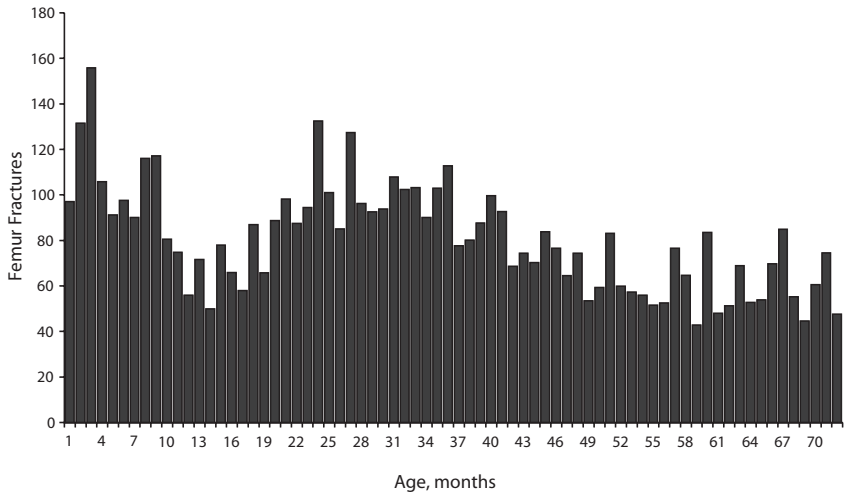
RESULTS

The rate of femur fracture was highest during the first year of life and in 2-year olds (Table 1). One-year-olds were less likely to sustain a fractured femur than those aged younger than 1 year. While the ratio of boys to girls was nearly equal in those aged younger than 1 year, all older age groups had more boys.

In children for whom the age in months was known, the greatest number of fractures occurred during the third month of life (Figure 1). There were slightly fewer fractures in children aged 4 to 11 months, and fewer still in children aged 12 to 20 months. After the first peak during infancy, there was a second peak in children aged 20 to 40 months. In children older than 40 months but younger than 72 months, the number of fractures was lower and relatively constant.

DISCUSSION

Previous studies of femur fractures in childhood have identified a peak in incidence at



Source. Kids' Inpatient Database, 1997.[6]

FIGURE 1—Estimated number of femur fractures among children in the United States, by month of age.

TABLE 1—US Population Estimates for Femur Fractures in Children, by Year of Age

	No. of Femur Fractures			Femur Fractures/100 000 (95% CI)		
Age, y	Male	Female	Total	Male	Female	Total
<1	849	763	1612	44 (39, 49)	41 (37, 46)	43 (39, 46)
1	759	485	1244	40 (35, 44)	26 (22, 30)	33 (30, 36)
2	1126	449	1575	58 (53, 63)	24 (21, 28)	42 (38, 45)
3	889	349	1238	45 (40, 50)	19 (15, 22)	32 (29, 35)
4	680	307	987	34 (29, 38)	16 (13, 19)	25 (23, 27)
5	622	342	964	30 (26, 34)	17 (14, 20)	24 (22, 26)

Note. CI = confidence interval.
Source. Kids' Inpatient Database, 1997.[6]

age 2 to 3 years. By contrast, femur fractures in children younger than 1 year of age are thought to be less common and, when they occur, to be highly suggestive of abuse.[7,8] We found that femur fractures were as common in children younger than 1 year as in those aged 2 years and older, with the greatest number of fractures occurring during the third month of life. There are few plausible explanations for a femur fracture in this age group other than intentional injury. These data suggest that an infant has as great a chance of sustaining a femur fracture from physical abuse as an older child does from all causes.

The reason for the rise in incidence at age 2 to 3 years, and the subsequent fall, is less clear. Although most children are walking by age 15 months, femur fractures were infrequent at this age. The 2- to 3-year-old may be at increased risk of injury owing to changes in gait,[9] increased mobility, greater climbing ability, and exposure to vehicular traffic. The decline in femur fractures after age 3 may be due to improvements in gait and judgment, as well as to increased bone strength. Although child abuse is thought to be a less common cause for femur fracture in children who are walking,[10] there are widely varying estimates of its occurrence, reflecting the difficulty of establishing the diagnosis of abuse with certainty.[11]

Our study, based on an administrative database, lacks the clinical detail of a case series. The sample size is large, however, and the coding of femur fractures and age are likely to be accurate.[12] The rate of femur fracture in children younger than 2 years of age was 38.0 per 100 000; this is greater than the rate of 25.5 per 100 000 reported by Hinton and colleagues for femoral shaft fractures in this age group in Maryland.[5] We included fractures of the proximal and distal femur, which may contribute to the higher rate we report.

We cannot determine how often fractures were due to abuse or neglect, but child abuse is thought to be common in children younger than 1 year old with femur fractures.[7,8] Other possible causes include heritable disorders of connective tissue such as osteogenesis imperfecta[13] and motor vehicle accidents. Short falls, as occur when a child rolls off a bed or table, are unlikely to cause a femur fracture in an infant.[14,15] The equal number of boys and girls younger than age 1, and the predominance of boys among those older than 1 year, may signify a shift from intentional to accidental injury.

Although not as specific for abuse as the metaphyseal corner fracture or rib fracture, a single long-bone fracture may be the most common type of fracture due to abuse.[16] Abuse should be suspected if caretakers provide inconsistent or implausible accounts of how a femur fracture occurred, or if there are additional unexplained injuries. A skeletal survey may provide evidence of occult injuries and may support a diagnosis of abuse. Efforts to prevent femur fractures in children should focus on preventing physical abuse in infants and accidental injury in the 2- and 3-year-old children at greatest risk. ■

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Contributors

D. Brown conceived the study, performed the analyses, and wrote the brief. E. Fisher assisted in the design of the study and statistical analyses and contributed to the design of the tables and the writing of the brief.

Human Participant Protection

Institutional review board approval was not required for this study.

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Asthma, Wheezing, and Allergies in Russian Schoolchildren in Relation to New Surface Materials in the Home

Jouni J. K. Jaakkola, MD, DSc, PhD, Helen Parise, PhD, Victor Kislitsin, MSc, Natalia I. Lebedeva, MD, DSc, and John D. Spengler, PhD

In a cross-sectional study of 5951 Russian 8–12-year-old schoolchildren, risks of current asthma, wheezing, and allergy were related to recent renovation and the installation of materials with potential chemical emissions. New linoleum flooring, synthetic carpeting, particleboard, wall coverings, and furniture and recent painting were determinants of 1 or several of these 3 health outcomes. These findings warrant further attention to the type of materials used in interior design. (*Am J Public Health*. 2004;94:560–562)

The Soviet era has been followed by increased activity in construction and renovation of housing in the Russian Federation, as well as an introduction of new building technology and new materials used in interior design, furniture, and textiles. Two recent studies indicated that exposure to plastic flooring and wall materials may increase the risk of respiratory conditions in children.[1,2] As part of a cross-sectional study of air pollution and respiratory health in Russia in 1996 to 1997,[3,4] we tested a hypothesis that the risks of children's asthma and allergic diseases are related to recent renovation, especially newly installed synthetic surface materials, furniture, and painting.

METHODS

The study population included 5951 children in second to fifth grade (8–12 years old) in 8 Russian cities in the Sverdlovsk Oblast region and the city of Cherepovets in the Upper Volga Oblast.[3] The participation rate in schools varied from 96% to 98%. The questionnaire, modified from previous European and North American questionnaires for the Russian conditions,[5,6] inquired about the child's personal characteristics, health information, and socioeconomic factors. Local elementary schoolteachers were trained to conduct the interviews, and parents and guardians were invited to meetings after the school day was finished. After signing an informed consent form, a parent completed the questionnaire.

The current study focused on asthma, wheezing, and allergy. *Current asthma* was defined as a history of doctor-diagnosed asthma and symptoms, signs, or medication of asthma during the past 12 months. *Current wheezing* was defined as wheezing during the past 12 months. *Any allergy* was defined as any history of doctor-diagnosed allergy or parental-reported hay fever or pollinosis.

Exposure assessment was based on the following question: "Have you conducted any of the following renovations in your home within the past 12 months or earlier?" The choices were installation of linoleum floor, painting, particleboard, new furniture, synthetic carpet, wall covering, and suspended ceiling.

We used the odds ratio (OR) as a measure of effect and logistic regression analysis to adjust for age, gender, preterm birth, low birthweight, parental atopy, maternal smoking during pregnancy, exposure to environmental tobacco smoke at home (at ages 0–1 years, ages 2–6 years, and currently), and mother's and father's education.

RESULTS

Of the children, 1.5% had current asthma, 13.4% had current wheezing, and 33.2% had an allergy. Table 1 shows the occurrence of the potential sources of emissions.

The risks of current wheezing (adjusted OR=1.36; 95% confidence interval [CI]=1.00, 1.86) and allergy (adjusted OR=1.31; 95% CI=1.05, 1.65) were significantly related to the installation of linoleum flooring during the past 12 months (Table 2). The corresponding risk estimates were slightly lower when focusing on exposure earlier than 12 months ago. There was a general pattern of positive as-

TABLE 1—New Surface Materials, Furniture, and Recent Painting in Russian Homes

Emission Source	Past 12 Mo, %	Earlier, %
New linoleum flooring	9.9	34.0
New synthetic carpet	6.5	22.0
New wall covering	35.9	38.2
Recent painting	32.9	39.9
New particleboard	4.7	20.7
New furniture	12.9	39.3

sociation between installation of synthetic carpet during the past 12 months and the 3 outcomes (adjusted ORs from 1.39 to 1.84), although for asthma, the association was not statistically significant. The effect estimates for the past 12 months were greater than those for earlier installation. The adjusted odds ratios for new wall covering during the past 12 months (from 1.20 to 1.25) and earlier (from 1.12 to 1.22) were lower. The odds ratios for recent painting were elevated for current wheezing and allergy. The odds ratios for new particleboard were substantially elevated for all the studied relations except for recent installation of particleboard and the risk of current asthma. The adjusted odds ratios for current asthma (1.33; 95% CI=0.57, 3.06), current wheezing (1.32; 95% CI=0.99, 1.77), and any allergy (1.43; 95% CI=1.16, 1.75) were increased in relation to new furniture during the past 12 months but weaker in relation to new furniture installed earlier.

DISCUSSION

Consistent with our hypothesis, the risks of current asthma and wheezing and allergic diseases were related to installation of materials with potential chemical emissions.

Two previous studies provided evidence of the role of polyvinyl chlorides and other plastic surface materials.[1,2] We asked about installation of linoleum flooring to identify polyvinyl chloride materials. In line with the Norwegian study, the risks of asthma and wheezing in the current study were related to installation of linoleum floors. Linoleum in colloquial Russian represents a large heterogeneous group of synthetic floor materials with an unknown proportion of polyvinyl chlorides; therefore, the exposure parameter was rather nonspecific.

Substantial evidence indicates that in the working-age population (13–65 y), painters have an increased risk for developing asthma and asthma-related and other respiratory symptoms.[7–9] Paints used in home renovation are likely to emit similar chemical substances as the paints used by professional painters, although exposure levels in occupational settings are much higher than in the home environments after renovation. In the homes, the exposure levels are the highest during and shortly after painting, but low levels of exposure may remain for several months. Wooden furniture and also painted or varnished and new furniture are likely to emit chemical substances. Also, synthetic carpets, furniture, painting, and wall covering used as exposure indicators constitute a heterogeneous group of potential emitting materials.

The current findings warrant further attention to the type of materials used in interior design. ■

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TABLE 2—Adjusted Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Current Asthma, Current Wheezing, and Presence of Any Allergy According to Recent Installation of Surface Materials and Furniture

	Current Asthma		Current Wheezing		Any Allergy	
	Crude OR	Adjusted OR[a] (95% CI)	Crude OR	Adjusted OR[a] (95% CI)	Crude OR	Adjusted OR[a] (95% CI)
New linoleum flooring						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	1.44	1.13 (0.44, 2.04)	1.36	1.36 (1.00, 1.86)	1.47	1.31 (1.05, 1.65)
Yes, earlier	1.64	1.39 (0.69, 2.77)	1.31	1.25 (0.99, 1.59)	1.41	1.22 (1.04, 1.45)
New synthetic carpet						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	2.70	1.84 (0.73, 4.65)	1.81	1.70 (1.21, 2.40)	1.56	1.39 (1.07, 1.80)
Yes, earlier	1.60	1.26 (0.58, 2.72)	1.29	1.24 (0.96, 1.61)	1.44	1.22 (1.02, 1.46)
New wall covering						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	1.60	1.25 (0.63, 2.51)	1.28	1.20 (0.95, 1.52)	1.40	1.25 (1.06, 1.48)
Yes, earlier	1.61	1.22 (0.62, 2.43)	1.19	1.12 (0.88, 1.41)	1.32	1.16 (0.99, 1.37
Recent painting						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	1.45	1.09 (0.53, 2.22)	1.34	1.25 (0.99, 1.58)	1.40	1.25 (1.05, 1.47)
Yes, earlier	1.58	1.29 (0.65, 2.53)	1.19	1.11 (0.88, 1.40)	1.29	1.16 (0.99, 1.37)
New particleboard						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	1.10	0.60 (0.13, 2.77)	1.44	1.33 (0.89, 2.00)	1.72	1.49 (1.12, 2.00)
Yes, earlier	1.78	1.38 (0.65, 2.94)	1.50	1.39 (1.07, 1.80)	1.48	1.28 (1.07, 1.54)
New furniture						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes, past 12 mo	1.65	1.33 (0.57, 3.06)	1.32	1.32 (0.99, 1.77)	1.59	1.43 (1.16, 1.75)
Yes, earlier	1.57	1.27 (0.64, 2.51)	1.22	1.16 (0.92, 1.47)	1.39	1.24 (1.05, 1.46)

[a]From logistic regression, adjusted for age, gender, preterm birth, low birthweight, parental atopy, maternal smoking during pregnancy, exposure to environmental tobacco smoke at home (at ages 0–1 years, ages 2–6 years, and currently), and mother's and father's education.

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Contributors

J.J.K. Jaakkola conceived the hypothesis, participated in the planning of the study and statistical analyses and in the interpretation of the results, and wrote the brief. H. Parise conducted the statistical analyses and contributed to the interpretation of the results. V. Kislitsin and N.I. Lebedeva participated in the planning of the study and contributed to the writing of the brief. J.D. Spengler participated in planning of the study and statistical analyses and in the interpretation of the results and provided input on the writing of the brief.

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Human Participant Protection

Parents were informed that participation was voluntary. Questionnaires were completed by parents and returned in sealed envelopes. No personal identifiers were used in our data files and all questionnaires have been destroyed. Data were not collected from children.

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The Impact of the SARS Epidemic on the Utilization of Medical Services: SARS and the Fear of SARS

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Using interrupted time-series analysis and National Health Insurance data between January 2000 and August 2003, this study assessed the impacts of the severe acute respiratory syndrome (SARS) epidemic on medical service utilization in Taiwan. At the peak of the SARS epidemic, significant reductions in ambulatory care (23.9%), inpatient care (35.2%), and dental care (16.7%) were observed. People's fears of SARS appear to have had strong impacts on access to care. Adverse health outcomes resulting from accessibility barriers posed by the fear of SARS should not be overlooked. (*Am J Public Health*. 2004;94:562–564)

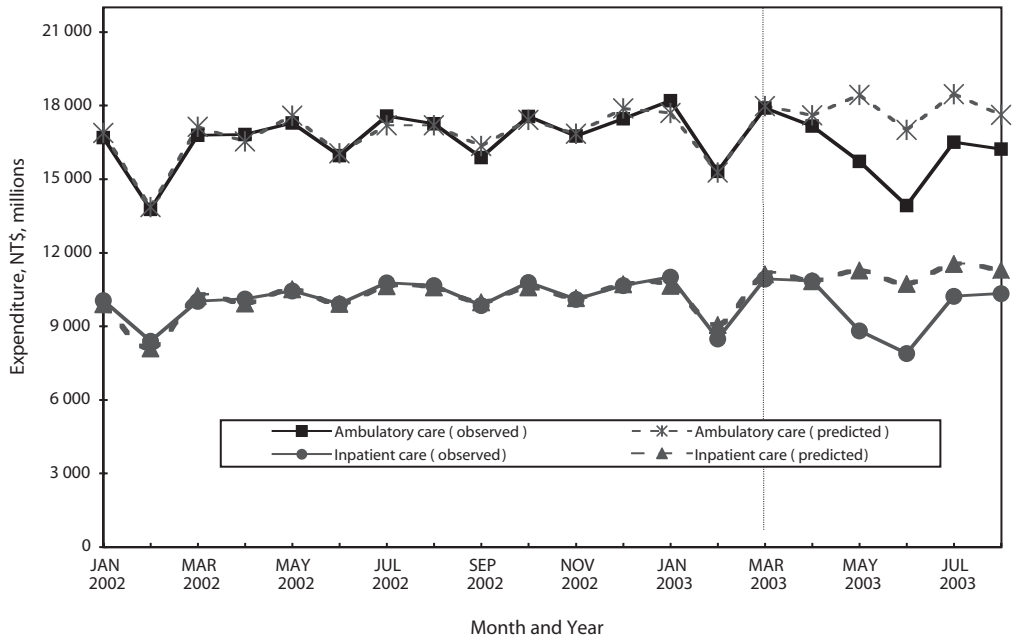
Since the outbreak of severe acute respiratory syndrome (SARS), its etiology, transmission routes, treatments, and outcomes have received much research attention.[1–5] SARS has low mortality and morbidity; however, the health consequences of the SARS epidemic are not limited to people who have been infected.[6] The potentially serious impact of SARS on people's accessibility to medical services should not be overlooked.[7–10] However, no study has systematically evaluated the impact of the fear of SARS on the general population.

People's fears of SARS were mainly caused by its novel, rapid nosocomial transmission, and the vulnerability of hospitals and health care workers. Many wondered whether the fears of SARS among patients and health care workers alike deterred people from seeking care or providers from offering services. Therefore, a critical challenge is to determine how public health agencies should respond to utilization changes and possible accessibility barriers to the general population created by the SARS epidemic. In this study, we aimed to assess how people's fears of SARS influenced their utilization patterns of medical services in Taiwan.

METHODS

The SARS epidemic in Taiwan started in mid-March 2003 and lasted for almost 4 months. The epidemic was effectively contained during the initial SARS period (March 14 to April 21, 2003).[11] However, multiple clusters of hospital outbreaks among patients and health care workers initially struck at the end of April and extended to May and June, dramatically exacerbating the epidemic. As a result, overwhelming fears of SARS spread over the entire island along with the SARS epidemic. The situation persisted until July 5, when Taiwan was officially removed from the World Health Organization's list of SARS-affected countries.[11,12]

We retrieved all claims made to the National Health Insurance program between January 1, 2000, and August 31, 2003, including inpatient care, Western medicine ambulatory care, Chinese medicine services, and dental services. An interrupted time-series design was used. Trends for different types of services were analyzed separately to determine whether utilization changes were specific to certain services. The time-series autoregressive-moving average (ARIMA) analysis[13] was applied to determine whether the SARS epidemic was significantly associated with changes in medical service utilization rates. Relative differences between observed and ARIMA-predicted values were expressed in percentages. All analyses were performed



Note. NT$ = New Taiwan dollars.

FIGURE 1—Observed and predicted expenditures for ambulatory and inpatient care in the preepidemic, epidemic, and postepidemic periods, January 2002 through August 2003. The date of the initial outbreak is marked with a vertical line.

using SAS for Windows, Version 8.2 (SAS Institute Inc, Cary, NC) and Stata 8.0 (Stata Corp, College Station, Tex).

RESULTS

Figure 1 compares the observed trends in expenditures for ambulatory and inpatient care in Taiwan with the predicted trends estimated by the ARIMA model that assumes the absence of the SARS epidemic. During the epidemic, the figure shows significant reductions in observed expenditures compared with those expected. The general patterns for both ambulatory and inpatient services were quite similar and corresponded to each transition period of the SARS epidemic. Correspondingly, virtually no impact was observed before the first hospital cluster in late April, when the epidemic was effectively contained. A significant reduction was observed in May and continued to expand significantly in June, when the fears of SARS grew after the expansion of the epidemic to all of Taiwan. Finally, the expenditures increased gradually in July and August after the SARS epidemic was over. Compared with ambulatory care, inpatient care experienced larger reductions in expenditure at the peak period and rebounded to levels closer to usual values toward the end of the epidemic. This suggests that the SARS epidemic had a stronger influence on inpatient services than on ambulatory services.

Although the responses of medical service expenditures were similar to those of medical service utilization, reductions in utilization were relatively larger. Inpatient services experienced the largest reduction (35.2%), followed by dental services (23.9%) and Western medicine ambulatory services (16.7%) at the peak of the SARS epidemic (Table 1). On the other hand, unlike other types of medical services, Chinese medicine services experienced an increase in utilization (1.8%) during the SARS epidemic. One plausible explanation may be that Chinese medicine services served as a substitute for Western medicine ambulatory services.

DISCUSSION

Over the study period, we observed significant utilization reductions at the peak of the SARS epidemic. Overall, this short-term impact on utilization reductions translated into an approximate $18.8 billion new Taiwan dollars decrease (approximately 6% of the annual National Health Insurance expenditure) in health care expenditure during the SARS epidemic from April 2003 through August 2003. The results strongly suggest that the fears of SARS significantly influenced people's care-seeking behavior and that this fear seriously compromised their accessibility to quality care.

Although all the international attention is focused on the direct causes of SARS, serious health consequences resulting from people's fears of SARS should not be overlooked. The results presented here could provide public health agencies with a more complete picture of overall health impacts of the SARS epidemic, so that when SARS re-emerges, it can guide public health officials to prevent avoidable health consequences because of the fears people have regarding SARS. ■

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TABLE 1—Observed and Predicted Monthly Medical Expenditures and Utilizations, by Type of Medical Service: Taiwan, January 2003 through August 2003

	Pre-SARS and Initial SARS Period			Peak SARS Period				Post-SARS Period			
	Jan–Mar, Avg $/No.	April, $/No.	Difference, %	May, $/No.	Difference, %	June, $/No.	Difference, %	July, $/No.	Difference, %	August, $/No.	Difference, %
					Expenditures						
Inpatient care											
Observed	10 143	10 845	0.1	8808	−21.9	7888	−26.5	10 228	−11.3	10 334	−8.3
Expected	10 269	10 839		11 278		10 729		11 533		11 272	
Ambulatory care											
Observed	17 131	17 161	−2.5	15 726	−14.7	13 922	−18.1	16 503	−10.6	16 226	−7.9
Expected	16 977	17 601		18 427		17 006		18 467		17 623	
Dental care											
Observed	2297	2339	−9.1	1991	−23.5	2083	−16.3	2538	−7.2	2495	−2.8
Expected	2323	2572		2603		2488		2736		2568	
Chinese medicine											
Observed	1343	1458	0.8	1418	−8.3	1331	−1.4	1454	−4.7	1450	2.0
Expected	1362	1446		1546		1351		1525		1421	
					Utilization						
Inpatient care											
Observed	241	245	−6.3	180	−32.4	167	−35.2	227	−16.8	228	−15.7
Expected	245	262		266		258		272		271	
Ambulatory care											
Observed	23 117	22 525	1.3	18 665	−22.2	15 744	−23.9	18 668	−18.1	19 043	−11.1
Expected	22 323	22 245		23 979		20 692		22 783		21 418	
Dental care											
Observed	2040	2055	−9.7	1717	−25.3	1833	−16.7	2269	−5.6	2231	−1.4
Expected	2073	2275		2299		2201		2404		2262	
Chinese medicine											
Observed	2454	2654	5.4	2558	−5.8	2359	1.8	2575	−1.4	2606	7.2
Expected	2430	2519		2715		2319		2612		2431	

Note. Avg = monthly average; $ = new Taiwan dollars in millions; No. = number of visits per admissions in thousands; Difference = [(observed value − predicted value)/predicted value] 100. The official exchange rate for 2003 published by the Central Bank of China is 1 US$ = 34.24 New Taiwan $. Available at: http://www.cbc.gov.tw. Accessed February 26, 2004.

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Contributors

H.J. Chang planned the study and supervised all aspects of its implementation. N. Huang assisted with the study and led the writing. C.H. Lee synthesized analyses and contributed to the writing of the article. C.J. Hsieh assisted with the data management and the study. Y.J. Hsu assisted with the study and analyses. Y.J. Chou planned the study, completed the statistical analysis, and supervised the study implementation. All authors helped to conceptualize ideas, interpret findings, and review drafts of the brief.

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Human Participant Protection

No protocol approval was needed for this study.

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Health Effects Associated With Recreational Coastal Water Use: Urban Versus Rural California

Ryan H. Dwight, PhD, Dean B. Baker, MD, MPH, Jan C. Semenza, PhD, MPH, and Betty H. Olson, PhD

We compared rates of reported health symptoms among surfers in urban North Orange County (NOC) and rural Santa Cruz County (SCC), California, during 2 winters (1998 and 1999) to determine whether symptoms were associated with exposure to urban runoff. NOC participants reported almost twice as many symptoms as SCC participants during the 1998 winter. In both study years, risk increased across symptom categories by an average of 10% for each 2.5 hours of weekly water exposure. Our findings suggest that discharging untreated urban runoff onto public beaches can pose health risks. (*Am J Public Health*. 2004;94:565–567)

Coastal waters along public beaches can be polluted by urban runoff, which is water that carries non–point-source pollution via surface waterways to the ocean.[1] A variety of illnesses have been associated with exposure to polluted recreational coastal waters.[2–4] In this study, which involved 2 geographic watersheds differing in terms of urbanization, we measured reported health effects on individuals with high levels of exposure to coastal waters.

METHODS

North Orange County (NOC), California, was the "urban" site because its watershed is 1 of the most developed areas in the world and generates highly polluted runoff waters.[5–9] We selected Santa Cruz County (SCC), California, as the comparison "rural" site because of its coastal water quality indicators (Figure 1) and watershed characteristics.

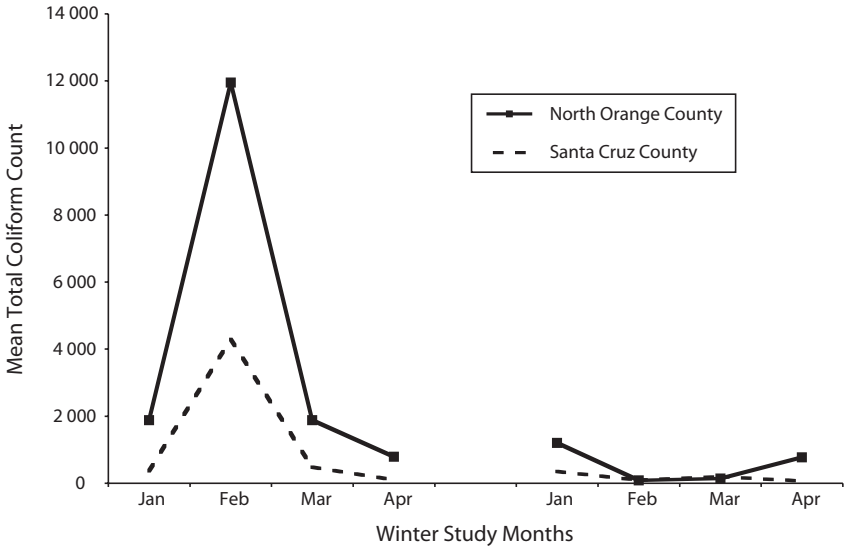

FIGURE 1—Mean monthly total coliform counts (per 100 mL) during 1998 El Niño and 1999 La Niña winters: North Orange County and Santz Cruz County coastal waters (data provided by Orange County Health Care Agency and Santa Cruz Health Agency).

We conducted 2 cross-sectional surveys of surfers from NOC and SCC, 1 in April 1998 and 1 in April 1999, and gathered data on reported health symptoms (e.g., vomiting, diarrhea, sore throat) experienced during the previous 3 months. The 1998 El Niño winter had led to record high precipitation throughout California, while the 1999 La Niña winter had led to record low precipitation in NOC. NOC had lower total rainfall than SCC in both years, yet the former had higher coastal water coliform (a water quality measure of pollution) levels (Figure 1).

Surfers were selected as the study population because of their regular exposure to coastal waters. Interviewers at surfing beaches recruited participants by approaching all individuals who had wetsuits and surfboards. Those who reported surfing at least once a week and were 18 years or older were eligible to be interviewed. Demographic information was collected, as well as information on symptoms experienced during the previous 3-month period. Participants also reported the amount of time they were exposed to coastal waters.

Multiple reports of 1 symptom were combined, allowing only 1 symptom report per participant, equivalent to a 3-month period prevalence. Logistic regression analysis was used to estimate adjusted odds ratios (ORs) comparing symptom reporting rates between the 2 counties, stratified by year. The final logistic model included the following variables: county, water exposure, gender, age, occupation, educational level, annual income, political outlook, and level of concern about water quality. The latter 2 variables were included to control for potential reporting bias associated with perspectives about the potential health effects of environmental pollution and water quality.

RESULTS

In 1998, investigators interviewed 479 participants in NOC and 374 in SCC. In 1999, investigators interviewed 662 participants in NOC and 358 in SCC. At each site, response rates were above 80% in both 1998 and 1999. The mean age of the participants was 30 years, and 93% were male.

The urban versus rural analysis showed that NOC participants reported almost twice

TABLE 1—Odds Ratios for Reported Symptoms: North Orange County and Santa Cruz County, 1998 and 1999

	1998 (El Niño Winter)		1999 (La Niña Winter)	
	OR	95% CI	OR	95% CI
Any symptom	1.85	1.36, 2.52	1.17	0.87, 1.57
SRD	1.29	0.91, 1.82	0.75	0.53, 1.05
HCGI	2.32	1.27, 4.25	0.97	0.62, 1.51
Fever	1.63	1.08, 2.44	0.89	0.61, 1.28
Nausea	1.18	0.74, 1.90	0.89	0.58, 1.36
Stomach pain	2.51	1.45, 4.32	0.90	0.60, 1.37
Vomiting	2.13	0.95, 4.78	0.84	0.46, 1.53
Diarrhea	2.10	1.33, 3.31	1.06	0.69, 1.63
Sinus problems	1.41	1.05, 1.91	1.25	0.93, 1.68
Cough	1.36	0.96, 1.91	1.10	0.80, 1.51
Phlegm	1.33	0.92, 1.92	0.52	0.35, 0.76
Sore throat	1.96	1.42, 2.70	1.55	1.13, 2.14
Eye redness	2.44	1.20, 4.93	1.42	0.60, 3.33
Ear pain	1.36	0.89, 2.09	1.55	0.98, 2.46
Skin infection	1.93	1.12, 3.33	0.71	0.42, 1.21

Note. Odds ratios (ORs) were adjusted for water exposure, gender, age, occupation, education, income, political outlook, and level of concern about coastal water quality. CI = confidence interval; SRD = significant respiratory disease (fever and sinus problems, fever and sore throat, or cough and phlegm); HCGI = highly credible gastrointestinal illness (vomiting, diarrhea and fever, or stomach pain and fever).

as many symptoms overall as SCC participants (OR=1.85; 95% confidence interval [CI]=1.4, 2.5) during the 1998 El Niño winter (Table 1). In that year, NOC participants reported higher rates of every symptom. During the 1999 La Niña winter, NOC participants reported only slightly more symptoms than SCC participants (OR=1.17; 95% CI=0.9, 1.6) and reported slightly higher frequencies in regard to 6 of the 12 symptoms. Odds ratios decreased consistently across all symptoms between the 2 winters. In both study years, risk increased across almost every symptom category by an average of about 10% (OR=1.1) for each additional 2.5 hours of water exposure per week.

DISCUSSION

Results from this investigation and other studies[9,10] suggest that discharging untreated urban runoff onto public beaches can pose health risks. This conclusion is supported by the higher reporting rates of symptoms among urban NOC participants during the rainy 1998 El Niño winter, after controling for possible confounding (due to demographic characteristics) and reporting bias (due to concern about coastal water quality). The exposure–response relationship demonstrated for most of the symptoms further supports this conclusion. Direct associations have been reported between pollution levels in runoff waters and urban land use, population levels, and amount of impervious surface area in the watershed.[11–15]

Research on the health consequences of urban runoff represents a relatively new area of investigation, despite decades of urban runoff contaminating coastal waters.[7,12] Most previous epidemiological studies focused on waters contaminated with domestic sewage, and the majority found associations between water pollution levels and incidence levels of symptoms.[2–4,16–21] Most epidemiological studies of recreational water use have focused on single exposure events rather than exposure over time.[2,3] Our study assessed 3-month prevalence rates of symptoms and demonstrated that average symptom prevalence was associated with different levels of water pollution.

To reduce the potential for confounding, we sampled from the same source population during both years and used comparable groups of surfers with relatively similar social characteristics. The high participation rates (above 80%) lowered potential bias due to selective participation. The study was cross sectional, which represents a limitation in terms of assessment of symptoms over a 3-month period, but any recall bias was likely to be nondifferential and toward the null. To reduce potential differential reporting bias, we adjusted for participants' level of concern about coastal water quality. Another limitation is that we did not measure water quality at the sites, so we were unable to determine the specific nature of the pollutants associated with symptoms.

In summary, this study suggests that discharging untreated urban runoff onto public beaches can pose health risks. These potential health risks warrant greater public health surveillance, as well as greater efforts to reduce pollutants discharged onto public beaches. Large-scale prospective investigations are needed to further characterize the health risks of people exposed to untreated urban runoff in coastal waters. ■

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All authors contributed to the design, writing, and final approval of the article.

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Human Participant Protection

The use of human participants in this study was approved by the institutional review board of the Univer-

sity of California at Irvine. The participants provided verbal informed consent.

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Raised Speed Limits, Speed Spillover, Case-Fatality Rates, and Road Deaths in Israel: A 5-Year Follow-Up

Elihu D. Richter MD, MPH, Paul Barach MD, MPH, Lee Friedman, BA, Samuel Krikler, MS, Abraham Israeli, MD, MBA

Objectives. We assessed the 5-year, nationwide impact on road deaths of the raise in the speed limit (November 1, 1993) on 3 major interurban highways in Israel from 90 to 100 kph.

Methods. We compared before–after trends in deaths as well as case fatality—an outcome independent of exposure (defined as vehicle-kilometers traveled).

Results. After the raise, speeds rose by 4.5%–9.1%. Over 5 years, there was a sustained increase in deaths (15%) and case fatality rates (38%) on all interurban roads. Corresponding increases in deaths (13%) and case fatality (24%) on urban roads indicated "speed spillover."

Conclusions. Immediate increases in case fatality predicted and tracked the sustained increase in deaths from increased speeds of impact. Newtonian fourth power models predicted the effects of "small" increases in speed on large rises in case fatality rates. Countermeasures and congestion reduced the impact on deaths and case-fatality rates by more than half. (*Am J Public Health*. 2004;94:568–574)

On November 1, 1993, the government of Israel increased the enforced speed limit for all vehicles, including trucks, from 90 to 100 kilometers per hour (55.9 to 62.1 mph) on segments (115 km, or 71.4 miles) of 3 major interurban highways connecting its 4 major cities: Tel Aviv, Jerusalem, Haifa, and Beersheba. The government made major improvements on these highways and many other roads and declared the increased speed limit a 1-year "experiment."[1] Simultaneously, it mandated the use of rear seat belts and daytime running lights.

Lower travel speeds and fewer deaths usually follow lowered speed limits.[2,3] Higher travel speeds and more deaths follow increased speed limits.[4–10] Recent data demonstrate a 17% increase in deaths after a 4% increase in speeds on US interstate highways.[11] High-speed driving on highways induces speed adaptation (a situation in which vehicle speed is influenced by the speed and duration of recent travel in the vehicle) on connecting interurban roads, and even urban roads. This so-called spillover effect may persist for 5 to 6 years.[12–15] Yet there is still worldwide controversy over the impact of increased speed limits.[16–18] One view holds that increased speed limits not only shorten travel time but also are protective when increased vehicle mileage is used to correct for increases in death tolls.[16] The US Centers for Disease Control and Prevention, despite 41 967 road deaths in 1997, has not cited higher speed limits as contributing to the high number of deaths.[19] The British government, by contrast, is committed to strategies to reduce speeds.[20]

Israel, with a size of 21 501 km^2, provides an ideal setting for observing the effects of speed limits. Israel has a fairly modern car fleet and roads and relatively low drunk-driving rates and is isolated from traffic from neighboring states.[21] All its highways are interurban. The 3 highways on which the enforced speed limit was raised serve as the major conduits of Israel's interurban traffic.

We examined the suddenness, size, distribution, and persistence of nationwide changes in death and injury tolls after the increase in the speed limit on these highways, with specific attention to speed spillover and its nationwide effect on road deaths. We examined the utility of 2 empirically derived models that demonstrate the relations between speed and fatality risks; the models are based on Newtonian physics. The first model demonstrates that case-fatality rates (CFRs) vary to the fourth power of the velocity at vehicular impact with both unbelted[22] and belted[23] drivers; the second model demonstrates that the number of crashes, injuries, and deaths varies with the first, second, and fourth power, respectively, of increases in average traffic speeds.[24,25] We also examined whether an increase in the CFR, a crash-phase outcome (those variables which influence survival in the event of a crash, such as speed of impact, seat belt use, and trauma care) independent of exposure (billion vehicle-kilometers of travel [bvkmt]),[26] predicted and tracked sustained trends in increased road death tolls. Finally, we assessed the degree to which protective countermeasures and increased traffic congestion offset and conceal the full impact of increases in travel speeds on road deaths.

DATA SOURCES AND METHODS

Speed Trends

Data on speed trends on the 3 high-speed highways (Tel Aviv to Jerusalem [highway 1]; Tel Aviv to Haifa [highway 2]; Tel Aviv to Beersheba [highway 4]) came from sporadic roadside daytime monitoring in the years 1971–1994 using roadside radar and laser cameras.[27–30]

Road Deaths and Injuries

We collected data on road deaths (up to 30 days after crash injury), serious injuries (hospitalized more than 24 hours), and light injuries (not hospitalized, or hospitalized less than 24 hours), and exposure—as measured by billion vehicle-kilometers of travel—from the Central Bureau of Statistics.[27,29] We also used a surrogate measure for the CFR—the proportion killed among all seriously injured (hereafter CFRS, for "CFR surrogate")—to avoid biases from transient underreporting of light injuries, which the Central Bureau of Statistics estimated to be of the order of 10%.

Impacts Within Subgroups

We carried out a 1-year comparison of deaths and case-fatality rates (CFRs and CFRSs), before and after the increase to 100 kph on November 1, 1993, for high-speed roads, other interurban roads, and all

urban roads, and for major crash types and driver subgroups.

Sustained Impact of 100 kph

We analyzed changes in death rates and CFRs between 3 years before and 5 years after the increase in the speed limit with the Student *t* test and cumulative summing. This method involves subtracting the differences between monthly totals for deaths from the overall mean of the 3-year control period derived from a baseline of monthly average death totals and testing these differences with simple *t* tests.[31] We then estimated the specific effect of increased speed limits without countermeasures and congestion by comparing the observed change in the number of deaths per year with that attributable ($K_{(ATTRIB)}$) specifically to the change in CFRs. We made these estimates using the formula

$$(1) \quad K_{(ATTRIB)} = \{K_{(B)}(CFRS_{(A)}/CFRS_{(B)})\} - K_{(B)},$$

where $K_{(B)}$ represents persons killed per year 3 years before, and $CFRS_{(B)}$ and $CFRS_{(A)}$ are the proportion of those killed among all those seriously injured before and after the speed limit change, respectively.[19] Using models developed by Evans,[3] we also estimated the average change in speeds (V) of travel and crash impact, as well as the change in number of deaths per each 1% change in speed, using algebraic fourth-root models in which

$$(2) \quad K_{(A)}/K_{(B)} = V_{(A)}^4/V_{(B)}^4$$

where $K_{(A)}$ and $K_{(B)}$ are persons killed per year 5 years after and before the increase, respectively, and $V_{(A)}$ and $V_{(B)}$ are average speeds on roads after and before the speed increase, respectively.

(A separate autoregressive integrated moving averages analysis of observed–expected ratios for deaths, deaths per billion vehicle-kilometers of travel, and CFRs on interurban and urban roads of the first year after the increased speed limit is available from the authors on request. This analysis used monthly totals for these parameters going back 13 years as a baseline for predicting expected results during the first 6 and subsequent 8 months after the increase in the speed limit.)

RESULTS

Speed Trends: 1 Year

Sporadic monitoring from 1971 to 1994 (Table 1) indicated that right after the increase in the speed limit, travel speeds on high-speed roads increased by 4.5% on the slow lane of the Tel Aviv–Haifa road (highway 2), by 9.1% on the fast lane of the Tel Aviv–Jerusalem road (highway 1), and even more on a newly widened stretch of a major connecting road off the Tel Aviv–Jerusalem road (Table 1, highway 40)—all compared with the year before. Other data showed that speeds rose on all 3 highways after the speed limits were raised, and that the mean estimated increase in speeds on the high-speed roads later fell back to a net increase of approximately 4% (range: –4% to 13%) in 1995.[27–30]

Deaths and Case-Fatality Rates: Immediate Effects

A sudden increase in monthly nationwide death tolls and CFRSs followed the increase in the speed limit on November 1, 1993. The first month after speed limits were increased, deaths (n=61) increased 32.6% from October (n=46). Interurban deaths (n=38) and the CFRS (26.4%) were the highest since November 1990.

First-Year Trends: Subgroups

In the first year after the increase in the speed limit, deaths increased by 24%, from 257 to 319, and CFRSs increased by 29.5% on all interurban roads combined, compared with corresponding increases of 3%—from 230 to 236—and less than 10%, respectively, on urban roads (Table 2). On newly widened segments of the 3 high-speed highways (and extensions) with 100-kph limits (roads 1, 2, and 3), deaths increased by 67%, from 21 to 35, a reversal of downward trends from 1990, and CFRSs increased by 50%. Even so, 48 (77%) of the 62 added deaths on interurban roads in the 12 months after the increase in the speed limit occurred not on the 3 high-speed roads but on other interurban connecting roads. A separate autoregressive integrated moving averages analysis[29] verified that the abrupt, large jump in deaths in the first 6 months after the increase in the speed limit was especially marked on interurban roads and was directly attributable to increases in CFRs and offset the long-term drops in deaths per billion vehicle-kilometers of travel. Before and after the increase in the speed limit, 90% or more of those killed in truck crashes were occupants of passenger cars.[32] After the increase in the speed limit, much—60%—of the *increase* in the nationwide road death toll came from large increases in deaths from truck crashes, mainly on interurban roads. Table 2 also shows increased CFRs and deaths in 1-vehicle and motorcycle crashes nationwide and decreased deaths among pedestrians and cyclists. Despite retention of the 90-kph limit by the military, reported deaths involving soldiers—both drivers and occupants—increased 106% (from 15 to 31), and reported CFRs increased 30%.

Modifiers and Confounders

Exposure. In the first year following the increase in the speed limit to 100 kph, interurban traffic increased 5% from 11.4 to 12.0 bvkmt, whereas urban traffic increased much more—from 13.2 to 15.5 bvkmt (17%). The number of road deaths per year was weakly correlated ($r=0.15$) with billion vehicle-kilometers of travel annually from 1963 to 1995, but negatively correlated with the number of licensed drivers ($r=-0.46$) from 1970 to 1995. These results rule out more vehicle traffic and drivers as plausible explanations for the sudden large increase in deaths.

Precrash and crash countermeasures. The government introduced several countermeasures, including laws requiring rear seat belts and daytime running lights (both mandated on November 1, 1993), more capital investment in upgrading old roads, building of new roads, midline concrete barriers and flyovers, and nighttime lighting. Hospital trauma services increased from 1 to 5, and police enforcement, measured by issuance of speeding tickets, increased approximately fourfold in the years 1994 to 1998 (Cdr E. Efrat, Traffic Police Division, written communication, May 2001).

There were no changes in before–after ratios of billion vehicle-kilometers of travel for trucks to all vehicles (27.3%:27.5%), drivers aged 19 to 24 years–all drivers (17.2%:17.5%), or fuel costs or alcohol sales.

TABLE 1—Measured Speeds on 3 Main Highways and Other Roads Before and After the Speed Limit Increase (90 kph to 100 kph) of November 1993: Israel, 1971–1994

Highway	Date	No. of Sitings	No. of Vehicles	Weighted Mean Speed Traveled, kph[a]	Range of SDs (by Siting)	Weighted 90th Percentile[b]
			Right lane[c]			
Before November 1993						
Highway 2 (Tel Aviv–Haifa)	2/7/93–9/14/93	5	3533	90.7	9.9–12.7	108.7
After November 1993						
Highway 2 (Tel Aviv–Haifa)	1/11/94–3/23/94	3	3132	94.8	12.8–13.4	109.9
			Left lane[c]			
Before November 1993						
Highway 2 (Tel Aviv–Haifa)	1971–1990[d]	10	11974	94.4	7.7–13.9	NA
Highway 2 (Tel Aviv–Haifa)	9/7/93–9/9/93	7	8076	98.6	12.5–22.9	114.6
Highway 4 (Tel Aviv–Beersheba)	8/23/93–8/25/93	6	8340	96	13.6–20.0	111.7
Highway 1 (Tel Aviv–Jerusalem)	8/24/93–9/1/93	8	10000	98.9	11.0–14.7	114
After November 1993						
Highway 1 (Tel Aviv–Jerusalem)	4/27/94–5/30/94	11	2762	107.9	8.6–17.3	126.6
			Single-lane connecting road			
Before November 1993						
Highway 40 (off Tel Aviv–Jerusalem)	3/92	3	1461	72.2	11–15	84.8
After November 1993						
Highway 40	7/1/94–7/4/94	8	1901	90.7	11.2–15.3	113.6

Note. NA = not available.
[a]Weighted mean speed traveled: (sum of number of vehicles per siting multiplied by mean speed for individual siting) divided by total number of vehicles.
[b]Weighted 90th percentile for speed traveled: (sum of number of vehicles per siting multiplied by 90th percentile speed for individual siting) divided by total number of vehicles.
[c]Right lane is slow lane; left lane is fast lane.
[d]From 1971 to 1990, mean speeds (and number of sitings) were as follows: 1971: 92.3 (1537); 1975: 97.4 (1338); 1976: 97.3 (1446); 1977: 94.0 (1758); 1980: 88.3 (1860); 1981: 87.6 (1866), 1983: 95.9 (1974); 1988: 103.8 (NA); 1990: 102.8 (NA).

5-Year Trends in Deaths and Case-Fatality Rates

During the entire 5-year period following the increase in the speed limit (November 1, 1993, to October 31, 1998), there were substantial drops in the number of persons reported with serious injuries (Table 3). In July 1995, the monthly death toll (n=62) peaked. In the third year after the increase in the speed limit, the death toll on interurban roads began to fall from a peak in 1994–1995 (n=327), corresponding to indications of decreases in average interurban speeds (Table 1) but continued to increase on urban roads. During the entire 5-year period, there were mean increases of 39.2 (15%) and 27.2 (13%) deaths per year on interurban and urban roads, respectively. The corresponding increases in CFRs, which tracked trends in speeds of impact, were much greater: 38% (from 12.5% to 17.3%), and 24% (from 7.9% to 9.8%) (Table 3). Using validated Newtonian models,[3] we estimated that without countermeasures and congestion, increased speeds of travel and impact would have resulted in *increases* of 100.2, and 51.1 deaths per year on interurban and urban roads, respectively. Countermeasures and congestion would have resulted, if not for the increased speed limits, in corresponding *reductions* of 61 and 23.9 deaths per year. The 5-year nationwide increase in deaths per year (n=151.3) expected from increased speeds of impact greatly exceeded the observed increase (n=66.4) in total deaths per year (Table 4).

DISCUSSION

After the increase in the speed limit from 90 to 100 kph, sporadic data suggested that travel speeds increased on Israel's 3 major highways and other roads, later falling back somewhat. In the first year after the increase in the speed limit, there were abrupt increases in deaths from increases in travel speeds on the 3 major highways and spillover of these effects to other urban and interurban roads. More than three quarters of the first-year increase in deaths (n=62) on interurban roads occurred from a systemwide spillover effect from high-speed roads on which the speed limits were legally increased to other interurban roads. All these findings state the case for systemwide increases in real travel speeds.

The Case for a Cause-and-Effect Relation

Our observations state the case for a direct cause-and-effect relation between the increase in the speed limit and the increase in the death toll. First, the step function increase in deaths coincided with the increase in the speed limit. Second, the increase in deaths is attributable specifically to the increase in CFRs—in all vehicle and crash types—a find-

TABLE 2—Deaths and Case-Fatality Rates in Israel Before and After the Speed Limit Increase (90 kph to 100 kph) of November 1993: Israel, November 1992–October 1993 vs November 1993–October 1994

	No. Killed 1992–1993	No. Killed 1993–1994	Absolute Change, No.	Ratio of Nos. (1993–1994/1992–1993)	CFR 1992–1993, %	CFR 1993–1994, %	CFR Ratio	CFRS 1992–1993, %	CFRS 1993–1994, %	CFRS Ratio
Interurban Roads										
High Speed[a]	21	35	14	1.7	NA	NA	NA	14.0	21.0	1.5
Other[b]	236	284	48	1.2	2.5[f]	3.0[f]	1.2	12.8	16.1	1.3
All[b]	257	319	62	1.2	2.1	2.7	1.2	12.9	16.7	1.3
Urban roads[b]	230	236	6	1.0	0.9	0.9	1.2	8.7	9.3	1.1
Trucks[c]										
Interurban	48	74	26	1.5	3.6	5.5	1.5	NA	NA	NA
Urban	25	37	12	1.5	1.9	3.6	1.9	NA	NA	NA
Single vehicle[d]	85	108	23	1.3	1.80	2.7	1.5	8.8	11.9	1.4
Soldiers[e]										
Off-duty	14	20	6	1.4	10.6	7.5	0.7	19.4	26.0	1.3
On-duty	1	11	10	11.0	0.40	2.9	8.0	3.6	25.6	7.2
Motorcycles[b]	22	39	17	1.8	0.8	1.3	1.6	5.9	9.1	1.5
Pedestrians[b]	199	189	–10	1.0	3.7	3.8	1.0	13.7	14.2	1.0
Bicycles[b]	12	10	–2	0.8	1.2	1.1	0.9	6.6	6.5	1.0

Note. CFR = standard case-fatality rate: killed/(killed + seriously injured + lightly injured), expressed as percentage; CFRS = modified case-fatality rate: killed/(killed + seriously injured only), expressed as percentage; NA = not available.
[a]Data received from the Israel's Police National Headquarters; data comprising only killed and seriously injured on 100-kph and 90-kph sections of highways 1 (Tel Aviv–Jerusalem), 2 (Tel Aviv–Haifa), and 4 (Tel Aviv–Ashdod) from 1990 to 1994.
[b]Data from Israel Central Bureau of Statistics 1992–1994: *Road Accidents with Casualties: Part I.*
[c]Truck data received by Israel's Police National Headquarters; data comprising killed and injured (serious + light) for 11 months of 1993 and 11 months of 1994 (November through September).
[d]Single-vehicle crash data from Israel Central Bureau of Statistics 1992–1994 *Road Accidents with Casualties: Part I.* Single-vehicle crashes include categories skidding, overturning, running of the way, and collision with fixed object.
[e]Soldier data received by Israel Defense Forces. Data consists of 8-month periods (November 1992 to July 1993; November 1993 to July 1994). For the CFRS, we combine moderate and serious injuries (Israel Defense Forces have a different classification: slight, moderate, serious, and killed).
[f]CFR ratios for 1993–1994 were corrected using the correction factor provided by the Central Bureau of Statistics to correct for underreporting in 1994.

ing that suggests increased speeds of impact. Third, the increases in deaths and CFRs on the high-speed roads were proportionately much greater than on other interurban roads and on urban roads. Fourth, the degree of increase in CFRs and deaths matched that expected from the reported increases in travel speeds based on the validated models. Fifth, time trends in *all* the modifiers and confounders (enforcement, seat belts, trauma care) should have resulted in reductions, not increases, in death tolls.

Predictive Models for the Size of the Relation

We confirmed the utility and validity of predictive Newtonian models in which deaths and CFRs increase in proportion to the fourth power of increases in speeds of travel of all vehicles and impact speeds of crashing vehicles, respectively. In the first year after the 100-kph speed limit was implemented, the observed increase in average *travel* speeds of 4% to 4.5%, based on sporadic measurements, accords with the increase of 5.5% in average travel speeds predicted from the observed increase of 24% in deaths on all roads.[3,23–25] Increases in CFRs of 50% on highways and 26% on other interurban roads in the *first* year imply that average increases in speeds of *impact* on these roads were of the order of 11% and 5.6%, respectively. During the entire 5-year period after implementation of the 100-kph speed limit, similar calculations suggest that *impact* speeds of *crashing* vehicles increased on interurban and urban roads by some 8.3% and 5.5%, respectively. These increases exceeded the estimated increases of 3.6% and 3.1% in average *speeds of travel* for *all* vehicles (Table 4). These calculations imply that each 1% increase in average speeds of travel and impact resulted in increases of approximately 11 deaths per year on interurban roads, and 9 deaths per year on urban roads.

Subgroups at Risk for the Effect of the Increased Speed Limit

The large increases in the number of deaths and CFRs were seen in crashes involving trucks, motorcycles, single vehicles, and soldiers (both on and off-duty) but not pedestrians and bicyclists. Other evidence suggested that the fatal crash risk of truck drivers from higher speeds increased with longer hours, irregular shifts, and incentive premiums.[32,33]

The fact that increases in deaths from increases in CFRs occurred in all subgroups except pedestrians and bicyclists rules out changes in the case mix of crash types as the reason for the increase in total CFRs of all crash groups combined. Soldiers—both drivers and occupants—were at increased risk, despite

TABLE 3—Deaths and Case-Fatality Rates in Israel Before and After the Speed Limit Increase (90 kph to 100 kph) of November 1993: All Roads, Interurban, and Urban, Israel, 1990–1997

	Year[a]	Killed, No.	Seriously Injured, No.	CFRS, %	Range
			Interurban[b]		
Before increase					
	1990–1991	219	1718	11.8	6.99–17.73
	1991–1992	307	2078	12.9	10.04–18.07
	1992–1993	257	1735	12.9	7.50–19.33
	Mean	261	1843.7	12.5	
After increase					
	1993–1994	319	1596	16.7	11.36–26.39
	1994–1995	327	1596	17.0	11.58–21.09
	1995–1996	293	1407	17.2	12.99–23.53
	1996–1997	278	1259	18.1	12.82–24.37
	1997–1998	284*	1344	17.4**	12.10–24.42
	Mean	300.2	1440.4	17.3	
			Urban[b]		
Before increase					
	1990–1991	211	2448	7.94	5.49–12.28
	1991–1992	195	2592	7.00	3.46–9.65
	1992–1993	230	2410	8.71	4.74–13.86
	Mean	212	2483.3	7.9	
After increase					
	1993–1994	236	2303	9.3	7.39–11.27
	1994–1995	223	2376	8.6	6.15–11.77
	1995–1996	235	2231	9.5	6.35–12.76
	1996–1997	249	2156	10.4	5.47–14.04
	1997–1998	253*	2008	11.2**	7.11–14.42
	Mean	239.2	2214.8	9.8	

Note. CFR = case-fatality rate: the standard rate killed/all casualties; CFRS = modified case-fatality rate: killed/(killed + seriously injured). Student's *t* test compared monthly death and case-fatality rates of November 1990 through October 1993 versus November 1993 through October 1998 for all roads combined, interurban alone, and urban alone. Slightly Injured were corrected for underreporting in 1993 by 1% and in 1994 by 9% as recommended by the Central Bureau of Statistics. Change in 1996 reporting of slightly injured resulted in an increase in reported light injuries by more than 8000 during 1995–1996. Change in reporting ("attendance only" requirement) probably reduced number of seriously injured as well.
[a]Calendar year in this study is from November through October. The speed limit was raised officially on November 1, 1993.
[b]All data obtained from Israel Central Bureau of Statistics *Road Accidents with Casualties: Part I* for years 1990 to 1997.
* $P < .01$; ** $P < .001$.

the military's retention of the 90-kph limit during working hours. For pedestrians and bicyclists, the drop in death tolls may be explained by a trend seen in the United Kingdom, where there has been a reported decrease in walking and cycling on interurban roads.[34] The fact that CFRs from 1-vehicle crashes—which are not influenced by vehicle–vehicle interactions—were not less than those from other crash types undermines the claim that increased speed variance[35] and not increased speed is the real cause for the increase in deaths.

Speed Versus Congestion and Countermeasures

The observed increase in deaths per year following the increase in the speed limit to 100 kph substantially underestimated the increase in deaths directly attributable to the increase in CFRs. The exponential effect of "small" increases in speed and speed spillover on nationwide increases in CFRs over the next 5 years more than offset the decreases in death *risks* per vehicle-traveled from protective countermeasures, as well as congestion in urban areas. The persistence of high CFRs indicates that increased speeds of impact were negating the protective effects of newly widened roads, improved lighting, cloverleafs, air bags, rear seat belt laws, more speed enforcement and changes in trauma care, and other countermeasures, as well as increased congestion. Without protective countermeasures and increased traffic congestion, there would have been many more deaths, and without the increase in the speed limit, there would have been many fewer deaths (Table 4).

In Israel, increases in traffic congestion and road safety countermeasures have produced a long-term strong inverse relation between deaths per billion vehicle-kilometers of travel and both the number of vehicles (r=–0.88) and vehicles per population (r=–0.85).[19,27] In the United States, car occupant deaths dropped 11% between 1975 and 1997, despite a ninefold increase in cars.[36] But from 1992 to 1997, by which time US states were raising speed limits, deaths—and even deaths per billion vehicle-kilometers of travel—did not drop.[19] By contrast, in the years 1991 to 1998, road deaths per year fell by 25.2% in the United Kingdom with correspondingly larger decreases in deaths per billion vehicle-kilometers of travel.[37]

Risks for deaths per billion vehicle-kilometers of travel have always decreased with increases in billion vehicle-kilometers of travel[38] ("the soccer field is tilted downwards"[19]); therefore, before and after differences *following* increased speed limits will underestimate the increases in death tolls *from* increased speed limits over many years. Studies of the long-term impact of increased speed limits that "correct" for increases in billion vehicle-kilometers of travel may underestimate the full direct impact of increased speed limits and travel speeds on road deaths. Long-term follow-up in Washington State, for example, showed that when corrected for exposure, a 25% increase in deaths on interstates was reduced to a 10% increase.[39] These studies ignore the role of countermeasures and increased congestion in producing the falling trends in deaths per billion vehicle-kilometers of travel in urban areas. Congestion from increased billion vehicle-kilometers of travel during the so-called rush hours offsets the ef-

TABLE 4—Effect of Increase in Speed Limit With and Without Countermeasures and Congestion: Deaths, Estimated Change in Speeds, and Estimated Deaths per Year per 1% Increase in Speed

	Mean Deaths per Year November 1990 to October 1993 (Baseline), No.	Mean Deaths per Year November 1993 to October 1998 (Follow-up), No.	Absolute Change (Baseline to Follow-up), No.	Ratio Change (Baseline to Follow-up)	Estimated Change in Speeds (Nilsson[a] and Joksch[b] Formulas), %	Mean Change in Deaths per Year for Each 1% Increase in Speed, No.[c]
Interurban roads						
Observed[d]	261	300.2	39.2	1.15	3.6[a]	10.9
Expected						
Speed limit raised; without increase in countermeasures and congestion (expected scenario 1)[e]	261	361.2	100.2	1.38	8.3[b]	12.1
Speed limit not raised; with increase in countermeasures and congestion (expected scenario 2)[e]	261	200	−61	0.77	−6.4[a]	−9.53
Urban roads						
Observed[d]	212	239.2	27.2	1.13	3.1[a]	8.8
Expected						
Speed limit raised; without increase in countermeasures and congestion (expected scenario 1)[e]	212	263.3	51.3	1.24	5.5[b]	9.3
Speed limit not raised; with increase in countermeasures and congestion (expected scenario 2)[f]	212	188.1	−23.9	0.89	−2.9[a]	−8.24

[a]Change in travel speeds derived from fourth root of ratio of number of deaths after to deaths before increase in speed limit, using equation $K_{(a)}/K_{(b)} = V_{(a)}^4/V_{(b)}^4$ to solve for $V_{(a)}$, where $K_{(a)}$ and $K_{(b)}$ and $V_{(a)}$ and $V_{(b)}$ are deaths and average vehicular speeds, respectively, on roads after and before (Evans[3,23]).
[b]Change in impact speeds calculated as fourth root of ratio of case-fatality rate after increase in speed limit to that before increase in speed limit (see references 23 and 24).
[c]Derived by dividing absolute change in deaths by estimated percentage change in speeds.
[d]See the means for deaths per year before and after the increase in speed limit in Table 3.
[e]Derived from increase in mean case-fatality rate alone from 12.5% to 17.3% on interurban roads and from 7.9% to 9.8% on urban roads after the increase in the speed limit from 90 to 100 kph on November 1, 1993. We used the formula $K_{(ATTRIB)} = \{K_{(B)} \times (CFRS_{(A)}/ CFRS_{(B)})\} - K_{(B)}$, where $K_{(ATTRIB)}$ is persons killed per year attributable to the increase in speed 5 years after the speed limit increase, $K_{(B)}$ is persons killed per year 3 years before the increase in the speed limit, and $CFRS_{(B)}$ and $CFRS_{(A)}$ are the proportion of those killed among all those seriously injured before and after the speed limit increase. We estimated average changes in speed of travel and crash impact and number of deaths per each 1% change in speed, using fourth-root models.
[f]The difference in observed deaths in the 5-year follow-up period and the absolute change (baseline to follow-up) in scenario 1 gives us expected deaths for scenario 2.

fects of higher speeds during other hours, notably during nighttime.

Evans has shown how relations between increases (and decreases) in speed and the increases (and decreases) in death tolls are direct, obey algebraically defined laws derived from Newtonian physics, and are reversible.[23] Based on these premises, we suggest that a reduction by 10% in the average speeds of impact during the period we studied would have prevented 121 (or 40%) of the 300 interurban deaths per year, and 85 (or 35.4%) of the 240 urban deaths per year (Table 4). Support for this inference comes from the observation in the United Kingdom that there are even bigger decreases in death tolls—up to 70%—with *reductions* in speeds from massive use of roadside speed cameras.[40] Substantial reductions in deaths, injuries, and crashes from the use of speed cameras state the case for their use.[41]

We suggest, however, that achieving sustainable major reductions in road death tolls requires not only lower speed limits and increased detection and deterrence of high speeds, but also lower design speeds for cars, and a downward shift in speed distributions, in keeping with the principle of treating sick populations, not just sick individuals.[42] The public health stakes involved in applying this principle are enormous, given that globally, there are now more than 1 170 000 road deaths per year[43] and more than 40 000 deaths per year in the United States alone. We predicted *increases in death tolls* from new highways and spillover roads with even higher design speeds and speed limits, a trend now seen in many rapidly motorizing countries.[44] Elsewhere, we have suggested using the CFR to track the direct long-term impact of increased travel speeds on death tolls in the United States.[19] The CFR, the outcome of concern, is a parameter based on a universe; because it is extremely sensitive to small changes in speed well within the range of sampling and measurement errors, it paradoxically may be a more valid indicator of speed trends than sporadic speed measurements themselves.

Elsewhere, we have addressed the ethical and scientific lapses underlying the decision to increase the speed limit.[45,46] In retrospect, the sentinel increases in travel speeds on the

study's highways, CFRs, and deaths in the very first months of the 100-kph "experiment" predicted its subsequent 5-year nationwide impact and stated the case for its cancellation.

In 2002, despite an increase in deaths to 540 from 476 the year before, there were renewed pressures to increase the speed limit still further, to 110 kph–120 kph. ■

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Contributors

E.D. Richter collected preliminary data, conceived the study, and wrote the brief. P. Barach collected much of the data on the trends in the first year and reviewed the literature. L. Friedman did the data collection and statistical analysis for the 5-year follow-up. S. Krikler oversaw time series analyses connected with the short-term effects. A. Israeli served as co-tutor and adviser to P. Barach.

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Knee Pain and Driving Duration: A Secondary Analysis of the Taxi Drivers' Health Study

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Objectives. We explored a postulated association between daily driving time and knee pain.

Methods. We used data from the Taxi Drivers' Health Study to estimate 1-year prevalence of knee pain as assessed by the Nordic musculoskeletal questionnaire.

Results. Among 1242 drivers, the prevalence of knee pain, stratified by duration of daily driving (≤6, >6 through 8, >8 through 10, and >10 hours), was 11%, 17%, 19%, and 22%, respectively. Compared with driving 6 or fewer hours per day, the odds ratio of knee pain prevalence for driving more than 6 hours per day was 2.52 (95% confidence interval=1.36, 4.65) after we adjusted for socioeconomic, work-related, and personal factors in the multiple logistic regression.

Conclusions. The dose-related association between driving duration and knee pain raises concerns about work-related knee joint disorders among professional drivers. (*Am J Public Health.* 2004;94:575–581)

Knee pain is a common health problem worldwide. Data from the First National Health and Nutrition Examination Survey (NHANES I) suggest that in the 1970s, it was the second most common musculoskeletal symptom, affecting 13.3% of people aged 25 to 74 years.[1] Results of NHANES III (1988–1994) revealed that 18.1% of US men and 23.5% of US women aged 60 years or older suffered from significant knee pain.[2] During the same period surveyed by NHANES III, the estimated 1-year prevalence of persistent knee pain in England was 25% among those aged 55 years and older.[3] Similar statistics showing that knee pain is a prevailing public health problem can be derived from studies conducted in Europe.[4–7] Other research findings demonstrate that people who live in the nonindustrialized world are not exempt from this endemic problem, because estimates of knee pain prevalence from nonindustrialized countries either were comparable to those in industrialized countries[8–11] or were even higher,[12] partially because of the greater prevalence of heavy physical activities in nonindustrialized countries.

Knee pain is very likely a health problem with tremendous health care costs, despite the lack of direct cost estimates. In 1996–1997, more than 6 million Americans sought medical care for knee problems,[13] about 5 million of whom visited offices of orthopedic surgeons and 1.4 million of whom went to a hospital emergency room. A survey of US orthopedic surgeons conducted in 1997 found that the knee was the most often treated anatomic site, accounting for 26% of all orthopedic visits.[13] Pain relief remains one of the major reasons for joint replacement.[14] In 1999, 311 106 inpatient hospital stays involving total knee replacement in the United States accrued a "national bill" of more than $6.5 billion.[15] The annual rate at which patients request total knee replacements to ameliorate knee pain and restore mobility has increased since the early 1990s.[13] A similar trend also has been reported in Europe. After examining data from the Swedish Knee Arthroplasty Registry, researchers found that the number of knee arthroplasties per year between the periods 1976–1980 and 1996–1997 increased more than fivefold.[16] On the basis of the 1996 and 1997 data, it was projected that, from 2000 through 2030, in the absence of an effective preventive treatment, the number of knee arthroplasties per year will increase by at least one third.

Moreover, knee pain imposes a significant disability burden on modern societies.[3,17–20] Both cross-sectional and prospective studies have consistently shown that knee pain, rather than radiographically detectable abnormalities, is the major determinant of knee osteoarthritis–related physical disability.[6,21–26] Longitudinal studies have demonstrated that previous knee pain is associated with both the development of disease[27] and the progression of radiographically evident knee osteoarthritis.[28,29] In the NHANES Epidemiologic Follow-Up Study[30] on the relative risk of experiencing difficulty in ambulation and transfer (as from a chair to a standing position), the estimated relative risk for knee osteoarthritis patients (4.42 and 4.08, respectively) were twice those for heart disease patients (2.27 and 2.13). Framingham Osteoarthritis Study[31] researchers estimated that approximately 15% of the risk for the overall population of experiencing difficulty in walking—the highest attributable proportion for any single medical comorbidity—was attributable to knee osteoarthritis. Knee pain also may lead to accidental falls,[32–35] which, together with arthritis, account for more than 30% of all restricted-activity days among older US adults.[36] As the baby boom generation ages, the knee pain–related disability burden will become even more substantial; therefore, studying the multifaceted problem of knee pain is a public health task of fundamental importance.

Researchers should seek a better understanding of the mechanisms and the impacts on health of knee pain.[2,17,37] Because most musculoskeletal pain is chronic and recurrent,[38] studies of knee pain with onset at a younger age, such as knee pain precipitated by work-related injury or strain, and the contribution of knee pain to later disability will provide us with better information about the natural history of knee osteoarthritis. Such knowledge will help us to develop effective prevention strategies and management modalities tailored to different stages of the disease. A similar re-

search direction has been adopted in studies of other types of musculoskeletal pain.[39–43]

Descriptive results of 2 previous reports directed our attention to work-related knee pain among professional drivers. Anderson and Raanaas[44] conducted a survey of musculoskeletal complaints of taxi drivers in Norway. They used the Nordic musculoskeletal questionnaire[45] and found that the 1-year prevalence of knee pain among 703 full-time taxi drivers was higher than that among the reference group from the local community (29% vs 25%, respectively). A nationwide occupational health survey in Taiwan[46,47] that used a modified version of the Nordic musculoskeletal questionnaire also found that employed professional drivers had a knee pain prevalence slightly higher than the national average (11% vs 8.6%). However, no further data were available to explain the higher prevalence of knee pain among professional drivers observed in these 2 studies.

In 2000, the Taxi Drivers' Health Study (TDHS)[48]—an occupational, epidemiological study of cardiovascular disease risk, job stress, and low back pain—was launched in Taipei, Taiwan. The TDHS baseline data allowed us to test the hypothesis that prolonged driving is associated with increased knee pain prevalence among taxi drivers.

METHODS

The TDHS is integral to a medical-monitoring program sponsored by the Taipei city government that provides taxi drivers with free physical examinations each year.[48,49] From January 31 to May 31, 2000, 3295 taxi drivers participated in this program. From the 5 hospitals designated to provide free physical examinations (each hospital had a maximum number of taxi drivers it could serve), we selected the one with the largest assigned service volume as our study base for the TDHS. For drivers to be eligible for enrollment in our study, they had to (1) have been registered taxi drivers in Taipei for at least 1 year, (2) be voluntary participants, and (3) be able to read.

A standardized, self-administered questionnaire was delivered to each participant in the selected hospital. Its feasibility was tested among a volunteer sample of taxi drivers, who were recruited from cab companies, cooperative practices, local unions, and resting areas (a large parking area wher drivers can take a break, wash their cars, etc.), before the study began. In addition to questions about demographics and health behaviors, the questionnaire contained items regarding driver profiles (professional seniority in years, average number of driving days per month, and duration of daily driving in hours) and average frequency of physical activities (lifting and bending/twisting) during both work and leisure time. Previous studies[50,51] have shown that self-reporting is a relatively reliable and valid method to assess time spent driving a motor vehicle. In a small subset of baseline data from drivers who also participated in an exposure assessment study,[52] we found that 97% of self-reported daily driving times (grouped by periodic categories) agreed with data we retrieved from diary records and structured interviews. Although self-reported daily driving estimates exceeded actual measurements by an average of 0.9 hour, this measurement error was independent of knee pain (P=.73). The modified Nordic musculoskeletal questionnaire, the same questionnaire used in a previous nationwide survey,[47] presented a graph of 9 body parts and asked subjects to mark the anatomic sites at which they had experienced any pain in the past 12 months. (The Nordic musculoskeletal questionnaire has been demonstrated to possess acceptable validity and reliability.[45,53]) The modified questionnaire also included a job dissatisfaction subscale from the Job Content Questionnaire (Chinese version) and 5 questions about mental health from the Taiwanese version of the 36-item Medical Outcomes Study short form (SF-36).[54,55] Anthropometric and laboratory data were retrieved from annual free physical examination records.

We used multiple logistic regression analysis to estimate the odds ratio of knee pain prevalence associated with a change in duration of driving time. We grouped drivers by 4 categories according to duration of daily driving (≤6, >6 through 8, >8 through 10, and >10 hours) and calculated the crude odds ratio for knee pain prevalence in each group. Drivers who had driving times of 6 or fewer hours composed the reference group. We wanted to make a statistical inference about the effect of daily driving time on knee pain prevalence that controlled for biomechanically or biologically plausible risk factors for knee pain and osteoarthritis. We searched for these potential predictors before we examined the relationship between any covariate and knee pain prevalence in the univariate analysis. This process identified age, body mass index (BMI), education, smoking, lifting, bending/twisting, and psychosocial variables as predictors retained in the final model. We then fit the univariate model, driving time only (base model). All other variables had to cause at least a 10% change in the estimate of the odds ratio of knee pain prevalence associated with duration of daily driving in the base model to be included in the final logistic model, or they had to be significant in the univariate analysis (P=.25). We assumed no interactions among the potential predictors and included only subjects with complete data in the final analyses. The Hosmer–Lemeshow test[56] was used to assess the goodness of fit. Finally, we performed the jackknife dispersion test[57] to obtain an unbiased adjusted odds ratio of knee pain prevalence associated with a change in duration of daily driving. All of these statistical analyses were conducted with Stata 7.0 statistical software (Stata Corp, College Station, Tex).

RESULTS

Of the 1355 drivers who received medical examinations in the selected hospital, 1242 (92%) completed the 2 sets of questionnaires. The study population's mean age±SD was 44.5±8.7 years, drivers drove an average of 9.8 hours per day and 26 days per month, and 234 (19%) drivers had experienced knee pain in the past 12 months. Personal characteristics and occupational factors are shown in Table 1. We also tabulated the population reference statistics[58] and the demographic and other characteristics of the other 1940 drivers who were not enrolled in the TDHS but who had received physical examinations in other hospitals during the study period. With respect to the distribution of age, gender, professional seniority, daily driving duration, BMI, marital status, and registration type, the TDHS–enrolled drivers were not significantly

TABLE 1—Demographic and Occupational Characteristics of Participants in the Taxi Drivers' Health Study (TDHS) and Other Drivers[a]: Taipei, Taiwan, 2000

Characteristics	TDHS Participants (N = 1242)		Other Drivers (N = 1940)		
	n1	Mean ± SD or %	n2	Mean ± SD or %	Reference Group[b]
Age, y	1242	44.5 ± 8.7	1403	46.6 ± 8.7	43.9
Professional seniority, y	1234	11.4 ± 7.8	1890	11.0 ± 7.5	9.2
Total driving per month, days	1239	26.2 ± 2.6	1780	25.2 ± 3.6	26.8
Total driving per day, h	1238	9.8 ± 2.8	1889	9.9 ± 2.5	10
Body mass index, kg/m^2	1242	24.9 ± 3.6	1780	25.2 ± 3.6	…
Gender					
Male	1193	96%	1854	96%	97%
Female	49	4%	82	4%	3%
Education					
Less than high school	405	33%	770	40%	…
High school	782	63%	1067	56%	…
College or more	53	4%	69	4%	…
Marital status					
Single	201	16%	257	14%	…
Married	960	75%	1469	77%	…
Separated/divorced/widowed	116	9%	178	10%	…
Registration type					
Individual	497	40%	808	43%	…
Cooperative	395	32%	606	33%	…
Affiliated with taxicab company	341	28%	447	24%	…
Lifting activities					
Never/rare/seldom	604	49%	…	…	…
Often/sometimes	508	41%	…	…	…
Very frequently	122	10%	…	…	…
Bending/twisting					
Never/rare/seldom	643	52%	…	…	…
Often/sometimes	482	39%	…	…	…
Very frequently	111	9%	…	…	…
Leisure-time physical exertion					
Never/rare/seldom	602	49%	…	…	…
Often/sometimes	506	41%	…	…	…
Very frequently	126	10%	…	…	…
Perceived job stress					
None	282	23%	…	…	…
Mild	639	52%	…	…	…
Moderate to severe	311	25%	…	…	…
Mental health score (0–100)	1218	63.1 ± 16.8	…	…	…
Job dissatisfaction index (0.01–1.00)	1225	0.61 ± 0.17	…	…	…
Low back pain in past 12 months	628 (1241)	51%	988 (1798)	55%	…
Knee pain in past 12 months	234 (1241)	19%	395 (1798)	22%	…

Note. n1 = number of subjects in TDHS group; n2 = number of subjects not in study base. The total number summed up across each category varies slightly because of missing data.
[a]Other drivers received medical examinations at hospitals outside the study.
[b]Data from Dept of Statistics, Ministry of Transportation and Communication, Taiwan.[58]

different from drivers who were not enrolled, although they had a slightly lower prevalence of both knee pain and low back pain. We also noted that the demographic features of these 2 groups of drivers were comparable to the reference statistics.

Crude estimates of the 1-year prevalence of knee pain—stratified by duration of daily driving (≤6, 6–8, 8–10, and >10 hours)—were 11%, 17%, 19%, and 22%, respectively. Compared with drivers who drove 6 or fewer hours per day, the crude odds ratio of knee pain prevalence for drivers who drove more than 6 hours per day was 2.06 (95% confidence interval [CI] = 1.23, 3.43). Univariate analyses indicated that high frequency of bending/twisting activities during both work and leisure time, moderate to severe self-perceived job stress, a low mental health score, and high job dissatisfaction were significantly and positively associated with knee pain prevalence ($P<.05$).

The results of the multiple logistic regression analyses are shown in Table 2. After we adjusted for age, gender, BMI, income, education, marital status, smoking habit, frequency of regular exercise, mental health score, self-perceived job stress, job dissatisfaction index score, physical exertion during both work and leisure time, and professional seniority, taxi drivers with long driving times (>6 hours/day) had a significantly higher prevalence of knee pain than drivers with short driving times (≤6 hours/day): an adjusted odds ratio of 2.52 (95% CI = 1.36, 4.65). In contrast to the case for crude analyses, this increase in odds ratio estimate resulted mainly from the joint negative confounding by high physical exertion during leisure time, low income, and registration as an individual driver (as opposed to being in a cooperative practice or affiliated with a taxicab company). Those drivers with any 1 of these 3 characteristics tended to drive less than their counterparts.

The result of the Hosmer–Lemeshow test ($P=.74$) supported the goodness of fit of the multiple logistic model. The jackknifed odds ratio associated with long driving times (>6 hours/day) was 2.40 (95% CI = 1.24, 4.63). All of the jackknife estimates of the odds ratios for knee pain prevalence associated with each category of daily driving time were similar to estimates provided by all observations,

TABLE 2—Odds Ratios (ORs) and 95% Confidence Intervals (CIs) for Prevalence of Knee Pain in Past 12 Months (n = 1115): TDHS, Taipei, Taiwan, 2000

Characteristic	Crude OR (95% CI)	Adjusted[a] OR (95% CI)
Total driving per day, h		
≤6	1.00	1.00
6–8	1.70 (0.93, 3.11)	1.99 (1.00, 3.98)
8–10	1.95 (1.12, 3.40)*	2.55 (1.32, 4.94)**
>10	2.30 (1.35, 3.93)**	3.14 (1.62, 6.08)**
Bending/twisting		
Never/rare/seldom	1.00	1.00
Often/sometimes	1.25 (0.92, 1.29)	1.08 (0.75, 1.55)
Very frequently	1.75 (1.09, 2.80)*	1.56 (0.88, 2.75)
Leisure-time physical exertion[b]		
Never/rare/seldom	1.00	1.00
Often/sometimes	1.48 (1.09, 2.01)*	1.35 (0.94, 1.93)
Very frequently	1.78 (1.12, 2.82)*	1.94 (1.12, 3.34)*
Perceived job stress		
None	1.00	1.00
Mild	1.58 (1.05, 2.38)*	1.36 (0.85, 2.15)
Moderate to severe	2.49 (1.61, 3.84)**	1.78 (1.06, 2.99)*
Low mental health score[c]		
No	1.00	1.00
Yes	2.12 (1.57, 2.88)**	1.77 (1.26, 2.50)**
High job dissatisfaction[d]		
No	1.00	1.00
Yes	1.50 (1.07, 2.11)*	1.31 (0.90, 1.91)
Registration type		
Affiliated with taxicab company or cooperative practice	1.00	1.00
Individual practice	1.22 (0.91, 1.62)	1.60 (1.09, 2.35)*

[a]Adjusted for age, gender, education level, body mass index, marital status, income, smoking, professional seniority in years, days of driving per month, full-time status, frequency of heavy lifting activities, regular exercise, and all the other covariates in the table.
[b]The frequency of bending/twisting and/or heavy lifting when not at work.
[c]Low mental health score is defined as standardized mental health score lower than the first quartile as measured by the Taiwanese version of the 36-item Medical Outcomes Study short form (SF-36).
[d]High job dissatisfaction is defined as those whose job dissatisfaction index are in the highest quartile as measured by the job dissatisfaction subscale in the Job Content Questionnaire.
*P < .05; **P < .01.

suggesting that no observations were overly influential.

DISCUSSION

To our knowledge, analytic studies that show the association between knee pain and long driving times have not been reported in the literature in English. Our study indicated a likely association between long driving times and increased knee pain prevalence, both in the crude analysis and after adjustment for a large set of potential confounders and risk factors for knee pain and knee osteoarthritis.

Few previous studies have examined this interesting association. In a nationwide survey of musculoskeletal symptoms among post office pensioners in England,[59] Sobti et al. found that having driven more than 4 hours per day in previous occupations was common (15%) among post office employees. About 43% of pensioners reported experiencing knee pain or stiffness in the past month, but a significant association between driving and knee pain was not reported. In a survey of musculoskeletal pain in 12 groups of newly hired young workers (median age=23 years), Nahit et al.[60] examined whether the 1-month prevalence of knee pain (22%) was related to daily driving duration. The odds ratio associated with driving 15 minutes or more per day was found to be nonsignificant (odds ratio [OR]=1.0; 95% CI=0.6, 1.7). Because both study populations in Sobit et al. and Nahit et al. consisted of members of occupation groups with different background risks for musculoskeletal disorders, the limited variability in driving duration may not have provided the investigators with sufficient power to detect a significant association between long driving times and knee pain.

Our finding of a significant association between daily driving duration and knee pain was in accord with the results of previous studies. In an earlier study by Jajic et al.,[61] a significant increased concentration of ^{99m}Tc-polyphosphate in bone scans of knee joints (indicating increased bone rebuild, an early sign of degenerative changes referred to as a "preosteoarthrotic condition" in the report) was found among professional drivers. A recent study by Coggon et al.[62] showed an association (OR=2.3; 95% CI=1.4, 4.0) between long driving times (≥4 hours/day) and knee cartilage injuries in a community-based case–control study. Several survey results[63–65] have indicated that the knee is one of the joints most frequently injured in motor vehicle accidents. Studies of musculoskeletal injuries among bus drivers also showed that injuries to the lower extremities, including the knees, were the most common musculoskeletal injuries.[66] In a subset of 893 drivers who had information on previous motor vehicle accident–related knee injuries, we found previous knee injury to be strongly associated with knee pain prevalence (adjusted OR=6.54; 95% CI=1.62, 26.4). However, after we adjusted for previous motor vehicle accident–related knee injuries, long driving times were still significantly associated with increased knee pain prevalence (adjusted OR=2.30; 95% CI=1.18, 4.47).

We further examined the associations between knee pain and vehicle characteristics to provide some mechanic implications of our

findings. Our analyses yielded no consistent association between knee pain and vehicle manufacturers or engine sizes. Interestingly, the crude knee pain prevalence among drivers who operated vehicles made in 1990 or earlier was 25%, but only 18% among those who operated vehicles made after 1990. This association was marginally significant (adjusted OR=1.63; *P*=.07) after we controlled for all variables retained in the final multiple logistic model (Table 2). In a previous exposure assessment study on back disorders,[52] we found that 37% of the taxicabs in Taipei had manual transmissions. Presumably, most taxicabs made before 1990 had manual transmissions; more repetitive motion in the lower extremities is required when driving such vehicles. Nevertheless, it is noteworthy that the association between duration of daily driving and knee pain remained statistically significant (OR=2.63; 95% CI=1.42, 4.88). Regardless of the potential measurement errors of this rough classification, our analyses imply that in addition to repetitive motions of lower extremities, the contribution of other physical factors associated with prolonged driving (e.g., strenuous knee postures, relative immobilization of the left knee when using an automatic transmission) should be investigated in future studies.

Other physical and psychosocial factors associated with knee pain in our study conform to previous observations. Physical activity during both work and leisure time has been found to be a risk factor for developing knee osteoarthritis.[67,68] Many studies have identified psychosocial variables, such as self-perceived job stress, job dissatisfaction, and mental health (all included in our study), that are important determinants of knee pain in both occupational and community settings.[4,19,69–71] Another interesting finding, which is probably related to psychosocial context as well, was that independent drivers had slightly higher knee pain prevalence (21%) than did drivers in a cooperative practice (18%) or those affiliated with taxicab companies (17%). This difference was statistically significant in the multiple logistic regression, which suggests that factors other than the physical and psychosocial variables retained in our model may be more common among independent drivers and may account for their higher knee pain prevalence. We posited that the social-network function (e.g., social support) could partially explain this observation, because independent taxi drivers may be more isolated than other taxi drivers. Detailed analysis of data from the Job Content Questionnaire is needed to support this speculation.

We wanted to be cautious about the observed association between driving and knee pain. Therefore, we took the following steps to rule out plausible alternative explanations of our finding. Because a few studies had found that clustering of musculoskeletal symptoms is very common,[59,70,72] we first examined whether the reported knee pain was merely a co-symptom of other more frequent musculoskeletal complaints, such as pain in the low back (51%), neck (50%), and shoulder (30%) in this group of taxi drivers. After adding these 3 variables into the final multiple logistic regression, we found that our data did support the clustering of musculoskeletal symptoms. Taxi drivers who reported musculoskeletal pain in these 3 sites had significantly higher knee pain prevalence, with a corresponding adjusted odds ratio of 1.88 (95% CI=1.35, 2.65) for those who had low back pain, 1.90 (95% CI=1.35, 2.71) for those who had neck pain, and 1.71 (95% CI=1.22, 2.41) for those who had shoulder pain. However, even after we adjusted for the clustering of musculoskeletal symptoms, the association between long driving times and knee pain remained statistically significant (adjusted OR=2.41; 95% CI=1.28, 4.50).

Our sensitivity analysis[73] (data not shown) was intended to examine the likelihood that our analyses had missed an important confounder not provided by the TDHS data. The sensitivity analysis was conducted to determine how severe an unmeasured confounder would have to be to affect our results. For a presumably confounded odds ratio to be depressed from 2.52, for example, to 1.50, we would have had to miss an unmeasured confounder. However, such a confounder either must be related to long driving times (>6 hours/day) with an odds ratio greater than 3 *and* associated with knee pain with an odds ratio of 4 or greater or must be related to long driving times with an odds ratio of 2 *and* associated with knee pain with an odds ratio greater than 5. Because no such strong factors have ever been documented, and because our association had been adjusted for a large set of variables retained in the multiple logistic model, we considered the odds of having missed such important factors to be small.

As a secondary analysis of existing data, our study had several limitations. First, the TDHS baseline data depended on subjective reporting to estimate the frequency of musculoskeletal disorders. No further objective information was available on the nature of the reported knee pain, such as the sidedness of knee complaints and the clinical significance of the observed association between driving and knee pain. Future studies need to include these distinctions, especially when investigating knee pain in relation to early knee osteoarthritis and the resultant disability among professional drivers.

Second, our study may have been limited by shortcomings of the cross-sectional design. Although we employed a widely used occupational study questionnaire to measure the prevalence of knee pain, the Nordic musculoskeletal questionnaire does not include detailed items that assess severity of musculoskeletal symptoms. In a small subset of 319 drivers who were administered questionnaire items on severity of musculoskeletal complaints, 61% of those who had knee pain recalled that they had lost at least 1 day of work in the past year because of knee pain. The average number of lost workdays likely related to knee pain was 4.4 days (range: 1–30 days). Because of the study's cross-sectional design, it is therefore arguable that the TDHS baseline data may overrepresent cases of knee pain with relatively longer symptomatic duration (and probably with less severe underlying knee joint disorders). Counteracting this length-biased sampling is the healthy worker effect, which may either have excluded former drivers who had more severe knee pain (and therefore were forced to retire or quit) from the TDHS baseline data or led to changes of driving duration among symptomatic drivers who remained in the taxicab business. A prospective study should provide a more appropriate design to address these complexities.

CONCLUSIONS

Our exploratory analyses of the TDHS baseline data revealed a strong and robust association between long driving times and knee pain. The public health impact of work-related knee pain among professional drivers could be substantial. For this reason, findings from our cross-sectional study need to be replicated in longitudinal studies and in biomechanical studies that examine the nature and the mechanisms of knee pain and its relationship with early osteoarthritis. ■

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Contributors

All of the authors conceptualized the study and interpreted the results. J.C. Chen, W.P. Chang, and Y. Cheng developed the survey instrument. J.C. Chen performed the analysis and led the writing of the article. L.M. Ryan supervised the data analysis. W.P. Chang and C.J. Chen were the principal investigators of the Taxi Drivers' Health Study.

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The study protocol was approved by the human subjects committee of the Harvard School of Public Health and by the institutional review board of the Taipei Veterans General Hospital, Taipei, Taiwan.

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Estimating Capacity Requirements for Mental Health Services After a Disaster Has Occurred: A Call for New Data

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Objectives. We sought to estimate the extended mental health service capacity requirements of persons affected by the September 11, 2001, terrorist attacks.

Methods. We developed a formula to estimate the extended mental health service capacity requirements following disaster situations and assessed availability of the information required by the formula.

Results. Sparse data exist on current services and supports used by people with mental health problems outside of the formal mental health specialty sector. There also are few systematically collected data on mental health sequelae of disasters.

Conclusions. We recommend research-based surveys to understand service usage in non–mental health settings and suggest that federal guidelines be established to promote uniform data collection of a core set of items in studies carried out after disasters. (*Am J Public Health.* 2004;94:582–585)

In the chaotic aftermath of a disaster, authorities are faced with the need to provide an extensive array of services to the affected population. Such a situation occurred after the terrorist attacks of September 11, 2001, when mental health and other related support systems mobilized to deliver services to persons who were psychologically or psychiatrically affected by the events.

Planning efforts required estimates of both anticipated mental health needs and the capacity required to respond to these needs. The New York State Office of Mental Health (NYSOMH), in conjunction with researchers from Columbia University's Joseph P. Mailman School of Public Health, conducted a mental health needs assessment. Their report focused on persons suffering from posttraumatic stress disorder (PTSD). For this group, they estimated the breadth of the need, the likely number of services required, and sources of payment for care.[1–3] Data were presented regarding the current capacity of the New York State mental health specialty sector, and a general formula to estimate the service capacity that would be required after a disaster appears in an appendix to that report.

The rationale for the formula and its formulation are presented in this article. By envisioning the formula being applied to cover the largest population likely to seek help, information that is currently available to numerically calculate the value of the formula was identified, as were gaps that limit the ability to provide realistic estimates. An examination of these gaps has led to recommendations for local and national data collection that would enhance the potential for appropriate capacity planning following disasters. The formula, when applied in limited scope, has immediate utility for estimating the service requirements of priority populations. An example of this use is given for persons living below 110th Street in Manhattan and who experienced PTSD after the September 11 disaster.

METHODS

Basic Formula

Extended capacity after a disaster has occurred is defined as the service capacity above the usual service delivery levels required; extended capacity is defined in terms of units of service. These units may be converted to monetary or staff requirements. Extended capacity may be required to provide services to disaster victims who experience emotional distress that is severe enough to require a mental health intervention. Victims could include both persons not currently receiving mental health services (new, or incidence, cases) and persons already receiving services whose problems have been exacerbated by the disaster (old, or prevalence, cases). New cases will require services at some rate to be agreed upon, whereas old cases may require services in addition to those they currently receive as a result of exposure to the disaster.[4] The extended capacity requirement for each group is simply the product of the number of persons in the group and the number of anticipated services required as a result of the disaster. The total extended capacity requirement is the sum of the requirements of the 2 groups.

More formally, extended capacity, ΔC, is based on the number of new cases requiring services postdisaster, N_{new}; the number of old cases requiring additional services postdisaster, N_{old}; the average number of services per person required by new cases, R_{new}; and the average number of additional services required per person for old cases, R^{+}_{old}. The equation is as follows:

$$(1) \qquad \Delta C = N_{new} R_{new} + N_{old} R^{+}_{old}.$$

A conservative estimate of extended capacity assumes that old cases will not require any additional services and that new cases will receive services at current or lower-than-current levels. That is, $R_{new} \leq R_{old}$, where R_{old} is the current service delivery rate, $R^{+}_{old} = 0$, and $\Delta C = R_{new} N_{new}$. At the other extreme, old cases may require new services, and new cases may require services at a rate that is higher than the exacerbated rate of old cases. That is, $R_{new} > R_{old} + R^{+}_{old}$, and $R^{+}_{old} > 0$. All other cases are intermediate to these 2 cases.

Range of Possibilities

Time frame. Service requirements will differ in the acute and postacute phases in the aftermath of a disaster. New cases may emerge over time, whereas distress may abate in some persons.

Population groups. Different population groups will have different diagnostic and care-seeking patterns, leading to different service requirements. Population groups can be defined in terms of geographical areas or exposure to the disaster (e.g., "first responders," adults living

closest to the scene of the disaster). They could be further classified demographically (e.g., by age group, by racial/ethnic categories).

Diagnoses/disorders. Victims of disasters are at risk for experiencing a gamut of mental health disorders ranging in type and severity. For new cases, the disorders expected to occur after a disaster include, but are not limited to, acute stress disorder, PTSD, depression, anxiety, panic disorder, and traumatic grief. Diagnoses for existing cases cover all their current diagnoses and possibly new ones similar to those of the new cases.

Services. The types of services that will be required are diagnostic specific and are likely to include assessments, crisis counseling, psychoeducation, psychotherapy, and pharmacotherapy. The number of services required to treat cases will change over time, with higher rates of treatment expected in the initial phases following the disaster.

Sector/service venue. It is well known that even under normal times, persons experiencing mental distress may seek help in service venues other than the organized mental health sector.[5,6] In particular, persons who experience nonpsychotic disorders are quite likely to seek services first from non–mental health specialists. For example, persons with depressive disorders will often turn to primary care physicians,[7] and persons from particular cultural groups may first seek services from traditional healers.[8] Many who experience distress at subclinical threshold levels seek services from the clergy or self-help groups. Regier[5] first noted this collection of providers from whom persons experiencing mental distress seek help and labeled them as members of a de facto mental health system. More than likely, these same providers will be approached by disaster victims for help. To model the extended mental health service requirement following a disaster, data are required on how many of those from different population and clinical groups will seek and receive services from these de facto venues.

Comprehensively, the sectors where persons are expected to seek services include mental health services provided by programs funded, certified, or operated by state offices of mental health; the Department of Veterans Affairs (VA); general hospital emergency rooms; and other non–mental health sectors such as schools, social service agencies, and family agencies (referred to hereafter as "non–mental health sectors"). Services may also be provided by individuals who are mental health specialists in private practices that are not part of a state office of mental health ("mental health specialists"); primary care physicians in private practices, clergy, and self-help groups ("other specialists"); and others in nonformal settings.

Persons with existing severe disorders or persons who experience distress that reaches clinical diagnostic thresholds are likely to be served by a state office of mental health, the VA, or mental health individuals. Persons with disorders that do not reach threshold levels are likely to use the remaining sectors. They may well account for the bulk of the new service needs, especially in the acute phase after a disaster has occurred. The provider list could be expanded to include sectors that serve persons with alcohol and substance abuse disorders, depending on the scope and purview of the capacity assessment.

General Formula

The general formula sums the basic formula in equation 1 over the range of possibilities to provide an estimate of total extended capacity requirements, ΔC. It relates to a length of time after the disaster, T; service sectors, S; population groups that use these sectors, g(S); disorders of the groups that seek services in the sector, d(g(S)); and units of service type u required for a disorder, u(d(g(S))). For a fixed T, total extended capacity requirements are expressed as

$$(2) \qquad \Delta C(T) = \Sigma_u \Sigma_d \Sigma_g \Sigma_S \Delta C\ (T, S, g, d, u),$$

where we have suppressed the notation indicating the sequential dependencies of groups on sectors, diagnoses on groups, and services on diagnoses.

To avoid double counting, the assumption is made that population groups do not overlap and that service requirements are distinct across diagnoses and sectors. (Note, however, that when the formula is used in limited scope, any population group can be singled out and its extended capacity requirement estimated.) The formula also assumes that no 2 diagnoses are associated with the same service requirement. Although a particular service requirement of a person with comorbidities might be counted with respect to each diagnosis, this will not happen if the range of service requirements is restricted to those closely connected to the diagnosis. If this is not possible, the comorbid condition itself could be introduced as a diagnosis and the service requirement could be attributed to the comorbid diagnosis.

It is also possible that service requirements cannot be distinctly ascribed to a sector. Use of multiple sectors for similar needs has been documented for veterans. It has been observed that a small percentage (<5%) of veterans use similar type services from both VA and non–federally funded providers (C. Siegel, PhD, S. Lin, PhD, E. Laska, PhD, unpublished data, 2003). If estimates of the usage of multiple venues for similar services were available, then a model-based adjustment to the total estimate could be made.[9]

Although the estimate of extended capacity does not depend on current service capacities, the ability to provide the services required clearly does. An important step is to estimate whether extended capacity requirements can be met, the projected shortfalls, and the concomitant budgetary and staffing requirements.[10]

Limited Versions

Most likely, however, estimates of extended capacity will be desired for high-priority situations that limit the range of possibilities covered in the general formula. For example, an estimate may be required of the extended capacity needs of a specific population group for special types of disorders with their service requirements, delivered in the service sectors in which these services are apt to be delivered. To obtain estimates limited in scope, $\Delta C\ (T, S, g, d, u)$ is summed over specific subsets that delineate the coverage of the capacity estimate. For example, if a sector S* requires an estimate of its total extended capacity requirement for T months after the disaster, it is $\Sigma_u \Sigma_d \Sigma_g \Delta C\ (T, S^*, g, d, u)$. An estimate of high and immediate priority might consider the extended capacity required to treat, in the formal mental health sector S*, within the first 6 months postdisaster, first responders (population group g*) experiencing PTSD (say disorder d*), where they would require within 6 months an amount u* of specialized treatment *u**. The estimate formula is $\Delta C\ (6, S^*, g^*, d^*, u^*)$. Estimates restricted to geographical areas most directly affected by

the attacks would also be of high priority. In this case, g is held fixed to represent the geographic area, and all else in equation (2) is summed.

RESULTS

Obtaining Data to Valuate the Formula

Little of the information required to estimate extended capacity is available or can be extrapolated from studies on disasters before September 11. Recent studies conducted after September 11[11–13] do provide some new data that are useful for budget justifications and planning for increased staffing requirements.

N_{old} may be obtained directly for some sectors from utilization data related to recent time frames before the disaster. These data are available in New York State for the NYSOMH and for the VA. Some sector usage data can be extrapolated from the Epidemiologic Catchment Area Study[5] and the National Comorbidity Survey.[6] Both studies provide an estimate of the proportion of persons with mental disorders who seek services in these other sectors, and the latter study provides some limited data on actual utilization.

Studies of other disasters and studies mounted soon after a disaster has occurred do provide information on the risk of a disease, given exposure to the new disaster. The risk times the population size is N_{new}.

There are few estimates of R^{+}_{old} and R_{new} available from studies conducted of other disasters. R^{+}_{old} could be informed by the current service delivery rate$=C_{old}/N_{old}$, where C_{old}=current capacity. Local providers and other key informants can be asked to estimate exacerbation rates, but they might find it difficult to make guesses specific to diagnostic groups or service types. Consensus approaches would need to be used to avoid overinflated estimates.

A Valuation of the Formula

One of the most likely estimates to be required immediately after a disaster is the capacity requirements for populations that are close to the disaster and that experience severe emotional distress. Some data have appeared since September 11 that enable an estimate to be made of extended capacity requirements for the New York City adult population living below 110th Street (close to the disaster site) who experienced PTSD. Galea et al.[11] estimated the percentage of new cases with PTSD among this group. They randomly sampled adult persons from the area 5 to 8 weeks after September 11 and administered a telephone interview to assess their psychiatric symptoms. They found that 7.5% of the sample had symptoms severe enough to classify them as having PTSD related to the September 11 attacks.

Persons with this serious diagnosis would most likely require services within the formal mental health specialty sector, but prior studies suggest that not all of these persons will seek services. Boscarino et al.[12] reported that 19.4% of those interviewed in the Galea sample had mental health visits, but estimates for utilization specific to PTSD and specific to symptoms related to the disaster were not provided. Kessler et al.[14] reported that 28% of persons with PTSD sought services, and we used this higher estimate in our calculation.

The population size of adult persons living in Manhattan below 110th street is 919 000. Assuming no exacerbation of symptoms in old cases, a conservative estimate of extended capacity requirements is based on the number of new cases, N_{new}, that will emerge and their rate, R_{new}, of service usage. The calculation of N_{new} is $.075 \times .28 \times 919\,000 = 19\,299$. Jack and Glied[3] concluded that in a 6-month period, treatment among those with a diagnosis of PTSD should consist on average of 7 outpatient visits and 6 monthly medication visits at a cost in New York City of approximately $1500 per person. Using these data for R_{new} in the conservative version of the formula provides an estimate of an extended capacity requirement of the formal mental health sector for this population/diagnostic group in the 6-month period after September 11 of $R_{new}\,N_{new} = 135\,093$ PTSD visits and 115 794 medication visits, with a total cost of $28 948 500.

CONCLUSIONS

Information that is currently available includes epidemiological estimates of mental disorders and mental health service utilization after disaster incidents,[11,15,16] epidemiological information on the incidence of mental disorders in the general population and the naturalistic use of the various service sectors predisaster,[5,6] and sector-specific administrative service utilization data sets on pre- and postdisaster utilization (e.g., state data sets, VA data sets, county-level data sets).

These data, however, neither adequately cover the scope nor provide the comprehensive information required to accurately estimate the full range of extended capacity requirements. The information available from other disaster incidents does not span all manifestations nor all venues in which persons seek help. Further, these data may be only partially applicable to those affected by a new disaster because of differences in population demographics and service system characteristics.

Sector-specific data sets on utilization are limited to the organized specialty mental health services and are unavailable for the sectors most likely to be used after a disaster has occurred (e.g., general hospital emergency rooms, non–mental health sectors, mental health individuals, private primary care physicians, other non–mental health individuals). Data that are reported on these latter sectors in epidemiological studies conducted to date[5,6] provide information on the likelihood of using the sector and offer only limited data on actual utilization, with estimates based on small sample sizes. Other administrative data sets on utilization that are available are payer specific (e.g., Medicaid data sets, behavioral health care data sets). Although useful for examining the disaster impact on payers, they are less useful for planning for services in the locations where they are needed.

Finally, although there are resource data on the number of persons in a given profession who are capable of providing mental health services, these numbers are not readily convertible to estimates of current mental health capacity. Persons in these professions (e.g., social workers) may already be included in other sector counts or may not provide mental health services. If they do provide such services, the amount provided is unknown.

There are 2 classes of information that would be useful for estimating service requirements related to disasters. The first is current service usage of the various sectors of the de facto mental health system. This information would enable natural pathways to care to be identified, current capacities to be documented better, and multiple use of sectors to be understood better. The second class of information is data that are collected more systematically

about future disaster incidents and their mental health sequelae. These 2 data sets would enable better modeling of needs that would emerge after a disaster occurs.

One effective way to collect sector usage data would be through a 2-part survey, a provider inventory and a survey of usage of a provider by persons with mental health problems. The first part would inventory, within a sector, individuals and agencies that are capable of providing both formal and informal mental health services, producing in effect a resource directory for that sector. The coverage area of the survey should coincide with geographical areas that have been designated as service areas for disaster response. Mounting such a survey in a large urban area such as New York City would be a daunting task, but once computerized mechanisms are in place, the resource could serve as an invaluable management tool if a disaster were to occur (as well as in normal times). It would increase the ability to coordinate services and also provide a basis for estimating training and recruiting requirements, should enhanced capacity be required.

The inventory is needed to conduct the second part of the survey in which data are collected to enable estimation of the number and types of persons with mental health problems who are seen by each provider.

The NYSOMH Patient Characteristics Survey provides one approach that could be followed. Providers of mental health services that are funded, contracted, or operated by New York State are surveyed on the characteristics and services used by persons seen in a representative week. If data are collected during periods of normalcy, the 1-week counts can be annualized or inflated to other time periods using a statistical method currently employed by the NYSOMH.[17,18] Analogously, for a particular sector, all providers in the sector could be surveyed using a 1-week time frame to ascertain the number of persons using their services for mental problems, their characteristics, and their service use. For greater precision, screening of persons seen by the provider for mental disorders could also be part of the survey.

Because full census surveys might not be feasible, sampling strategies could be employed, especially if details on the types of mental distress that manifest themselves are to be collected. During times of normalcy, these data can be adjusted to establish base capacity rates. If the survey is repeated after a disaster, perhaps at several different time points, more could be learned about manifesting problems and the new capacities that have emerged to deal with them. Data collected in this manner would facilitate the parsing of government budget allocations for disaster situations to the sectors in proportion to the assistance they provide to the population in need.

Other information needs to be extrapolated from data of studies of disaster situations. Currently, investigators collect data according to their own protocols, resulting in studies having limited commonality in data elements. Guidelines are needed for data collection of at least a core set of items. This could include specific details of the disaster, specified time frames, delineated population groups, specification of problems, specific treatment system variables, time frames to report duration of service needs, specific outcomes, and bases for cost estimates. Having such data would facilitate synthesis and extrapolation to other disaster incidents.

Developing such guidelines will require a federally sponsored effort and mandates to establish a core set of items to be uniformly collected. With such data, when new disasters occur, needs assessment models could be used to relate the nature of the disaster to the nature and extent of the problems that would be expected to arise and the capacity required to deal with them. ■

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The Economic Burden of Hospitalizations Associated With Child Abuse and Neglect

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Objectives. This study assessed the economic burden of child abuse–related hospitalizations.

Methods. We compared inpatient stays coded with a diagnosis of child abuse or neglect with stays of other hospitalized children using the 1999 National Inpatient Sample of the Healthcare Costs and Utilization Project.

Results. Children whose hospital stays were coded with a diagnosis of abuse or neglect were significantly more likely to have died during hospitalization (4.0% vs 0.5%), have longer stays (8.2 vs 4.0 days), twice the number of diagnoses (6.3 vs 2.8), and double the total charges ($19 266 vs $9513) than were other hospitalized children. Furthermore, the primary payer was typically Medicaid (66.5% vs 37.0%).

Conclusion. Earlier identification of children at risk for child abuse and neglect might reduce the individual, medical, and societal costs. (*Am J Public Health.* 2004;94:586–590)

In 1999, an estimated 826 000 children were abused or neglected nationally.[1] This represents a victimization rate of 11.8 per 1000 children. It was further estimated that 1100 children died as a result of abuse and neglect. Such estimates are surely conservative because victims of abuse and neglect are often not identified, and even if suspected, abuse and neglect are underreported.[2–7] Although the personal costs in pain and suffering to victims and their families cannot be calculated, in this study we assess the burden of hospitalizations associated with child abuse and neglect. By demonstrating the substantial costs of tertiary care for victims of child abuse and neglect, we can justify increased support for primary prevention.[8,9]

Several studies have demonstrated that hospitalized children who are identified as abused or neglected have longer hospital stays, more severe injuries, worse medical outcomes, and higher hospital charges, and such children are more likely to die during the current hospitalization compared with other hospitalized children.[8–12] Most of this research is based on reviews of medical records conducted in pediatric tertiary care hospitals with trauma centers. One study found significant differences of more than $2000 in daily hospital charges for child abuse patients compared with other children admitted to a pediatric intensive care unit, with mean charges for hospitalized victims of $30 684.[8] Another study conducted at a regional pediatric trauma center reported an average hospital charge of $20 359 for victims of child abuse, and this was significantly higher than the hospital charges for other injured children except those for burn victims.[12] To better understand the scope of the problem, we should look at nationally representative data. However, we know of only 2 studies that relied on national data, and neither reported on the costs of hospitalization.[2,10]

Our research objective was to determine 1 aspect of the economic burden of child abuse and neglect on the health care system using a national probability sample of US community hospitals. Specifically, we compared children hospitalized with a diagnostic code of abuse or neglect with other hospitalized children in terms of mean hospital charges, length of hospital stay, and the numbers of diagnoses and deaths during hospitalizations. Although research has demonstrated that diagnostic codes for child and adult abuse are likely underutilized,[13,14] analyses based on these codes can provide valuable information on the medical response to victims of abuse and neglect. Thus, for hospitalizations coded with a diagnosis of abuse or neglect, we expect higher hospital charges, longer hospital stays, and more comorbidities compared with other hospitalized children, as well as more deaths during hospitalization.

METHODS

We conducted secondary data analyses of the 1999 Nationwide Inpatient Sample of the Healthcare Costs and Utilization Project (NIS-HCUP).[15] The sampling design for the NIS-HCUP was a stratified random sample of hospitals with all discharges included from each selected hospital. The 1999 NIS-HCUP provides data on 7 198 929 hospital inpatient stays at 984 hospitals in 24 states, thereby approximating a 20% stratified sample of US community hospitals. Sample weights are provided in the NIS-HCUP to enable data users to produce national estimates.

Identification of Cases

All inpatient hospital stays of children aged 18 years and younger were selected (n=1 371 835). Of these, two thirds (64.8%) had neonatal/maternal diagnoses/procedures. Because these patients were less likely to have diagnoses of abuse or neglect, all new mothers and neonates aged less than 1 day were omitted, thereby resulting in 636 802 inpatient hospital stays for these analyses. Among the excluded cases, there were 19 coded with child abuse or neglect: 15 were neonatal/maternal, and 4 were 0 days old.

To identify cases of child abuse and neglect, we used diagnostic codes from the *International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM).*[16] In 1996, the single code for child maltreatment syndrome was expanded to better specify the forms of abuse: physical, sexual, emotional/psychological, neglect, and shaken infant syndrome (see the list of codes presented in Table 1).[17] The NIS-HCUP provides a primary diagnosis and up to 14 secondary diagnoses

TABLE 1—Estimated 1999 US Hospital Inpatient Stays Coded With a Diagnosis of Child Abuse or Neglect

ICD-9 Code	Types of Abuse	Percentage With Diagnosis of Abuse	Weighted n[a]
99550	Child abuse, unspecified	4.6	217
99551	Child emotional/psychological abuse	0.5	25
99552	Child neglect (nutritional)	16.0	761
99553	Child sexual abuse	8.9	426
99554	Child physical abuse	38.8	1849
99555	Shaken infant syndrome	21.1	1008
99559	Other child abuse and neglect	6.5	311
	Two or more types of abuse coded	3.6	173
Totals		100	4771

Note. ICD-9 = The International Classification of Diseases, Ninth Revision, Clinical Modification.
[a]Represents population estimates based on weighted data for n = 966.

for each inpatient stay. We selected inpatient stays with any of the *ICD-9-CM* diagnostic codes of child abuse or neglect as the subset of cases of abuse-related hospitalizations. Five cases coded as adult abuse but involving patients younger than 18 years were recoded to child abuse. Remaining hospitalizations constituted our comparison group.

Demographic Variables

Age at the time of admission, gender, race, and income are presented for both groups (see Table 2 for breakdown of variables). Age was provided in years, with zero indicating infants younger than 1 year. For race, Hispanic, Asian/Pacific Islander, Native American, and other were collapsed into 1 category called Other, and because race was not provided for nearly 20% of patients, we created a separate category labeled Unknown for inclusion in analyses. Income is based on 4 categories representing the median income for the patient's zip code. Despite caveats about the use of zip codes as proxies for income,[18] this variable is the best approximation available in these data. Another proxy for income is the expected primary payer for the hospitalization, which is also reported.

Medical Discharge Variables

Admission source (e.g., emergency vs routine), whether or not the patient died during hospitalization, the length of hospital stay in days, the number of diagnoses and procedures, and total charges are compared and reported for both groups.

Hospital Variables

The location of the hospital (i.e., urban or rural), whether or not it was a teaching hospital, and region of the country are also reported for both groups.

Analysis

Using sample weights provided in the NIS-HCUP data, we produced national estimates of hospitalizations coded with a diagnosis of abuse or neglect. Analyses were done with SAS (SAS Institute Inc, Cary, NC), and SUDAAN (Research Triangle Institute, Research Triangle Park, NC) was used for calculating variance and assessing statistical significance that takes into account the sampling design. Unless otherwise specified, only weighted data are reported. We estimated the overall percentage of US hospitalizations coded with a diagnosis of child abuse or neglect, along with a breakdown of the types of abuse. Statistical comparisons between child abuse–related and other hospitalizations are presented; we used χ^2 tests for categorical variables and *t* tests for means. Significance tests of total charges and length of stay were based on analyses that used log transformations to adjust for skew. Odds ratios with confidence intervals are reported when appropriate. One-way analysis of variance was used to compare total charges between groups and among the types of abuse.

RESULTS

In the 1999 NIS-HCUP, there were 966 cases of children hospitalized with 1 of the diagnostic codes for abuse or neglect, providing a national estimate of 4771, or 0.15% of US hospitalizations of children aged 18 years and younger, after neonatal or maternal diagnoses/procedures and neonates younger than 1 day were omitted. Physical abuse was coded most often (38.8%), followed by shaken infant syndrome (21.1%), child neglect (16.0%), sexual abuse (8.9%), child abuse unspecified (4.6%), and emotional/psychological abuse (0.5%) (Table 1). Two or more diagnoses of abuse were coded in 3.6% of cases. Abuse or neglect was the primary diagnosis 40.2% of the time.

Overall, we found significant differences between hospital stays coded with a diagnosis of abuse or neglect and other hospital stays in the demographics of the child/parent, medical utilization, and hospitals sampled.

Demographically, children whose hospital stays were coded with a diagnosis of abuse or neglect tended to be younger on average (2.7 vs 5.2 years; $P<.0001$) (Table 2). In analyses not shown, 49.2% of those coded with abuse or neglect were younger than 1 year compared with 40.8% of those not coded as such. Nearly one half of hospitalized children were White, but they represented less than one third of the group coded as abused or neglected ($P<.0001$). Hospitalizations of Black children and those hospitalized without a racial classification were proportionally more likely to be coded as abused or neglected. The median income based on the patient's zip code indicates that those coded as abused or neglected were significantly more likely to be in the lower income categories ($P=.0027$). Race continued to discriminate between the abused and not-abused groups even after we controlled for income categories. Medicaid was the expected primary payer for two thirds (66.5%) of the hospitalizations of the abused or neglected group compared with almost one third (37.0%) of the hospitalizations of other groups, and the opposite tendency was observed for private payers (24.0% vs 54.5%; $P<.0001$). The gender of the hospitalized children did not differ between the 2 groups.

TABLE 2—Estimated 1999 US Hospital Inpatient Stays Coded With a Diagnosis of Abuse or Neglect Compared With Those Not Coded With Abuse or Neglect

Variables	Abuse/Neglect Coded	No Abuse/Neglect Coded	*P* Value
Patient/Family Demographic Characteristics			
Mean age, y (SE)	2.7 (0.18)	5.2 (0.13)	<.0001
Gender (% male)	54.5	54.4	.9375
Race, %			<.0001
Black	20.1	16.0	
White	30.9	46.6	
Other (Hispanic, Asian/Pacific Islander, Native American, and other)	14.3	17.6	
Unknown	33.9	19.9	
Median income for patient's zip code, %			.0027
$1–$24 999	10.6	8.4	
$25 000–$34 999	38.3	32.9	
$35 000–$44 999	30.5	30.9	
≥$45 000	20.6	27.9	
Expected primary payer, %			<.0001
Medicaid	66.5	37.0	
Private including HMO	24.0	54.5	
Other	9.5	8.5	
Medical/Hospital Stay Characteristics			
Admission source, %			<.0001
Emergency room	58.5	37.6	
Routine/birth/other	27.3	56.9	
Another hospital or facility	14.2	5.3	
Court/law enforcement	0.11	0.22	
In-hospital deaths (% died) (OR = 8.82, 95% CI = 6.19, 12.60)	4.0	0.47	<.0001
Mean length of stay, d (SE)	8.2 (0.59)	4.0 (0.08)	<.0001
Mean number of diagnoses (SE)	6.3 (0.19)	2.8 (0.05)	<.0001
Mean number of procedures (SE)	1.3 (0.092)	0.8 (0.04)	<.0001
Mean total charges, $ (SE)	19 266 (1646)	9513 (458)	<.0001
Hospital Characteristics			
Region, %			.0004
Northeast	17.5	18.9	
Midwest	28.7	17.2	
South	33.9	46.7	
West	19.9	17.4	
Location of hospital (% urban) (OR = 1.63; 95% CI = 1.13, 2.34)	91.2	86.4	.0029
Status of hospital (% teaching) (OR = 2.94; 95% CI = 2.33, 3.71)	81.8	60.5	<.0001

Notes. HMO = health maintenance organization; OR = odds ratio; CI = confidence interval. Analyses are based on weighted data: population estimate = 3 123 626.

Hospitalized children whose stays were coded with a diagnosis of abuse or neglect were significantly more likely to be admitted through the emergency room than routinely ($P<.0001$) compared with children not coded as abused or neglected; and they were nearly 9 times more likely to die during hospitalization (odds ratio [OR]=8.82, 95% confidence interval [CI]=6.19, 12.60), resulting in an estimated 190 deaths. On average, those coded with abuse or neglect compared with those not coded as abused or neglected spent twice the number of days (8.2 vs 4.0), had twice the number of diagnoses (6.3 vs 2.8), had more procedures (1.3 vs 0.8), and had double the total charges ($19 266 vs $9513).

A higher percentage of hospitalizations with a diagnosis of child abuse and neglect were coded in the Midwest (28.7% vs 17.2%) and a lower percentage in the South (33.9% vs 46.7%). Medical staff at hospitals located in urban areas were significantly more likely to have coded abuse or neglect compared with hospitals located in rural areas ($P=.0029$); and teaching hospitals were nearly 3 times more likely to have coded abuse or neglect compared with nonteaching hospitals (OR=2.94, 95% CI=2.33, 3.71).

Comparisons of the mean total charges for each type of abuse or neglect and when no abuse was coded are presented in Table 3 and show that the abused or neglected children, *regardless of type of abuse,* had significantly higher average charges. Compared with the mean total charges for hospital stays in which no abuse or neglect was coded ($9513), the highest mean charges were for shaken infant syndrome ($30 311), followed by children who had experienced multiple types of abuse or neglect ($22 070), and then "other child abuse and neglect," which includes multiple forms ($20 267). Because costs at teaching hospitals are known to be higher,[19] we reanalyzed the mean total charges for abuse-related hospitalizations compared with those without abuse or neglect while controlling for teaching status, and the significant differences in average total charges, *regardless of type of abuse,* remained.

DISCUSSION

Our analyses demonstrate that the financial costs for children hospitalized with a diagnosis of child abuse or neglect are considerable compared with those for other hospitalized children. The average total charges were nearly $10 000 more per hospitalization for the abused or neglected group, with an estimated total 1999 cost of nearly $92 million for fewer than 5000 children. In addition to the diagnosis of abuse or neglect, they had twice the number of diagnoses/comorbidities. Sadly, these children were also nearly 9 times more likely to die during hospitalization.

Possible explanations for higher charges for children hospitalized with a diagnosis of abuse

TABLE 3—Mean Total Charges for 1999 US Hospitalizations Coded With an Abuse/Neglect Diagnosis Compared With Those Not Coded With Abuse or Neglect

Types of Abuse	Mean Total Charges, $	SE	n
Not coded as abused: comparison group	9 513	458	3 024 581
99550: Child abuse unspecified	12 163	1774	207
99551 Child emotional/psychological abuse	9 875	2509	25
99552: Child neglect	14 292	2664	729
99553: Child sexual abuse	11 285	2420	421
99554: Child physical abuse	17 593	2003	1 719
99555: Shaken infant syndrome	30 311	2928	979
99559: Other child abuse and neglect	20 267	4747	301
≥ 2 types of abuse coded	22 070	4743	163

Note. Analysis of variance between each type of abuse or neglect and the comparison group is significant at $P < .05$ ($F = 59.56$, $df = 8$).

or neglect include the following: (1) Those so coded may represent the most severe cases, and certain types of abuse may get coded more than other types (e.g., physical abuse and shaken infant syndrome).[2,6] (2) Children with preexisting disabilities may be overrepresented among the abused and neglected[20,21] and also more likely to have higher charges associated with their hospitalizations. (3) Children identified as abused may need to stay in the hospital longer for additional diagnostic evaluations/investigations of the circumstances of the injuries or neglect.[8] None of these explanations, however, negate the fact that child abuse and neglect are costly for both victims and society. Moreover, by excluding hospital stays with neonatal/maternal diagnoses or procedures and neonates aged less than 1 day, which we argued would be less likely to be coded with a diagnosis of abuse or neglect, a bias may have been introduced. But in fact, when these cases were included in the analyses, the difference in mean total charges between the 2 groups was even greater, lowering the mean total charges for the group with no abuse or neglect diagnostic codes to $6879.

We also found children hospitalized with a diagnosis of child abuse or neglect to be more likely to be younger, to be Black, to live in lower-income areas, and to be insured by Medicaid. Other studies support findings of differences in the recognition and identification of abuse according to racial/socioeconomic status.[2–4,22,23] It remains unclear whether some social groups may be at greater risk for abuse or neglect or whether they may be more likely identified/coded as abused because of social conceptions about abuse and about reporting it. National incidence studies of child abuse and neglect have found no racial differences in the incidence of maltreatment, but lower incomes are related to higher incidence rates.[24] Actual demographics of abuse and neglect require additional study to assist in identification of at-risk children.

The limitations of these analyses include the fact that these data reflect only hospitalizations coded with a diagnosis of child abuse or neglect, and therefore these analyses most likely underestimate the numbers of hospitalized children who experienced abuse or neglect. Unfortunately, it is very unlikely that fewer than 5000 children nationally were admitted to community hospitals for medical care associated with child maltreatment.[2,24] These data only provide the total charges incurred for the hospitalization and do not include other costs related to services, medical and otherwise, that victims or their families may incur after discharge—and as such reflect an underestimate of the overall financial burden. The charges also do not include physician services. One study estimates an additional 25% for inpatient physician services,[25] thus increasing the total estimated charges for these hospitalizations in 1999 to nearly $115 million. Additionally, we know that abused and neglected children often experience a lifetime of poorer mental and physical health, requiring more medical and social services.[8,10–11] Therefore, our estimate of the charges for 1 hospitalized victim of abuse or neglect does not reflect the lifetime of health care costs that can result in such cases. Despite these limitations, our research demonstrates the substantial medical bill for the maltreatment of children and sheds light on the social costs of such maltreatment. Furthermore, these analyses provide unique descriptions of the current use of the diagnostic codes for child maltreatment and may provide important information for future efforts to develop guidelines for the appropriate use of these codes.[26]

Child abuse and neglect are underidentified, underdiagnosed, and undercoded. Targeting interventions for those already being abused or neglected as well as medical education for health care providers and interventions for children at risk of abuse and neglect can reduce the individual, medical, and social costs. Notably, teaching hospitals were thrice as likely to code abuse, thereby confirming the importance of medical training for addressing the problem of child abuse and neglect. Future analyses of the diagnoses or comorbidities associated with inpatient stays coded with a diagnosis of abuse or neglect may help prevent future harm to children by providing potential indicators or red flags of present abuse. Our findings provide the economic rationale for policies and programs to prevent child abuse and neglect. ■

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Contributors

S. Rovi and M.S. Johnson conceived of the study. S. Rovi and P.-H. Chen conducted data analyses. All authors interpreted findings and reviewed drafts of the article.

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Human Participant Protection

The University of Medicine and Dentistry of New Jersey's institutional review board approved this research as exempt from review.

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Somali and Oromo Refugees: Correlates of Torture and Trauma History

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Objectives. This cross-sectional, community-based, epidemiological study characterized Somali and Ethiopian (Oromo) refugees in Minnesota to determine torture prevalence and associated problems.

Methods. A comprehensive questionnaire was developed, then administered by trained ethnic interviewers to a nonprobability sample of 1134. Measures assessed torture techniques; traumatic events; and social, physical, and psychological problems, including posttraumatic stress symptoms.

Results. Torture prevalence ranged from 25% to 69% by ethnicity and gender, higher than usually reported. Unexpectedly, women were tortured as often as men. Torture survivors had more health problems, including posttraumatic stress.

Conclusions. This study highlights the need to recognize torture in African refugees, especially women, identify indicators of posttraumatic stress in torture survivors, and provide additional resources to care for tortured refugees. (*Am J Public Health.* 2004; 94:591–598)

Historically, refugees and asylum seekers have had a high probability of experiencing politically motivated torture.[1] The United States has resettled many groups of refugees, including these study populations from Somalia and Ethiopia. Having experienced civil war and a lack of formal government for more than a decade,[2] Somalis have often suffered traumatic events. Oromos claim ongoing political oppression since their territory was incorporated into the country of Ethiopia at the end of the 19th century.[3–5]

Estimating the prevalence of torture in community samples of refugees is extremely difficult, often impossible, and rarely attempted. The political and emotional sensitivity of torture makes it difficult to study, and refugees are challenging groups for research.[6] Between 5% and 35% of refugees have been tortured, according to the most frequently cited review.[7] Existing studies of torture and associated factors typically conducted in refugee clinics and in other treatment settings,[1,6,8–16] report that posttraumatic stress, anxiety, depression, and somatization are common.[17,18] However, these studies have rarely included control groups, generally have had small samples, and cannot address the prevalence of torture survival in communities. Any consequences specifically associated with torture, compared with other traumatic events that refugees commonly experience, still need to be identified and the effects quantified.[19,20]

Only a few studies with large samples ($n > 500$) have examined torture prevalence and posttraumatic stress disorder (PTSD) rates, the focus of our article. From national samples, De Jong et al.[21] studied postconflict populations in Algeria, Cambodia, Gaza, and Ethiopia, finding rates of PTSD ranging from 16% to 37%. The prevalence of PTSD in the 1200 Ethiopians surveyed was 16%, higher for torture survivors ($P < .001$) than for those not tortured. Modvig et al.[22] randomly surveyed 1033 household representatives in East Timor and found a torture prevalence rate of 30%.

Symptom levels tend to be higher in refugee camps than in resettlement populations.[23–26] Mollica et al.,[27,28] studying 993 Cambodians in a Thai refugee camp, found that a third had PTSD. Comparing 526 Bhutanese torture survivors in a Nepalese refugee camp with matched controls, Shrestha et al.[29] found higher posttraumatic stress and anxiety. Van Ommeren et al.[30] subsequently randomly sampled 810 (418 tortured and 392 nontortured) Bhutanese refugees from the same frame (the general source from which the sample population is selected), finding that torture survivors had more occurences of PTSD (43% vs 4%).

Among refugees living in nearby countries, Iacopino et al.[31] found 4% torture prevalence among 1180 randomly sampled households of Kosovars living in Macedonia and Albania. Smaller samples included Senegalese refugees in Gambian camps (16% torture prevalence),[32] Tibetan nuns and lay students tortured in Tibet but living in India (54% anxiety vs 29% in controls),[33] and Burmese political dissidents in Thailand (23% PTSD).[34]

In smaller prisoner populations in Turkey, Paker et al.[35] estimated that tortured Turkish prisoners had significantly more PTSD compared with other prisoners, and Basoglu et al.[36] showed higher rates of lifetime (33%) and current (18%) PTSD than controls.

In Western resettlement populations, Thonneau et al.[37] found 8% torture prevalence among 1194 refugee applicants to Canada. Smaller samples have shown PTSD rates among Cambodians of 12%,[38] 50%,[39] and even 86%.[40]

Our 5-year, multiphased, community-based epidemiological study aimed to identify demographic characteristics, pre- and postmigration factors, torture prevalence, and the association of torture survival with health and social problems in 2 resettled refugee communities. The findings of a quantitative survey of 1134 adult participants are presented here. Two subsequent surveys, using subsets from this sample, will compare torture survivors with nontortured refugees using (1) structured instruments to assess symptoms, disability, coping, social support, and family function and (2) a brief neurological screen to identify soft signs of impairment and the Schedules for Clinical Assessment in Neuropsychiatry[41] to make *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*,[42]

and *International Classification of Diseases, 10th Revision*[43] psychiatric diagnoses.

METHODS

Questionnaire Development and Interview Administration

Questionnaires were administered by 8 Somali and Oromo staff with experience in health care or interviewing and backgrounds in medicine, law, engineering, biology, or social services.

Investigators provided training in survey methods and research protocols. To prevent participants from associating the study with torture, we used the terms *health problems* and *physical or psychological abuse* instead of the word *torture* in the informational documents and consent forms used. Three authors (M.S., C.R., D.J.) held biweekly sessions with staff, providing debriefing and ongoing supervision during data collection.

Because using structured assessment instruments for the entire study was too expensive and impractical for a large sample, we chose a multiphased design. We developed a comprehensive closed-ended questionnaire for this first interview phase. Although questionnaires to assess trauma and torture are described in the literature,[44–47] their validity for East African refugees has not been documented. Our questionnaire consisted of 188 questions with 537 response items adapted from published studies and the authors' clinical and research experiences. The questionnaire elicited biographical information; current and earlier life circumstances; health status; and experiences of violence, deprivation, and physical or psychological trauma and torture. Questions were asked in a variety of formats, including yes/no, Likert scale, and checklist. The most sensitive trauma and torture questions were asked at the end of the questionnaire, after staff had the opportunity to gain participants' trust. Checklists, an accepted assessment method for torture techniques,[46] were used, and scales were developed to estimate physical, psychological, and social problems.

Because PTSD is one of the most common and controversial diagnoses associated with torture, the PTSD Checklist (PCL-C) was administered in this interview phase and, for comparison, in the next 2 phases. The PCL-C, a self-report Likert scale with 17 items, has shown high internal consistency and reliability and strong correlation with PTSD diagnosis using the Clinician Administered PTSD Scale.[48,49] Translated versions of the PCL-C into Oromo and Somali produced high reliability with Cronbach's α (.93).

Staff members were matched with participants by gender and ethnicity. The questionnaire was self-administered for literate participants (50.4%) and interviewer administered for those who were illiterate (49.6%). These percentages were not significantly different across the groups exposed and unexposed to torture ($\chi^2=0.36$, $df=1$, $P=.55$). Staff remained with all participants during questionnaire administration to ensure that the participants understood the items and to observe for signs of distress. The questionnaire was translated from English into the Somali and Oromo languages and back-translated using standard techniques.[50–60]

Sampling and Classification

We estimated that a sample size of 1200 would allow us to detect a doubling of the rate of PTSD in the tortured group from a base prevalence of 10% (power=80%, $\alpha=.05$, 2-tailed).

Oromos or Somalis living in the Minneapolis/St. Paul metropolitan area and who were at least 18 years old were eligible. Exclusion criteria included psychological inability to participate or residence in a household where a relative had already been interviewed. Unrelated persons living in the same household were eligible.

Census data were not available for our study because most Oromo and Somali refugees arrived after the 1990 census, and interviewing for this sample (which took place between July 14, 1999, and September 3, 2001) began before the Year 2000 census. Even if census data had been available, the census has historically undercounted refugees and minorities.[61] State data were kept only for initial resettlement of refugees, and community organizations and agencies lacked complete data.

Because a random sampling frame was unavailable, a combination of nonrandom sampling approaches[62–64] and lengthy recruitment (more than 25 months) was used. Sampling approaches included targeting persons associated with community organizations and geographic locations (62%) and sampling by linkage (38%). The participation rate was 97.1% of all invited.

Lacking the ability to assess true representativeness of the sample, we compared the sample demographics with newly available outside data in order to identify any differences between our sample and the underlying populations.[65] The outside data were used to estimate community size, geographic distribution, and demographic feaures and included public school enrollment reports, birth records, and state refugee resettlement data. No large differences were found between the sample and the underlying populations, suggesting that the sample was representative. The outside data allowed us to estimate the Minneapolis/St. Paul populations as 641 Oromos and 6538 Somalis aged 18 years or older. Consequently, the Oromo sample (n=512) may have represented 80% of potential participants, and the Somali sample (n=622) may have represented only 8%.

The United Nation's (UN) definition of torture[66,67] formed the basis for classifying participants as tortured. Key components of this definition include physical or psychological pain and suffering, intentionally inflicted for any reason, based on discrimination and perpetrated by persons acting officially. Although torture can occur in many nonpolitical settings, such as domestic violence or satanic cults, "official" perpetration differentiates the UN definition from others. During administration of the questionnaire, staff clarified the context of traumatic events to ensure compatibility with the UN definition. For Somalis, who have had no formal government since 1991, "official" perpetrators included opposing clans who had taken power.

Participants were classified as torture survivors if they (1) responded in the positive to any of 3 items directly asking whether they had been tortured (Have you been tortured in prison? [Y/N]; Was tortured [marked off on a checklist]; Were you tortured in prison or jail? [Y/N]) and reported experiencing at least 1 identified torture technique item (details available from authors) or (2) reported experiencing 1 of the subsets of torture techniques that investigators considered could be used

only during torture sessions (details available from authors), even if participants responded in the negative to all of the questions about torture exposure.

Data Collection, Management, and Analysis

Staff explained the purpose, procedures, risks, and benefits of the study to potential participants and their rights to refuse participation at any time throughout the interview process. Literate persons read the consent form in the language of their choice, and interviewers read it to those who were illiterate. Staff obtained signed or oral informed consent from subjects before they began the interviews.

Two strategies were undertaken to appraise the level of cooperation and credibility of participants. (1) Staff rated participants after completion of interviews using a 1 to 5 Likert scale, with 1 being the most positive. The median rating was 2 (very cooperative or credible). (2) The questionnaire contained 4 internal validity measures identifying participants with response inconsistency on item pairs,[68] and, for more than 95% of the sample, more missing items,[69] a greater number of unusually virtuous responses,[70](i.e., responses designed to assess if respondents presented themselves in an overly favorable manner) and a greater number of extreme responses.[71]

Those participants identified according to these 4 validity measures were classified as "suspect." On analysis, the suspect group of participants was found to be associated with torture exposure, Oromo ethnicity, and younger age. Results for all participants were compared with a subset that excluded the suspect records. Because subjective ratings of cooperation and credibility were not different between the suspect and nonsuspect groups, results were presented for the entire sample. Data were double entered, and periodic error checks were made. Summary data for each variable were extensively checked, and out-of-range responses were compared with the original paper questionnaires.

The trauma count for each participant was controlled in order to assess any additional effect of torture beyond other types of trauma. Several specific items to measure social, psychological, and physical problems were incorporated into the questionnaire, combined into summative scales, and refined using an item analysis. These scales, meant to provide only a preliminary indication of problem areas, are presented with the Cronbach αs in Table 1.

Descriptive statistical analysis revealed the distribution of variables and the appropriateness of statistical tests for the data. Stepwise logistic regression was used to compare suspect and nonsuspect groups. Bivariate comparisons between torture groups were conducted using χ^2 and t tests. Major outcomes of interest included social, physical, and psychological problems, and total PCL-C scores. For the interval measures, multivariable analysis was accomplished using stepwise multiple linear regression. Regression diagnostics were performed for each model to assess any possible violations of assumptions. Analysis was performed using SPSS version 8 (SPSS Inc, Chicago, Ill) and SAS version 8 (SAS Institute Inc, Cary, NC).

RESULTS

The final sample of 1134 included 622 Somalis and 512 Oromos, 605 men and 529 women. Table 2 describes characteristics of the ethnic/gender subgroups. On average, participants reported experiencing 21 of 61 possible nontorture traumatic events. All but 6 participants reported experiencing traumatic experiences.

Problem Scales and PCL-C

Table 3 displays results of the analyses assessing variables associated with the problem scales and PCL-C. Trauma count and exposure to torture were significant at $P<.0001$ for all these measures. In addition, the number of social problems was greater among

TABLE 1—Problem Scales and Cronbach αs: Minneapolis, Minn, July 14, 1999–September 3, 2001

Social Problems	Psychological Problems/Source of Problems	Physical Problems
Does not speak English easily	Had child die	Starvation before leaving homeland
Does not read English easily	Had death in family in last 6 months	Currently taking medication
Does not have job	Has no good work opportunities in United States	On journey from homeland:
Has less than high school education	Feels stress living in United States	Life-threatening illness
Is separated from family	Bothered by things done in home country	Life-threatening lack of food
Has less money to spend than in homeland	Has trouble sleeping	Life-threatening lack of water
Does not anticipate opportunities for work	Has loss of appetite	Life-threatening serious injury
Does not have good friends	Hears voices	Life-threatening physical assault
Has difficulty caring for monthly expenses	Has thoughts of killing self	Head injuries from torture
Feels alone	Has been to doctor for mental health problem	Physical problems resulting from torture
Has problems getting job	Has intense memories of torture	Still has physical reactions from torture
Family has stayed behind	Has frequent headaches	Has faintness or dizziness
Has problems learning English	Has had changes in appetite	
Has hard time understanding American life		
Likes home food better than American food		
α = .77	α = .63	α = .77

TABLE 2—Characteristics of the Sample by Ethnic/Gender Group

	Total (n = 1134)	Oromo Men (n = 282)	Oromo Women (n = 230)	Somali Men (n = 323)	Somali Women (n = 299)	F Statistic	*P* Value
Age, y, mean (SD)	35.1 (13.9)	31 (12.4)	34.8 (13.9)	37.4 (15.9)	36.8 (14.7)	$11.6_{3,1123}$	<.001
Age at time of leaving home, mean (SD)	27.5 (14.5)	24.6 (12.1)	27.8 (14.4)	28.9 (16.0)	28.4 (14.7)	$5.0_{3,1123}$	.002
Number of traumatic events endorsed, mean (SD)	20.8 (11)	25.3 (10.4)	25.2 (8.2)	14.1 (8.5)	20.2 (11.8)	$83.5_{3,1130}$	<.001
Years between leaving home and arriving in United States, mean (SD)	4.1 (3.2)	3.3 (3.0)	3.1 (2.8)	4.9 (3.2)	4.8 (3.3)	$25.91_{3,1100}$	<.001
Years in United States, mean (SD)	3.4 (3.6)	3.1 (4.2)	3.9 (4.4)	3.4 (3.2)	3.2 (2.8)	$1.9_{3,1108}$	.12
Marital status, no. (%)							
Married, living with partner	319 (29)	68 (25)	93 (41)	69 (22)	89 (30)		
Separated by immigration	264 (24)	78 (29)	34 (15)	86 (27)	66 (23)		
Single	342 (31)	122 (45)	58 (25)	115 (37)	47 (16)		
Separated, divorced, widowed	180 (16)	2 (1)	44 (19)	43 (14)	91 (31)	156.3_{9}	<.001
Education, no. (%)							
No formal education	167 (15)	14 (5)	62 (27)	14 (4)	77 (26)		
Less than high school	418 (37)	104 (38)	103 (45)	117 (36.5)	94 (31)		
High school diploma	292 (26)	58 (21)	31 (14)	115 (36)	88 (30)		
Post high school	245 (22)	99 (36)	33 (14)	75 (23.5)	38 (13)	167.2_{9}	<.001
Speaks English, no. (%)	633 (56)	198 (70)	101 (44)	224 (69)	110 (37)	104.8_{3}	<.001
Receives government aid, no. (%)	83 (7)	3 (1)	28 (12)	12 (4)	40 (13)	46.6_{3}	<.001
Employed, no. (%)	577 (51)	198 (72)	103 (45)	188 (58)	88 (30)	114.1_{3}	<.001
Owns home, no. (%)	64 (6)	25 (9)	26 (11)	6 (2)	7 (2)	34.2_{3}	<.001
Has no permanent address, no. (%)	35 (3)	5 (2)	8 (4)	9 (3)	13 (4)	3.4_{3}	.33
Muslim, no. (%)	990 (87)	204 (72)	185 (80)	313 (97)	288 (96)	115.5_{3}	<.001
Christian, no. (%)	105 (9)	62 (22)	43 (19)	0	0	142.2_{3}	<.001

TABLE 3—Multivariable Analysis of Characteristics and Problems

	Social Problems		Physical Problems		Psychological Problems		Total PCL-C Score	
	β (SE)	*P* Value	β (SE)	*P* Value	β (SE)	*P* Value	β (SE)	*P* Value
Gender: female	1.54 (0.13)	<.0001	(1.0 (0.13)	<.0001	0.16 (0.10)	.0899	(3.35 (0.88)	.0001
Ethnicity: Oromo	(0.71 (0.14)	<.0001	(0.78 (0.14)	<.0001	0.02 (0.10)	.8650	2.29 (0.94)	.0149
Age	(0.01 (0.03)	.7023	(0.02 (0.03)	.5347	(0.02 (0.02)	.3209	0.04 (0.19)	.8336
Married or living with partner	(0.39 (0.13)	.0036	(0.40 (0.14)	.0035	(0.18 (0.10)	.0764	(1.66 (0.91)	.0685
High school graduate	(1.94 (0.13)	<.0001	(0.33 (0.14)	.0159	0.01 (0.10)	.8877	(0.85 (0.93)	.3615
Speaks English	(2.01 (0.14)	<.0001	(0.22 (0.15)	.1289	(0.04 (0.11)	.6848	–0.57 (0.98)	.5606
Has job	(1.27 (0.13)	<.0001	(0.44 (0.14)	.001	(0.18 (0.10)	.0709	(0.55 (0.90)	.5436
Owns home	(0.84 (0.26)	.0013	(0.07 (0.27)	.8027	(0.29 (0.19)	.1381	0.95 (1.85)	.6048
Age at which left home country	0.04 (0.03)	.0979	0.02 (0.03)	.4239	0.05 (0.02)	.0189	(0.01 (0.19)	.9752
Years between leaving home and arriving in United States	0.06 (0.03)	.0472	0.02 (0.03)	.5977	0.02 (0.02)	.3423	(0.35 (0.22)	.1046
Years in United States	0.01 (0.03)	.6347	0.03 (0.03)	.2778	0.06 (0.23)	.0163	(0.20 (0.23)	.3795
Religious practices increased	(0.21 (0.18)	.2570	0.18 (0.19)	.3144	0.08 (0.14)	.5740	3.19 (1.28)	.0131
Religious practices decreased	0.53 (0.24)	.0293	0.25 (0.25)	.3398	0.76 (0.18)	<.0001	4.67 (1.67)	.0051
Trauma count	0.08 (0.01)	<.0001	0.15 (0.01)	<.0001	0.09 (0.01)	<.0001	0.52 (0.05)	<.0001
Exposure to torture	(0.59 (0.14)	<.0001	0.60 (0.15)	<.0001	0.61 (0.11)	<.0001	7.18 (0.98)	<.0001
Adjusted R^2	0.7170		0.5091		0.4962		0.4122	

Note. PCL-C = Posttraumatic Stress Syndrome Checklist.

women, Somalis, and those who had decreased their religious practices since immigrating. English language fluency, employment, high school graduation, marriage, home ownership, and longer residency in the United States were associated with fewer social problems. Male gender and Somali ethnicity were associated with more physical problems, whereas employment, marriage, and high school graduation were associated with fewer physical problems.

None of the assessed factors were associated with fewer psychological problems. More psychological problems were associated with leaving home at an older age, longer residency in the United States, and decreased religious practices since immigrating. Female gender was inversely associated with PCL-C score, whereas Oromo ethnicity and any change in religious practices since immigrating were associated with increased PCL-C scores.

Associations with fewer problems on at least 2 problem scales/PCL-C included employment, high school graduation, marriage, and continued religious practices, whereas more problems were associated with decreased religious practices, high trauma count, and torture exposure. Despite high scores on the scales, less than 1% of the participants requested or accepted referral to mental health services.

Prevalence of Torture and Characteristics of Survivors

Prevalence rates for each torture category were calculated (Table 4). Of the 1134 participants, 44% met criteria for torture exposure and 56% did not. Criteria for classification are described in the Methods section. Only 15% (n=92) met criteria solely by reporting that they experienced 1 or more of the techniques occurring only during torture (false negatives). Conversely, 40 participants were excluded from the torture group because, although they gave a positive answer to a torture question, they did not report experiencing any torture techniques (false positives).[72] Had we accepted, as the only criterion, an affirmative response when asking participants whether they had been tortured, 132 (11.6%) would possibly have been misclassified according to our definition. Fifty torture survivors (10%) reported experiencing only physical or only psychological techniques, whereas the other 90% reported experiencing both.

Torture history varied by gender and ethnicity: men (n=272, 45% of the men) and women (n=228, 43% of the women) had approximately equal exposure to torture, and more Oromos were exposed to torture (n=286, 55%) than Somalis (n=224, 36%). Among ethnic/gender groups, those most often exposed to torture were Oromo men (n=194, 69%) and Somali women (n=141, 47%), followed by Oromo women (n=85, 37%) and Somali men (n=81, 25%). Gender differences were not statistically significant (Table 3), but differences by ethnicity (χ^2= 42.6, *df*=1, *P*<.001) and by ethnic/gender group (χ^2=126.0, *df*=3, *P*<.001) were significant.

Participants in the tortured and nontortured groups differed in several characteristics and in the degree of reported problems (Table 3). Torture survivors were less often married and were older when they left their home countries. No significant between-group differences were found for men or for those employed, more educated, with permanent addresses, or with longer times between leaving their home countries and arriving in the United States.

For both groups, a higher number of traumatic events correlated positively with scores on all problem scales and total PCL-C. The tortured group averaged 13 more traumatic events than the nontortured group (28 vs 15), adding an average of 1 social and 1 psychological problem, 2 physical problems, and 7 points to the PCL-C scale for those exposed to torture. Beyond the general trauma, torture exposure added small but significant increases to the number of psychological and physical problems but not to the number of social problems. For social problems, the estimated adjusted means were 6.46 (SE = 0.11) for those exposed and 7.03 (SE= 0.09) for those unexposed to torture, showing no significant statistical difference.

The most striking correlate of torture exposure was the increase in total PCL-C score. Those exposed to torture averaged an addi-

TABLE 4—Demographic Characteristics of the Sample by Torture Classification

	Exposed to Torture	Unexposed to Torture	χ^2	*P* Value
Entire sample (n = 1134)	502 (44)	632 (56)		
Ethnicity			42.6_{1}	<.0001
Somali, no. (%) (n = 622)	221 (36)	401 (64)		
Oromo, no. (%) (n = 512)	261 (55)	231 (45)		
Gender			0.74_{1}	.3900
Male, no. (%) (n = 605)	272 (45)	333 (55)		
Female, no. (%) (n = 529)	228 (43)	302 (57)		
Married or living with partner, no. (%)	120 (25)	199 (32)	7.79_{1}	.0052
Graduated from high school, no. (%)	230 (47)	307 (49)	0.60_{1}	.4387
Employed, no. (%)	243 (49)	334 (53)	1.55_{1}	.2128
Owns home, no. (%)	30 (06)	34 (05)	0.19_{1}	.6656
Has no permanent address, no. (%)	19 (04)	16 (03)	1.47_{1}	.2255
Current age, mean (SD)	36.78 (14)	33.78 (15)	3.45_{1125}	.0006
Age on leaving home, mean (SD)	29.50 (14)	25.90 (15)	4.19_{1109}	<.0001
Years between leaving home and arriving in United States, mean (SD)	4.04 (03)	4.12 (03)	0.66_{1102}	.5119
Years in United States, mean (SD)	3.04 (04)	3.66 (04)	2.82_{1110}	.0048
Number of traumas endorsed, mean (SD)	28.07 (10)	14.94 (08)	25.02_{1132}	<.0001
Number of social problems, mean (SD)	7.09 (04)	6.43 (03)	3.26_{1132}	.0012
Number of psychological problems, mean (SD)	3.67 (02)	1.74 (01)	19.14_{1132}	<.0001
Number of physical problems, mean (SD)	4.32 (03)	1.72 (02)	18.88_{1132}	<.0001
PCL-C scores, mean (SD)	42.53 (15)	27.17 (10)	18.53_{919}	<.0001

Note. PCL-C = Posttraumatic Stress Syndrome Checklist.

tional 7-point (>10%) increase in PCL-C score beyond the increase due to trauma alone. Using a cutoff PCL-C score of greater than 50 (range 17 to 85) to indicate suspected PTSD, 123 (25%) of those exposed to torture met this criterion, but only 23 (4%) of the unexposed did.

DISCUSSION

Our study interviewed 1134 East Africans, the largest refugee community sample conducted in a resettlement country. As expected, social problems were common throughout the sample, because the early stages of resettlement for all refugees are characterized by many problems in daily living and adjustment.[9,73,74] Factors associated with fewer social problems included speaking English and graduating from high school, which presumably increased the chances for employment and home ownership. Marriage can provide social stability, and longer residency in the United States can allow time for adjustment. High school education, employment, and marriage were associated with fewer physical problems, perhaps because those with fewer physical problems could adjust more successfully. However, none of these factors were associated with fewer psychological problems.

Low scores on at least 2 of the problem scales/PCL-C were associated with employment, high school graduation, marriage, and continuing religious practices since immigration. These factors indicate the importance of education, economic stability, social support, and religion for the well-being of refugees. High scores for trauma and torture, as expected, were associated with more problems.

Torture rehabilitation centers have historically claimed that the more educated men were most often targeted as leaders who would serve as examples to their communities.[1] Our study, in contrast, showed that men and those with higher education levels were no more likely to be tortured than women or those with less education. Although more time in transition to the United States, usually in refugee camps, might be expected to increase the risk of trauma, our study did not find any increase for those experiencing longer transitions.

Except for Somali men (25% of whom were tortured), the prevalence of torture exposure in this community sample was significantly higher than the 5% to 35% often reported.[7] This high prevalence was found despite the expectation that participants would underreport.[8] If these results can be replicated in other refugee communities, 400 000[75] is likely an underestimation of the number of torture survivors living in the United States.

Women were as likely to experience torture as men, an exposure rate not previously reported. Because civilians are increasingly affected by modern warfare and terrorism, it is not surprising that women frequently experience torture.[76,77]

Significant differences in torture exposure by ethnicity and ethnic/gender group were found. Oromos were tortured more often than Somalis, whereas Oromo men and Somali women were the ethnic/gender groups most often tortured. A number of possible explanations can be posited. The very high rates in the Oromo community may reflect long-standing interethnic conflicts. Somali women were more often tortured than Somali men. Anecdotally, Somali men were either killed in their home country or able to escape unharmed, whereas women and children had a more difficult time leaving the country.

Our results suggest that torture's effect may be additive to other forms of trauma. Torture survivors were more likely than other refugees to experience physical and psychological problems, even after we analyzed the group differences using nontorture trauma as a covariate. The most striking correlate of torture exposure in our study was the increase in total PTSD symptoms. Of those tortured, 25% had suspected PTSD compared with only 4% of those not tortured. These rates are comparable to those cited in earlier studies. In addition, PTSD also showed the greatest additive effect of torture. In another study, Silove et al.[19] found an additive effect of PTSD in Tamil torture survivors in Australia after they accounted for other traumatic events. Although the published literature is contradictory, our findings also support a possible dose–response relationship between torture and PTSD.

Several limitations were inherent in the study design. Because we could not use true random sampling methods, our prevalence results are estimates. However, our analyses indicate that an estimated 80% of the Oromo community, nearly the entire Oromo "village," may have been sampled. With such a comprehensive sample of Oromo, the extremely high torture prevalence of Oromo men (69%) is plausible. The study variables were measured at the same time, a limitation shared by all cross-sectional studies. Therefore, inferring causality between torture history and problems was not possible. Although the present analyses strongly support associations between torture history and key problem areas, our 2 follow-up studies will further elucidate these findings. The final clinician-administered diagnostic phase of our research will also assess the duration and frequency of torture experiences in greater depth.

Our study of 2 communities represents only a small proportion of refugees and displaced persons but contributes to the enormous gap in understanding of the effects of torture and trauma on refugee populations worldwide. The capacity to care for survivors falls far short of the need. This study suggests that the shortfall is even greater than previously thought. The 31 torture rehabilitation centers in the United States and the more than 200 worldwide[78] cannot treat all torture survivors.

From a public health perspective, our findings warrant screening refugees for a history of torture at least among East African and women refugees. However, less than 1% of our highly traumatized sample either requested or followed up a referral to Western mental health services. This highlights the need to investigate the reasons for underutilization and to adequately address the needs of torture survivors living not only in Minnesota but throughout the world. ■

About the Authors

At the time of the study, James M. Jaranson was with the HealthPartners Division of Behavioral Health and the departments of Epidemiology and Psychiatry at the University of Minnesota, Minneapolis. James Butcher was with the Psychology Department, University of Minnesota. David Robert Johnson and Joseph Westermeyer are with the Veterans Administration Medical Center and the Psychiatry Department, University of Minnesota. David Rob-

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Contributors

All authors contributed to the conceptualization of ideas, interpretation of findings, and review of the article. J. Jaranson (principal investigator) conceived the study, drafted the article, and supervised operations and personnel. J. Butcher contributed to design and developed the questionnaire. L. Halcon contributed the epidemiological perspective for the research. D.R. Johnson contributed to design and provided insights from his work at the Center for Victims of Torture. C. Robertson contributed to design from the public health nursing perspective. K. Savik supervised the analyses of the data and the research assistants. M. Spring was project coordinator, supervised the interview staff, ensured the quality of the data, and provided a cross-cultural perspective. J. Westermeyer contributed to design with his extensive background in cross-cultural psychiatry.

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Human Participant Protection

The University of Minnesota institutional review board and human subjects protection program and the HealthPartners Foundation institutional review board approved the study before interviewing began and subsequently approved protocol changes, consent forms, and annual progress reports.

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Gender Differences in Long-Term Health Consequences of Physical Abuse of Children: Data From a Nationally Representative Survey

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Objectives. This study investigated the effects of physical abuse in childhood on health problems in adulthood and assessed gender differences in these associations.

Methods. We used data from 8000 men and 8000 women who were interviewed in the National Violence Against Women Survey. We used multivariate logistic regression to test for main and interactive effects and conducted post hoc probing of significant moderational effects.

Results. Men were more likely than women to have experienced physical abuse during childhood. Whereas abuse had negative consequences for both boys and girls, it was generally more detrimental for girls.

Conclusions. Findings suggest the need to consider gender differences and long-term adverse health consequences in the development of intervention strategies to address physical abuse in childhood. (*Am J Public Health.* 2004;94:599–604)

The public health significance of physical abuse in childhood is manifested by its incidence and prevalence[1,2] and also its long-term psychological and physical health consequences. In its most recent report, the Administration on Children, Youth, and Families estimated physical abuse incidence rates for boys to be 2.1 per 1000 and for girls to be 2.2 per 1000.[1] In terms of prevalence, national data reveal that approximately 1.5 million children have experienced physical abuse.[2] Psychological and behavioral problems that have been found to be associated with physical abuse in childhood include poorer academic and intellectual outcomes, posttraumatic stress disorder, depression, substance abuse, personality disorders, suicidal behavior, and aggression.[3–15] Physical health problems associated with physical abuse in childhood include gastrointestinal problems, greater physical functional disability, more physical health symptoms, and more hospitalizations.[16–20]

According to the National Research Council's Panel on Child Abuse and Neglect, there is little research on gender differences in the consequences of child abuse. The panel recommended that research be conducted to determine whether there are differential consequences of child abuse for boys and girls.[21] The lack of research on gender differences is likely because most studies on the consequences of child maltreatment have focused on females.[22] Studies that have included males have typically examined the consequences of maltreatment for males and females separately and have not tested for the interaction between gender and maltreatment, or compared magnitudes of associations across gender. Further, most of these studies have not focused on the effects of physical abuse per se, but rather on maltreatment in general.[10,15,23–25]

Studies that have examined gender differences in the long-term consequences of child abuse have produced mixed findings. Some of these studies have focused on sexual abuse,[26] some on physical abuse,[27] and some on maltreatment in general.[15,24] Although findings have been somewhat inconsistent, by and large, the results suggest that females are more affected by child abuse than males. For example, although both men and women who had experienced physical abuse in childhood were more likely to have higher lifetime prevalence rates of anxiety disorders and alcohol abuse/dependence than their nonabused counterparts, only female victims were at increased risk for a major depressive disorder or drug abuse/dependence.[27] Similarly, in a sample of adult prisoners, child maltreatment (combined sexual, physical, and emotional abuse) was more strongly related to depression, suicidal behavior, and substance abuse in women than in men.[24] Consistent with this, in one of the most rigorous longitudinal studies of child maltreatment to date, Widom and White[15] found that abused and neglected females, but not males, were at significantly higher risk for substance abuse/dependence than their nonabused counterparts.

The purpose of the current study was to test for gender differences in the associations of physical abuse in childhood with health problems in adulthood. Data are from a large, nationally representative survey conducted in the United States with 8000 men and 8000 women. This study extends the literature on child abuse by including a large sample of men and by specifically testing the differential effects of physical abuse on health problems for males and females.

METHODS

Sample and Procedures

Data for this study came from the National Violence Against Women Survey. Respondents were told that the survey was about personal safety. The study was conducted by the Center for Policy Research and was jointly funded by the National Institute of Justice and the Centers for Disease Control and Prevention. Data were collected between November 1995 and May 1996. The sample of 8000 men and 8000 women was derived using random-digit dialing among households with telephones in all 50 states and the District of Columbia. The participation rates for women and men were 72% and 69%, respectively. Before the interview, respondents were informed that their participation was voluntary and that their answers would be kept confidential. Computer-assisted telephone interviewing was used. A Spanish-language version of the survey was used for Spanish-speaking respondents. For more information on the sampling procedures, see Tjaden and Thoennes.[28]

Measures

Predictor Variable: Physical Abuse in Childhood. Physical abuse in childhood was assessed using 12 questions from the Conflict Tactics Scales.[29] Respondents were asked if they had experienced as a child (*child* was not defined) any of 12 violent behaviors perpetrated by a parent, stepparent, or guardian. The violent behaviors included having something thrown at [them]; being pushed, grabbed, or shoved; having hair pulled; being slapped or hit; being kicked or bitten; being choked or experiencing attempted drowning; being hit with some object; being beaten; being threatened with a gun; being threatened with a weapon other than a gun; having a gun used on [them]; and having another type of weapon used on [them]. Respondents were classified as victims (1=yes; 47%) or nonvictims (0=no; 53%) based on whether they had experienced any of these violent behaviors as a child.

Dependent Variables: Health Problems in Adulthood. We included in the analyses only those health problems for which we were able to determine the onset and thus could create the correct temporal sequence (i.e., occurrence of childhood abuse preceded onset or occurrence of health problems). *Physical injury* assessed whether after the age of 17 years respondents had sustained a serious injury (e.g., head injury) that was disabling or interfered with their normal activities (8% said yes). *Chronic physical health condition* assessed whether after the age of 17 years respondents had acquired a chronic physical health problem (e.g., high blood pressure) that was disabling or interfered with their normal activities (11% said yes). *Chronic mental health condition* assessed whether after the age of 17 years respondents had acquired a chronic mental health problem (e.g., depression) that was disabling or interfered with their normal activities (2% said yes). *Alcohol use* assessed whether respondents drank alcohol every day or nearly every day during the past 12 months (6% said yes). *Drug use* assessed respondents' past-month use of tranquilizers (5% said yes), prescription pain killers (10% said yes), antidepressants (4% said yes), and illegal drugs such as marijuana, crack, heroin, or angel dust (3% said yes). *Current perceptions of unfavorable physical health* assessed whether respondents perceived their health to be fair or poor (12% said yes) rather than good, very good, or excellent.

Demographics and Other Violence Experienced. We controlled for 6 demographic variables: gender, marital status, employment status, education, race, and age. We also controlled for sexual abuse occurring in childhood. This variable assessed whether respondents had experienced completed or attempted forced vaginal, oral, or anal intercourse before the age of 18 years.

Interaction Term. We computed an interaction term to represent the cross-product of gender and physical abuse in childhood.

Statistical Analysis

First, we examined the demographic composition of the total sample, as well as for males and females separately. Second, we examined differences between males and females on the reported prevalence of physical abuse in childhood. Third, we conducted bivariate logistic regression analyses to test the main effects of physical abuse in childhood on health problems in adulthood. Fourth, we conducted multivariate logistic regression analyses that tested for the main effects of physical abuse in childhood on health problems in adulthood while controlling for the demographic variables and child sexual abuse. In this way, we could assess the unique contribution of physical abuse in childhood on health problems in adulthood while holding constant other factors (e.g., age) that have also been shown to be related to health problems. Fifth, to determine whether physical abuse in childhood had differential effects on the health measures based on the respondent's gender, we conducted moderational analyses using Baron and Kenny's[30] criteria for testing moderation. Specifically, the interaction term between the predictor (physical abuse in childhood) and the hypothesized moderator (gender) must be significantly related to the dependent variables (health measures) after we controlled for the main effects of both the predictor and hypothesized moderator variable. We conducted the moderator analyses for all of the outcome variables. Last, we conducted post hoc probing of significant moderational effects using Holmbeck's[31] recommended procedures.

RESULTS

Descriptive Statistics

The sample comprised 50% men aged 18 years and older and 50% women aged 18 years and older. Approximately two thirds (64.9%) of the sample were married (men=66.9%, women=62.9%), 68.4% were employed (men=78.2%, women=59.0%), 89.6% had at least a high school education (men=89.9%, women=89.3%), 82.5% were White (men=82.8%, women=82.2%), 9.2% were Black (men=8.5%, women=9.9%), and 8.3% (men=8.7%, women=7.9%) were of other races (e.g., Native American/Alaska Native; Asian/Pacific Islander). The mean age of the sample was 43.33 years (SD=15.76) (men=42.47 (SD=15.33), women=44.19 (SD=16.13)).

Prevalence of Physical Abuse in Childhood by Gender

Table 1 presents descriptive data for males and females on each of the abuse items (these data also appear in Tjaden and Thoennes[28(p38)]). Men were significantly more likely than women to have experienced 7 of the 12 violent behaviors perpetrated by a parent, stepparent, or guardian. Specifically, men were more likely than women to have had something thrown at them that could hurt; to have been pushed, grabbed, or shoved; to have been slapped or hit; to have been kicked or bitten; to have been beaten up; to have been hit with some object; and to have been threatened with a weapon other than a gun during their childhood. No gender differences were found on the other items.

Bivariate Associations Between Physical Abuse in Childhood and Health Problems in Adulthood

Results from bivariate analyses are presented in Table 2 and indicated that respondents who had experienced physical abuse in childhood were significantly more likely than their nonabused counterparts to have sustained a serious injury in adulthood (crude odds ratio [COR]=1.73, 95% confidence interval [CI]=1.54, 1.95), acquired a mental health condition in adulthood (COR=2.05, 95% CI=1.57, 2.67), used alcohol daily in the past year (COR=1.51, 95% CI=1.32,

TABLE 1—Descriptive Data on the Reported Prevalence of Physical Abuse in Childhood for Men and Women Interviewed in the National Violence Against Women Survey: 1995–1996[a]

Violent Act	Males, % (n = 8000)	Females, % (n = 8000)
Had something thrown at you[b]	8.4	6.1
Were pushed, grabbed, or shoved[b]	25.1	15.7
Had hair pulled	12.2	11.4
Were slapped or hit[b]	43.8	33.3
Were kicked or bitten[b]	3.8	3.0
Were choked or experienced drowning	1.2	1.5
Were beat up[b]	6.3	5.5
Were hit with object[b]	25.9	16.9
Were threatened with gun	0.8	0.9
Were threatened with another weapon[b]	2.1	1.4
Had gun used on you	0.4	0.4
Had another weapon used on you	1.3	1.1
Experienced any violence[b]	53.8	40.0

[a]These data also appear in Tjaden and Thoennes.[28(p38)]
[b]Differences between males and females are statistically significant.

1.73), and used the following substances in the past month: tranquilizers (COR=1.74, 95% CI=1.51, 2.01), pain killers (COR=1.45, 95% CI=1.31, 1.61), antidepressants (COR=1.84, 95% CI=1.55, 2.17), and illegal drugs (COR=2.78, 95% CI=2.25, 3.42). There were no significant differences between abused and nonabused respondents in their likelihood of acquiring a physical health condition in adulthood or reporting perceptions of unfavorable health.

Multivariate Results for Effects of Gender and Physical Abuse in Childhood on Health Problems in Adulthood

Findings from the multivariate analyses are also presented in Table 2. Results for the demographic statistical controls are presented although we only describe the main effects for gender and physical abuse in childhood.

Gender. When we controlled for the other demographic variables, childhood sexual abuse, and childhood physical abuse, men were significantly more likely than women to have sustained a serious injury in adulthood (adjusted odds ratio [AOR]=1.33, 95% CI=1.17, 1.52), used alcohol daily in the past year (AOR=2.68, 95% CI=2.29, 3.13), and used illegal drugs in the past month (AOR=2.80, 95% CI=2.21, 3.56). Women were significantly more likely than men to have acquired a physical health condition in adulthood (AOR=0.79, 95% CI=0.71, 0.89), acquired a mental health condition in adulthood (AOR=0.69, 95% CI=0.51, 0.94), used tranquilizers in the past month (AOR=0.61, 95% CI=0.51, 0.71), and used antidepressants in the past month (AOR=0.38, 95% CI=0.31, 0.46).

Physical Abuse in Childhood. When we controlled for demographic variables and childhood sexual abuse, childhood physical abuse was significantly associated with all of the assessed health problems in adulthood. Specifically, respondents who had experienced physical abuse in childhood were significantly more likely than their nonabused counterparts to have sustained a serious injury in adulthood (AOR=1.74, 95% CI=1.53, 1.97); acquired a physical health condition in adulthood (AOR=1.23, 95% CI=1.10, 1.38); acquired a mental health condition in adulthood (AOR=2.36, 95% CI=1.75, 3.18); used alcohol daily in the past year (AOR=1.42, 95% CI=1.24, 1.63); used tranquilizers (AOR=2.10, 95% CI=1.80, 2.46), painkillers (AOR=1.58, 95% CI=1.42, 1.76), antidepressants (AOR=2.13, 95% CI=1.78, 2.54), and illegal drugs (AOR=2.38, 95% CI=1.91, 2.96) in the past month; and reported perceptions of unfavorable health (AOR=1.25, 95% CI=1.13, 1.39). The largest effects were found for mental health problems, tranquilizer use, antidepressant use, and illegal drug use.

Test of Gender × Childhood Physical Abuse Interaction Terms. The interaction term was statistically significant for acquiring a chronic mental health condition in adulthood (Wald=6.48, $P<.01$) and reporting perceptions of unfavorable health (Wald=4.02, $P<.05$). These significant interaction terms indicate that the effects of physical abuse in childhood on health problems in adulthood differed for men and women. However, they did not tell us in what way the effects differed.

To interpret the nature of the interaction terms (i.e., for which group—men, women, or both—physical abuse in childhood was significantly associated with health problems in adulthood), we conducted post hoc probing of the moderational effects. This entailed computing 2 conditional moderator variables and then running 2 more regression models, 1 for each conditional moderator variable. In the first model, men were assigned a score of zero, and in the second model, women were assigned a score of zero. On the basis of these post hoc analyses, we derived adjusted odds ratios, 95% confidence intervals, and *t* values for the associations between physical abuse in childhood and the health measures for both men and women. In this way, we were able to determine whether physical abuse in childhood was significantly associated with acquiring a mental health condition in adulthood and reporting perceptions of unfavorable health for men only, for women only, or for both genders but in differing magnitudes.

Results from these analyses indicated that physical abuse in childhood was significantly related to acquiring a mental health condition in adulthood for women (AOR=3.10; 95% CI=2.13, 4.50; $t=5.92$), but not for men (AOR=1.40; 95% CI=0.86, 2.28; $t=1.36$). Physical abuse in childhood also was significantly related to reporting current perceptions of unfavorable health for women (AOR=1.38; 95% CI=1.20, 1.59; $t=4.44$), but not for men (AOR=1.12; 95% CI=0.96, 1.30; $t=1.38$).

TABLE 2—Odds Ratios and 95% Confidence Intervals for Predicting Health Problems in Adulthood: National Violence Against Women Survey, November 1995 to May 1996

Predictors	Injury		Chronic Physical		Chronic Mental		Alcohol Use		Tranquilizers		Painkillers		Antidepressants		Illegal Drugs		Perceived Health	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Bivariate results[a]																		
Physical abuse in childhood	1.73	1.54, 1.95[b]	0.97	0.88, 1.08	2.05	1.57, 2.67[b]	1.51	1.32, 1.73[b]	1.74	1.51, 2.01[b]	1.45	1.31, 1.61[b]	1.84	1.55, 2.17[b]	2.78	2.25, 3.42[b]	1.09	1.00, 1.20
Multivariate results[c]																		
Unmarried vs married	1.17	1.03, 1.33[b]	0.97	0.86, 1.09	1.95	1.46, 2.60[b]	1.01	0.87, 1.17	1.36	1.17, 1.59[b]	1.14	1.02, 1.28[b]	1.42	1.19, 1.69[b]	2.52	1.99, 3.18[b]	1.27	1.14, 1.41[b]
Unemployed vs employed	1.39	1.21, 1.60[b]	1.37	1.20, 1.56[b]	2.69	1.97, 3.66[b]	0.94	0.79, 1.12	1.76	1.49, 2.09[b]	1.58	1.40, 1.78[b]	1.39	1.15, 1.68	1.29	1.02, 1.62[b]	2.18	1.95, 2.45[b]
Education < high school vs ≥ high school	1.30	1.08, 1.55[b]	1.45	1.25, 1.70[b]	1.35	0.93, 1.95	0.56	0.43, 0.74[b]	1.29	1.04, 1.59[b]	1.47	1.26, 1.72[b]	0.99	0.75, 1.31	1.24	0.91, 1.68	2.56	2.24, 2.91[b]
Black vs White	1.32	1.08, 1.62[b]	2.06	1.72, 2.45[b]	1.22	0.79, 1.90	0.55	0.40, 0.77[b]	0.59	0.42, 0.82[b]	1.48	1.24, 1.75[b]	0.46	0.31, 0.68[b]	1.04	0.77, 1.42	1.49	1.26, 1.77[b]
Other vs White	1.34	1.08, 1.66[b]	1.16	0.92, 1.46	1.05	0.64, 1.74	0.88	0.66, 1.16	1.30	1.00, 1.69	1.35	1.16, 1.62[b]	0.72	0.51, 1.03	0.92	0.66, 1.26	1.70	1.43, 2.02[b]
Age	1.01	1.01, 1.02[b]	1.06	1.05, 1.06[b]	1.01	1.01, 1.02[b]	1.00	0.70, 1.45[b]	1.02	1.02, 1.03	1.01	1.01, 1.02[b]	1.01	1.01, 1.02[b]	0.93	0.92, 0.94[b]	1.03	1.02, 1.03[b]
Male vs female	1.33	1.17, 1.52[b]	0.79	0.71, 0.89[b]	0.69	0.51, 0.94[b]	2.68	2.29, 3.13[b]	0.61	0.51, 0.71[b]	0.96	0.86, 1.07	0.38	0.31, 0.46[b]	2.80	2.21, 3.56[b]	0.99	0.88, 1.10
Sexual abuse in childhood	1.61	1.27, 2.05[b]	1.44	1.25, 1.83[b]	2.68	1.79, 4.00[b]	1.00	0.70, 1.45	1.92	1.48, 2.48[b]	1.46	1.89, 1.78[b]	1.81	1.38, 2.38[b]	2.21	1.57, 3.13[b]	1.63	1.33, 2.00[b]
Physical abuse in childhood	1.74	1.53, 1.97[b]	1.23	1.10, 1.38[b]	2.36	1.75, 3.18[b]	1.42	1.24, 1.63[b]	2.10	1.80, 2.46[b]	1.58	1.42, 1.76[b]	2.13	1.78, 2.54[b]	2.38	1.91, 2.96[b]	1.25	1.13, 1.39[b]

Note. OR = odds ratio; CI = confidence interval.
[a]Odds ratios presented are crude odds ratios.
[b]The 95% confidence interval does not include 1.
[c]Odds ratios presented are adjusted odds ratios.

DISCUSSION

Using data from a large, nationally representative sample, we found that physical abuse in childhood was more prevalent among men than women. We also found that physical abuse in childhood was related to health problems in adulthood for the sample as a whole and adversely affected the mental health and general perceptions of health of women more than men.

The inclusion of a large sample of men and women allowed testing how the association between physical abuse in childhood and health problems in adulthood might vary by gender. Our results are consistent with other studies that have examined gender differences in the effects of child abuse. That is, child abuse is generally detrimental for both males and females. However, female abuse victims appear to be at greater risk for some health problems than their male counterparts. As with prior studies, we found this to be the case with mental health problems.[24,27] However, unlike prior studies,[15,24,27] we did not find gender differences in the effects of child abuse on alcohol or drug use.

Although this study had several strengths, including its large national sample and the statistical test of the interaction between physical abuse in childhood and gender, there were some limitations. First, even though most of the examined relations were statistically significant, the magnitudes of the odds ratios were small. This is not surprising given the amount of time that elapsed between the occurrence of physical abuse in childhood and the occurrence of health problems in adulthood. The elapsed time allowed a large and varied number of experiences that could have affected the associations between child abuse and health problems in adulthood. Second, although we took efforts to ensure that the temporal order between physical abuse in childhood and health problems in adulthood was correct, our findings do not provide firm conclusions that child abuse is causally related to health problems in adulthood. Third, data were retrospective, so recall bias was possible. For 2 reasons, recall bias may be particularly problematic when the stressor under study is child victimization. First, because the amount of time that

elapsed since the childhood victimization allows a great and varied number of experiences, recall bias may be more likely as people perceive past events in light of their later and current experiences.[32] Second, research suggests that emotional trauma might cause memory impairment.[33]

Although our data suggest that physical abuse in childhood is related to adverse health outcomes in adulthood, they do not address why childhood physical abuse would lead to later health problems, or why some of these associations differed by gender. Future research should address variables that might explain or mediate the child abuse–adult health problems relation. Physical abuse in childhood has been found to be related to several potential mediators of the child abuse–adult health problem association, including insecure attachment patterns and more aggression (interpersonal problems), deficits in receptive and expressive language and poor academic achievement (cognitive problems), and an increased likelihood of risky sexual behavior, physical inactivity, and smoking (risky health behaviors).[7,34,35] These potential mediators, in turn, have been found to be associated with health problems.

Our data also do not address why physical abuse in childhood was associated with poor mental health and perceived physical health in adulthood for women but not for men. Some researchers have speculated that child abuse may be a marker for other negative childhood experiences that are more common for girls than for boys.[27] Although we were able to control for childhood sexual abuse, we were not able to control for other early childhood experiences that may account for the observed gender differences. In 1 study, abused and neglected girls were at increased risk of substance abuse and arrests for violent crimes compared with their counterparts even after researchers controlled for such family background characteristics as parental substance abuse, parental arrest, and family welfare status. For boys, however, previously significant bivariate relations between abuse and these outcomes were reduced to nonsignificance after researchers controlled for these family background variables.[15] Some researchers have speculated that females may be more likely than males to engage in self-blame after child abuse, and that this accounts for females' increased risk for mental health problems in adulthood.[36,37] Others have speculated that males and females react to stress differently, with females being more likely to internalize stress symptoms (e.g., depression) and males being more likely to externalize stress reactions (e.g., aggressive behaviors).[15,38] Because our study did not include measures of externalizing behaviors, we could not test this hypothesis. It has also been suggested that females may be more likely than males to evidence problems after abuse because the abuse was more persistent or severe.[36] Data from the current study indicate that males actually experience more forms of physical abuse in childhood than females (men had higher mean score on sum of Conflict Tactics Scale items than women did), so differences in the magnitude of abuse did not account for our finding of gender differences. However, Widom and White[15] suggest that abuse may be tolerated more for boys than girls, and hence nonvictim comparison groups for boys may have more false negatives than nonvictim comparison groups for girls. More research is needed to determine in what ways and why males and females differ in the consequences of physical abuse in childhood.

Although this study did not directly address why there may be gender differences in the association between physical abuse in childhood and health problems in adulthood, the findings suggest the importance of considering potential long-term adverse health consequences in the development of intervention strategies to address physical abuse in childhood. Health care providers should be aware that physical abuse in childhood might be associated with health problems in adulthood, especially among females. Intervening at an early stage may reduce a child's likelihood of developing long-term health sequelae and also reduce the public health burden of child abuse by preventing future health problems. Attention also should be paid to the primary prevention of physical abuse of children. Some efforts to prevent the initial occurrence of child abuse have shown promising results.[39–41]

In sum, little research has focused on differences between males and females in the consequences of physical abuse in childhood. This study helps to address this research gap by examining the moderating role of gender in the associations between physical abuse in childhood and health problems in adulthood. We found that childhood physical abuse was more prevalent among males, and although it was related to adverse health outcomes for both genders, the effect was generally greater for females. These findings can help inform intervention strategies by alerting public health and medical practitioners of the potential for physical abuse in childhood to be related to health problems in adulthood. ■

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Contributors

M.P. Thompson took the lead role in conceptualizing the study, conducting the analyses, and writing the article. J.B. Kingree and S. Desai assisted with study conceptualization and manuscript preparation.

Human Participant Protection

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Prevalence and 3-Year Incidence of Abuse Among Postmenopausal Women

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Objectives. We examined prevalence, 3-year incidence, and predictors of physical and verbal abuse among postmenopausal women.

Methods. We used a cohort of 91 749 women aged 50 to 79 years from the Women's Health Initiative. Outcomes included self-reported physical abuse and verbal abuse.

Results. At baseline, 11.1% reported abuse sometime during the prior year, with 2.1% reporting physical abuse only, 89.1% reporting verbal abuse only, and 8.8% reporting both physical and verbal abuse. Baseline prevalence was associated with service occupations, having lower incomes, and living alone. At 3-year follow-up, 5.0% of women reported new abuse, with 2.8% reporting physical abuse only, 92.6% reporting verbal abuse only, and 4.7% reporting both physical and verbal abuse.

Conclusions. Postmenopausal women are exposed to abuse at similar rates to younger women; this abuse poses a serious threat to their health. (*Am J Public Health.* 2004; 94:605–612)

Abuse, including physical, sexual, financial, or psychological mistreatment, is a serious problem for adults aged 65 years and older.[1] According to the National Elder Abuse Incidence Study, approximately 450 000 older adults in domestic settings were abused, neglected, or both during 1996.[2] This number increases to approximately 551 000 when older adults who experienced self-neglect are included. In a population-based survey of metropolitan Boston, Pillemer and Finkelhor found a rate of elder abuse of 3.2%.[3] In the long-term care setting, 23% of older adults either have been or still are victims of abuse.[4–6]

The public health implications of abuse are its associations with premature mortality and morbidity.[7–13] Lachs and colleagues found that among older adults who were victims of abuse, only 9% were alive 2 years later compared with 40% of older adults who had not been abused.[11] Other studies have found a risk of death for older abuse victims that is 3 times higher than for nonvictims.[12,13] The direct medical costs associated with these violent injuries are estimated to add over $5.3 billion to the nation's annual health expenditures (K. Fullin et al., unpublished data, 1994).

Gender is an important factor in abuse exposure. Worldwide, between 10% and 50% of women report being physically assaulted at some point in their adult lives; 14% to 25% of women seen at ambulatory medical clinics and 20% of women seen in emergency departments have been physically abused.[7–10] Older, postmenopausal women (65 years or older) are more likely than older men to be the victims of all forms of abuse, except for abandonment, even when taking into account the fact that they make up a larger proportion of the aging population.[3,4,14,15] While females made up about 57.6% of the total national population aged 65 years and older in 2000, women were the victims in 76.3% of reports of emotional or psychological abuse, 71.4% of physical abuse, 63.0% of financial or material exploitation, and 60.0% of neglect.[2] Women in the early postmenopausal ages (aged 50–65 years) are exposed to abuse by intimate partners at a rate of 0.5 per 1000 and account for 30% of homicides committed by an intimate partner.[16] Cognitive or physical impairment, or both, is an additional factor in abuse exposure. In a study of mortality due to mistreatment of elders, over 85% of victims of elder abuse had some impairment of their activities of daily living.[2,11]

Unfortunately, most studies examining the associations with abuse exposure have focused on younger women in their childbearing years or on frail, functionally dependent older adults. To date, no study has examined the associations with physical and verbal abuse in functionally independent, cognitively intact, older women. We conducted this study to (1) describe the 1-year baseline prevalence and 3-year incidence of physical and verbal abuse in a cohort of functionally independent older women and (2) examine the sociodemographic factors and health behaviors associated with this prevalence and incidence of abuse.

METHODS

Subjects

We analyzed survey responses from 93 205 women enrolled in the observational study arm of the Women's Health Initiative (WHI). The study design of the WHI and its observational study arm has been described in detail previously.[17] In brief, the WHI is a large, multicenter study with 2 components, an observation study and a clinical trial. Postmenopausal women, aged 50 to 79 years old at baseline, were recruited through targeted mass mailings to voter registration lists, vehicle registration lists, and driver's license lists and invited to participate in the clinical trial. Subjects who were eligible and interested enrolled in 1 or more of the 3 WHI clinical trials: (1) hormone replacement therapy to prevent cardiovascular disease, (2) a low-fat, high-fiber diet to prevent breast and colorectal cancer, and (3) calcium and vitamin D to prevent osteoporosis-related fractures.

Subjects who were ineligible or unwilling to participate in the clinical trials were invited to participate in the observational study, a longitudinal study of health outcomes. In general, women were ineligible for any clinical trial if they had a medical condition with a predicted survival of less

than 3 years, cancer within the last 10 years, or dementia rendering them unable to answer study questions. Women were excluded from the hormone replacement therapy clinical trial study if they were taking hormone replacement therapy and were unwilling to stop use. Women were ineligible for the low-fat diet clinical trial study if they had a baseline body mass index of less than 18 kg/m^2 or if they consumed more than 6000 kcal per day. Women were ineligible for the vitamin D/calcium clinical trial study if they had a history of an osteoporosis-related fracture or medical contraindications to taking study medication. All observational study participants completed several study questionnaires at the time of enrollment, including questions about abuse in the previous year. Three years after enrollment, participants were scheduled for a follow-up clinic visit and administered the same study questionnaires.

To determine the occurrence of physical abuse at baseline, the following question was asked: "Over the past year, were you physically abused by being hit, slapped, pushed, shoved, punched or threatened with a weapon by a family member or close friend?" Subjects could choose from the following responses: (1) no, (2) yes, and it upset me not too much, (3) yes, and it upset me moderately (medium), or (4) yes, and it upset me very much. We classified women who answered yes (responses 2–4) as having been exposed to physical abuse.

To determine the occurrence of verbal abuse at baseline, the following question was asked: "Over the past year, were you verbally abused by being made fun of, severely criticized, told you were a stupid or worthless person, or threatened with harm to yourself, your possessions, or your pets, by a family member or close friend?" Subjects could chose from the following responses: (1) no, (2) yes, and it upset me not too much, (3) yes, and it upset me moderately (medium), or (4) yes, and it upset me very much. We classified women who answered yes (responses 2–4) as having been exposed to verbal abuse. Women who fell into either the physical or verbal abuse categories at baseline determined the exposure group for our abuse prevalence estimates.

Using these questions, women were screened for physical and verbal abuse again 3 years after enrollment. Women who responded no at baseline but who answered yes 3 years after enrollment determined our 3-year incidence estimates of abuse. Any woman who screened positive for physical or verbal abuse at baseline or follow-up was given information about the Domestic Violence Hotline, self-help information about domestic violence, and information about the nearest battered women's shelter. They were also urged to seek help from adult protective services and receive psychological counseling for domestic violence.

Responses to these abuse questions determined 3 mutually exclusive variables: physical abuse only, verbal abuse only, and physical and verbal abuse. These 3 variables became our main outcomes of interest. Our baseline predictor variables included age, race/ethnicity, occupation, marital status, income, education, smoking, alcohol intake, and living arrangement. These predictor variables were chosen on the basis of previous literature suggesting an association of sociodemographics (age, race/ethnicity, education, occupation, and income) and health behaviors (smoking and alcohol use) with elder abuse and intimate partner violence.[18–20]

Data Analysis

We first examined the descriptive statistics of the predictor variables and the abuse variables (at baseline and year 3): no abuse, physical abuse only, verbal abuse only, and combined physical and verbal abuse. Chi-square tests were then performed to examine the bivariate association of the various variables with reports of physical, verbal, and combined physical and verbal abuse vs no abuse. The bivariate analyses examined the association of each variable without adjusting for other factors.

We considered abuse to be the outcome variable and our sociodemographic and health behavior variables to be covariates. Two sets of multivariate regression models were developed for both baseline abuse prevalence data and 3-year abuse incidence data. Complete case analysis was used for all modeling and all explanatory variables were kept in each model, regardless of statistical significance. Thus, estimates of odds ratios for each predictor variable were adjusted for all other variables in the model. Continuous variables were included as linear covariates and categorical variables as indicator levels. Logistic regression models were developed to examine the association of study covariates with each level of abuse status versus no abuse (i.e., a separate model for each level of abuse vs no abuse). All analyses were performed with the *SAS System, Version 8* (SAS Institute Inc, Cary, NC).

RESULTS

Of the 91 749 subjects responding to survey questions on abuse at baseline, 10 199 (11.1%) reported exposure to abuse within the preceding 12 months. Most women in our sample were non-Hispanic White (82.9%), well educated (40.3% had at least a college degree), and married (64.9%) (Table 1). While most women in our sample were not currently employed, those who were employed tended to work in managerial or professional occupations. Of those women who were married, most reported that their spouse was not currently employed. Most women reported drinking less than 1 alcoholic beverage per week and were not currently smokers.

Of the 10 199 women exposed to abuse, 218 women (2.1%) were exposed to physical abuse only, 9083 (89.1%) to verbal abuse only, and 898 (8.8%) to physical and verbal abuse sometime during the year before the baseline interview. Exposure to abuse was associated with being in the younger age cohort (<58 years), being of non-White race/ethnicity, having less than a high school education, having a family income of less than $20 000, being divorced or separated, being a past or current smoker, and drinking more than 1 drink per week (all P values $<.01$) (Table 1).

The associations with exposure to physical abuse at baseline only, after control for other covariates, are shown in Table 2. Black women were 2.84 times more likely (95% confidence interval [CI] = 1.89, 4.26) to report exposure to physical abuse only at baseline than non-Hispanic White women. Other ethnic minority subgroups were also

TABLE 1—Baseline Abuse Prevalence in Cohort of Postmenopausal Women, by Subjects' Characteristics (N = 91 749)

Characteristic	No Abuse, No. (%) (n = 81 550)	Any Abuse (n = 10 199)					
		Physical Abuse Only, No. (%)	*P*	Verbal Abuse Only, No. (%)	*P*	Physical and Verbal Abuse, No. (%)	*P*
Overall		218 (2.1)		9 083 (89.1)		898 (8.8)	
Age, y			.19		<.001		<.001
< 58	22 136 (27.1)	73 (33.5)		3 229 (35.5)		384 (42.8)	
59–64	20 620 (25.3)	57 (26.1)		2 408 (26.5)		210 (23.4)	
65–69	18 367 (22.5)	43 (19.7)		1 821 (20.0)		162 (18.0)	
70–74	14 052 (17.2)	29 (13.3)		1 122 (12.4)		102 (11.4)	
> 74	6 375 (7.8)	16 (7.3)		503 (5.5)		40 (4.5)	
Ethnicity			<.001		<.001		<.001
American Indian/Alaska Native	339 (0.4)	4 (1.8)		56 (0.6)		14 (1.6)	
Asian/Pacific Islander	2 393 (2.9)	8 (3.7)		219 (2.4)		35 (3.9)	
African American	6 682 (8.2)	55 (25.2)		639 (7.0)		137 (15.3)	
Hispanic/Latino	2 950 (3.6)	19 (8.7)		458 (5.0)		103 (11.5)	
White	69 186 (84.8)	132 (60.6)		7 711 (84.9)		609 (67.8)	
Education			<.001		<.001		<.001
0–8 y	1 284 (1.6)	13 (6.0)		150 (1.7)		47 (5.3)	
Some HS/HS diploma/GED	16 141 (20.0)	62 (28.8)		1 541 (17.1)		193 (21.7)	
School after high school	29 242 (36.1)	77 (35.8)		3 527 (39.2)		381 (42.9)	
College graduate or higher	34 232 (42.3)	63 (29.3)		3 784 (42.0)		267 (30.1)	
Family income, $			<.001		<.001		<.001
< 20 000	11 730 (15.5)	65 (33.0)		1 496 (17.8)		283 (34.5)	
20 000–34 999	17 567 (23.2)	39 (19.8)		2 002 (23.8)		204 (24.9)	
35 000–49 999	15 287 (20.2)	40 (20.3)		1 676 (19.9)		118 (14.4)	
50 000–74 999	15 378 (20.3)	29 (14.7)		1 653 (19.7)		114 (13.9)	
> 75 000	15 655 (20.7)	24 (12.2)		1 580 (18.8)		101 (12.3)	
Occupation			<.001		<.001		<.001
Managerial/professional	33 991 (43.7)	66 (33.0)		3 655 (42.4)		260 (31.1)	
Technical/sales/administrative	22 155 (28.5)	48 (24.0)		2 526 (29.3)		254 (30.4)	
Service/labor	13 151 (16.9)	65 (32.5)		1 596 (18.5)		221 (26.4)	
Homemaker only	8 489 (10.9)	21 (10.5)		852 (9.9)		101 (12.1)	
Currently employed (yes)	28 018 (35.4)	69 (32.9)	.44	3 340 (38.1)	<.001	330 (38.5)	.06
Marital status			<.001		<.001		<.001
Never married	3 940 (4.9)	8 (3.7)		335 (3.7)		12 (1.3)	
Divorced/separated	12 379 (15.3)	59 (27.4)		1 665 (18.4)		279 (31.2)	
Widowed	14 717 (18.1)	38 (17.7)		999 (11.1)		127 (14.2)	
Presently married	50 133 (61.8)	110 (51.2)		6 032 (66.8)		475 (53.2)	
Partner's main job			<.001		<.001		<.001
Homemaker	152 (0.3)	2 (1.9)		22 (0.4)		4 (0.9)	
Managerial/professional	26 926 (56.3)	37 (35.2)		2 986 (51.9)		168 (37.5)	
Technical/sales/administrative	6 697 (14.0)	15 (14.3)		815 (14.2)		67 (15.0)	
Service/labor	8 371 (17.5)	33 (31.4)		1 219 (21.2)		140 (31.3)	
Other	5 715 (11.9)	18 (17.1)		716 (12.4)		69 (15.4)	
Partner currently employed (yes)	18 446 (38.0)	36 (34.6)	.003	2 228 (38.4)	<.001	186 (41.1)	<.001
Smoking			.09		<.001		<.001
Never smoked	41 115 (51.0)	100 (46.3)		4 410 (49.3)		420 (47.7)	
Past smoker	34 568 (42.9)	96 (44.4)		3 844 (43.0)		354 (40.2)	
Current smoker	4 865 (6.0)	20 (9.3)		694 (7.8)		107 (12.1)	

Continued

TABLE 1—*Continued*

Alcohol intake			<.001		<.001		<.001
Nondrinker	9 139 (11.3)	24 (11.3)		881 (9.8)		138 (15.5)	
Past drinker	14 879 (18.3)	65 (30.5)		1 975 (21.9)		242 (27.2)	
<1 drink/wk	25 516 (31.5)	56 (26.3)		3 034 (33.6)		266 (29.9)	
1–6 drinks/wk	21 122 (26.0)	43 (20.2)		2 145 (23.7)		156 (17.5)	
≥7 drinks/wk	10 430 (12.9)	25 (11.7)		999 (11.1)		89 (10.0)	
Living alone (yes)	21 940 (27.1)	52 (24.0)	.31	1 884 (20.9)	<.001	233 (26.2)	.56

Note. HS = high school; GED = general equivalency diploma.

TABLE 2—Multivariate Associations With Baseline Reports of Abuse vs No Abuse Among Postmenopausal Women

	Physical Abuse Only OR (95% CI)	Verbal Abuse Only OR (95% CI)	Physical and Verbal Abuse OR (95% CI)
Age, y (vs 50–58 y)			
59–64	0.81 (0.55, 1.19)	0.79 (0.75, 0.84)	0.56 (0.47, 0.68)
65–69	0.76 (0.49, 1.17)	0.68 (0.64, 0.73)	0.49 (0.39, 0.60)
70–79	0.67 (0.42, 1.06)	0.57 (0.53, 0.62)	0.38 (0.30, 0.48)
Race (vs non-Hispanic White)			
American Indian/Alaska Native	2.54 (0.62, 10.45)	1.34 (0.98, 1.83)	3.10 (1.73, 5.54)
Asian/Pacific Islander	2.04 (0.98, 4.24)	0.79 (0.68, 0.92)	1.52 (1.04, 2.24)
African American	2.84 (1.89, 4.26)	0.73 (0.66, 0.80)	1.26 (0.99, 1.59)
Hispanic American	1.74 (0.93, 3.26)	1.08 (0.96, 1.22)	1.95 (1.49, 2.54)
Education (vs college graduate)			
≤HS diploma	1.45 (0.90, 2.33)	0.70 (0.65, 0.76)	0.82 (0.64, 1.04)
Some college/technical school	1.10 (0.72, 1.66)	0.98 (0.93, 1.05)	1.14 (0.94, 1.38)
Income, $ (vs > $75 000)			
<20 000	2.72 (1.43, 5.18)	2.12 (1.86, 2.42)	5.15 (3.75, 7.06)
20 000–34 999	1.64 (0.93, 2.89)	1.72 (1.56, 1.88)	3.14 (2.40, 4.11)
35 000–49 999	1.18 (0.73, 1.90)	1.43 (1.33, 1.53)	1.94 (1.54, 2.44)
50 000–75 000	1.42 (0.91, 2.22)	1.22 (1.14, 1.30)	1.29 (1.01, 1.64)
Employment (vs managerial)			
Technical	0.95 (0.61, 1.49)	1.03 (0.97, 1.10)	1.16 (0.94, 1.43)
Service	1.68 (1.08, 2.62)	1.08 (1.00, 1.17)	1.40 (1.12, 1.75)
Homemaker	1.03 (0.57, 1.86)	0.96 (0.87, 1.05)	1.04 (0.78, 1.40)
Marital status (vs married)			
Never married	0.83 (0.35, 1.99)	0.71 (0.62, 0.82)	0.28 (0.15, 0.52)
Divorced	1.55 (0.97, 2.49)	1.05 (0.96, 1.14)	1.42 (1.13, 1.79)
Widowed	1.06 (0.63, 1.78)	0.64 (0.58, 0.71)	0.75 (0.57, 0.99)
Smoking status (vs never smoked)			
Past smoker	1.34 (0.97, 1.84)	1.06 (1.01, 1.12)	1.07 (0.91, 1.26)
Current smoker	1.30 (0.74, 2.26)	1.30 (1.18, 1.43)	1.69 (1.33, 2.16)
Alcohol use (vs past/never drank)			
<1 drink/wk	0.79 (0.54, 1.17)	0.97 (0.92, 1.03)	0.86 (0.72, 1.03)
≥1 drink/wk	1.02 (0.70, 1.50)	0.80 (0.76, 0.86)	0.73 (0.60, 0.89)
Living alone (vs no)	0.61 (0.39, 0.95)	0.75 (0.69, 0.81)	0.76 (0.61, 0.95)

Note. OR = odds ratio; CI = confidence interval; HS = high school.

more likely to report physical abuse exposure than non-Hispanic White women, although these associations did not reach statistical significance. When other variables are controlled for, women who had incomes of less than $20 000 (odds ratio [OR] = 2.72; 95% CI = 1.43, 5.18) and who worked in service-type occupations (OR = 1.68; 95% CI = 1.08, 2.62) were more likely to report exposure to physical abuse. Women who were living alone were nearly half as likely to report exposure to physical abuse at baseline.

Table 2 also demonstrates the multivariate associations with exposure to verbal abuse only at baseline. When other variables are controlled, women in the 3 older age categories were less likely than women aged 50 to 58 years to report verbal abuse only at baseline. Black and Asian/Pacific Islander women were less likely to report verbal abuse only at baseline than non-Hispanic White women (OR = 0.73 and 0.79, respectively), as were women who were never married/widowed, drank less than 1 drink per week, or who lived alone. Women who had incomes of less than $75 000 annually or who were current smokers were more likely to report verbal abuse only.

For women reporting both physical and verbal abuse, those in the older age categories were less likely to report abuse at baseline than women aged 50 to 58 years, as were women who were never married, were widowed, or lived alone. Ethnic minority women, those with incomes of less than $75 000, those employed in service-type jobs, and those who were current smokers were more likely to report both physical and verbal abuse.

Of the 48 522 women with follow-up data at year 3 and who reported no exposure to domestic violence at baseline, 2431 women (5.01%) reported exposure to abuse at their follow-up visit 3 years later. Of these 2431 women, 67 (2.8%) reported physical abuse only, 2250 (92.6%) verbal abuse only, and 114 (4.7%) both physical and verbal abuse (Table 3). Ethnicity was associated with all 3 abuse categories, while education and income were associated with both physical abuse only and verbal abuse only. Age and marital status were associated with verbal abuse only and the combined abuse category.

The associations with 3-year incident exposure to physical and verbal abuse, after control for other covariates, are demonstrated in Table 4. Women in the 2 older age categories were less likely to have been exposed to either physical or verbal abuse at the 3-year follow-up visit than women aged 50 to 58 years. Non-White women were more likely to report exposure to either physical or verbal abuse at the 3-year follow-up visit than non-Hispanic White women, as were women who had lower annual household incomes (i.e., incomes of less than $75 000 annually). Women who were past or current smokers were more likely to report 3-year incident exposure to verbal abuse only. Women who were living alone were less likely to report incident exposure to verbal abuse only.

DISCUSSION

In this study, we found that many functionally independent, older women are exposed to physical and verbal abuse. Our finding that 1.2% of the women in our self-selected, postmenopausal cohort were physically abused is similar to the prevalence estimates reported in other population-based surveys.[21–23] However, our finding that 10% of women reported verbal abuse is 3 to 10 times higher than population-based results showing a 1.1% to 3.2% prevalence of verbal abuse.[21–24] These findings suggest that even for nondependent older women, physical and verbal abuse is occurring at rates similar to, or higher than, those for younger women. Perhaps more importantly, we found that 3.7 per 1000 older women reported new exposure to physical abuse and 46 per 1000 older women reported becoming new victims of verbal abuse. This result compares with population estimates that show the annual incidence of abuse ranging from 735 000 to 2 million out of an estimated 31 million older women.[24] To our knowledge, our findings are the first estimate of incidence of physical and verbal abuse in a large sample of postmenopausal women.

Exposure to abuse among these postmenopausal women is associated with younger age and lower income. These findings are comparable to data in intimate partner abuse research but contrast with elder abuse data. Studies demonstrate that victims of intimate partner abuse are more likely to be younger than 35 years old, not to be college educated, and to have lower socioeconomic status.[11,18,19,25–28] Studies on abuse among older adults, however, show that advanced age (>75 years old), functional dependency, shared living arrangement, social isolation, depression, personality disorder, cognitive impairment, and excessive use of drugs or alcohol place an older adult at risk for abuse.[20,28]

The discrepancies between our findings and previous research with regard to age and living situation may be related to the fact that all the women in our sample were functionally independent. Given the high level of physical functioning in our sample, it is unlikely that abuse by caregivers, neglect, or self-neglect was a predominate cause of abuse in our study. By focusing on the frail elderly, most of the previous research on the abuse of older adults was influenced by issues of caregiver abuse and neglect. These findings suggest that there is a transition in abuse risk factors for women as they age. If a woman remains functionally independent, the risk factors for abuse mirror those for intimate partner violence. If she becomes dependent functionally, and perhaps more vulnerable, the risk factors for abuse mirror those of caregiver abuse and neglect.

One interesting finding was the relationship between race/ethnicity and abuse. Non-Hispanic White women reported more exposure to verbal abuse than their minority counterparts, while African American women reported more exposure to physical abuse. Our 3-year incidence results show a similar pattern for African American women, with less verbal abuse in this group, although the results did not reach statistical significance. The 3-year incidence results, however, show a stronger association of all 3 types of abuse exposure among Hispanic women.

These results are in contrast to the findings on elder abuse and abuse in younger women that show non-Whites as being more likely to be victimized by all types of abuse. Previous research demonstrates a 4-fold influence of ethnicity on reports of abuse.[19] There has not been any distinction demonstrated in the types of abuse experienced across racial subgroups. Since intimate relationships have strong culturally specific meanings, the interpretation of what constitutes abuse across cultures may influence the association of racial/ethnic group with certain types of abuse. Perhaps race/ethnicity is a factor for abuse exposure that has more specific targets in older, functionally independent women as contrasted with more broad categories of race/ethnicity in more frail older women. Thus, despite their older age, functionally independent victims of abuse in our study seem to be similar to younger victims of intimate partner violence.

In addition to race/ethnicity, other lifestyle factors are associated with abuse exposure. Current smoking seems to be associated with greater exposure to abuse, particularly for verbal abuse. However, alcohol use seems to be less likely among those who were exposed to abuse, particularly verbal abuse. The associations with verbal abuse are consistent for both our prevalence and 3-year incidence results. While previous research has not examined smoking behaviors in women exposed to violence, our findings regarding alcohol use are in contrast with most previous research. Research on intimate partner violence and elder abuse suggests that abuse victims in both groups have a higher rate of alcohol and substance use.[20,29] Our results may reflect the fact that the functionally independent older women in our study did not perceive a need to "escape" an abusive relationship through alcohol use. Another possibility may be that these women perceived alcohol use as increasing their vulnerability and thus escalating their potential of being victimized by greater violence.

TABLE 3—Three-Year Abuse Incidence in Cohort of Postmenopausal Women, by Subjects' Characteristics (N = 48 522)

Characteristic	No Abuse (n = 46 091)	Any Abuse (n = 2 431)					
		Physical Abuse Only, No. (%)	*P*	Verbal Abuse Only, No. (%)	*P*	Physical and Verbal Abuse, No. (%)	*P*
Overall		67 (2.8)		2 250 (92.6)		114 (4.7)	
Age, y			.12		<.001		<.001
<58	14 272 (31)	27 (40.3)		940 (41.8)		53 (46.5)	
59–64	10 903 (23.7)	20 (29.9)		533 (23.7)		32 (28.1)	
65–69	9 860 (21.4)	8 (11.9)		420 (18.7)		14 (12.3)	
70–74	7 700 (16.7)	7 (10.4)		260 (11.6)		10 (8.8)	
>74	3 356 (7.3)	5 (7.5)		97 (4.3)		5 (4.4)	
Ethnicity			<.001		<.001		<.001
American Indian/Alaska Native	156 (0.3)	0 (0.0)		8 (0.4)		2 (1.8)	
Asian/Pacific Islander	1 320 (2.9)	4 (6.0)		81 (3.6)		2 (1.8)	
African American	2 831 (6.1)	11 (16.4)		139 (6.2)		14 (12.3)	
Hispanic/Latino	1 119 (2.4)	8 (11.9)		109 (4.8)		14 (12.3)	
White	40 665 (88.2)	44 (65.7)		1 913 (85)		82 (71.9)	
Education			.04		<.001		.31
0–8 y	509 (1.1)	3 (4.5)		28 (1.3)		2 (1.8)	
Some HS/HS diploma/GED	8 611 (18.8)	14 (21.2)		369 (16.6)		23 (20.7)	
School after high school	16 228 (35.5)	25 (37.9)		879 (39.4)		46 (41.4)	
College graduate or higher	20 405 (44.6)	24 (36.4)		953 (42.8)		40 (36.0)	
Family income, $			.01		.007		.06
<20 000	6 073 (14.1)	18 (29.5)		356 (16.9)		22 (20.6)	
20 000–34 999	9 989 (23.2)	12 (19.7)		469 (22.3)		32 (29.9)	
35 000–49 999	8 770 (20.4)	12 (19.7)		432 (20.5)		17 (15.9)	
50 000–74 999	8 949 (20.8)	10 (16.4)		410 (19.5)		21 (19.6)	
≥75 000	9 264 (21.5)	9 (14.8)		436 (20.7)		15 (14.0)	
Occupation			.08		.06		.36
Managerial/professional	19 732 (45.1)	20 (31.7)		967 (45.7)		45 (41.7)	
Technical/sales/administrative	12 425 (28.4)	18 (28.6)		579 (27.4)		35 (32.4)	
Service/labor	7 028 (16.1)	16 (25.4)		375 (17.7)		21 (19.4)	
Homemaker only	4 550 (10.4)	9 (14.3)		193 (9.1)		7 (6.5)	
Currently employed (yes)	16 675 (37.6)	19 (29.2)	.16	929 (43.6)	<.001	46 (41.1)	.45
Marital status			.51		<.001		.02
Never married	2 220 (4.8)	4 (6.0)		99 (4.4)		4 (3.6)	
Divorced/separated	6 804 (14.8)	14 (20.9)		417 (18.6)		27 (24.1)	
Widowed	7 750 (16.9)	11 (16.4)		267 (11.9)		12 (10.7)	
Presently married	29 155 (63.5)	38 (56.7)		1 457(65.0		69 (61.6)	
Partner's main job			<.001		<.001		.08
Homemaker	67 (0.2)	1 (2.9)		5 (0.4)		0 (0.0)	
Managerial/professional	16 261 (58)	12 (34.3)		733 (52.8)		25 (39.1)	
Technical/sales/administrative	3 934 (14.0)	4 (11.4)		200 (14.4)		12 (18.8)	
Service/labor	4 540 (16.2)	13 (37.1)		279 (20.1)		16 (25.0)	
Other	3 232 (11.5)	5 (14.3)		171 (12.3)		11 (17.2)	
Partner currently employed (yes)	11 242 (40.2)	16 (43.2)	.052	604 (43.6)	.02	37 (53.6)	.08
Smoking			.97		.003		.14
Never smoked	23 332 (51.2)	35 (52.2)		1 070 (48.2)		56 (50.5)	
Past smoker	19 710 (43.2)	28 (41.8)		995 (44.8)		44 (39.6)	
Current smoker	2 556 (5.6)	4 (6.0)		155 (7.0)		11 (9.9)	

Continued

TABLE 3—*Continued*

Alcohol intake			.11		.04		<.001
Nondrinker	4 698 (10.2)	9 (13.4)		226 (10.1)		17 (15.2)	
Past drinker	7 849 (17.1)	17 (25.4)		431 (19.2)		35 (31.3)	
<1 drink/wk	14 511 (31.6)	21 (31.3)		717 (32.0)		29 (25.9)	
1–6 drinks/wk	12 525 (27.3)	17 (25.4)		597 (26.6)		18 (16.1)	
≥7 drinks/wk	6 291 (13.7)	3 (4.5)		271 (12.1)		13 (11.6)	
Living alone (yes)	12 087 (26.3)	17 (25.4)	.86	520 (23.3)	<.001	26 (23.2)	.45

Note. HS = high school; GED = general equivalency diploma.

TABLE 4—Multivariate Associations With 3-Year Incidence of Abuse vs No Abuse Among Postmenopausal Women

	Physical Abuse Only OR (95% CI)	Verbal Abuse Only OR (95% CI)	Physical and Verbal Abuse OR (95% CI)
Age, y (vs 50–58 y)			
59–64	1.17 (0.62, 2.19)	0.75 (0.67, 0.85)	0.67 (0.41, 1.10)
65–69	0.44 (0.18, 1.06)	0.61 (0.53, 0.70)	0.33 (0.17, 0.64)
70–79	0.46 (0.20, 1.09)	0.50 (0.43, 0.57)	0.33 (0.17, 0.64)
Race (vs non-Hispanic White)			
American Indian	...	1.03 (0.48, 2.21)	5.40 (1.29, 22.65)
Asian/Pacific Islander	2.11 (0.63, 7.00)	1.45 (1.14, 1.84)	0.70 (0.17, 2.91)
African American	1.66 (0.66, 4.13)	0.87 (0.71, 1.06)	1.39 (0.71, 2.73)
Hispanic American	4.50 (1.90, 10.66)	1.65 (1.31, 2.08)	3.56 (1.77, 7.15)
Education (vs college graduate)			
≤HS diploma	0.52 (0.22, 1.22)	0.87 (0.75, 1.02)	1.21 (0.64, 2.30)
Some college/technical school	0.86 (0.44, 1.67)	1.13 (1.01, 1.27)	1.13 (0.67, 1.90)
Income, $ (vs > $75 000)			
<20 000	2.18 (0.58, 8.18)	2.05 (1.57, 2.66)	1.37 (0.42, 4.52)
20 000–34 999	2.74 (1.08, 6.95)	1.65 (1.38, 1.98)	2.46 (1.18, 5.15)
35 000–49 999	1.24 (0.53, 2.89)	1.34 (1.17, 1.54)	2.02 (1.12, 3.62)
50 000–75 000	1.48 (0.69, 3.19)	1.25 (1.10, 1.42)	1.11 (0.59, 2.09)
Employment (vs managerial)			
Technical	1.47 (0.68, 3.14)	0.89 (0.78, 1.00)	0.90 (0.53, 1.52)
Service	2.18 (1.00, 4.77)	0.95 (0.82, 1.10)	0.67 (0.35, 1.28)
Homemaker	2.23 (0.88, 5.63)	0.90 (0.75, 1.08)	0.47 (0.19, 1.20)
Marital status (vs married)			
Never married	0.87 (0.19, 4.05)	0.85 (0.66, 1.10)	0.63 (0.18, 2.19)
Divorced	1.34 (0.56, 3.25)	1.09 (0.92, 1.29)	1.39 (0.73, 2.65)
Widowed	1.45 (0.58, 3.62)	0.79 (0.65, 0.96)	1.08 (0.51, 2.26)
Smoking status (vs never smoked)			
Past smoker	0.88 (0.50, 1.54)	1.16 (1.05, 1.28)	0.98 (0.64, 1.51)
Current smoker	0.72 (0.22, 2.39)	1.22 (1.01, 1.48)	1.58 (0.78, 3.17)
Alcohol use (vs past/never drank)			
<1 drink/wk	0.84 (0.43, 1.63)	0.96 (0.85, 1.08)	0.55 (0.34, 0.90)
≥1 drink/wk	0.85 (0.43, 1.68)	0.90 (0.79, 1.01)	0.50 (0.30, 0.83)
Living alone (vs no)	0.76 (0.34, 1.72)	0.83 (0.70, 0.97)	0.73 (0.39, 1.36)

Note. HS = high school.

This study has important limitations. The detection of exposure to physical and verbal abuse relies on the self-report of the victims. Subjects may have been reluctant to admit to abuse, resulting in an underestimate of the prevalence and 3-year incidence. This underestimate may also diminish the differences found in the association of abuse with our predictor variables. Also, the subjects recruited for the WHI are drawn from a volunteer sample of older healthier women. These women may differ from other women of their age in exposure to abuse and its effects on their health status.

Despite these limitations, our finding that 11.1% of women aged 50 to 79 years reported exposure to abuse in the past year, and that an additional 5% in this age group reported exposure to abuse over a 3-year interval, reveals an important problem for older women. While it is unclear if this abuse is a continuation of a lifelong cycle of violence or the result of late-life onset of violence, these results suggest that abuse is occurring at rates too great to ignore. If abuse of older women yields the same untoward morbidity and mortality seen in younger women and fragile elders, there is a great threat to public health. Although a recent article by Ramsay et al. challenges the effectiveness of screening for domestic violence,[30] screening these postmenopausal women may trigger an investigation by agencies like Adult Protective Services that can provide help to abuse victims. Our results suggest that additional investigations regarding the impact of abuse in this population and the impact of screening for abuse in postmenopausal women should be encouraged. ■

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Contributors

C.P. Mouton conceived of the study, developed the study design, and supervised the data acquisition and analysis, and drafting of the manuscript. R.J. Rodabough retrieved study data and completed data analysis. S.L.D. Rovi assisted in the study design. S.K. Burge assisted in defining the categories of abuse. All authors assisted in the interpretation of the data analysis and drafting of the manuscript.

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Human Participants Protection

Protocol and consent forms were approved by the institutional review boards of all the Women's Health Initiative participating institutions, including the University of Texas Health Science Center at San Antonio. All women provided written informed consent.

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Protection Orders and Intimate Partner Violence: An 18-Month Study of 150 Black, Hispanic, and White Women

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Objectives. We compared types and frequencies of intimate partner violence experienced by women before and after receipt of a 2-year protection order.

Methods. Participants were 150 urban English- and Spanish-speaking Black, Hispanic, and White women who qualified for a 2-year protection order against an intimate partner.

Results. One woman committed suicide 6 weeks into the study. The remaining 149 women completed all interviews. Results showed significant reductions in threats of assault, physical assault, stalking, and worksite harassment over time among all women, regardless of receipt or nonreceipt of a protection order.

Conclusions. Abused women who apply and qualify for a 2-year protection order, irrespective of whether or not they are granted the order, report significantly lower levels of violence during the subsequent 18 months. (*Am J Public Health.* 2004;94:613–618)

Abused women use a variety of methods in seeking assistance to halt violence inflicted upon them, including court orders of protection. Such orders restrict the access of 1 person (e.g., a male abuser) to another person (e.g., an abused woman) for a specified time. (The synonym "restraining order" is used in some jurisdictions.) Protection orders, both temporary and permanent, represent public documentation that abuse has occurred, and if the order is violated, the assailant is subject to prosecution. A protection order offers the victim legal action when the victim does not want the abuser charged criminally or jailed for an offense. However, choice of this action does not preclude other civil or criminal action.

Results of research on the effectiveness of protection orders are inconsistent. We identified 8 recent longitudinal studies that measured additional intimate partner violence committed against women after a protection order had been filed. Six of the studies reported positive results,[1–6] meaning that the respondents felt the protection order helped to end or reduce the violence. The remaining 2 studies reported high reassault rates after filing of the protection order.[7,8] In the case of most of these studies, low response rates, short follow-up periods, and lack of comparison groups do not allow generalizations to be made.

In addition, we did not identify any studies that included non–English-speaking women or measures of worksite harassment. To test the effectiveness of protection orders, we entered into a partnership with a local district attorney's office in a large urban city in an attempt to determine whether women who are granted a 2-year protection order experience lower levels of violence than women who apply and qualify for such an order but are not granted one.

METHODS

Our study was conducted from January 2001 to June 2002 at a special family violence unit of the Houston, Tex, district attorney's office that serves an ethnically diverse population of 3 million citizens. The primary service of the family violence unit is processing of protection orders. During the 12 months preceding this study, 2932 women applied to the unit for a protection order; 1980 (68%) met qualifying criteria, and 962 (49%) were granted the protection order. Qualification criteria for protection orders are set by state law and include applicants providing evidence (i.e., police or witness report, visible injury) that the respondent (e.g., abuser) has been violent with them and is likely to continue this violence toward them. In addition, the applicant must have previously lived with the abuser in the same household, or they must be the biological parents of the same child.[9]

If the applicant's case is accepted, the attorneys file the case with the family law court and ask for a court date to be set for a hearing. After the case has been filed, the court issues a temporary protection order. A copy of this order is sent to the applicant by mail, and a copy is served to the abuser in person. The temporary protection order is similar to the final 2-year protection order in that it informs the abuser that he or she must stay 200 ft (60 m) away from the applicant's home and workplace and prohibits the respondent from assaulting the applicant, from threatening the applicant directly or through another person, and from harassing or stalking the applicant.

However, the temporary protection order differs from the final 2-year protection order in that a violation of the temporary order cannot be charged as a criminal offense; it can be filed only as a civil contempt of court. Furthermore, the temporary protection order is valid for only 20 days. The court date is set within those 20 days, and the order expires whether or not the abuser is served or the hearing takes place. However, the temporary protection order may be extended if the abuser is not served by the hearing date.

The applicant is not responsible for any fees in association with the protection order. The order is granted for 2 years and can result in both criminal and civil penalties if violated. Applicants are informed at the time of application as to whether they do or do not qualify to receive the order. All qualifying applicants are assigned to a case worker who provides them with educational information about violence and safety planning as well as information regarding community resources (e.g., emergency shelters, counseling, legal and medical assistance). Applicants are encouraged to contact the case worker for further questions about the protection order process.

All women who presented to the special family violence unit at the district attorney's office to apply for a protection order, completed the application process, qualified for the protection order, and met our inclusion criteria (e.g., female, 18 years or older, English or

Spanish speaker) were invited into the study by 1 of the 6 investigators until 150 women agreed to participate and were entered into the study. Four women refused to participate. One woman committed suicide 6 weeks into the study. All of the remaining 149 women completed the 3-month, 6-month, 12-month, and 18-month follow-up interviews, resulting in a retention rate of 99%.

Instruments

Demographic data form. This form was used to document information on participants' age, education, income, self-identified race/ethnicity, employment status, relationship to the abuser, and primary language.

Severity of Violence Against Women Scales (SVAWS). This 46-item instrument is designed to measure threats of physical violence (19 items) and physical assault (27 items).[10] Examples of behaviors that threaten physical violence are threats to destroy property, do bodily harm, or harm other family members. Examples of behaviors that represent physical violence are kicking, choking, beating up, and engaging in forced sex. For each item, respondents use a 4-point scale to indicate how often the behavior occurred (1=never, 2=once, 3=2–3 times, 4=4 or more times). Possible score ranges were 19 to 76 for the threat of abuse dimension and 27 to 108 for the physical abuse dimension. The higher the score was, the more violence that was reported.

Internal consistency reliability estimates in studies of abused women have ranged from 0.89 to 0.91 for the threat of abuse dimension and from 0.91 to 0.94 for the physical abuse dimension.[5,11,12] In the present study, reliabilities (measured with Cronbach α coefficients) were 0.91 for the threat of abuse dimension and 0.94 for the physical abuse dimension.

Stalking Victimization Survey. This 17-item yes/no questionnaire was used to document the frequency and type of stalking engaged in by the perpetrator. The initial stalking survey instrument consisted of 7 items (e.g., being followed or spied on, being sent unsolicited letters or written correspondence, or finding the perpetrator standing outside one's home, school, or workplace) developed by Tjaden and Thoennes[13]; 10 items were added from the Sheridan[14] HARASS instrument to form the overall 17-item instrument used here. Examples of items added include threats by the abuser to harm the children or to commit suicide if the woman left the relationship, leaving threatening notes on the woman's car, and threatening her family. The possible score range was 0 to 17. In this study, reliability (Cronbach α coefficient) was 0.83.

Danger Assessment Scale. This instrument, which consists of 15 items with a yes/no response format, assists women in determining their potential risk of becoming a femicide victim.[15] All of the items refer to risk factors that have been associated with murder in situations involving abuse. Examples of risk factors include the abuser's possession of a gun, use of drugs, and violent behavior outside the home. The possible score range was 0 to 15. Scale reliability coefficients have ranged from 0.60 to 0.86 in several studies.[16] In this study, the reliability (Cronbach α coefficient) was 0.67.

Worksite harassment. Eight yes/no questions were asked about worksite harassment. Questions were derived from a congressional report[17] that reviewed studies of worksite harassment of women by intimate partners. Questions focused on, for example, repeated calls/visits to the woman's worksite and difficulties experienced by the woman in regard to going to work. The possible score range was 0 to 8. Reliability (Cronbach α coefficient) was measured as 0.76.

Procedure

Data collection began after institutional review board approval had been received and consent had been obtained from the district attorney's office. Women meeting the study criteria were escorted to a private room in the offices of the family violence unit where the investigators provided an explanation of the study's purpose, protocol, instruments, administration time, and follow-up schedules. Women who agreed to take part in the study signed an informed consent form, and the investigators administered the study instruments. Instruments were offered in English and Spanish according to women's language preference. All measures focused on women's reports of violence and health status during the preceding 3 months.

To assist in maintaining contact with each of the women, we formed a safe contact list of at least 6 persons the woman granted permission for us to contact in the event she could not be reached. This list consisted of close relatives (i.e., mother, grandmother, sister, and adult children), neighbors, friends, work colleagues, and other acquaintances. In each case, name, relationship, address (home and work), and contact telephone numbers (i.e., home, work, and cellular) were listed. When contacted, the person was told that the woman was involved in a health study and had given permission for the researcher to contact individuals who may know of her current address/telephone number(s). During all subsequent interviews with the women, both their contact information and that of each safe contact were reviewed and updated.

The safe contact list proved the best method for maintaining contact with the women over the 18-month study period. We ensured women's safety in completing the follow-up telephone interviews by establishing a convenient, private, and safe time for these interviews. A safety protocol was used for each follow-up telephone interview. Women were reimbursed $20 for the first interview; $30 for the 3-month interview; $40 for the 6-month interview; $50 for the 12-month interview; and $60 for the 18-month interview. They were reimbursed an extra $40 for completing all of the interviews.

Data Analyses

Means, standard deviations, and frequencies were used in descriptions of the demographic characteristics of the 150 women who applied for protection orders. We conducted independent *t* tests to determine whether the women who were granted an order differed significantly in terms of age or years of education from the women who were not granted an order. Chi-square analyses were used to determine whether the groups of women differed significantly with respect to race/ethnicity, income, employment status, or status of relationship with abuser.

Using Cohen's power analyses and tables,[18] we calculated the a priori power of our between-groups repeated measures analysis of variance to produce a small-to-moderate multi-

variate effect size by conducting a multivariate analysis of variance with 4 dependent variables (i.e., the 4 score differences from intake scores). Given a significance level of .05, 150 participants, 4 dependent variables, 2 groups, and the goal of a small-to-moderate effect size, we calculated the power of the analysis as 91%. Assumptions of independent observations, normality, and homogeneity of (co)variance were examined. Results indicated that the study's robustness, procedure, number of participants, and sample size ratio satisfied these assumptions.

Violence scores for women who were granted or not granted the protection order were subjected to repeated measures analyses. We initially considered as covariates demographic characteristics that exhibited significant between-groups differences at intake and were shown to be univariately associated with the dependent variables. However, we retained only significant covariates in the final analyses. We conducted a 1-factor repeated measures multivariate analysis of covariance on SVAWS scores to accommodate the 2 dependent subscales (threats of violence and physical violence scores). We performed 1-factor repeated measures analyses of covariance (ANCOVAs) on danger, stalking, and work harassment scores.

We calculated adjusted means, standard deviations, and multivariate effect sizes (0.02 = small, 0.10 = small to moderate, 0.15 = moderate, 0.35 = large).[18] To achieve a balance between type I and type II error, we set the significance level at .025 for each SVAWS subscale. In the case of within-group (time, Group × Time interaction) contrasts, we set significance levels at .006 for subscale scores and .0125 for stalking, danger, and worksite harassment scores.

RESULTS

The women were stratified into 2 groups: those who were granted a 2-year protection order (n=81) and those who were not granted such an order (n=69). Reasons for nonreceipt of the protection order were as follows: the woman dropped the order (n=40), inability to locate the abuser and serve papers to appear in court (n=18), and dismissal of cases (n=11).

Reasons for Not Being Granted a 2-Year Protection Order

Forty women dropped the protection order before their court date. Most did so because they returned to the relationship with the abuser or because the protection order process was "too much of a hassle" or "inconvenient." To obtain a protection order, applicants must be willing to arrive at the district attorney's office with proper photo identification and complete paperwork, and they are required to complete an interview with a caseworker, be photographed, and sign an affidavit. This process requires about 2 to 3 hours. Applicants must wait approximately 6 weeks for a court date and then appear in court in front of a judge, at which time the abuser may contest the protection order.

In addition, many women need to return to the district attorney's office at a later date with additional required paperwork/witnesses to the abuse. For some women, these trips to the district attorney's office mean work absences and loss of income. We did not ask the participants in our study who had dropped the protection order when they did so; however, at the 3-month interview, many women reported dropping the order within the first 2 weeks after application.

Eighteen women were not granted a protection order because the abuser could not be found and served papers to appear in court. Eleven women were not granted the order because their case was dismissed. Six cases were dismissed by the district attorney's office owing to incomplete applications (e.g., required documents not being supplied). Seven cases were dismissed by the judge, 2 because the protection order was contested by the abuser and the remaining 5 because the women did not appear in court. One of these women committed suicide. As mentioned, the remaining 149 women completed the 4 follow-up interviews, for a retention rate of 99%.

Between-Groups Differences in Demographic Characteristics and Violence Scores

Frequencies, percentages, and the results of tests assessing demographic differences among women who were and were not granted a 2-year protection order are shown in Table 1. Relationship status was significantly ($\chi^2_1 = 4.407$, $P = .036$) associated with receipt of a protective order. Slightly more than half of the women who were granted a protection order were involved in relationships, as compared with 71% of women who were not granted the order. No other significant differences were found.

Adjusted means and standard deviations for violence scores at intake and at 3, 6, 12, and 18 months among women who were granted (n=81) and not granted (n=69) the 2-year protection order are shown in Table 2. After adjustment for age, race/ethnicity, and relationship status, results of the multivariate analysis of variance focusing on SVAWS scores yielded a significant ($F_{8,1144} = 16.123$, $P < .001$) multivariate main effect for time. The magnitude of this multivariate effect size was in the small-to-moderate range (0.10). Univariate tests revealed a significant main effect for both SVAWS subscales: threats ($F_{4,572} = 19.077$, $P < .001$) and physical abuse ($F_{4,572} = 36.261$, $P < .001$). The group main effect and the Group × Time interaction were not significant. The effect size between the groups was small (0.02). Examination of within-subject contrasts showed that intake scores were significantly ($P < .001$) higher than subsequent scores.

After adjustment for age and race/ethnicity, repeated measures ANCOVAs showed significant ($F_{4,141} = 16.17$, $P < .001$, and $F_{4,141} = 18.33$, $P < .001$, respectively) effects over time of stalking and danger scores. The time effect size for stalking was in the medium-to-large range (0.31), and the effect size for danger was moderate (0.18). There was no significant group main effect or significant Group × Time interaction. Between-groups effect sizes were zero or small (0.02). After adjustment for relationship status, the repeated measures ANCOVA of work harassment scores also showed a significant ($F_{4,80} = 13.88$, $P < .001$) effect over time. There was no significant group main effect or significant Group × Time interaction. The between-groups effect size was zero. Examination of within-subject contrasts for the main effect of time showed that intake scores were signifi-

TABLE 1—Demographic Characteristics and Results From χ^2 Tests of Independence Assessing Differences Between Women Who Were Granted (n = 81) and Not Granted (n = 69) a 2-Year Protection Order

Characteristic	Protection Order	No Protection Order	Total No.	Test Statistic (χ^2 or *t*) (*P*)
Age, y, mean (SD)	33.5 (9.2)	31.2 (9.1)	150	1.503[a] (.135)
Education, y, mean (SD)	11.7 (3.0)	11.9 (2.6)	150	0.503[a] (.615)
Race/ethnicity, No. (%)				3.320[b] (.190)
African American	31 (38.3)	18 (26.1)	49	
White	22 (27.2)	18 (26.1)	40	
Latino/Hispanic	28 (34.6)	33 (47.8)	61	
Relationship status, No. (%)				4.407[b] (.036)
Current spouse/boyfriend	44 (54.3)	49 (71.0)	93	
Ex-spouse/friend	37 (45.7)	20 (29.0)	57	
Family income, $, No. (%)				0.672[b] (.412)
≥19 000	22 (32.8)	22 (40.0)	44	
>19 000	45 (67.2)	33 (60.0)	78	
English speaking, No. (%)				0.831[b] (.362)
No	15 (18.5)	9 (13.0)	24	
Yes	66 (81.5)	60 (87.0)	126	
Employed, No. (%)				2.916[b] (.088)
No	8 (9.9)	2 (2.9)	10	
Yes	73 (90.1)	67 (97.1)	140	

[a] *t* test.
[b] χ^2 test.

cantly ($P<.001$) higher than subsequent scores.

Protection Order Violations

Finally, women were asked, at each interview, whether a violation of the 2-year protection order had occurred since the previous interview. Among the 81 women granted a protection order, 36 (44%) reported at least 1 violation over the 18 months of the study. Violations were reported by 17 women (21%) at 3 months, 16 women (20%) at 6 months, 20 women (25%) at 12 months, and 19 women (23%) at 18 months. Four women (5%) reported a violation during each of the 4 time periods measured. Most violations involved nonadherence to the order to stay 200 ft from the woman's home or workplace; stalking, threats of violence, and a combination of these infractions were other examples of violations. Women reporting a violation also were asked whether they had called the police. Among these 36 women, 21 (58%) had called the police at least once to report a violation.

DISCUSSION

The 149 women who took part in this study reported significantly lower levels of intimate partner violence, including worksite harassment, up to 18 months after applying for a protection order. Whether women were granted or not granted the protection order made no significant difference in terms of the amount of violence they reported at the time of application for the order or during the subsequent 3, 6, 12, or 18 months. Forty-four percent of the women granted a 2-year protection order reported at least 1 violation over the 18-month study period, and half of these women reported the violation to the police.

This study followed women after they had qualified for a protection order, irrespective of whether or not they were granted the order. Our results agree with those of others[5,19] reporting significantly lower levels of violence experienced by women seeking assistance from the justice system, irrespective of the justice system outcome. One other study, to our knowledge, involved the use of victim interviews to measure levels of violence toward women granted and not granted a protection order.[20] Although this study reported that violence frequency was not significantly decreased by receipt of a protection order, the study's low response rate and short follow-up period limited the generalizability of the findings. Other researchers have focused only on women who received an order of protection against the abuser[1–4,7] or have relied solely on police reports.[6] Because fewer than half of abused women ever report intimate partner violence to law enforcement personnel,[21] relying on police reports may severely underrepresent levels of violence experienced by women both with and without a protection order.

Our findings of significant reductions in violence scores over time among all of our participants, regardless of receipt or nonreceipt of the protection order, are consistent with abuse intervention findings reported by social and health researchers. In one study, abused women exiting a shelter and receiving home social support were compared, at 6 months, with abused women not receiving such support; women in both groups reported decreases in physical abuse.[22] In 2 health clinic studies involving comparisons of abused women receiving intensive counseling and outreach support and abused women offered a wallet-sized card listing community abuse resources, women in both groups reported significantly lower levels of abuse at 6, 12, and 18 months postintervention.[23,24] Although we found no other studies with which to compare our results, the economic implications of the significant decline in worksite harassment experienced by abused women after contact with the justice system merit further research.

Do these findings indicate that the justice intervention of a protection order and the health and social service interventions of counseling, support, and referrals are no more of a deterrent to future violence than an abused woman's contact with assistance agencies? When an abused woman decides to contact a criminal justice, civil justice, health, or social service agency, information about the abuse is shared, and contact is made. Just as the privatization of domestic violence contributes to its

TABLE 2—Adjusted Means and Standard Deviations for Violence Scores at Intake and 3, 6, 12, and 18 Months: Women Who Were Granted (n = 81) and Not Granted (n=69) a 2-Year Protection Order

Measure and Group	Intake, Mean (SD)	3 Months, Mean (SD)	6 Months, Mean (SD)	12 Months, Mean (SD)	18 Months, Mean (SD)
SVAWS[a]					
Threats of abuse[a]					
No order	44.7 (13.7)	21.6 (7.7)	20.7 (7.1)	21.4 (8.6)	21.9 (8.1)
Order	47.5 (13.7)	23.1 (7.8)	22.9 (7.2)	24.9 (8.7)	22.7 (8.1)
Physical abuse[b]					
No order	49.2 (17.0)	27.7 (5.5)	27.54 (4.6)	27.2 (7.4)	28.3 (7.6)
Order	48.5 (17.0)	29.2 (5.5)	28.46 (4.6)	31.1 (7.5)	29.2 (7.6)
Stalking[b]					
No order	7.7 (4.0)	2.2 (3.3)	1.4 (2.8)	1.9 (3.0)	2.1 (3.0)
Order	7.0 (4.0)	3.0 (3.3)	1.8 (2.8)	2.4 (3.0)	1.6 (3.0)
Danger[b]					
No order	7.1 (3.0)	1.5 (2.2)	1.2 (2.0)	1.1 (2.1)	1.4 (2.1)
Order	7.1 (3.0)	2.2 (2.2)	1.7 (2.0)	2.0 (2.1)	1.6 (2.1)
Worksite harassment[c]					
No order	3.7 (1.8)	2.1 (1.5)	1.5 (1.1)	1.4 (1.1)	1.3 (0.8)
Order	4.3 (1.8)	2.1 (1.5)	1.2 (1.1)	1.6 (1.1)	1.3 (0.8)

Note. One participant committed suicide; analyses were performed on a sample of 149 participants. SVAWS = Severity of Violence Against Women Scales.
[a]Adjusted for age, race/ethnicity, and relationship.
[b]Adjusted for age and race/ethnicity.
[c]Adjusted for relationship status.

continuation, perhaps the contact and public knowledge stemming from justice encounters can prevent reoccurrence of violence. Perhaps just as legal sanctions (e.g., requirements involving the use of helmets and seat belts) have proven effective in reducing unintentional injuries, such sanctions can reduce the occurrence of intentional intimate partner violence.

An earlier qualitative study focusing on why women seek civil orders of protection revealed a desire among women to regain some measure of control in their lives by making the abuse public.[25] These women discussed using the application for a protection order as a "loudspeaker" to notify the abuser that the law knew about his behavior. They viewed the legal system as a force larger than themselves and as having power over the abuser that they themselves had lost as a result of the abuse. Moreover, they felt a need to have the legal system both approve and reinforce their decision to leave the abuser. The protection order becomes an announcement that the abused woman refuses to "take it" anymore and is acting on her own behalf. Our results appear to quantify these qualitative findings. Once a woman applied and qualified for a protection order, a rapid and significant decline in violence scores occurred and was sustained for 18 months.

Our study involved limitations that are important to the generalizability of the findings. Our sample was small and limited to women from a single urban agency who were seeking assistance. Furthermore, we relied exclusively on self-reports, possibly leading to underreporting as a result of inadequate recall or lack of voluntary disclosure. If we are to learn more about the occurrence of intimate partner violence in the absence of justice system contact, there is a need for future research with larger, representative samples of abused women that include those who are victimized but do not apply for a protection order. In addition, replication is essential in rural settings with diverse ethnic groups. Despite these limitations, our urban sample of English- and Spanish-speaking women demonstrates the important effect of justice system contact in terms of reductions in future episodes of violence.

CONCLUSIONS

Ensuring the safety of victims of intimate partner violence is of utmost importance to health care providers, justice agencies, shelter workers, and other service providers. This study clearly demonstrates that, irrespective of whether or not a 2-year protection order was granted, abused women who sought a protection order reported significantly lower levels of threats of abuse, physical abuse, stalking, work harassment, and risk factors for femicide at 3, 6, 12, and 18 months after their initial contact with the justice system. ■

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Contributors

J. McFarlane conceived the study, supervised all aspects of its implementation, and wrote the first draft of the article. A. Malecha assisted with supervision of all aspects of the study, synthesized the analysis, and edited the drafts. J. Gist assisted with data collection and managed the study. K. Watson completed the data analyses. E. Batten translated all instruments and collected and coded data on Spanish speakers. I. Hall and S. Smith collected data, coded data, and synthesized analyses. All of the authors helped to conceptualize ideas, interpret findings, and review drafts of the article.

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Note. *The points of view offered in this article are those of the authors and do not necessarily represent the official positions or policies of the US Department of Justice.*

Human Participant Protection

This study was approved by the Texas Woman's University institutional review board. All participants provided informed consent according to the guidelines specified by the Texas Woman's University institutional review board.

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Assessing the Long-Term Effects of the Safe Dates Program and a Booster in Preventing and Reducing Adolescent Dating Violence Victimization and Perpetration

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Objectives. This study determined 4-year postintervention effects of Safe Dates on dating violence, booster effects, and moderators of the program effects.

Methods. We gathered baseline data in 10 schools that were randomly allocated to a treatment condition. We collected follow-up data 1 month after the program and then yearly thereafter for 4 years. Between the 2- and 3-year follow-ups, a randomly selected half of treatment adolescents received a booster.

Results. Compared with controls, adolescents receiving Safe Dates reported significantly less physical, serious physical, and sexual dating violence perpetration and victimization 4 years after the program. The booster did not improve the effectiveness of Safe Dates.

Conclusions. Safe Dates shows promise for preventing dating violence but the booster should not be used. (*Am J Public Health.* 2004;94:619–624)

Adolescent dating violence is a public health problem.[1–12] The Safe Dates Project is a randomized controlled trial for testing the effects of a school-based intervention on the prevention and reduction of dating violence among adolescents. Findings reported earlier suggested that 1 month after intervention, Safe Dates prevented and reduced dating violence and positively changed cognitive mediating variables that were based on program content.[13] One year after the intervention, cognitive risk factor effects were maintained, but behavioral effects disappeared.[14] These findings are consistent with those from prevention trials aimed at other adolescent problem behaviors that measured long-term effects: behavioral effects faded whereas effects on cognitive risk factors persisted.[15–18]

Three years after Safe Dates was implemented, a booster was implemented with a random half of the original treatment group adolescents. Boosters are intended to reinforce the content of original programs so as to maintain or regain initial program effects. They are typically a briefer version of the original program but with the same theoretical base and are administered at least 1 year after the original intervention. Boosters that have been used with school-based programs for preventing other adolescent problem behaviors have included newsletters followed by telephone contact with the adolescent,[19] magazines handed out to the adolescents in school,[20] and a reduced number of classroom sessions.[21,22] Our booster was a newsletter mailed to the adolescents and a personal contact from a health educator by telephone.

The purposes of this article are to (1) examine the 4-year postintervention effects of Safe Dates on dating violence perpetration and victimization and (2) determine whether the booster improved the effectiveness of Safe Dates. Findings from evaluations of other adolescent problem behavior interventions support the potential for long-term program effects[23] and booster effects[20,22,24] even after original program effects have faded. This is the first study to test the long-term effects of an adolescent dating violence prevention program and to test whether a booster prevents adolescent dating violence.

We examined the effects of Safe Dates and the booster on psychological, physical, serious physical, and sexual dating violence victimization and perpetration. Because the effects of programs for preventing other adolescent problem behaviors have been found to vary by gender,[25] race,[19] and preprogram involvement in the problem behavior,[15,20,21,26] we also determined if the effects of Safe Dates and the booster were moderated by these variables.

METHODS

Design

Adolescents were eligible for this study if they were enrolled in the 8th grade in the fall of 1994 in 1 of the 10 public schools in a rural North Carolina county. Baseline data were collected in October 1994 (wave 1) from 85.1% (n=957) of eligible adolescents. The 10 schools were then matched by school size. One member of each matched pair was randomly assigned to receive either Safe Dates or to serve as a control. Adolescents in the 5 treatment schools were exposed to Safe Dates from November 1994 through March 1995.

Safe Dates included a theater production performed by students, a curriculum comprising 10 45-minute sessions taught by health and physical education teachers, and a poster contest based on curriculum content. Process data suggested high program fidelity in treatment schools.[13,14] For details on program development, content, and theoretical base, see the 1996 report by Foshee et al.[27]

Follow-up data were collected from treatment and control adolescents at 1 month (wave 2) and 1 year (wave 3) after Safe Dates was completed. After wave 3, parents of adolescents who provided baseline data collection were recontacted to solicit permission for continued adolescent participation, and 65% (n=620) of the parents consented to have their child do so. Adolescents who had parental consent for continued participation completed questionnaires 2 years after Safe Dates (wave 4), and then the original treatment group adolescents were randomly allocated to

booster and nonbooster conditions. Hence, the study design changed from 2 groups (treatment and control) to 3 groups (treatment only, treatment plus booster, control). Adolescents completed questionnaires again 3 years (wave 5) and 4 years (wave 6) after Safe Dates was completed.

The booster was an 11-page newsletter mailed to the adolescents' homes and a personal contact by a health educator by telephone approximately 4 weeks after the mailing. The newsletter included information and worksheets based on content from the Safe Dates school curriculum. Examples of information presented include red flags that a relationship is abusive, effective communication strategies, and tips for safe dating. Five worksheets were included. As 1 example, in a large paper heart, adolescents wrote down how they want to be treated by dating partners (e.g., respected, listened to, treated equally), and in a circle with a line through it, they wrote how they did not want to be treated (e.g., lied to, threatened, ignored, humiliated). In another example, adolescents considered the short- and long-term consequences of various abusive behaviors for the victims and perpetrators.

Approximately 4 weeks after the mailing, a health educator made a personal contact with the adolescent by telephone. At that contact, the health educator answered the adolescent's questions related to the newsletter, provided additional information when needed, and determined if the adolescent read each informational component and completed the worksheets. The adolescent was mailed $10 after the health educator determined that the newsletter activities were completed. Approximately 82% of the adolescents assigned to receive the booster read the newsletter and completed the worksheets.

The analyses for this article are limited to the adolescents who completed baseline (wave 1) and both wave 4 and wave 6 questionnaires (n=460). Wave 4 data are required to assess whether booster effects differ by prior involvement in dating violence, and wave 6 data are required to assess booster and 4-year follow-up effects of Safe Dates. Of the 460 adolescents, 201 were in the control group, 124 were in the group that received only Safe Dates, and 135 were in the group that received Safe Dates and the booster. The only statistically significant difference between the study sample (n=460) and the 957 8th graders who completed baseline questionnaires was gender; there were significantly more females in the study sample (58.5%) than in the baseline sample (51.2%) (P=.01) (Table 1).

TABLE 1—Baseline Characteristics of the Baseline Sample and the Study Sample: North Carolina, 1994

	Baseline Sample (n = 957)		Study Sample (n = 460)	
	Percentage or Mean	Standard Deviation	Percentage or Mean	Standard Deviation
Female, %	51.20		58.50*	
White, %	72.80		75.60	
Mean perpetration scores				
Psychological	2.15	4.19	1.67	4.11
Physical	0.79	3.94	0.69	3.46
Serious physical	0.19	1.34	0.14	1.15
Sexual	0.06	0.50	0.05	0.38
Mean victimization scores				
Psychological	3.75	6.72	3.30	6.65
Physical	1.40	4.36	1.22	3.87
Serious physical	0.25	1.25	0.21	1.01
Sexual	0.16	0.66	0.17	0.72

Note. Satterthwaite's approximation for the degrees of freedom for the appropriate *t* test was used.
*P<.01

Measures

The 8 behavioral outcomes measured, 4 pairs of parallel perpetration and victimization outcomes, were anchored to the previous year. The frequency of perpetrating each of 14 psychologically abusive acts (e.g., "damaged something that belonged to them," "insulted them in front of others") was summed to form a composite score for psychological abuse perpetration. A parallel procedure was used to create a composite score for psychological abuse victimization. The frequency of perpetrating each of 18 physically and sexually violent acts (e.g., "slapped them," "kicked them," "hit them with my fist") was summed to form a composite score for physical violence perpetration. Serious physical violence perpetration was defined by the sum of responses to a subset of 6 serious acts (i.e., choked, burned, hit with a fist, hit with something hard besides a fist, beat up, and assaulted with a knife or gun). Sexual violence was defined by the sum of a subset of 2 acts (i.e., forced them to have sex, and forced them to do something sexual that they did not want to do). Parallel questions were used to measure physical, serious physical, and sexual violence victimization. Adolescents were asked to report acts perpetrated or received that were not in self-defense.

Attrition Analyses

The outcome in our attrition analysis was whether adolescents who completed a baseline questionnaire also completed wave 4 and wave 6 questionnaires. Our attrition analysis indicated that there were no significant interactions between treatment condition and baseline characteristics when predicting dropout status and that the amount of attrition did not differ for treatment and control groups. Gender and serious physical violence victimization were associated at P<.05 with dropout status in both treatment and control groups; males were more likely than females to drop out of the study (odds ratio [OR]=1.69; 95% confidence interval [CI]=1.13, 2.53), and the odds of dropping out decreased with increased serious physical violence victimization (OR=0.51 per unit; 95% CI=0.30, 0.89).

Analysis Strategy

Linear regression models were used to assess Safe Dates' effects and booster effects,

and effect modifiers. Each of the 8 wave 6 outcome variables was regressed on treatment condition (0=control and 1=Safe Dates but no booster), booster condition (0=Safe Dates and 1=Safe Dates + booster), and 4 covariates: gender (0=male, 1=female), race (0=White and 1=non-White), the wave 1 (baseline) value of the outcome variable, and the wave 4 value of the outcome variable. The interactions of the treatment and booster variables with the 4 covariates were included. The interactions with gender and race assessed whether program effects were moderated by gender and race, respectively. The interaction between the wave 1 value of the outcome variable and treatment condition assessed whether the effects of Safe Dates were moderated by prior (i.e., in the previous year) involvement in dating violence. The interaction between the wave 4 value of the outcome variable and booster condition assessed whether the effects of the booster were moderated by prior (i.e., in the year before the booster) involvement in dating violence. Models were reduced using a backward elimination procedure.

When statistically significant interactions remained in the reduced models, we calculated the predicted mean of the outcome for each intervention condition based on the parameters of the reduced models, and then calculated the difference in those predicted means at each level of the moderator variable. For these analyses, prior involvement in dating violence was reduced to 3 strata: no prior involvement, the mean level of involvement (average prior involvement), and the mean level of involvement plus 1 SD (high prior involvement). Statistical tests were computed to determine whether there were statistically significant differences in predicted means between the intervention conditions for each level of the moderator.

TABLE 2—Reduced Models When Predicting Perpetration of Dating Violence

	Psychological		Physical		Serious Physical		Sexual	
	β	SD	β	SD	β	SD	β	SD
Intercept	2.33**	0.55	0 .08	0.53	-0.01	0.17	-0.01	0.05
Treatment (Safe Dates vs control)	-1.07	0.72	-1.11*	0.49	-0.42**	0.16	-0.10*	0.05
Booster (Safe Dates + booster vs Safe Dates)	0.40	0.61	0.70	0.46	0.21	0.14	0.05	0.05
Gender	-0.25	0.42	-0.35	0.35	-0.18	0.11	-0.08*	0.04
Race	-0.18	0.49	-0.22	0.41	-0.07	0.13	-0.02	0.04
Wave 1 outcome	0.04	0.07	0.14**	0.05	-0.02	0.05	-0.00	0.05
Wave 4 outcome	0.13*	0.06	0.02	0.03	-0.01	0.03	-0.05	0.03
Wave 1 outcome by treatment	0.31*	0.14						
Wave 1 outcome by booster	-0.16	0.16						
Wave 4 outcome by treatment	-0.14	0.10						
Wave 4 outcome by booster	0.34**	0.12						

Note. The wave 4 outcome-by-treatment and the wave 1 outcome-by-booster interactions are included in the models as required because of the dummy coding of the treatment and booster variables, but they are conceptually meaningless. Analyses controlled for the correlation between individuals in the same school by using SAS PROC MIXED with school specified as a random effect.
*$P < .05$
**$P < .01$

RESULTS

We first present results concerning the long-term effects of Safe Dates, followed by results concerning the effects of the booster. For each, we present the effects on perpetration followed by the effects on victimization. Because neither race nor gender moderated either Safe Dates or booster effects on any of the 8 outcomes, these interactions are not further considered in this article.

Safe Dates' Effects on Perpetration

As shown in Table 2, adolescents who received only Safe Dates reported perpetrating significantly less physical (β=–1.11, P=.02), serious physical (β=–.42, P=.01), and sexual (β=–.10, P=.04) dating violence perpetration at the 4-year follow-up than those in the control group. Safe Dates' effects on psychological abuse perpetration are moderated by prior (wave 1) involvement in dating violence (β=.31, P=.02). As noted in Table 3, in all 3 strata of prior psychological abuse perpetration, the Safe Dates group reported less psychological abuse perpetration than the control group at follow-up. However, none of those differences were statistically significant. The likely reason for the significant interaction is that the difference in the Safe Dates and control group predicted means is progressively less as prior psychological abuse perpetration status increases.

Safe Dates Effects on Victimization

As shown in Table 4, Safe Dates had a significant main effect on sexual victimization (β=–.23, P=.01) in the expected direction but no effect on psychological abuse victimization (β=–.35, P=.68), and the effects of Safe Dates on physical and serious physical victimization were moderated by prior (wave 1) involvement with the behavior (β=.34, P=.02; β=.59, P=.003, respectively). As noted in Table 3, in all 3 strata of prior physical abuse victimization, the Safe Dates group reported less physical abuse victimization at follow-up than the control group. These differences were statistically significant when prior physical victimization was average (P=.01) and high (P=.002) and close to significant when there was no prior physical victimization (P=.07). The pattern was similar for serious victimization: in all 3 strata of prior serious physical victimization, adolescents exposed only to Safe Dates reported significantly less victimization from serious dating violence than adolescents in the control group did.

Booster Effects on Perpetration

As shown in Table 2, the booster did not improve the effectiveness of Safe Dates in preventing physical (β=.70, P=.12), serious physical (β=.21, P=.14), or sexual (β=.05, P=.26) dating violence perpetration, and prior (wave 4) involvement in psychological abuse perpetration moderated the effect of the booster on psychological abuse perpetration (β=.34,

TABLE 3—Differences in the Predicted Means on the Follow-Up Outcomes Between Specified Intervention Groups and Significance Levels, Stratifying by Prior Involvement in Dating Violence

Follow-Up Outcome	Prior Involvement[a] None	Average	High
Psychological abuse perpetration			
Safe Dates mean minus control mean	−1.07	−0.86	−0.33
Safe Dates + booster mean minus Safe Dates mean	0.40	0.88	2.02*
Physical abuse victimization			
Safe Dates mean minus control mean	−1.12	−1.53**	−2.74**
Safe Dates + booster mean minus Safe Dates mean	0.42	0.69	1.49
Serious physical victimization			
Safe Dates mean minus control mean	−0.45*	−0.50**	−0.66*
Safe Dates + booster mean minus Safe Dates mean	0.08	0.22	0.82*
Sexual violence victimization			
Safe Dates + booster mean minus Safe Dates mean	0.05	0.14	0.52***

Note: Predicted means for each treatment condition were calculated based on the reduced models in Tables 2 and 4. The differences in predicted means in the treatment conditions are presented in this table.
[a]Prior involvement refers to involvement in the same types of dating violence as the follow-up outcome.
*$P<0.05$
**$P<0.01$
***$P<0.001$

TABLE 4—Reduced Models When Predicting Victimization of Dating Violence

	Psychological		Physical		Serious Physical		Sexual	
	β	SD	β	SD	β	SD	β	SD
Intercept	3.67***	0.80	0.47	0.64	0.01	0.21	−0.16	0.09
Treatment (Safe Dates vs control)	−0.35	0.86	−1.12	0.62	−0.45*	0.20	−0.23**	0.08
Booster (Safe Dates + booster vs Safe Dates)	0.68	0.91	0.42	0.59	0.08	0.19	0.05	0.08
Gender	−0.46	0.69	−0.48	0.42	−0.11	0.14	0.02	0.06
Race	−1.30	0.80	−0.74	0.49	−0.29	0.16	−0.11	0.07
Wave 1 outcome	0.15**	0.06	0.04	0.08	−0.04	0.08	0.10*	0.04
Wave 4 outcome	0.30***	0.05	0.43***	0.06	0.37***	0.06	0.28***	0.06
Wave 1 outcome by treatment			0.34*	0.14	0.59**	0.20		
Wave 1 outcome by booster			−0.10	0.16	−0.24	0.24		
Wave 4 outcome by treatment			−0.44***	0.08	−0.42***	0.08	−0.28***	0.08
Wave 4 outcome by booster			0.21*	0.11	0.47**	0.15	0.50***	0.11

Note: The wave 4 outcome-by-treatment and the wave 1 outcome-by-booster interactions are included in the models as required because of the dummy coding of the treatment and booster variables, but they are conceptually meaningless. Analyses controlled for the correlation between individuals in the same school by using SAS PROC MIXED with school specified as a random effect.
*$P<.05$
**$P<.01$
***$P<.001$

$P=.003$). As can been seen in Table 3, those adolescents high in prior psychological abuse perpetration who were exposed to the booster reported significantly more psychological abuse perpetration at follow-up than those exposed only to Safe Dates ($P=.03$).

Next we compared the booster to the control group. Because of the way the treatment and booster variables were dummy coded, we were able to determine the differences in the predicted means between the control and the booster group from the estimates in Table 2. We determined if those differences were statistically significant using linear contrasts with SAS (SAS Institute Inc, Cary, NC). There were no significant differences between the booster and the control group in follow-up physical ($P=.38$), serious physical ($P=.16$), or sexual dating violence perpetration ($P=.28$). There were also no significant differences between those 2 groups in follow-up psychological abuse perpetration in any of the strata of prior (wave 4) psychological abuse perpetration. Thus, there were no situations in which the booster group reported significantly more perpetration at follow-up than controls.

Booster Effects on Victimization

We first compared the booster to the Safe Dates–only group. As shown in Table 4, there were no effects of the booster on psychological abuse victimization ($\beta=.68$, $P=.46$), and the effects of the booster on physical ($\beta=.21$, $P=.05$), serious physical ($\beta=.47$, $P=.002$), and sexual victimization ($\beta=.50$, $P<.0001$) were all moderated by prior (wave 4) victimization. As noted in Table 3, in all 3 strata of prior physical abuse victimization, adolescents exposed to the booster reported more physical victimization at follow-up than those exposed only to Safe Dates; however, none of these differences were statistically significant. A similar pattern emerged when considering serious physical and sexual victimization in that in all 3 strata of prior victimization, adolescents exposed to the booster reported more serious physical and sexual victimization at follow-up than adolescents who received only Safe Dates. Those differences were statistically significant only when prior involvement in dating violence was high.

Next we compared the booster to the control group. There were no significant differences between the booster and the control group in follow-up psychological abuse victimization ($P=.70$). Within the strata of prior (wave 4) physical, serious physical, and sexual violence victimization, the only significant differences in the booster and control groups were in serious victimization when there was no prior serious victimization ($P=.05$) and

sexual victimization when there was no prior sexual victimization (P=.03). In both cases, those exposed to the booster reported significantly less victimization than controls. Thus, there were no comparisons in which the booster group reported significantly more victimization at follow-up than controls, and in 2 comparisons, the booster group reported significantly less victimization at follow-up than controls.

DISCUSSION

In this 4-year follow-up of Safe Dates, we found significant treatment and control group differences in the expected direction in physical, serious physical, and sexual dating violence perpetration and victimization. Although prior victimization moderated program effects on physical and serious physical victimization, there were statistically significant program effects on those 2 victimization variables at almost all strata of prior victimization. The program was equally effective for males and females and for Whites and non-Whites. Compared with controls, adolescents exposed to Safe Dates reported from 56% to 92% less dating violence victimization and perpetration at follow-up.

It is unlikely that these favorable effects are due to differential attrition, because we found no evidence of greater attrition of high-risk adolescents from our Safe Dates group than from the control group, and the amount of attrition was the same in both groups.[28] Because of the long period since program exposure, it is also unlikely that these changes were the result of more socially desirable reporting of the outcomes by the treatment than the control group. A likely explanation for the favorable changes is that Safe Dates caused the changes observed. Long-term effects may have been realized because Safe Dates was offered at the beginning of the adolescents' dating careers (8th grade) and included information and skills that could be incorporated into individual dating practices that continued through the high school years. For example, adolescents were asked to actively consider how they wanted to be treated by their dating partners, they analyzed the negative consequences of being a perpetrator and a victim of dating abuse, they learned effective ways of communicating with their partners and for dealing with anger toward a partner, and they learned how having unfair gender-based expectations of partners could lead to abuse. Specific to the prevention of sexual dating violence, they analyzed verbal and nonverbal cues that a partner is not ready to have sex, were encouraged to be clear with partners about sexual boundaries, and discussed dating tips for protecting themselves from sexual dating violence and for respecting their partners.

The booster did not improve the effectiveness of Safe Dates. In fact, adolescents exposed to Safe Dates and the booster reported significantly more psychological abuse perpetration and serious physical and sexual victimization at follow-up than those exposed only to Safe Dates, but only when prior involvement in those forms of dating violence was high. It is possible that the booster prompted adolescents who were already being victimized to leave abusive relationships. Studies report that partner violence escalates when victims try to leave the abusive relationship.[29–31] Boosters, because of their low intensity, may be inappropriate for the secondary prevention of dating violence. Leaving an abusive dating partner can be complicated and dangerous, and adolescents doing so may need support from their family, friends, and community agencies. A booster may motivate a victim to leave the relationship but may need to be paired with additional support to do that safely and successfully.

Boosters have received substantial prominence. For example, both the National Cancer Institute and the Center for Substance Abuse Prevention list boosters as essential and effective elements of adolescent substance use prevention programs.[32,33] However, only 3 studies on adolescent substance use prevention rigorously evaluated the impact of a booster with an experimental design that allowed assessment of booster effects independent of original treatment effects,[20,22,24] and there have been no prior studies testing the effectiveness of a booster in preventing dating violence or other forms of youth violence. Our findings suggest that boosters could have negative effects. However, there were no situations in which the booster group reported significantly more victimization or perpetration at follow-up than the control group.

Attrition is the primary potential limitation of this study. However, as mentioned earlier, our analyses suggest that differential attrition did not threaten the internal validity of the study. It is also unlikely that attrition affected external validity given the similarity of the study sample to the baseline sample, which because of the high response rate should approximate the characteristics of 8th graders in the county. The study sample did have significantly more females than the baseline sample, but given that program effects did not vary by gender, this finding should not reduce the generalizability or external validity of the findings. These findings can be generalized with a fair amount of confidence to other rural counties with similar demographic characteristics. Relative to the United States as a whole, when the study was conducted, the county had an overrepresentation of minority residents, lower-income households, and more individuals with limited education.

Another potential limitation is reliance on self-reports of dating violence. Previous analyses of these data, however, suggest that our measures of dating abuse have high construct validity: they correlate as expected with theoretically based constructs[34]; also as expected, the prevalence of psychological abuse was larger than the prevalence of physical abuse, which was larger than the prevalence of serious physical and sexual abuse; and the prevalences of the various forms of dating abuse were comparable to those found in other adolescent dating abuse studies.[2,6] Also, consistent with almost all other studies of adolescent dating violence, gender was not associated with physical dating violence victimization[1,5,9] or perpetration[3,6,35] but was associated with sexual dating violence victimization, with females reporting more sexual dating violence victimization than boys.[9]

Safe Dates is being used in many geographically diverse areas, including inner-city urban areas, rural areas, and countries besides the United States. However, the only published evaluations of the Safe Dates program have been in this rural US sample.[13,14] Future studies are needed to determine the effectiveness of Safe Dates for adolescents living in other locales. Also, from anecdotal reports we know

that the program is not always used in its entirety (the play, curriculum, and poster contest), yet the design of our evaluation did not allow assessment of the effectiveness of individual components. Future evaluations need to incorporate designs that allow assessment of individual components and fewer curriculum sessions.

In conclusion, this is the first experimental study to test the long-term effects of an adolescent dating violence prevention program and to test the efficacy of a booster for preventing adolescent dating violence. Safe Dates reduced dating violence as many as 4 years after the program. The booster did not improve the effectiveness of Safe Dates. Neither gender nor race moderated program effects, but prior behavior moderated some effects. These findings suggest that implementation of the Safe Dates program to reduce dating violence is indicated but that the booster should not be used. ■

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Contributors

V. Foshee conceived of the study, supervised all aspects of its implementation, and prepared drafts of the manuscript. K. Bauman assisted with all aspects of designing and conducting the study. S. Ennett contributed to the analysis strategy, presentation of results, and interpretation of the findings. F. Linder managed all aspects of the study. T. Benefield completed all the analyses for the article with direct supervision by C. Suchindran and V. Foshee. C. Suchindran designed the analysis strategy. All authors contributed by conceptualizing ideas, interpreting findings, and reviewing drafts of the article.

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Human Participant Protection

This study was reviewed and approved by the University of North Carolina, School of Public Health, institutional review board for the protection of human subjects. Active parental consent and adolescent assent were obtained from all study adolescents.

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Community Violence and Asthma Morbidity: The Inner-City Asthma Study

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Objectives. We examined the association between exposure to violence and asthma among urban children.

Methods. We obtained reports from caretakers (n=851) of violence, negative life events, unwanted memories (rumination), caretaker-perceived stress, and caretaker behaviors (keeping children indoors, smoking, and medication adherence). Outcomes included caretaker-reported wheezing, sleep disruption, interference with play because of asthma, and effects on the caretaker (nights caretaker lost sleep because of child's asthma).

Results. Increased exposure to violence predicted higher number of symptom days ($P=.0008$) and more nights that caretakers lost sleep ($P=.02$) in a graded fashion after control for socioeconomic status, housing deterioration, and negative life events. Control for stress and behaviors partially attenuated this gradient, although these variables had little effect on the association between the highest level of exposure to morbidity, which suggests there are other mechanisms.

Conclusions. Mechanisms linking violence and asthma morbidity need to be further explored. (*Am J Public Health.* 2004;94:625–632)

In the United States, recent trends of increasing childhood asthma morbidity disproportionately affect urban children who are poor and non-White. Known risk factors (e.g., air pollutants, environmental and in utero tobacco smoke, viral infections, indoor allergens) do not fully explain these trends.[1]

Geographic variation in asthma outcomes among large cities[2] and among neighborhoods within cities[3–5] has been observed. Variation in asthma morbidity across urban neighborhoods cannot be explained by socioeconomic factors alone. Many New York City communities do not have elevated asthma morbidity in spite of the fact that they are comparably low on many socioeconomic indicators and have physical environmental exposures seemingly similar to other high-risk neighborhoods. These findings indicate that other factors may mediate the effects of living in low–socioeconomic status (SES) neighborhoods.

Health disparities research points to the influence of specific community characteristics, conceptualized as neighborhood disadvantages, on residents' health and well-being.[6] Neighborhood disadvantage—characterized by the presence of a number of community-level stressors, including poverty, underemployment, limited social capital, substandard housing, and high crime and violence rates[7]—is prevalent in many US urban communities.[8]

Studies of minority and low-income populations have shown a high prevalence of children who experience[9] and witness violence in the inner city.[10–12] A prevalence study in a Boston, Mass, pediatric primary care clinic found that 10% of children younger than 6 years had witnessed a knifing or a shooting and that 47% had heard gunshots in their neighborhoods.[11] In Chicago, Ill, investigators found that 42% of children between the ages of 7 and 13 years had witnessed a shooting.[13]

Exposure to violence may affect asthma through many pathways.[14] It may be related to psychological stress experienced by those who witness or are victims of violence,[15] which may have an impact on asthma.[16] Evolving research is exploring adverse psychological consequences among children who grow up in violent neighborhoods.[17,18] Health behaviors may be influenced by environmental factors, including high levels of stress, violence, and unpredictable daily life experiences. Exposure to violence (and other determinants of neighborhood disadvantage) may influence impulse control and risk-taking behavior, resulting in the adoption of coping behaviors (e.g., smoking) and leading to increased exposure to a known environmental trigger of asthma, (tobacco smoke).[19] Families who live in a violent environment may develop a fatalistic outlook that undermines their ability to invest in the future by complying with prescribed asthma treatment.[20]

High crime rates are correlated with other indicators of social disadvantage, including poor-quality housing. Deteriorated housing has been linked to high household cockroach allergen levels,[21] which in turn may increase asthma morbidity.[22] Exposure to community violence may influence behaviors that could result in increased exposure to other known environmental risk factors. Parents who live in high-violence communities may restrict their children's outdoor activities, causing increased indoor-allergen exposure and higher asthma morbidity. Individuals who live in low-SES neighborhoods with high violence rates also may experience other adverse life events more frequently than their higher-SES counterparts.[23,24]

We examined the association between exposure to community violence and caretaker-reported asthma symptoms and behaviors in the Inner-City Asthma Study (ICAS). We hypothesized that families with children who lived with higher levels of violence would have increased asthma morbidity. We examined factors that might be correlated with violence (SES and other adverse life events) and hypothesized mediating pathways, including measures of the psychological experience of stress (perceived stress and intrusive memories), poor compliance with medication regimens, and other caretaker behaviors (keeping children indoors and smoking).

METHODS

The ICAS was conducted from August 1998 to July 2001. This study enrolled 937 children with asthma (aged 5 to 12 years) and their caretakers to an intervention study to reduce symptoms. Families were recruited from 7 cities: Boston; Chicago; New York City (Manhattan and the Bronx); Dallas, Tex; Seattle, Wash; and Tucson, Ariz. The study design has been detailed elsewhere.[25] Eligibility required that the child had at least 1 hospitalization or 2 emergency department visits for asthma during the 6 months before screening. Census tracts with 20% to 40% of households below federal poverty guidelines were targeted. Many census tracts also were racially segregated (i.e., Black–White): Boston (39.4% vs 39%), Chicago (65.2% vs 18.5%), Manhattan (47.9% vs 22.1%), the Bronx (38.7% vs 19.3%), Dallas (44.5% vs 30.1%), Seattle (8.6% vs 69.4%), and Tucson (3.5% vs 59.1%).

After informed consent was obtained, trained bilingual interviewers administered a baseline survey to the child's primary caretaker that included questions about demographics, asthma morbidity, home environmental characteristics, exposure to tobacco smoke, the child's medication regimen and any problems with adherence, and psychosocial well-being measures described elsewhere.[26]

Assessment of Exposure to Violence

A community violence survey[27] was administered to caretakers. Caretakers were asked whether any of the following events had occurred in their neighborhoods during the past 6 months: (1) a fight in which a weapon was used, (2) a violent argument between neighbors, (3) a gang fight, (4) a sexual assault or rape, and (5) a robbery or mugging. Answers to these 5 items were summed to produce the Adult Violence Score. Additionally, caretakers were asked (1) whether the caretaker was afraid that the child would be hurt by violence in the neighborhood, and (2) whether the caretaker did not let the child play outside because of fear of violence in the neighborhood.

Additional Measures of Stress

The Negative Life Events (NLE) instrument is a modified version of the List of Recent Experiences[28,29] that has been shown to have good test–retest reliability for the scale (0.89 to 0.94) and for specific items (0.70).[30] Participants indicated whether they had undergone any of the enumerated experiences during the past 12 months and whether the experience in question had a positive or a negative impact. A few items (e.g., death of a family member) were assumed to be consensually negative. Total NLE score was derived by adding the number of negative experiences (either consensually rated or participant-rated).

The experience of unwanted thoughts and memories (rumination) was ascertained for each reported negative life event with this follow-up question: "In the last month, how often did you experience unwanted thoughts, memories, or images about this event?" Each item was scored on a 5-point frequency scale of "never" (0) through "very often" (4). A maximum score was based on the highest frequency of unwanted thoughts and memories reported for any experience (other than violence). Thus, if an individual reported 2 negative life events but experienced unwanted thoughts and memories only in connection with 1 of the events, the participant was classified on the basis of the higher frequency.

The 4-item Perceived Stress Scale (PSS4)[31] measured the degree to which respondents had felt that their lives were unpredictable, uncontrollable, and overwhelming in the preceding month (reliability = 0.85). Each item was scored on a 5-point frequency scale of "never" (0) through "very often" (4), and an overall/total score was obtained by summing the items (maximum = 16). Higher scores indicated greater stress.

Sociodemographic Indicators

Socioeconomic indicators included household income, the presence of at least 1 employed adult in the household, and caretaker level of education. Housing deterioration was assessed by summing a number of problems including water damage on walls or ceilings; other evidence of leaks; damaged or rotting windows; cracks or holes in floors; and chipped, cracked, or peeling paint on walls or windows. Race/ethnicity was categorized as Hispanic, Black, or White/other.

Outcome Measures

Measures of morbidity included caretaker-reported wheezing, sleep disruption, or interference with play activities caused by asthma during the preceding 2 weeks and the impact of the child's asthma on the caretaker (number of nights caretaker lost sleep because of child's asthma). A measure of maximum symptom days during the preceding 2 weeks was defined as the number of days that the child experienced wheezing, sleep disturbance, or disruption of play activities because of asthma.

Analyses

A total of 851 children and their caregivers had complete data for all covariates. We used analysis of variance (ANOVA) to examine mean outcome measures ([1] maximum symptom days and [2] nights caretakers lost sleep) by level of community violence. All analyses were adjusted for study center (site adjusted). A test for linear trend that used orthogonal polynomial coefficients was used to determine the relationship between (1) the Adult Violence Score and the mean asthma morbidity score and (2) the Adult Violence Score and the mean caretaker impact score.[32] Control variables were added in a stepwise fashion. We first added standard control variables, including SES, race/ethnicity, and a composite measure of general condition of the home, to ascertain whether associations we found were spurious (i.e., is level of exposure to violence merely a marker of low SES, race/ethnicity, or substandard housing stock, each of which may increase exposure to physical environmental factors related to morbidity?). We then added total NLE score to test whether the influence of violence on asthma morbidity was in part caused by greater exposure to other adverse events. Next we added hypothesized mediating variables. We introduced covariates into the linear model—individually or in sets—to determine whether they modified the effect of violence on morbidity. As covariates were added, we examined the change in the ANOVA model sums of squares related to the violence indicator. A substantial decrease in the effect size of the association between violence and the asthma morbidity

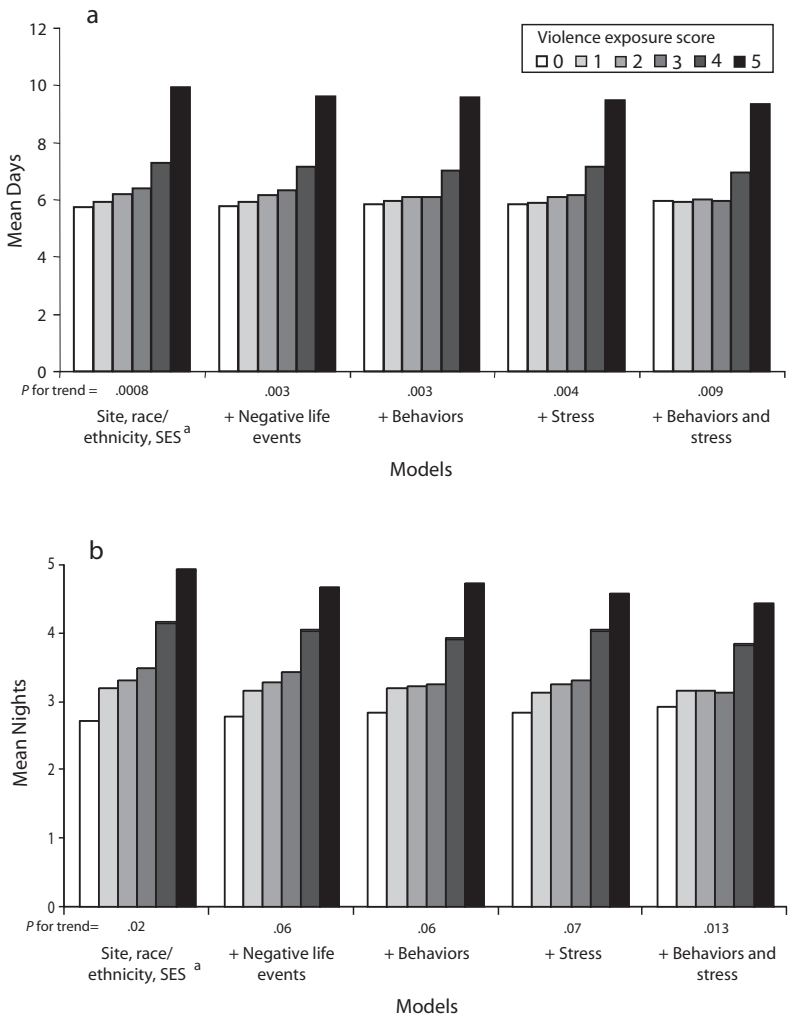
measure (i.e., percentage decrease in the violence sums of squares) would support 1 or more of these mediating pathways. We identified 2 mediating pathways—caretaker behaviors and the psychological experience of stress. Behaviors included (1) presence of smokers in the household, (2) caretakers skipping medications, and (3) caretakers not allowing the child to play outside. The stress-related predictors included (1) PSS4 score, and (2) unwanted thoughts about stressful life events (i.e., rumination over adverse events), which is another measure of coping. A final model was adjusted for site, SES, and all potential mediating variables. Mean outcome measures adjusted for other covariates were produced for each level of the violence score.

RESULTS

The frequency of caretaker-reported community violence varied across the sites: the highest mean scores were reported in Chicago and Manhattan (1.3 and 1.4, respectively); Boston (0.9), the Bronx (1.2), and Dallas (0.89) were close behind; and the lowest mean scores were reported in Seattle (0.84) and Tucson (0.65). Caretaker reports of violent events occurring in their neighborhoods during the past 6 months were quite prevalent for certain categories of events: a fight in which a weapon was used (28%), a violent argument between neighbors (33%), a gang fight (15%), a sexual assault or rape (9%), or a robbery or mugging (21%). More than one-third of caretakers (38%) reported being afraid their child would be hurt by violence in the neighborhood and reported keeping their children indoors owing to fear of violence (34%). Table 1 shows the mean exposure-to-violence scores and the outcome measures stratified by sociodemographic factors and by control variables. Those caretakers who had higher exposure-to-violence scores were more likely to be minorities, were less likely to report at least 1 employed adult in the household, had more housing problems, had greater perceived stress, ruminated more about adverse life events, smoked more often, kept their children indoors more often, and skipped medications more often than caretakers who had lower scores.

Site-adjusted analyses showed a gradient increase in mean maximum symptom days with increasing exposure to violence (P=.0006) (data not shown). Figure 1 shows the associations between violence exposure and mean (a) maximum symptom days or (b) nights caretaker lost sleep, adjusted for control and hypothesized mediator variables. We found no meaningful attenuation of the relationship between violence and caretaker-reported symptoms among children after we controlled for sociodemographic factors (we simultaneously adjusted for annual household income, presence of at least 1 employed adult in the home, caretaker education, housing deterioration score, and race/ethnicity) (P=.0008). A similar graded relationship for exposure to violence and caretaker impact was seen in these adjusted analyses (P=.02).

To assess whether level of exposure to violence was a marker for exposure to other adverse events, total NLE score was added to the model. The graded relationship between



Note. Behaviors = caretaker behaviors, including smoking, keeping children indoors, and skipping medications. Stress = Perceived Stress Scale and unwanted thoughts and memories. Each set of bars represents mean maximum symptom days (or nights caretaker lost sleep) adjusted for control variables. All models are adjusted for site, race/ethnicity, and socioeconomic status (SES) in addition to listed variates. *P*s are for trends.
[a]SES includes adjustment for household income, employment, caretaker education, and housing deterioration.

FIGURE 1—Mean (a) maximum symptom days and (b) nights caretaker lost sleep, by level of adult exposure to violence: adjusted analyses.

TABLE 1—Mean Caretaker Violence Exposure Scores and Maximum Symptom Days, by Potential Control, Confounding, and Mediating Variables: Inner-City Asthma Study (n = 851, baseline assessment), August 1998–July 2001

Characteristic	n (%)	Mean Violence Exposure Score	*P*	Mean Maximum Symptom Days	*P*	Mean Nights Caretaker Lost Sleep	*P*
Caretaker race/ethnicity							
Hispanic	364 (42.8)	0.96		5.93		3.15	
African American	340 (40.0)	1.16		6.48		3.19	
White/mixed/other	146 (17.2)	1.05	.1238	5.52	.1181	2.74	.5238
Household income, $							
< 15 000	512 (60.2)	1.11		6.40		3.33	
≥ 15 000	339 (39.8)	0.96	.0973	5.58	.0203	2.72	.0360
At least 1 employed adult in household							
No	207 (24.3)	1.33		6.77		3.81	
Yes	644 (75.7)	0.96	.0003	5.85	.0206	2.86	.0042
Caretaker education							
High school graduate	600 (70.5)	1.07		6.12		3.07	
Not a high school graduate	251 (29.5)	1.02	.5746	5.96	.6821	3.14	.8096
No. of housing problems							
0	355 (41.7)	0.88		5.86		2.76	
1	267 (31.4)	1.14		5.94		3.02	
2	82 (9.6)	1.35		6.32		4.04	
3	60 (7.0)	1.32		6.55		3.80	
4	38 (4.5)	1.18		7.42		3.50	
5	23 (2.7)	0.78		6.30		3.22	
6	26 (3.1)	1.08	.0106	6.35	.6134	3.00	.1964
Smoking in household							
No	432 (50.8)	0.97		5.66		3.01	
Yes	419 (49.2)	1.14	.0583	6.50	.0133	3.17	.5745
NLEs score (quartiles)							
1st	368 (43.2)	0.79		5.70		2.77	
2nd	130 (15.3)	0.85		5.58		2.58	
3rd	184 (21.6)	1.32		6.74		3.69	
4th	169 (19.9)	1.50	<.0001	6.54	.0435	3.51	.0218
Unwanted thoughts about adverse life events							
Never	296 (34.8)	0.74		5.55		2.76	
Almost never	53 (6.2)	0.81		5.74		2.64	
Sometimes	191 (22.4)	1.07		5.90		2.80	
Fairly often	135 (15.9)	1.04		5.82		2.79	
Very often	176 (20.7)	1.65	<.0001	7.44	.0017	4.33	.0006
PSS4 score (quartiles)							
1st	229 (26.9)	0.84		5.43		2.73	
2nd	262 (30.8)	1.11		6.11		3.14	
3rd	194 (22.8)	0.96		6.12		3.19	
4th	166 (19.5)	1.37	.0004	6.85	.0493	3.41	.4241
Ever skip medications							
No	550 (64.6)	0.97		5.57		2.83	
Yes	301 (35.4)	1.21	.0088	7.00	<.0001	3.56	.0146
Afraid to let child play outside							
No	559 (65.7)	0.82		5.71		2.66	
Yes	292 (34.3)	1.51	<.0001	6.76	.0037	3.90	<.0001

Note. NLE = Negative Life Events instrument; PSS4 = 4-item Perceived Stress Scale.

violence and morbidity markers remained significant.

Caretaker behaviors as mediating variables were then considered. Adjustments for smoking, keeping the child indoors, and skipping medications attenuated the gradient relationship between violence and morbidity markers. A significant trend remained for symptoms (P=.003), although the association was borderline significant for caretaker impact (P=.06). The decrease in impact of violence was not uniform across the gradient. The greatest absolute attenuation occurred in groups with lower levels of exposure to violence (i.e., violence exposure score ≤3).

When we adjusted for measures of psychological stress, including the PSS4 score and the frequency of unwanted thoughts and memories, exposure to violence remained an independent predictor of mean maximum symptom days (P=.004). We found attenuation of the graded relationship between violence and morbidity markers after we added these other measures of stress. Again, the greatest attenuation occurred in the groups with the lowest levels of exposure to violence. Notably, when frequency of other negative life events and the unwanted thoughts and memories were added together in models predicting asthma morbidity, negative life events were no longer significant (P=.5), suggesting that chronic reexperiencing of adverse life events may have a greater impact than discrete events.

In a final model adjusted for SES, other negative life events, perceived stress, unwanted thoughts and memories, and caretaker behaviors, increased exposure to violence was still associated with greater mean maximum symptom days (P=.009) and caretaker's losing sleep (P=.13), with more attenuation of the gradient in the groups with the lowest levels of exposure to violence.

To determine the relative contribution of the standard control variables and the purported mediators in explaining the effect of exposure to violence on morbidity, we examined the differences in the sums of squares associated with the violence exposure score alone in site-adjusted models and the sums of squares related to violence, and we adjusted for each covariate as it was added. The percentage decrease in the violence exposure sums of squares when each control variable was added is shown in Table 2. Socioeconomic indicators and smoking in the home explained relatively little of the effect of violence. Conversely, experiencing other negative life events and the occurrence of unwanted thoughts and memories of adverse events individually explained the greatest proportion of change in the violence exposure sums of squares. An intermediate proportion of change in the violence exposure sums of squares was explained by caretaker-perceived stress, skipping medications, and keeping the child indoors more often.

In subsequent models that adjusted for multiple variables, we again assessed by determining the percentage decrease in the violence exposure sums of squares in the respective multivariate models the relative contribution of the combined covariates in explaining the effect of exposure to violence. After we controlled for site, socioeconomic indicators, and race/ethnicity, 6% of the violence exposure effect was explained for symptoms and 11% for caretaker impact (i.e., on the basis of a 6% and 11% decrease in the violence exposure sums of squares in the respective models). After all behaviors were added with standard control variables (site, SES, race/ethnicity), 34% of the effect of exposure to violence was explained for symptoms and 50% for caretaker impact. After the other measures of stress were added with standard controls, 36% of the violence exposure effect was explained for symptoms and 51% for caretaker impact. A fully adjusted model including standard control variables, other negative life events, stress measures (perceived stress, unwanted thoughts and memories), and the behavior variables accounted for 46% of violence exposure effect for mean maximum symptom days and 70% for nights caretaker lost sleep.

TABLE 2—Percentage Decrease in Violence Exposure Sums of Squares After Each Variable Is Added Individually to the Model With Violence

Added Variable	Decrease in Violence Exposure Sums of Squares, %: Exposure to Violence Predicting Maximum Symptom Days	Decrease in Violence Exposure Sums of Squares, %: Exposure to Violence Predicting Nights Caretaker Lost Sleep
Socioeconomic status indicators		
Income category	3.0	2.6
Household employment	7.0	10.6
Caretaker education	0.37	-0.31
Housing problems	1.2	1.9
Caretaker behaviors		
Smoking at home	7.7	1.7
Skipping medications	14.0	12.5
Caretaker will not let child play outside	18.1	37.3
Negative life events	25.2	30.6
Experience psychological stress		
Unwanted thoughts	31.4	40.7
Perceived stress	18.1	16.7

DISCUSSION

As in previous studies,[10–13] a high prevalence of exposure to violence among the inner-city families was found. Greater exposure to violence was independently associated with asthma morbidity after simultaneous adjustment for income, employment status, caretaker education, housing problems, and other adverse life events, which suggests that exposure to violence was not merely a marker for these other factors. Psychological stress and caretaker behaviors (keeping children indoors, smoking, and skipping medications) partially explained the association between higher exposure to violence and increased asthma morbidity, although the greatest attenuation occurred among caretakers who re-

ported lower levels of exposure to violence. These findings suggest that other mechanisms are operating between high-level exposure to violence and childhood asthma morbidity.

The impact of exposure to violence on asthma morbidity was, in part, attenuated through psychological experiences of stress (i.e., the degree to which participants felt that their lives were uncontrollable, unpredictable, or overwhelming and the occurrence of unwanted thoughts and memories in connection with other adverse advents), supporting the notion that exposure to violence is a pervasive stressor that adds to environmental demands imposed on an already vulnerable population.[33] Living in a violent environment is associated with a chronic, pervasive atmosphere of fear and the perceived threat of violence.[34,35] Families who live with violence are more likely than those not exposed to violence to view their world and their lives as being out of their control.[36] Facing daily life experiences in an unpredictable or an uncontrollable environment may predispose these populations to suffer more deleterious effects from stress.[37]

Psychological stress has been associated with disturbed regulation of the hypothalamic-pituitary-adrenal (HPA) axis. An optimal level of mediators is needed to maintain a functional balance, and the absence of appropriate levels of glucocorticoids and catecholamines may allow immune mediators to overreact, thereby increasing the risk of inflammatory disorders, such as asthma.[38,39] In this framework, exposure to violence may be a psychosocial environmental factor that can "get into the body" and result in long-term biological changes that contribute to asthma morbidity.

Life events can have long-term effects on stress through lasting psychological, behavioral, and physiological responses maintained by recurrent unwanted thoughts about past events.[40] Caretakers who reported higher levels of exposure to violence were more likely to ruminate. Ongoing rumination may have an impact on problem-solving skills, may erode perceived control, and may decrease motivation to manage ongoing challenges, including management of a chronic illness such as asthma.[41] Caregivers who use ruminative coping strategies may experience greater stress and psychological comorbidity[42] that may more directly influence a child. Growing evidence links caregiver stress to the stress responses of their offspring. Animal and human studies suggest that caregiver stress may influence the stress response of the child and may modify infant neuroendocrine function during early development.[43–45] It also is possible that caretaker exposure to violence resulting in posttraumatic stress symptoms (e.g., avoidance, rumination) may cue their children to adopt less effective coping strategies, so that the children themselves experience greater stress.[46] This area of study warrants further research.

Poor adherence to medication regimens partially explained the relationship between exposure to violence and asthma. Coping with a violent environment may have an impact on compliance with therapy and with medical follow-up. Living in a violent community has been conceptualized as a barrier to keeping appointments and to following prescribed exercise programs.[47] Fearing to make a trip to a pharmacy or a medical facility may lead to lapses in prophylactic medication use, delayed intervention, and higher morbidity. Ruminative coping may influence problem-solving behaviors, which may impede compliance. Other unmeasured barriers to medication adherence may exist; for example, pharmacies may be reluctant to remain open 24 hours a day in high-crime communities. Exposure to violence may have an impact on access to medical care by diverting limited funds away from primary care and asthma specialty clinics.[48] Future research exploring other potential mediating pathways may contribute to more effective intervention strategies targeting high-risk urban populations.

Keeping children indoors also mediated the violence exposure and asthma relationship in our study. Children who are kept indoors will be more sedentary than those who go outside. This sedentariness may be linked to obesity, which has increased among US families who live in poverty[49] and has been linked to asthma.[50,51] Another reasonable hypothesis is that children who are restricted from going outside may have greater exposure to aeroallergens and increased likelihood of sensitization. Further research is needed to systematically examine this hypothesis.

Unexpectedly, smoking had little impact on the association between exposure to violence and asthma, perhaps because smoking is a strategy to cope with stress[52] that is related to violence.[19] This finding may reflect the fact that smoking was considered a dichotomous predictor, and we did not account for dose (i.e., number of cigarettes per day) or misclassification of self-reported smoking.

Exposure to violence and asthma morbidity were related in a graded fashion, even after we adjusted for socioeconomic indicators. The greatest absolute attenuation of the gradient occurred at the lowest level of exposure to violence, after we controlled for potential mediators. The relationship between the highest level of exposure to violence and increased asthma morbidity was not influenced by caretaker behaviors, perceived stress, or recurrent memories. Other factors important in explaining the association at the highest level of exposure to violence (i.e., that covary with high rates of exposure to violence) may not have been measured or cannot be fully adjusted for when accounting for individual-level SES factors. Crime and violence (or their absence) can be thought of as indicators of collective well-being or social cohesion within a community,[53,54] constructs increasingly linked to health.[55]

Neighborhood disadvantage, including higher crime rates and community violence, is enhanced in more racially segregated communities.[56] Segregated minority group status may predispose individuals to other pervasive stressors (e.g., discrimination, institutionalized violence, police injustice), a lack of infrastructure of the sort facilitating healthy living (e.g., fewer facilities for healthy physical recreation or purchase of healthy foods), and other societal factors that link minorities with neighborhood disadvantage.[57,58] Thus, individuals in these communities may face multiple social challenges simultaneously. Whereas individual psychosocial stressors may have small effects, cumulative stressors (at the individual and ecological levels) can enormously increase the likelihood of adverse health outcomes.[59]

Marginalized groups are disadvantaged not only in their vulnerability to adverse events but also in their access to coping resources.[60,61] Parents who are worried about their children's safety may restrict their social

behavior; thus, the family's ability to develop support networks may be compromised (i.e., exposure to violence may lead to diminished stress-buffering factors). These additional supports are especially important to the well-being of populations faced with the cumulative effects of many ecological stressors.

CONCLUSIONS

High crime rates and, thus, the real or perceived threat of violence are aspects of the inner-city environment that have an impact on the psychological and physiological functioning, as well as the health-promoting behaviors, of the inhabitants. Exposure to violence contributes to the environmental demands that tax both individuals and the communities in which they live. Systematic exploration of an association between exposure to violence (an urban stressor) and asthma may help us understand the rise in morbidity and further our understanding of the disproportionate asthma burden among poor urban children. ■

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Contributors

H. Mitchell, J. Stout, and R. Evans participated in the conceptualization of the study and the implementation of the Inner-City Asthma Study as principal investigators at their respective study sites. H. Mitchell, S. Cohen, D.R. Gold, and R.J. Wright guided the inclusion of stress measures in the parent study. H. Mitchell and C.M. Visness conducted the analyses and were guided by input from all of the authors. R.J. Wright synthesized the analyses and led the writing. All of the authors interpreted the findings and reviewed drafts of the article.

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This study was approved by the institutional review boards of all participating institutions.

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Social Disparities in Housing and Related Pediatric Injury: A Multilevel Study

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Objectives. We conducted an ecologic analysis to determine whether housing characteristics mediate the associations between concentration of poverty and pediatric injury and between concentration of racial minorities and pediatric injury and whether the association between housing conditions and pediatric injury is independent of other risks.

Methods. We created a hierarchical data set by linking individual-level data for pediatric injury with census data. Effect sizes were estimated with a Poisson model.

Results. After adjustment for owner occupancy and the percentage of housing built before 1950, the association between concentration of poverty and pediatric injury was attenuated. For concentration of racial minorities, only percentage of owner occupancy had some mediating effect. In hierarchical models, housing characteristics remained independent and significant predictors of pediatric injury.

Conclusions. The association between community characteristics and pediatric injury is partially mediated by housing conditions. Risk of pediatric injury associated with housing conditions is independent of other risks. (*Am J Public Health*. 2004;94:633–639)

It is commonly, but not universally, reported that children residing in areas with concentrated poverty or a high concentration of minorities suffer unintentional injury at higher rates than do other children.[1–7] However, concentrated poverty and concentration of minorities cannot be considered causal; they are correlates of more proximal determinants of injury. Because young children often suffer injuries at home,[8] at least some of these more proximal determinants are likely to be related to housing conditions. To the extent that housing conditions reflect conditions of the larger residential area,[9,10] they can be viewed as community-level determinants of injury, but more proximal community-level determinants than social conditions such as poverty. Moreover, because housing conditions are relatively more proximal to injury than social conditions, it is likely that the association between social conditions and injury is, at least partially, mediated by housing conditions. Other factors—particularly those at the individual level, such as parental supervision—are also among the important correlates of pediatric injury. However, the focus of this study was on housing conditions.

Age of housing is a likely determinant of injury.[8] Older houses are less likely to be in compliance with building or sanitary codes and may have substandard electrical and heating systems, narrow stairwells, or other safety hazards.[11,12] Another likely housing-related determinant of injury is whether a house is owner occupied or rented. Inadequate or deferred maintenance can be a common problem in low-income rental properties,[13,14] and high tenant turnover can increase the number of people exposed to these hazards over time.

To date, no studies have examined the association between housing factors and injury in which social conditions (e.g., concentrated poverty) and individual-level determinants (e.g., age, gender) have been simultaneously examined. We present the first multilevel, population-based study of pediatric injury. We also respond to calls for the study of nonfatal injuries,[15–17] the most severe of which require hospitalization. Injuries requiring hospitalization are often associated with high treatment and rehabilitation costs[18] and appear to have patterns and risks that are distinct from fatal injuries or injuries that do not require hospitalization.[16,19] As a consequence, the study of nonfatal, hospitalized injuries can provide information useful for their prevention. Falls and burns are among the most prevalent causes of pediatric injury.[20] In our sample, they accounted for 59% of all hospitalized pediatric injuries; after adjustment of hospital charges to true costs, the estimated median cost was $4670 for burns and $2760 for falls.[21] As a consequence, these types of injury were used to test the following hypotheses: community-level owner occupancy and age of housing (measured at the zip code level) mediate the association between concentration of poverty and concentration of minorities and risk for pediatric injury, and the association between pediatric injury and community-level housing conditions is independent of individual- and other community-level determinants of injuries requiring hospitalization.

METHODS

Sources of Data

Data from 2 different sources were used in this study. Information for all hospital discharges in the state of Illinois for 1990 through 2000 was abstracted from administrative hospital discharge data compiled as part of an Illinois state mandate[22] (as described elsewhere[23]), and the 1990 US census collected housing information by zip code, including number of owner-occupied units, number of residents living below the federal poverty limit, number of housing units built before 1950, and number of residents, by race. We linked these 2 data sets by zip code and created a hierarchical data structure with both individual- and zip code–level data.

Diagnostic codes were based on the *International Classification of Diseases, 9th Revision,*[24] in which E-codes reflect external causes of injury, poisoning, or other adverse events. An observation was defined as a fall if 1 of the principal or secondary diagnosis codes contained an E-code between 880 and 888. An observation was defined as a burn if it had an E-code between 890 and 899, 924.0 and 924.9, or 925.0. E-codes 925.1, 925.2, 925.8, and 925.9 were defined as nondomestic burns and were excluded. Sixty-

nine observations had 2 types of injury E-codes, in which case the first recorded E-code was used. Only children 6 years old and younger were included in the analysis. A total of 241 (2%) observations were excluded because they could not be linked to the 1990 US census. Of these, 96 observations were in zip codes created after 1990, and 29 observations were in zip codes that were either exclusively for post office boxes or for commemorative postal issues. The zip codes of 116 (1%) observations could not be identified in the *Postal Bulletin* archives, and these codes were also removed. Of the 1240 zip codes listed for the state of Illinois in 1990, 12 had no households or no children 6 years old or younger. Thus, 11 735 observations in 1228 zip codes remained for analysis.

Statistical Analyses

Data for this study involved counts of discharged injuries nested within a zip code. Because counts of injuries at the zip code level are bounded at zero and approximate a binomial distribution with a large number of trials and a small probability of success, the data assumed the shape of a Poisson distribution. Therefore, Poisson regression was used to model the rate of injury as a function of individual and community variables. An assumption of the Poisson distribution is that each individual observation is an independent event. In this study, some observations may have represented the same person or perhaps children from the same family, thus violating this assumption. We examined the extent of this violation, and our analysis indicated that multiple discharges for the same child were rare. Random samples of 1000 falls and 1000 burns were matched by birth date (month, day, and year), gender, and insurance type. Only 2 falls and 10 burns matched with 1 other observation on all 3 characteristics. When we used only birth date and gender, 3 falls and 13 burns had multiple observations. Thus, only about 1% of the falls and burns might have been repeat discharges of the same person. Moreover, the low probability of falls or burns in the population further limited the likelihood that the assumption of independence was violated. For these reasons, we assumed that the events were independent. Also, whereas violation of independence may have led to overestimation of injury rates, lack of independence will not bias rate ratios so long as the extent of overestimation is comparable across subgroups. Finally, offsets for rate calculations were based on the 1990 census population of children by age and gender.

The hierarchical structure of the data led to correlation between counts that were nested within zip codes. Thus, estimates of rates and rate ratios of injuries and their associated 95% confidence intervals were calculated using Generalized Estimating Equations, a method that allows for specification of the Poisson distribution and accommodates correlated data.[25] An exchangeable correlation structure was assumed, and standard errors for the parameters were based on the empirical estimator of the covariance matrix of the estimated coefficients. Rate ratios and their 95% confidence intervals were calculated in SAS with the GENMOD[26] procedure.

Chi-squared goodness-of-fit tests[27] indicated some lack of fit, so the standardized Pearson residuals were examined, and 3 outlying zip codes that were exerting undue influence on the coefficients were removed. Next, residual plots versus the predicted values and the covariates were examined. No systematic biases were detected that would indicate nonlinearity.

The distributions of concentrated poverty and concentration of minorities were also divided into decile indicator variables, and saturated models were fit. Because plots of the point estimates and confidence intervals evinced a slight curvature, both of these variables were divided into tertiles. This procedure was informed by data from the 1990 US Census. In 1990, approximately 12% of the population was African American[28]; this proportion was unchanged in 2000.[29] Between 1990 and 2000, the percentage of children living in poverty declined from approximately 20% to 16%.[30] Thus, the 3 categories for each of the census indicators corresponded roughly to below average, average, and above average for concentrated poverty and percentage African American in the United States.

Multivariate analyses followed the study aims. First, hierarchical models were fit to examine whether owner occupancy and age of housing mediated the association between concentration of poverty or concentration of minorities and pediatric injury. For example, the mediating effect of owner occupancy was examined by entering this variable in a model that, in addition to the individual-level variables, included either zip code–level concentrated poverty or concentrated minority. A significant change in the coefficient for concentrated poverty or concentrated minority indicated that its association with injury was mediated by owner occupancy (see D'agostino[31] for the significance test). Multivariate hierarchical models also were developed both with and without interactions.

RESULTS

In the state of Illinois, from January 1, 1990, through September 30, 2000, there were 11 735 hospital discharges of children with nonfatal injuries coded as occurring at home. The annual incidence rates for the 2 most prevalent types of injury, falls (43.5%) and burns (15.2%), were 3.93/10 000 population aged 6 years or younger (95% confidence interval [CI]=3.83, 4.04) and 1.37/10 000 population aged 6 years or younger (95% CI=1.31, 1.44), respectively. Fifty-eight percent of burns occurred among children aged 1 through 2 years, whereas falls were fairly evenly distributed across age groups. The median length of hospital stay for burns and falls was 5 and 2 days, respectively.

Bivariate analyses of individual-level variables indicated that compared with children aged 5 through 6 years, infants (<1 year) were more likely to suffer an injury resulting from a fall, and toddlers (aged 1 through 2 years) were most likely to suffer a burn. Males were at a higher risk for both types of injury. Bivariate analyses of the zip code–level variables for tenancy and age of housing demonstrated the change in risk for falling or being burned following a 10% increase in the continuous explanatory variable. For every 10% increase in the proportion of owner-occupied units, risk for falling decreased by 16% and risk for being burned decreased by 27%. A 10% increase in the proportion of housing built before 1950 was associated with a 17% increase in risk for falling and a 34% increase in risk for being burned. Chil-

TABLE 1—Hierarchical Fall and Burn Models

	Fall Rate Ratio	(95% CI[a])	Burn Rate Ratio	(95% CI[a])
Individual				
Age, y				
<1	2.13	(1.94, 2.34)	8.16	(6.77, 9.83)
1–2	1.11	(1.01, 1.23)	8.28	(6.92, 9.92)
3–4	0.97	(0.88, 1.07)	2.02	(1.64, 2.49)
5–6 (reference group)	1.00	. . .	1.00	. . .
Gender				
Male	1.45	(1.35, 1.55)	1.32	(1.18, 1.48)
Female (reference group)	1.00	. . .	1.00	. . .
Zip Code				
Percentage owner-occupied housing[b]	0.94	(0.90, 0.99)	0.92	(0.84, 1.00)
Percentage housing built before 1950[b]	1.10	(1.06, 1.15)	1.11	(1.04, 1.18)
Concentrated poverty				
High	1.05	(0.86, 1.27)	2.10	(1.56, 2.83)
Middle	1.02	(0.83, 1.24)	1.79	(1.36, 2.36)
Low (reference group)	1.00	. . .	1.00	. . .
African American population				
High	1.92	(1.55, 2.36)	2.64	(1.84, 3.79)
Middle	1.43	(1.17, 1.74)	1.24	(0.88, 1.74)
Low (reference group)	1.00	. . .	1.00	. . .

[a]Wald confidence interval.
[b]Rate ratios per 10% increase in the value of the independent variable.

dren residing in zip codes with the highest concentrations of poverty, compared with residents of zip codes with the lowest concentrations of poverty, were significantly more likely to sustain a fall or burn. Similarly, children residing in zip codes with the highest concentrations of minorities were significantly more likely to sustain a fall or burn than residents of zip codes with the lowest concentrations of minorities.

Next we examined the mediating effect of housing conditions on risk for injury associated with concentrated poverty and a concentration of minorities (Figures 1a–2b). The association between concentrated poverty and risk for falling or being burned was mediated by both owner occupancy and age of housing; the mediation was considerably larger and significant for the top tertile. When both housing conditions were included in the model for falls, the risk ratio for areas with a medium concentration of poverty became insignificant, and the risk ratio for areas with a high concentration of poverty remained only marginally significant. For burns, the risk ratios for middle and high levels of poverty were reduced but remained significant when both housing conditions were included in the model.

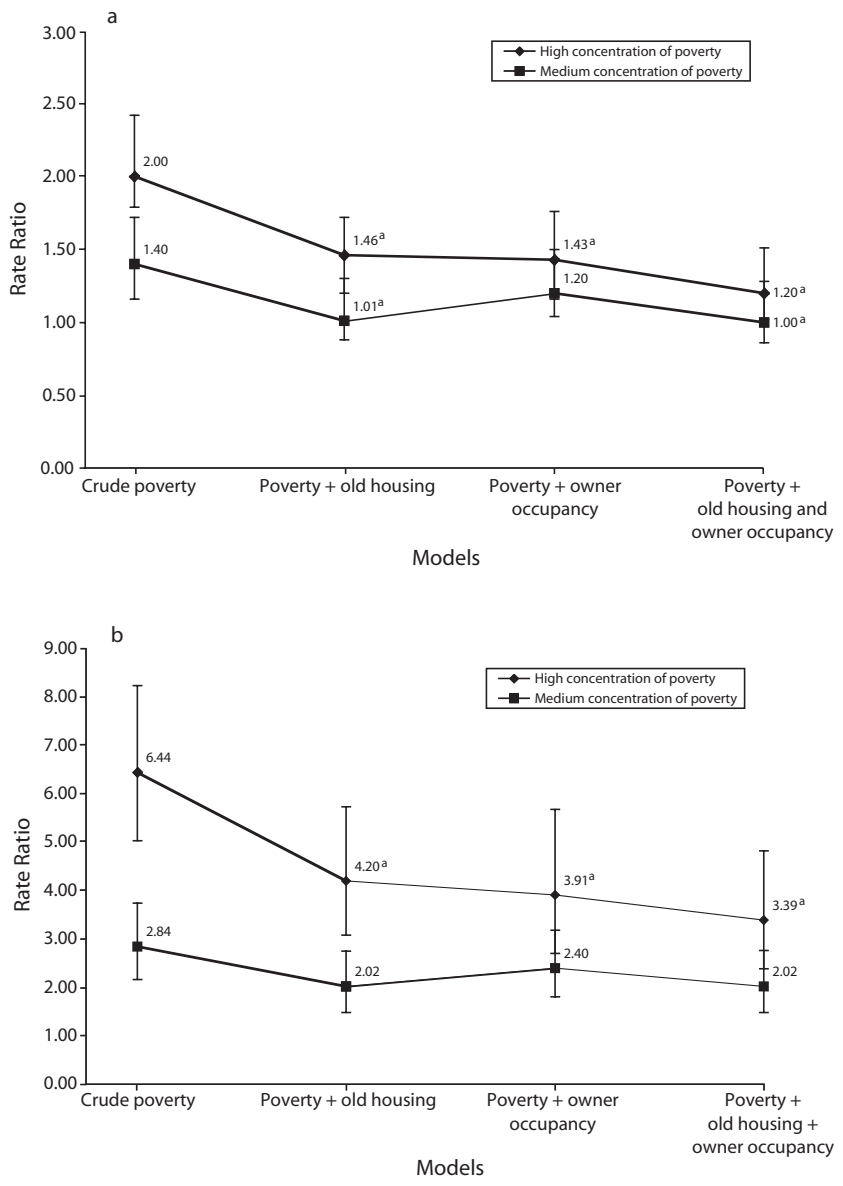
For concentrated minority populations, only owner occupancy appeared to be a mediator. Again, the mediation was larger and significant for the top tertile. Inclusion of age of housing actually increased the risk ratio for the middle tertile and only slightly reduced the risk ratio for the top tertile. The full model for falls (i.e., including both housing conditions) rendered significant the associations for areas with both medium and high minority concentration. The full model for burns evinced a significant association for areas with a high minority concentration, but the risk ratio for areas with a medium concentration became insignificant.

In the final hierarchical models (Table 1), both owner occupancy and age of housing remained significant predictors of both types of injury. We also found that owner occupancy modified risk for injury associated with concentration of poverty and minority concentration. In particular, owner occupancy was more protective in areas with higher concentrations of poverty and was less protective in areas with higher minority concentration and old housing.

DISCUSSION

Our findings indicate that the community-level concentration of owner-occupied housing and age of housing are significantly associated with rates of nonfatal hospitalized pediatric injury. This is in line with earlier findings that a variety of negative health outcomes, including fatalities caused by house fires, all-cause mortality, and elevated lead blood levels, are significantly more prevalent among renters than among homeowners.[1,2,32,33] This study further illuminates the association between housing conditions and health in 2 important ways. First, our results indicate that housing conditions mediate the association between community characteristics, such as concentrated poverty, and pediatric injury. Second, the results of our hierarchical analyses demonstrate that the association between housing conditions and pediatric injury is independent of both individual- and other community-level determinants of injury.

However, the association between these community characteristics and injury does remain significant after control for housing conditions. That the association between concentrated poverty and injury is only partially mediated by housing conditions may be in part the result of the fact that during most of the period covered by this study, some of the zip codes with concentrated poverty also had a concentration of federally owned or subsidized housing, which must meet minimum maintenance standards. Partial mediation of the association between concentration of minorities and injury is consistent with the findings of previous studies of racial disparities in health. These studies found that a significant proportion of health-related racial disparities remain unexplained even after control for social factors, probably because of the myriad of pathways by which race relations can influence health and because of the difficulties in capturing all of these influences in 1 study.[34–39]



[a]The estimate is significantly different from that of the crude model.

FIGURE 1—Old housing and owner occupancy as mediators of risk of (a) falls and (b) burns associated with poverty.

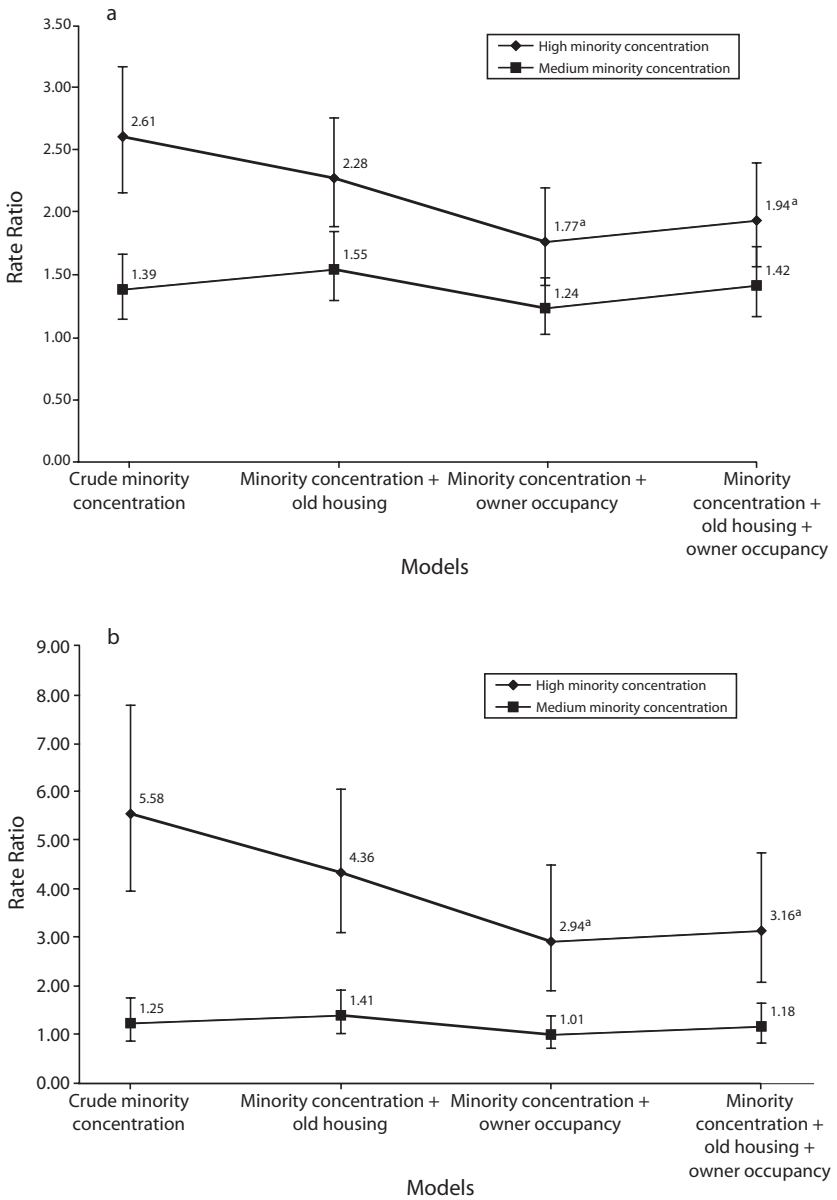
The implications of these findings should be considered in light of the study's limitations and strengths. A common shortcoming of ecologic studies such as this is the use of broadly defined and heterogeneous geographic areas (zip codes in our case) as the unit of analysis.[40,41] This misspecification can dilute the effect of interest and result in underestimation of the true effects. This misspecification also can compromise the accuracy by which community-level variables allow measurement of the underlying construct of interest. Given the heterogeneity of zip codes, underestimation of the relative risks and a degree of residual confounding are both possibilities in this study.

The main threat to validity of any ecological study is ecological fallacy, and we addressed this issue both methodologically and logically. In addition to controlling for key confounders, we limited our inferences at the ecological level (i.e., inferences regard group rates and not individuals' risk).[42,43] As outlined below, the validity of our conclusions are bolstered by their logical plausibility, an important criterion of validity for any study.[44] Arguably the most prominent source of ecological fallacy that remains unaccounted for in this study is parental child supervision and its correlates such as race/ethnicity and income. Clearly the link between poor supervision of children and injury (e.g., placing high chairs close to windows without screens) can masquerade as an effect of housing conditions. (It can be logically argued that given the same degree of parental supervision, children residing in well-maintained homes would be less likely to suffer injuries.) It is also reasonable to question whether housing conditions are themselves proxies for poverty and compromised ability to supervise children or are proxies for racial/ethnic norms about parenting.

In regard to poverty, although several developmental models evince a negative correlation between income and the quality of parenting,[45,46] material deprivation is not uniformly linked with poor child health outcomes or parenting practices. For example, low-income Hispanic women can have better pregnancy outcomes than wealthier residents of the same area, a paradox that may be attributable to social cohesion and the presence of extended family members among this population.[47–49] We suggest that social cohesion and the presence of extended family members also can promote better child supervision.

In regard to racial/ethnic differences in parenting, the literature generally indicates that correlates of poor parenting skills and supervision are not differentially distributed by race/ethnicity. Although certain parenting practices are more prevalent among various racial/ethnic groups, these differences are not necessarily correlated with poor developmental outcomes.[50] Moreover, the influence of race/ethnicity is often confounded by socioeconomic status and recency of immigration.[51,52] However, it appears that the effect of socioeconomic status is stronger and absorbs that of race/ethnicity.[53,54]

Another persuasive piece of evidence for the independent contribution of housing conditions to risk for pediatric injury is the signif-



[a]The estimate is significantly different from that of the crude model.

FIGURE 2—Old housing and owner occupancy as mediators of risk of (a) falls and (b) burns associated with minority concentration.

icant and sustained reduction in pediatric falls and burns that is attributed to the installation of window guards and sprinklers.[55,56] These passive measures address building defects and not parenting skills. In these cases, even when a high chair is placed next to a window, the window guard will prevent a fall regardless of the parents' income or skills. Thus, we suggest that the reported mediation by housing conditions is not simply a reflection of ecological fallacy. Future studies could be more persuasive by adjusting for more individual-level correlates of injury.

Finally, because our results are from racially segregated geographic areas, the generalizability of our findings may be limited to such areas. However, racial segregation is unfortunately a defining characteristic of many US cities.[35,39] Subsequently, our findings are likely to be generalizable to a broad cross-section of residential areas in the United States.

A strength of this study is our use of both individual- and community-level data in a hierarchical design. To our knowledge, this is the first study of injury that has used such a design. Another strength is our analysis of incident cases of acute health outcomes. This feature discounts 2 competing explanations that often limit inference regarding the effect of place on health. First is the argument that the health outcomes attributed to a place may be simply long-term consequences of individual-level characteristics.[57] Although this argument can be persuasive in the case of prevalent chronic illnesses, the argument that acute events such as injury among children age 6 years or younger are long-term consequences of individual-level characteristics is far less compelling. The second competing explanation, "social drift,"[57] suggests that those in poor health, through loss of economic status, "drift" to poorer residential areas. In this study, by examining incident cases among young children, we have rendered social drift an unlikely explanation for the disparate concentration of injury in poor and wealthy areas.

The association between housing conditions and pediatric injury has both immediate and long-term implications. The immediate implications regard the efficacy of intervention and prevention programs.[58] These programs can increase their efficiency and efficacy by considering both housing and socioeconomic characteristics of the community. Although not the focus of this study, we did find evidence of some interesting interactions. For example, we found that owner occupancy is less protective in areas with a high proportion of old housing. We also found owner occupancy to be more protective in areas with a higher concentration of poverty. This indicates that where a high proportion of homes were built before 1950, interventions should target old housing, whereas in high-poverty areas, the first priority should be non–owner-occupied homes.

The long-term implications are predicated on replication of these findings in longitudinal studies that consider more individual-level

variables, including parenting practices, conducted with smaller units of analysis. If such studies, as we suspect, further support our conclusions, then the long-term implications pertain to remediation of social disparities in health through remediation of differences in housing. Housing is an indicator (at times even a leading indicator) of conditions prevalent in the larger residential area. For example, age of housing reflects neighborhood economic conditions and can foretell future economic patterns. In 1990, housing units in high-poverty US census tracts, compared with low-poverty census tracts, were on average 11 years older and had correspondingly lower market value.[9] The units' low market values are a disincentive for maintaining them and a harbinger of worse physical conditions and further reductions in value. Concentration of units with low market value can initiate a cycle of private and institutional divestments, such as "redlining" by financial institutions, which can lead to the deterioration of neighborhoods' social, financial, and physical resources.[9,10] In turn, poor housing conditions have important and wide-ranging health implications.[9,33,59–61]

Vigorous application of already existing programs[62] to improve housing conditions may have benefits beyond the immediate residences and their occupants. This study indicates that social disparities in health may be addressed, at least partially, through remediation of social disparities in housing—a remediation that also can benefit future generations.[63,64] ■

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Contributors

E.D. Shenassa contributed to the conceptual development of the study, the data analysis plan, and the writing of the article. A. Stubbendick contributed to the development of the data analysis plan, the analysis of data, and the Methods and Results sections of the article. M.J. Brown contributed to the conceptual development of the study, the data analysis plan, and the writing of the article.

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Human Participant Protection

This study was exempt from human subject review because the secondary data used for the analysis did not contain personal data.

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Parental Social Determinants of Risk for Intentional Injury: A Cross-Sectional Study of Swedish Adolescents

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Objectives. We investigated the effect of family social and economic circumstances on intentional injury among adolescents.

Methods. We conducted a cross-sectional register study of youths aged 10 to 19 years who lived in Sweden between 1990 and 1994. We used socioeconomic status, number of parents in the household (1- or 2-parent home), receipt of welfare benefits, parental country of birth, and population density as exposures and compiled relative risks and population-attributable risks (PARs) for self-inflicted and interpersonal violence–related injury.

Results. For both genders and for both injury types, receipt of welfare benefits showed the largest crude and net relative risks and the highest PARs. The socioeconomic status–related PAR for self-inflicted injury and the PAR related to number of parents in the household for interpersonal violence–related injury also were high.

Conclusions. Intentional-injury prevention and victim treatment need to be tailored to household social circumstances. (*Am J Public Health.* 2004;94:640–645)

The contribution of intentional injury to the overall burden of trauma, mortality, and morbidity increases substantially during adolescence. In Sweden, for example, the incidence of self-inflicted injury among girls aged 15–19 years is close to that of traffic-related injury.[1] Additionally, strong inverse relationships have been found between household (parental) socioeconomic status (SES) and injury risk among adolescents[2–10]—in particular, interpersonal violence–related injury[2,3,10] and self-inflicted injury.[4,6]

Apart from household SES, few household social and economic characteristics have been documented in relation to intentional injuries during adolescence. However, it seems that living in a single-parent home[11–13] and being in a family that receives welfare benefits[14] both have an impact on risk level. By contrast, the effect of parental country of birth is unclear.[3] Furthermore, population density has been found to be associated with injury caused by interpersonal violence among young people.[15]

The fact that studies have not commonly considered several family-related social characteristics simultaneously limits our understanding of the true effect of any particular family social circumstance on injury risk during adolescence.[2] A recent Swedish national study revealed that among adolescents, the combination of living in a single-parent home, receiving welfare benefits, and not having a parent born in Sweden reduces the association of household SES with risk of intentional injury but not with risk of traffic-related injury.[1] Yet, the manner in which these characteristics operate separately and in association with one another remains unclear.

Our study investigated this question more closely. We considered family-related social attributes in conjunction with population density, and we measured the individual and the combined effects of these factors on risk of self-inflicted and interpersonal violence–related injury among Swedish adolescents.

METHODS

Creation of the Data Set

A cross-sectional design was employed and the study was based on a data set we created by linking records from 14 Swedish national registers (the population register, 2 censuses, 5 annual income registers, 5 annual hospital discharge registers, and the causes-of-death register). The study population consisted of all adolescents aged 10 to 19 years who lived in Sweden at some point between 1990 and 1994. Subjects were identified through the Swedish National Population Register, and gender and age were established through the national census of 1990.

Adolescents were linked to parents using the national censuses of 1985 and 1990 to document parental social and economic attributes and population density. Subjects were matched with the adult or adults they lived with (including biological and nonbiological parents); those subjects who could not be linked to a parent and whose parents did not reside in Sweden at the time of the 1990 census were excluded (about 5.2%). All household information was taken from the 1990 census, with the exception of information on receipt of welfare benefits, which was extracted from the annual income registers.

Adolescent SES was determined on the basis of the highest parental SES in the household in accordance with the dominance principle.[16] The Swedish socioeconomic classification provides a measure of class on the basis of occupation.[17,18] It divides individuals in the labor force into self-employed and employed. The former group is further divided into farmers and other self-employed persons; the latter group is divided into manual workers and nonmanual workers, who in turn are subgrouped according to the average educational level required for any particular occupation. In our study, all adolescents were allocated to 1 of 6 categories of household SES: high/intermediate-level nonmanual workers, assistant nonmanual workers, skilled manual workers, unskilled manual workers, self-employed persons (farmers and other self-employed), and other (students, housewives, persons living on early-retirement pensions, and the long-term unemployed).

With regard to number of parents in the household (1- or 2-parent home), we assigned the single-parent home characteristic to adolescents who were living with a parent who was not cohabiting with another adult. During the study period, approximately 15% to 20% of all children in Sweden lived with a single parent, and approximately 30% to 50% of these children were born to single-parent families.[19]

A household was regarded as having received welfare benefits if anyone in the household received benefits at least once during the study period.

We also included parental country of birth to assess whether subjects had at least 1 Swedish-born parent. An adolescent was considered to have a parent born in Sweden if 1 or both parents were born in the country.

Population density was calculated as the number of inhabitants (in 1990) within a 30-kilometer radius of the most heavily populated district within a particular municipality. For our study, 2 categories were created: high–population density areas (i.e., the 3 main Swedish urban areas of Stockholm/Södertälje, Gothenburg, and Malmö/Lund/Trelleborg), and low–population density areas (i.e., the rest of Sweden).

This material was then linked to the annual National Hospital Discharge registers for the years 1990 to 1994 and to the national causes-of-death registry for the years 1991 to 1994. Non-fatal (but requiring 1 or more nights of hospitalization) and fatal (2.6%) intentional injuries were examined together. We avoided double counting of subjects in both types of registers by excluding from the hospital discharge registers any subject who had the same diagnosis in both register data sets within 2 months. Coverage of the hospital discharge registers was estimated to be nearly complete; however, about 4.5% of subjects either lacked information on the external cause of injury or had no personal identification number.[20]

In accordance with the *International Classification of Diseases, 9th Revision* (*ICD-9*),[21,22] injuries were divided into 2 categories: interpersonal violence–related injury (E960–E969) and self-inflicted injury (E950–E959). Person-years were compiled as follows: subjects who lived in Sweden a whole year contributed 1 person-year; those who moved from Sweden, or who were born or died, contributed 1 half year for that year. Person-years (denominator in the relative-risk calculations) and injuries (numerator) were summed for the 5-year study period (1990–1994). Table 1 shows the distribution of person-years across categories of social characteristics. It also shows injury incidences per 100 000 person-years across social characteristics by diagnostic group and by gender. As expected, boys experienced more interpersonal-violence injuries and girls experienced more self-inflicted injuries.[1,23] For boys and girls together, self-inflicted injuries outnumbered injuries caused by interpersonal violence. Additionally, a large proportion of the interpersonal violence–related injuries were likely to have been perpetrated by strangers or acquaintances (i.e., nonfamily members.)[10]

TABLE 1—Injury Incidence per 100 000 Person-Years, by Selected Household Characteristics: Sweden, 1990–1994[a]

Characteristic	Person-Years		Injury Incidence per 100 000 Person-Years			
			Self-Inflicted		Interpersonal Violence–Related	
	Girls	Boys	Girls	Boys	Girls	Boys
Household socioeconomic status						
High/intermediate-level nonmanual workers	843 705	885 876	111	26	9	57
Assistant nonmanual workers	275 404	285 019	129	30	13	74
Skilled manual workers	282 805	298 899	145	30	17	92
Unskilled manual workers	292 772	305 868	210	44	27	99
Self-employed persons	154 774	164 411	133	34	14	69
Other	13 853	146 576	288	74	41	150
No. parents in household						
2-parent home	1 553 839	1 739 833	121	28	13	68
1-parent home	33 415	346 816	277	62	31	128
Receipt of welfare benefits						
No	1 745 102	1 830 954	110	26	11	64
Once or more	242 887	255 694	414	91	53	176
Parental country of birth						
1 or both parents born in Sweden	1 809 384	1 894 430	137	32	15	73
No parent born in Sweden	178 606	192 218	251	48	27	128
Population density						
Low	1 544 749	162 097	140	33	15	67
High	443 241	465 678	170	38	19	116
Total	1 987 990	2 086 648	147	34	16	78

[a]Fatal injuries were measured for 1991 to 1994 only; the proportion of fatal injuries (2.6%) and the injury incidence are therefore somewhat underestimated.

Data Analysis

We performed all analyses separately for boys and for girls, and we controlled for age category (10–14 years and 15–19 years) in all instances. Relative risks (RRs) with 95% confidence intervals (CIs) were computed for each social characteristic independently. High/intermediate-level nonmanual workers, 2-parent home, not having received welfare benefits, having at least 1 parent born in Sweden, and living in a low–population density area were used as reference categories.

We then performed multivariate regression analyses, with all social characteristics in a single model, to establish the importance of each measure when we controlled for the others. Population density was included only when RRs had been found to be significant in the former set of analyses. Logistic regression was used to compute the RRs.

Finally, population-attributable risks were calculated to assess the reduction (percentage) in injury risk that would be achieved assuming all groups on 1 variable had the same risk level as the group with the lowest risk level.[24,25] For our study, population-attributable risks were calculated with the RRs from the multivariate regressions.

TABLE 2—Relative Risk (RR) for Intentional Injury, by Selected Household Characteristics, Adjusted for Age: Sweden, 1990–1994

Characteristic	Self-Inflicted Injury, RR (95% CI)		Interpersonal Violence–Related Injury, RR (95% CI)	
	Girls	Boys	Girls	Boys
Household socioeconomic status				
High/intermediate-level nonmanual workers	1.00	1.00	1.00	1.00
Assistant nonmanual workers	1.16 (1.02, 1.31)	1.13 (0.88, 1.45)	1.39 (0.94, 2.06)	1.30 (1.11, 1.53)
Skilled manual workers	1.32 (1.18, 1.48)	1.17 (0.92, 1.49)	1.79 (1.24, 2.56)	1.63 (1.41, 1.89)
Unskilled manual workers	1.90 (1.72, 2.10)	1.69 (1.37, 2.09)	2.88 (2.11, 3.94)	1.75 (1.52, 2.02)
Self-employed persons	1.19 (1.02, 1.38)	1.30 (0.97, 1.73)	1.51 (0.94, 2.42)	1.21 (0.99, 1.48)
Other	2.69 (2.39, 3.02)	2.91 (2.32, 3.66)	4.48 (3.18, 6.29)	2.73 (2.33, 3.20)
No. of injuries	2917	702	320	1622
No. parents in household				
2-parent home	1.00	1.00	1.00	1.00
1-parent home	2.31 (2.13, 2.50)	2.29 (1.95, 2.69)	2.42 (1.91, 3.06)	1.94 (1.74, 2.16)
No. of injuries	2805	664	310	1538
Receipt of welfare benefits				
No	1.00	1.00	1.00	1.00
Once or more	3.98 (3.68, 4.30)	3.80 (3.25, 4.45)	5.03 (4.02, 6.29)	2.95 (2.65, 3.29)
No. of injuries	2917	702	320	1622
Parental country of birth				
1 or both parents born in Sweden	1.00	1.00	1.00	1.00
No parent born in Sweden	1.92 (1.74, 2.12)	1.56 (1.25, 1.94)	1.83 (1.35, 2.49)	1.85 (1.62, 2.12)
No. of injuries	2917	702	320	1622
Population density				
Low	1.00	1.00	1.00	1.0
High	1.22 (1.12, 1.32)	1.16 (0.97, 1.37)	1.21 (0.94, 1.55)	1.76 (1.58, 1.95)
No. of injuries	2917	702	320	1622

Note. CI = confidence interval.

RESULTS

Main Effects

Table 2 shows important differences in RRs with regard to all characteristics. Results for self-inflicted injury and for injury caused by interpersonal violence were fairly comparable. For both diagnosis groups, and for both boys and girls, the differences were greatest for receipt of welfare benefits, with the highest RR for interpersonal violence among girls whose families received welfare benefits (RR=5.03; 95% CI=4.02, 6.29).

Particularly high RRs were found for both male and female adolescents whose families were classified as "other" for household SES (students, housewives, persons living on early-retirement pensions, and the long-term unemployed) compared with adolescents whose parents were high/intermediate-level nonmanual workers, but RRs also were high among adolescents from unskilled-manual-worker families and, to a lesser degree, from skilled-manual-worker families.

Compared with living in a low–population density area, living in a high–population density area entailed an excess risk (although a lower risk than for other attributes) of interpersonal violence–related injury for teenaged boys and self-inflicted injury for teenaged girls.

Combined Effects

The RRs derived from the multivariate analyses are shown in Table 3. The expected reductions in RR were comparable in size for the 2 diagnostic groups, with the important exception of household SES. In spite of these reductions, RR for both types of injury remained higher for adolescents whose families received welfare benefits than for adolescents whose families did not. Interestingly, the RR for girls was higher than that for boys in the case of interpersonal violence–related injury (3.71 vs 2.24), although the CIs did overlap. Furthermore, the net effect of living in a single-parent home, as opposed to living with 2 adults, remained significantly higher for both boys and girls, with an excess risk of about 60% for self-inflicted injury and about 40% for interpersonal violence.

Decreases in RRs were most considerable for household SES and parental country of birth. With regard to household SES, only girls from unskilled-manual-worker families remained at noticeably higher risk for self-inflicted injury than did the comparison group, whereas girls and boys both from families classified as "other" and from unskilled-manual-worker families and skilled-manual-worker families remained at higher risk of injury caused by interpersonal violence. Having no parent born in Sweden was no longer a risk factor for intentional injury for boys, although it remained a risk factor for girls in the case of self-inflicted injury (albeit substantially lower than before controlling for the other social and economic characteristics).

Living in a high–population density area, as opposed to a low–population density area, still entailed excess risk of interpersonal violence–related injury for boys and excess risk of self-inflicted injury for girls.

Population-Attributable Risks

Population-attributable risks (expressed as percentages) are shown in Table 4. The risks varied from 0 to 29.8 for self-inflicted injury and from 0 to 32.1 for interpersonal violence–related injury. Population-attributable risks were quite similar across diagnoses for all characteristics except household SES.

Receipt of welfare benefits was the characteristic with the highest population-attributable risk for the 2 types of injury. It was closely followed by household SES for interpersonal violence and by number of parents in the household for self-inflicted injury. Interestingly,

TABLE 3—Relative Risk (RR) for Intentional Injury, by Selected Household Characteristics, Adjusted for All Other Household Characteristics and Age: Sweden, 1990–1994

Characteristic	Self-Inflicted Injury, RR (95% CI)		Interpersonal Violence–Related Injury, RR (95% CI)	
	Girls	Boys	Girls	Boys
Household socioeconomic status				
High/intermediate-level nonmanual workers	1.00	1.00	1.00	1.00
Assistant nonmanual workers	0.99 (0.87, 1.12)	0.99 (0.77, 1.27)	1.21 (0.81, 1.80)	1.19 (1.01, 1.40)
Skilled manual workers	1.08 (0.96, 1.22)	0.98 (0.76, 1.25)	1.46 (1.01, 2.10)	1.53 (1.31, 1.78)
Unskilled manual workers	1.19 (1.06, 1.33)	1.11 (0.88, 1.40)	1.81 (1.29, 2.54)	1.36 (1.16, 1.58)
Self-employed persons	1.14 (0.98, 1.32)	1.24 (0.92, 1.66)	1.41 (0.88, 2.26)	1.22 (0.99, 1.50)
Other	1.16 (1.00, 1.35)	1.26 (0.93, 1.70)	2.29 (1.51, 3.47)	1.38 (1.12, 1.71)
No. of parents in household				
2-parent home	1.00	1.00	1.00	1.00
1-parent home	1.56 (1.43, 1.71)	1.64 (1.37, 1.96)	1.36 (1.04, 1.76)	1.38 (1.22, 1.56)
Receipt of welfare benefits				
No	1.00	1.00	1.00	1.00
Once or more	3.20 (2.92, 3.50)	2.99 (2.47, 3.62)	3.71 (2.84, 4.84)	2.24 (1.96, 2.55)
Parental country of birth				
1 or both parents born in Sweden	1.00	1.00	1.00	1.00
No parent born in Sweden	1.17 (1.04, 1.31)	0.92 (0.72, 1.18)	0.91 (0.66, 1.28)	1.11 (0.95, 1.29)
Population density				
Low	1.00	. . .	. . .	1.00
High	1.14 (1.04, 1.24)	. . .	. . .	1.72 (1.54, 1.92)
No. of injuries	2805	664	310	1538

Note. CI = confidence interval.

TABLE 4—Population-Attributable Risk for Intentional Injury, by Selected Household Characteristics: Sweden, 1990–1994[a]

Characteristic	Self-Inflicted, Injury, %		Interpersonal Violence–Related Injury, %	
	Girls	Boys	Girls	Boys
Receipt of welfare benefits	29.8	26.6	32.1	22.6
Household socioeconomic status	8.5	8.1	31.1	21.1
No. of parents in household	13.1	14.4	9.5	9.7
Parental country of birth	3.5	. . .	. . .	2.4
No. of injuries	2805	664	310	1538

[a]The population-attributable risk for each household characteristic was based on relative risks obtained after we controlled for all other household characteristics and age.

for both diagnostic groups, the population-attributable risk related to parental country of birth was extremely low.

DISCUSSION

Main Findings

We found considerable differences in risk for intentional injury among Swedish adolescents for each household characteristic we investigated, which is in line with the findings of earlier studies.[2–4,6,10–15] For both boys and girls, receipt of welfare benefits showed the largest RR differences and was followed by household SES, number of parents in the household, and parental country of birth. In general, girls had somewhat higher RRs than did boys, but CIs overlapped.

As expected, simultaneous consideration of all characteristics led to RR reductions for all characteristics and for both diagnostic groups. The most remarkable reductions occured for parental country of birth (RRs became negligible for both types of intentional injuries) and for household SES (mainly for self-inflicted injury). After we controlled for other characteristics, only adolescent girls from unskilled-manual-worker families showed a higher risk of self-inflicted injuries as compared with the reference group. Nevertheless, adolescent boys and girls from both unskilled-manual-worker and skilled-manual-worker families, and also those from families classified as "other," still showed an excess risk of injury caused by interpersonal violence.

As might be expected, population-attributable risks were highest for receipt of welfare benefits—for both boys and girls and for both types of intentional injuries. The risks of self-inflicted injury and interpersonal violence–related injury could be reduced by 23% to 30% (depending on gender and diagnosis) if adolescents from families who received welfare benefits lived with circumstances similar to those of families who did not. Alternatively, the risk of self-inflicted injury could be reduced by 13% to 14% if the living circumstances of adolescents from single-parent homes mirrored those of 2-parent homes. Likewise, injuries related to interpersonal violence could be reduced by at least 21% if adolescents from all household SES categories lived with circumstances similar to those of children whose parents were high/intermediate-level nonmanual workers.

The finding of a large net effect of receipt of welfare benefits after we controlled for all other attributes may be surprising, because the Swedish welfare system is designed in such a way that welfare allocations are sufficiently high to prevent individuals and families from living in poverty. As a consequence, receipt of welfare benefits is generally not strongly related to individual financial poverty in Sweden.[26] Furthermore, compared with children in many other European countries, few Swedish children are considered to be poor in absolute or relative terms.[27,28] Nevertheless, the fact that wage earners can count on a reallocation of wealth to compensate for economic shortfall does not eliminate the dis-

comfort and the uncertainty they experience when faced with any such shortfall. Additionally, there are good reasons to believe that receipt of welfare benefits, as well as indicating financial strain on a family, also may signal the presence of a variety of related dysfunctional conditions in the household, such as alcohol abuse, depression, and aggression.

It is important to note that during the study period, Sweden was facing an economic recession. This recession meant that more people were dependent on welfare benefits and, among these people, more beneficiaries were newly exposed to such a situation because of unemployment.[29] Accordingly, it was difficult to assess whether the effects we observed were circumstantial or were intrinsic to being in a family in need of state subsidy.

Furthermore, in light of our results, it may be hypothesized that receipt of welfare benefits mediates household SES and intentional injury and does so to a greater extent for self-inflicted injuries than for interpersonal violence–related injuries. Additionally, the fact that RRs and the population-attributable risk for household SES remained high in the case of injuries caused by interpersonal violence indicates that household SES had a true impact on injury risk that cannot fully be explained by the confounding effects of welfare benefits or other family characteristics. A large proportion of the injuries caused by interpersonal violence during adolescence were not sustained in the household.[10]

Welfare benefits also may have mediated some of the effect of number of parents in the household on the risk of self-inflicted injury. However, single parents in Sweden need less financial support than in many other countries because of Sweden's labor market policies and subsidized public child care.[30] The presence of this "safety net" may explain, in part, why the net RRs of living with 1 parent remained significant for both boys and girls and for both diagnoses. This observation, in turn, suggests that single parenthood increased the risk for intentional injury among adolescents for reasons that cannot be reduced to the economic burden borne by parents. Still, it should be emphasized that population-attributable risks were quite low for that family characteristic.

One aspect highlighted by our results was that not having a Swedish-born parent had a low population-attributable risk when we controlled for other family social and economic characteristics. It is reasonable to suppose that factors such as receipt of welfare benefits, household SES, and population density were mediators of the originally observed excess risk of intentional injury. There is, in fact, evidence that Swedish immigrants are educationally overqualified for their work to a greater extent than are Swedish-born workers[31]; a high proportion of immigrants settle in the country's 3 largest city areas. However, the low population-attributable risk associated with not having a Swedish-born parent may be a reflection of a weak association between parental country of birth and the risk for intentional injury.

Our study is silent regarding the mechanisms that underlie the social patterning of intentional injury during adolescence. In particular, other intrafamilial risk factors that were not considered, or even the experience of earlier episodes of maltreatment within the family,[32–34] may have had an aggravating effect on the risk of self-inflicted and interpersonal violence–related injuries in social groups already at high risk.[10,32] Likewise, contextual factors related to adolescents' living circumstances outside the home (e.g. the school, peer groups, youth culture) also may have modified—either protected against or aggravated—the effect of family social characteristics.[35–38]

Study Strengths and Limitations

Our data have very good population coverage, and gaps in data caused by lack of information about social characteristics (5.2%) or injuries (4.5%) were few.

The first limitation of our study lies in the manner in which the household status of some adolescents was determined, which may have resulted in misclassification of SES and other social characteristics. Only 1 household was identifiable for adolescents who spent equal time living in the separate homes of each parent, because children in Sweden are registered at a single address. In the worst case, some adolescents were allocated to a different SES category than they should have been. Nevertheless, the number of cases is so small that it cannot significantly alter our results.[19]

Another concern lies in the underreporting inherent in register-based studies of intentional injury.[10] More importantly, because it is not possible to establish whether the degree of underreporting is comparable across categories of the social characteristics we considered, some uncertainty remains about the RRs we compiled.[1,23] For instance, if a lower propensity exists among adolescents from families with lower SES to seek hospital care when intentionally injured, RRs will be underestimated. And, in contrast, if the propensity is greater, RRs will be overestimated. The same reasoning applies to the other family characteristics. Unfortunately, we had no opportunity to assess the direction of underreporting bias in the various family characteristics, nor do we know whether the propensity on the part of hospital staff to keep injured adolescents in the hospital varies according to the adolescent's social group or whether there are diagnostic inaccuracies at the hospital (either the victim "does not tell" or the hospital "does not see") that are unevenly distributed across social groups.[39,40] However, it can be stated that there is no evidence of such discrimination by hospital staff in Sweden.[41]

Also, it should be stressed that intentional injuries, particularly self-inflicted ones, make up diagnosis-related groups in which individual victims can appear several times in hospital discharge registers. In our study, the number of injury occasions was used regardless of the number of so-called "repeaters." It was beyond the scope of our study to investigate whether repeaters were more prevalent in some social groups than in others. Nor did we investigate whether the likelihood of dying following an injury varied with social status. These questions are worth investigating in future studies.

CONCLUSIONS

Our study highlights the importance of a variety of social and economic characteristics of an adolescent's family when studying the association between parental SES and risk for self-harm or for violence perpetrated by others. Undeniably, the mechanisms that underlie the relationship between household SES and risk for intentional injury are complex. The relationship is likely to be mediated by the receipt of welfare benefits in the case of self-inflicted injuries.

For long-lasting effects to be achieved in the prevention of intentional injury—and for the treatment of victims to be successful—there may be a need to supplement population-based interventions with other interventions tailored to social circumstances particular to some households. ■

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Contributors

K. Engström conceived the study, built the data set, performed the analyses, and participated in the writing of the article. F. Diderichsen assisted in refining core ideas and helped with the analyses. L. Laflamme helped refine core ideas and interpret the results and participated in the writing of the article.

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Human Participant Protection

Ethical approval for this study was obtained from the Karolinska Institute's research ethics committee.

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A Matched Case–Control Study Evaluating the Effectiveness of Speed Humps in Reducing Child Pedestrian Injuries

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Objectives. We evaluated the protective effectiveness of speed humps in reducing child pedestrian injuries in residential neighborhoods.

Methods. We conducted a matched case–control study over a 5-year period among children seen in a pediatric emergency department after being struck by an automobile.

Results. A multivariate conditional logistic regression analysis showed that speed humps were associated with lower odds of children being injured within their neighborhood (adjusted odds ratio [OR]=0.47) and being struck in front of their home (adjusted OR=0.40). Ethnicity (but not socioeconomic status) was independently associated with child pedestrian injuries and was adjusted for in the regression model.

Conclusions. Our findings suggest that speed humps make children's living environments safer. (*Am J Public Health.* 2004;94:646–650)

Pedestrian injuries caused by automobile collisions are a leading cause of death among children aged 5 to 14 years.[1] The demographic characteristics of children injured by automobiles have remained the same over the past 20 years, with boys, children between the ages of 5 and 9 years, and children living in neighborhoods of low socioeconomic status (SES) at highest risk.[2–4]

Children en route to school or at play in front of their homes are exposed to roads and street traffic. Modifying traffic patterns is a passive and sustainable public health intervention that may make children's living environments safer.[5] Traffic patterns can be modified with a number of engineering strategies that fall under the rubric of "traffic calming." Distinct from speed limit signs or stop signs, traffic calming measures such as speed humps, street closures, median barriers, and traffic circles are successful in providing long-term safety for pedestrians and motorists because they are physical structures with designs that are self-enforcing rather than requiring police enforcement.[6–8]

For years, European countries such as Denmark, the Netherlands, and Great Britain, as well as Australia and New Zealand, have implemented and tested the effects of traffic calming.[6] A report published in British Columbia summarized 43 international studies that demonstrated reductions in collision frequency rates ranging from 8% to 100% after implementation of traffic calming measures.[6] A Danish study showed that, in comparison with control streets, 72% fewer injuries occurred on experimental streets incorporating a variety of traffic calming measures in addition to new speed zoning requirements.[9]

As a result of the successful efforts in other countries, there is developing interest in traffic calming in the United States, and the Federal Highway Administration, in cooperation with the Institute of Transportation Engineers, has initiated a national traffic calming technical assistance project.[6] However, the majority of safety studies focusing on traffic calming measures have assessed accident statistics before and after installation, and there is no available hospital-based information on the specific effects of these interventions on childhood pedestrian injury.

Oakland has historically been one of the most dangerous cities in California in which to be a pedestrian, exhibiting, for example, the highest rate of pedestrian fatalities among the state's cities in 1995.[10] In that year, after a series of child pedestrian deaths, the Oakland Pedestrian Safety Project was formed. This multidisciplinary alliance addressed child and senior pedestrian injuries occurring in the city of Oakland and advocated for installation of speed humps. Over the 5-year period 1995 to 2000, Oakland installed about 1600 speed humps on residential streets. In this study, we examined the effect of residing on a street with speed humps on the odds of child pedestrian injuries in Oakland.

METHODS

We conducted a matched case–control study among Oakland residents younger than 15 years over the 5-year period March 1, 1995, to March 1, 2000. Case patients were children who were seen in the emergency department at Children's Hospital Oakland after having been struck and injured by an automobile on a residential street. Since this hospital receives all pediatric ambulance trauma transports (including deaths on the scene) from the city of Oakland, it was considered an appropriate choice to target child pedestrians injured in Oakland. Case patients were each compared with 2 respective controls matched in regard to age and gender. The purpose of the study was to determine whether these children who had been struck by automobiles were any less likely to live near a speed hump than their peers who lived in the same city boundaries but visited the emergency room that day for a reason other than being hit by a car.

We identified case patients retrospectively from a trauma database using *International Classification of Diseases* (9th Revision)[11] E-code E814.7 (motor vehicle traffic accident involving collision with a pedestrian). Cases were limited to those involving children younger than 15 years who were residents of the city of Oakland and who were injured or died as a result of the collision. We reviewed charts and emergency medical service data sheets to eliminate parking lot injuries, injuries involving bicyclists who had been misclassified as pedestrians, and injuries suffered by children in driveway rollover collisions. In addition, we reviewed traffic report data from the Oakland Police Department, primarily to

confirm locations of collisions. When necessary, we reviewed original traffic reports for further clarification.

We also restricted our analysis to children injured or killed within 0.25 mi (0.4 km) of home and used a street atlas[12] to determine whether the injury occurred on the street block of the child's residence (defined by Mueller et al.[2] as the "index street"), within a 0.25-mi radius (about 5 blocks, considered the "surrounding neighborhood"[2]), or at a more distant location within Oakland. The type of street on which a child lived was classified with the street atlas as well.[12] Only children residing on minor roads (residential streets) were eligible for the study, because speed humps are installed only on such roads.

Living on a street with a speed hump, or within 1 block of a speed hump, was our principal predictor variable. We used data from the Department of Traffic Engineering in Oakland to determine the exact locations and dates of installation of speed humps (Department of Traffic Engineering, unpublished data, 1995–2000). Speed humps that were located on the other sides of primary or secondary roads (arteries) or were installed after the date of the injury were not considered.

As mentioned, we matched each case patient, according to age, gender, and date of emergency department visit, with 2 controls seen in the emergency department that same day for a reason other than being struck by a car. We identified all eligible controls of the same sex and with the same year of birth as the case patient from the daily log and randomly selected 2 such individuals. In situations in which there were fewer than 2 control patients born in the same year as the case patient, we made a random decision to search the 1 year above or below the age of the case patient, and then 2 years above or below and so on, until a suitable control was identified. Ninety-three percent of all controls were within 2 years of age of their respective case patients.

Controls were restricted to Oakland residents living on residential streets. We collected information on ethnicity and insurance status (classified as private, public, or self-pay) from medical records. In addition, we categorized the SES of patient and control households, using 1990 census data on median household income within the case patient or control's census tract, as low ($0–$15 736), medium ($15 737–$30 115), or high (more than $30 115).[13] Finally, we examined the records of case patients and controls to ascertain the presence of certain preexisting diagnoses, such as cerebral palsy, mental retardation, paraplegia, and developmental delay, that would have affected their walking ability and, thus, their potential to be exposed as pedestrians to automobile traffic.

Statistical analyses were performed with Stata software (Stata Corp, College Station, Tex). We used McNemar matched pairs analyses in examining the 200 case–control pairs (100 case patients each matched to 2 controls). When a factor is truly protective against disease, there are more case–control pairs in which the case lacks (and the control has) this protective factor than the converse. Separate univariate analyses focused on ethnicity, census tract household income, and insurance status to determine whether they were independent predictors of child pedestrian injuries. Once significant ($P<.05$) variables were determined, we constructed a multivariate conditional logistic regression model that included only these variables.

RESULTS

We identified 236 individuals who had been seen in the emergency department during the study period and had been assigned an E-code of E814.7. We eliminated 52 potential case patients because they (1) were not Oakland residents at the time of admission, (2) were injured outside Oakland, (3) were more than 14 years of age, (4) were bicyclists who had been misclassified as pedestrians, or (5) had been injured by an automobile backing up within a driveway or parking lot. We eliminated an additional 84 potential patients because they either lived on an artery street or had been injured outside of their neighborhood, yielding a final study sample of 100 case patients.

Case patients and controls were similar in terms of age, gender, insurance status, median household income, and proportion with an underlying premorbid neurodevelopmental disease (Table 1). Case patients were more likely to be Asian or of Hispanic ethnicity. The odds of Asian children having been involved as a pedestrian in an accident were 5.8 times as high as those for White children ($P=.018$), and the odds of Latino children having been involved were 4.3 times as high ($P=.038$). Admitting diagnoses of controls are available on request from the authors.

Unadjusted odds ratios (ORs) derived from a matched pairs analysis showed a protective effect of speed humps. In comparison with children living more than a block from a speed hump, those living within a block of a speed hump were significantly less likely to be injured as pedestrians within their neighborhood (14% vs 23%; OR=0.50; 95% confidence interval [CI]=0.27, 0.89) (Table 2). Among the 100 case patients, 49 were actually hit on the block in front of their home (index street). As a subset, these children were even less likely to have a nearby speed hump than their controls (12% vs 24%; OR=0.38; 95% CI=0.15, 0.90) (Table 2).

We performed multivariate logistic regression analyses using both predictor variables and included race and ethnicity in the model. After control for race and ethnicity, speed humps were associated with significantly lower odds of children being injured in their neighborhood (adjusted OR=0.47; 95% CI=0.24, 0.95) and being struck on the block immediately in front of their home (adjusted OR=0.40; 95% CI=0.15, 1.06) (Table 2).

DISCUSSION

In our observational study, we found that children who lived within a block of a speed hump had significantly lower odds of being struck and injured by an automobile in their neighborhood. Living within a block of a speed hump was associated with a roughly 2-fold reduction in the odds of injury within one's neighborhood (adjusted OR=0.47). This protective effect was even more pronounced among the subset of children who were injured on the block immediately in front of their house (index street). Children living within a block of a speed hump exhibited a 2.5-fold reduction in the odds of being injured on their street (adjusted OR=0.4). These results highlight the effectiveness of speed humps in reducing child pedestrian injuries.

TABLE 1—Demographic Characteristics of Case Patients and Controls

	Case Patients (n = 100)	Controls (n = 200)	Odds Ratio	P[a]
Male, No. (%)	68 (68)	136 (68)	. . .	. . .
Age, y, mean (SD)	6.8 (3.5)	6.6 (3.7)	. . .	.63
Ethnicity, %				
White	3 (3)	16 (8)	Reference	
Black	49 (49)	117 (58.5)	2.4	.187
Native American/other	11 (11)	21 (10.5)	3.2	.115
Hispanic	22 (22)	31 (15.5)	4.3	.038
Asian	15 (15)	15 (7.5)	5.8	.018
Insurance status				
Private insurance	17 (17)	43 (21.5)	Reference	
Public insurance	78 (78)	147 (73.5)	1.3	.366
Self-pay	5 (5)	10 (5)	1.3	.717
Household income, $ (census tract)				
High (> 30 115)	12 (12)	39 (19.5)	Reference	
Medium (15 737–30 115)	75 (75)	136 (68)	1.8	.105
Low (0–15 736)	13 (13)	25 (12.5)	1.7	.265
Premorbid diagnosis[b]				
Mild mental retardation	1 (1)	1 (0.5)	. . .	
Developmental delay	0 (0)	3 (1.5)	. . .	

Note. A univariate analysis of age, ethnicity, insurance status, household income, and presence of a premorbid diagnosis showed that only ethnicity was independently associated with child pedestrian injury.
[a]All *P* values were obtained from conditional logistic regression analyses, except for age, which was obtained with a 2-tailed test of means.
[b]Case patients and controls were screened for the presence of any of the following premorbid diagnoses: cerebral palsy, mental retardation, quadriplegia, paraplegia, and developmental delay.

Exposure to Traffic

Increased exposure to traffic (especially traffic at high volume and speed) is a known risk factor for child pedestrian injury. Stevenson and colleagues showed that an increase in volume of 100 vehicles per hour is associated with an incremental increase of about 2.0 in the odds of pedestrian injury.[14] Average speeds traveled on streets are also associated with risk of injury, and at least 2 studies have demonstrated that a higher proportion of vehicles exceeding the posted speed limit is associated with higher odds of child pedestrian injuries.[14,15] In addition to the type of street, the number of streets that children cross on their way to school seems to affect their risk.[16]

Need for Passive Environment Modification

Given the relationship between exposure to traffic and risk of child pedestrian injuries, we have essentially 2 prevention strategies at our disposal: we can protect children from fast-moving traffic by modification of either their behavior or their traffic environment. There have been multiple attempts to modify children's behavior, including school training programs,[17] "traffic clubs" designed to educate parents and children about safe behavior on streets,[18] simulation games,[19] and community-level interventions.[20] For the most part, however, these educational efforts have been unable to exert meaningful changes in the long-term behavior of children, largely owing to the developmental limitations of preschool-aged children.[20] As a result, a great deal of attention has shifted to environment modification and the promise it holds for affecting child pedestrian injury rates.

TABLE 2—Odds of Pedestrian Injury Within a Child's Neighborhood and Odds of Injury on a Child's Index Street of Residence When Child's Home Is Within 1 Block of a Speed Hump: Multivariate Model

	Case Patients (n = 100), No. (%)	Control Subjects (n = 200), No. (%)	OR (95% CI)[a]	Adjusted OR (95% CI)[b]
Neighborhood injury	14 (14)	46 (23)	0.50 (0.27, 0.89)	0.47 (0.24, 0.95)
Index street injury	6 (12)	24 (24)	0.38 (0.15, 0.90)	0.40 (0.15, 1.06)

Note. OR = odds ratio; CI = confidence interval.
[a]Calculated from McNemar matched pairs analysis.
[b]Calculated from multivariate model including ethnicity.

Focus on Neighborhood Injury

The deliberate focus of our study was on pedestrian injuries occurring in a child's own neighborhood (defined here as within a 0.25-mi radius of the child's home) as opposed to all injuries, including those occurring at more distant sites. We focused on such injuries because although children leave their neighborhoods with adults (and often in automobiles), most of their unsupervised time is likely to be near home. In addition, the traffic calming methods we examined can be applied only to residential streets. One 8-year study that examined fatal head injuries revealed that injuries to pedestrians were the most common cause of fatal head injuries and that 53% of those injured were playing in the street at the time of the injury. Of the 135 accidents that fell into this category, only 1 involved a child who had been under adult supervision at the time of the accident (the remaining children had been supervised by siblings or other children).

The same study showed that 80% of fatal pedestrian injuries had taken place within 1 mi (1.6 km) of the child's home.[21] Among the 184 children we initially identified for this study, 125 (68%) were eligible for the study because their injury occurred within 0.25 mi of home (the other children were eliminated because they lived on arterial streets). Therefore, our data suggests that roughly two thirds of injuries occur within the 0.25 mi surrounding a child's home. Passive interventions that

reduce child pedestrian injuries are likely to be of greater benefit in areas where children are prone to spend time without adults.

In our study, SES was not a significant independent predictor of child pedestrian injury. Mueller and colleagues found that living in a census tract with a median household income level below $20 000 was associated with 7.0-fold higher odds of injury than living in a census tract with a median income level above $30 000.[2] Other research points toward an association between increasing rates of pedestrian injury and lower SES, as approximated by census tract of residence,[4] spatial modeling of census tract and other data with a geographic information system,[22] and more indirect indicators of lower SES such as living near a convenience store, gas station, or fast food store.[15]

It is possible that, in our population, "overmatching" was the reason SES was not found to be an independent risk factor. Case patients were not matched with controls on SES, but if lower SES is associated with both increased odds of injury[2] and increased odds of an emergency department visit,[23] choosing controls from the emergency department may have resulted in overmatching in terms of SES.

Limitations

Our study involves potential methodological limitations. For example, limiting measurement to speed humps on a child's street ignores the potential protective effect of speed humps around the corner from a child's house. Thus, by measuring speed humps lateral to an index street (rather than in a 1-block radius), we may have underestimated the relevant rate of exposure to this intervention, which would have affected our estimation of the intervention's protective impact.

There are also limitations involved with our study sample. While relying on emergency department visits ensured that we incorporated higher severity injuries (including deaths), injuries that were not reported to the emergency medical services (and for which children may have been taken by their family to their regular doctor) would have been missed. This would mean that our sample underrepresented lower acuity injuries. It is also possible that our sample underrepresented younger children, in that children younger than 5 years are more likely to be hit in their driveway (often by a backing automobile)[24,25]; we excluded children in this age group from our study because such injuries are not related to the flow of street traffic.

Finally, it is possible that significant confounding factors were not addressed in this study. Some research suggests that the presence of sidewalks is not a significant contributor to odds of injury,[2,15] and other research suggests that the presence of sidewalks is a strong risk factor, with an odds ratio of 11.0.[14] We would have liked to control for the presence of sidewalks, but there were no reliable retrospective data on sidewalk or curb presence available to do so. Also, since much of the earlier literature points to lower SES as a risk factor for child pedestrian injury, the reason for our inability to reproduce this relationship may have been that the factors we used to approximate SES—census tract household income and medical insurance status—are inappropriate proxies for SES.

CONCLUSIONS

We found that speed humps were associated with a 53% to 60% reduction in the odds of injury or death among children struck by an automobile in their neighborhood. These findings invite additional research on the protective effects of traffic calming interventions and offer a framework for studying pedestrian injuries in relation to physical interventions implemented within a localized geographic region. Further confirmation of the protective effects of speed humps would be useful and could be augmented by additional information on stop signs or other factors that would affect slowing distances on either side of a speed hump. Our study provides direct observational evidence that speed humps are associated with a reduction in the odds of childhood pedestrian injuries and supports the installation of speed humps by traffic engineering departments. ■

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Contributors

J.M. Tester conceived the study, performed all analyses, and led the writing of the article. G.W. Rutherford assisted in data analyses, interpretation of findings, and revisions of the article. Z. Wald contributed to conceptualization of ideas as well as reviews of the article. M.W. Rutherford contributed to the study design and interpretation of the findings.

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Human Participant Protection

This study was reviewed and approved by the institutional review board of Children's Hospital and Research Center at Oakland. Informed consent was not required by the review board because patients did not need to be contacted for this retrospective data analysis.

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Housing First, Consumer Choice, and Harm Reduction for Homeless Individuals With a Dual Diagnosis

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Objectives. We examined the longitudinal effects of a Housing First program for homeless, mentally ill individuals' on those individuals' consumer choice, housing stability, substance use, treatment utilization, and psychiatric symptoms.

Methods. Two hundred twenty-five participants were randomly assigned to receive housing contingent on treatment and sobriety (control) or to receive immediate housing without treatment prerequisites (experimental). Interviews were conducted every 6 months for 24 months.

Results. The experimental group obtained housing earlier, remained stably housed, and reported higher perceived choice. Utilization of substance abuse treatment was significantly higher for the control group, but no differences were found in substance use or psychiatric symptoms.

Conclusions. Participants in the Housing First program were able to obtain and maintain independent housing without compromising psychiatric or substance abuse symptoms. (*Am J Public Health.* 2004;94:651–656)

Current rates of homelessness in New York City are the highest ever documented.[1] A small percentage of this population remains chronically homeless, either living on the streets or other public places or intermittently using emergency rooms, shelters, jails, and other short-term services, but never successfully ending their homelessness.[2] Members of this chronically homeless group typically have a history of mental illness,[3] compounded by substance use disorders.[4,5,6] Although much is known about the chronically homeless, these individuals continue to elude existing program efforts.

The predominant service delivery model designed to address the needs of this chronically homeless population, called the Continuum of Care, consists of several program components. It begins with outreach, includes treatment and transitional housing, and ends with permanent supportive housing. The purpose of outreach and transitional residential programs is to enhance clients' "housing readiness" by encouraging the sobriety and compliance with psychiatric treatment considered essential for successful transition to permanent housing. This approach assumes that individuals with severe psychiatric disabilities cannot maintain independent housing before their clinical status is stabilized. Furthermore, the model presumes that the skills a client needs for independent living can be learned in transitional congregate living. Research in psychiatric rehabilitation indicates, however, that the most effective place to teach a person the skills required for a particular environment is within that actual setting.[7]

Consumers' perception of the Continuum of Care offers another divergent perspective. Consumers experience the Continuum as a series of hurdles—specifically, ones that many of them are unable or unwilling to overcome. Consumers who are homeless regard housing as an immediate need, yet access to housing is not made available unless they first complete treatment. By leveraging housing on participation and treatment, continuum program requirements are incompatible with consumers' priorities and restrict the access of consumers who are unable or unwilling to comply with program terms.

In addition, most consumers prefer to live in a place of their own rather than in congregate specialized housing with treatment services onsite.[8,9] Most programs have rules that restrict clients' choices and that when violated are used as grounds for discharging the consumer from the program. For example, despite having attained permanent housing, clients who relapse and begin to drink mild or moderate amounts of alcohol, may be evicted if the program has strict rules about sobriety maintenance. The chronically homeless population is characterized by its frequent inability to gain access to existing housing programs. Individuals in this group often have multiple disabling conditions, especially psychiatric conditions and substance abuse.[10] Most programs are poorly equipped to treat people with dual diagnoses, let alone prepared to address their housing needs.[11] Treatment requires time and commitment and is often not available if a program is under pressure to move clients along a continuum.[12]

The loss of control over one's life resulting from housing instability, frequent psychiatric hospitalizations, and intermittent substance abuse treatment leaves some consumers mistrustful of the mental health system and unwilling to comply with demands set by providers.[13] Others prefer the relative independence of life on the streets to a fragmented treatment system that inadequately treats multiple diagnoses or addresses housing needs.[14,15] Paradoxically, consumers' reluctance to use traditional mental health and substance abuse services as a condition of housing only confirms providers' perceptions that these individuals are "resistant" to treatment, not willing to be helped, and certainly not ready for housing.[16]

The Housing First model was developed by Pathways to Housing to meet the housing and treatment needs of this chronically homeless population. The program is based on the belief that housing is a basic right and on a theoretical foundation that includes psychiatric rehabilitation and values consumer choice.[17] Pathways is designed to address the needs of consumers from the consumer's perspective.[18] Pathways encourages consumers to define their own needs and goals and, if the consumer so wishes, immediately provides an apartment of the consumers' own without any prerequisites for psychiatric treatment or sobriety. In addition to an apartment, consumers are offered treatment, support, and other services by the program's Assertive Community Treatment (ACT) team. ACT is a well defined community based inter-

disciplinary team of professionals that includes social workers, nurses, psychiatrists, and vocational and substance abuse counselors who are available to assist consumers 7 days a week 24 hours a day. The Pathways program has made two modifications to the standard ACT model: a nurse practitioner was included to address the considerable number of health problems, and a housing specialist was added to coordinate the housing services. Although housing and treatment are closely linked, they are considered separate domains, and consumers in the program may accept housing and refuse clinical services altogether without consequences for their housing status. There are 2 program requirements: tenants must pay 30% of their income (usually Supplemental Security Income [SSI]) toward the rent by participating in a money management program, and tenants must meet with a staff member a minimum of twice a month. These requirements are applied flexibly to suit consumers' needs.[21]

Consistent with the principles of consumer choice, Pathways uses a harm-reduction approach in its clinical services to address alcohol abuse, drug abuse, and psychiatric symptoms or crises. At its core, harm reduction is a pragmatic approach that aims to reduce the adverse consequences of drug abuse and psychiatric symptoms.[22] It recognizes that consumers can be at different stages of recovery and that effective interventions should be individually tailored to each consumer's stage.[23] Consumers are allowed to make choices—to use alcohol or not, to take medication or not—and regardless of their choices they are not treated adversely, their housing status is not threatened, and help continues to be available to them.

Continuum of Care supportive housing programs subscribe to the abstinence–sobriety model based on the belief that without strict adherence to treatment and sobriety, housing stability is not possible. But studies examining the model's effectiveness report only modest results in achieving housing stability for individuals who are chronically homeless and mentally ill.[24] Alternatively, the approach used by the Pathways program assumes that if individuals with psychiatric symptoms can survive on the streets then they can manage their own apartments. The program posits that providing a person with housing first creates a foundation on which the process of recovery can begin. Having a place of one's own may—in and of itself—serve as a motivator for consumers to refrain from drug and alcohol abuse.

The purpose of this study was to compare the effectiveness of the Housing First model with that of programs that used the Continuum of Care model for individuals who are chronically homeless and mentally ill.

We tested the following hypotheses: (1) the experimental (Housing First) group would report greater consumer choice over time than the control (Continuum of Care) group; (2) the experimental group would (a) exhibit lower rates of homelessness than the control group and (b) achieve and sustain greater residential stability than the control group; (3) the experimental group would exhibit rates of substance use similar to or lower than those of the control group; (4) the experimental group would participate in fewer substance-abuse treatments over time than the control group (i.e., because substance abuse treatment is not a precondition for the Housing First model, it is expected that there will be a lower rate of service utilization for the experimental group); and (5) the experimental group would exhibit rates of psychiatric symptoms similar to or lower than those of the control group.

METHODS

Participants

The 225 participants were randomized into 2 groups. One hundred twenty-six participants (56%) were assigned to the control group—and entered programs that followed the Continuum of Care model—and 99 (44%) were assigned to the experimental group and to a program that used the Housing First model. The control group was intentionally oversampled, anticipating that a higher number of control group participants may remain homeless and prove more difficult to locate for follow up interviews. The sample comprised 2 subgroups: an original street sample of 157 participants who met eligibility criteria, and a second group of 68 individuals recruited from 2 state psychiatric hospitals. To meet eligibility criteria, the first group had to have spent 15 of the past 30 days on the street or in other public places (shelters were not included), exhibited a history of homelessness over the past 6 months, and had an Axis I diagnosis[25] of severe mental illness. Diagnoses were based on previous records from service providers or, in cases in which records were unavailable, on an interview with an independent psychiatrist. Although a diagnosis or history of alcohol or substance abuse disorders was not an eligibility criterion, according to clinical records 90% of all the participants also had a diagnosis or history of alcohol or substance abuse disorders. The street sample was recruited through service agency staff referral of eligible clients who were interested in study participation. The second group met the same entry criteria for homelessness and mental illness immediately before hospitalization as did the street sample.

Because of administrative problems, 12 participants in the experimental condition were not assigned a Pathways apartment, and 7 control participants were erroneously assigned a Pathways apartment. Excluding these 19 participants reduced the number of control participants to 119 (58%) and the number of experimental participants to 87 (42%).

As can be seen in Table 1, the final sample consisted of 162 (79%) men and 44 (21%) women whose average age was 41.3 years. More than half of the participants (n=110, 53%) were diagnosed with a psychotic disorder. Seventeen percent (n=35) had become homeless before the age of 18 years. The longest period ever homeless, on average, was 4.5 years. Fifty-one percent (n=114) of the participants were literally homeless (staying in the streets or public spaces) at the time of the baseline interview. Another 36% entered the study from psychiatric institutions but had been homeless before hospitalization. After randomization, there were no significant differences between groups for baseline demographic characteristics such as gender, age, education, race, diagnosis, or amount of time homeless.

Procedures

After completing their baseline interviews, participants were interviewed every 6 months. Interviewers were blind to participants' assignment for baseline interviews but not for follow-up interviews. Data for the complete 24-month period were collected between December 1997 and January 2001. During each interim period, 5-minute telephone calls were conducted primarily to maintain contact with participants and establish their whereabouts. Par-

TABLE 1—Participant Characteristics at Baseline (n = 206)

	No. (%)
Study group	
Experimental	87 (42)
Control	119 (58)
Gender	
Female	44 (21)
Male	162 (79)
Age, y	
18–30	39 (19)
31–40	59 (29)
41–50	62 (30)
51–60	36 (17)
≥61	10 (5)
Education	
8th grade or less	21 (10)
Some high school	66 (32)
Finished high school	34 (17)
Completed general equivalency diploma	16 (8)
Vocational/trade/business school	5 (2)
Some college	49 (24)
College degree	10 (5)
Graduate degree	4 (2)
Race/ethnicity	
White (not Hispanic)	55 (27)
Black (not Hispanic)	84 (41)
Hispanic	30 (15)
Mixed/other/unknown	37 (18)
Diagnosis	
Psychotic	110 (53)
Mood–depressive	29 (14)
Mood–bipolar	29 (14)
Other	10 (5)
Unknown	28 (14)
Residence at baseline	
Streets/subways/parks/abandoned building/drop-in centers	114 (51)
Shelter/safe haven	13 (6)
Psychiatric hospital	80 (36)
Other	18 (8)

ticipants were paid for all interviews. Six-month interviews were conducted in a variety of locations, including the research office, the participant's apartment/residential location, or a public place such as a cafe or restaurant. When it was not possible for interviews to be conducted face-to-face (e.g., the participant had moved out of state), interviews were conducted by telephone. For participants in psychiatric hospitals and correctional facilities, research interviewers made onsite visits. The questions asked during each interview period remained the same. The follow-up rates by time period were as follows: 88% at 6 months, 87% at 12 months, 84% at 18 months, and 78% at 24 months. These follow-up rates do not include individuals who were missing at certain time points but who were located subsequently and for whom residential data was collected at a later point. Thus, the follow-up rates reported here are based on conservative calculations.

Measures

A modified version of Consumer Choice, a 16-item, 5-point scale developed by Srebnik, Livingston, Gordon, and King,[26] was used to determine (1) how important it was for the participant to have choice at baseline and (2) how much choice the participant actually had, at subsequent time points, in their location, neighbors and housemates, visitors, and so forth.

We measured residential status with a 6-month residential follow-back calendar developed by New Hampshire Dartmouth Research Center.[27] The interviewer assessed the participant's location for each day during the past 6 months on a day-by-day basis. From this information, we calculated the proportion of time spent homeless as well as the proportion of time spent in stable housing.

Following the interview, the interviewer coded the participant's residential location according to several distinct residential categories. For the purpose of analyses, homelessness was considered as living on the streets, in public places, or in shelter-type accommodations. Residential stability was defined as residing in one's own apartment; or having a room or studio apartment in a supportive housing program, a group home, a boarding home, or a long-term transitional housing program; or living long-term with parents, friends, or other family members. The number of days spent in any of the locations categorized as "homeless" or "stably housed" was summed and divided by the total number of days of residency reported at the interview.

We measured alcohol and drug use with the Drug and Alcohol Follow-Back Calendar.[28,29] Participants reported the number of drinks consumed each day, as well as the number of days that selected drugs were used during a 6-month period. We used an alcohol use variable (measuring the total number of drinks) and a drug use variable (measuring the total number of days of drug use) for each 6-month period in the analyses.

We measured substance abuse treatment service utilization with a modified shorter version of the Treatment Services Inventory.[30] In the interview, participants were asked whether they received any substance abuse treatment during the past 2 weeks. Drug and alcohol treatment services use was indicated by an average of 7 items including questions asking whether the participant had received treatment in a detoxification program or other program; consulted with a counselor to talk about substance problems; or attended Alcoholics Anonymous, Narcotics Anonymous, or any other self-help group.

Psychiatric symptoms were measured with the Colorado Symptom Index,[31] a 15-item questionnaire including items assessing psychotic symptoms as well as symptoms related to mood and suicidality.

Data Analysis

Repeated-measures analysis of variance (ANOVA) was used to examine group differences, during the 2-year follow-up period, for hypothesis 1 (consumer choice), hypothesis 2 (housing stability assessed as 2 separate outcomes: proportion of time stably housed and proportion of time homeless), and hypothesis 3 (substance abuse assessed as 2 separate outcomes: alcohol abuse and drug abuse). In cases in which repeated-measures ANOVAs yielded significant results, *t* tests were conducted to compare group differences at each time point. Group differences were then plotted and graphed for the 2 groups across time.

To appropriately examine differences in substance abuse treatment services use, hypothesis 4 was tested with a subsample of participants who were not on the streets but who were in some type of service-related program: namely, experimental participants who were currently housed by the Housing First program and control participants who were housed by one of the Continuum of Care programs. Control participants were included in this analysis if they reported that they lived most recently in one of the following places at the time of the interview: shelters, supportive housing programs,

drop-in centers, safe havens, detoxification facilities, crisis housing, intermediate care, boarding houses, transitional housing, group homes, alcohol/drug-free facilities, and treatment/recovery programs. Because participants' residential status changed from one time point to the next, the subsample also changed; we therefore had to conduct separate *t* tests for each time point. Because there were 5 time points, we used a Bonferroni adjusted α of .025 to account for Type I error.

Power Analysis

To retain 80% power to detect an effect that explains 4% of the variance in the context of an equation (with 5 covariates) that explains 25% of the variance, we needed to retain 68% of the original sample; moreover, power for repeated-measures analyses would be higher.[32] Our retention rates were substantially above this figure, so we did not anticipate any problems in the power to detect group differences.

RESULTS

Consumer Choice

Results from repeated-measures ANOVA showed that there was a significant time × group status effect, indicating that participants in the experimental condition perceived their choices to be more numerous than did participants in the control condition ($F_{4,11}2=8.91$, $P<.001$). Additionally, the experimental group's perceptions were more stable than were those of the control group. As can be seen from Figure 1, subsequent univariate analyses showed significant differences at 6, 12, 18, and 24 months, with the experimental group reporting significantly more choice than the control group.

Residential Stability

Repeated-measures ANOVA results showed a significant Time × Group status effect. Participants in the experimental condition had significantly faster decreases in homeless status and increases in stably-housed status relative to participants in the control condition ($F_{4,137}=10.1$, $P<.001$; $F_{4,137}=27.7$, $P<.001$). As can be seen from Figures 2 and 3, subsequent univariate analyses showed significant differences at 6, 12, 18, and 24 months, with the experimental group reporting less time spent homeless and more time spent stably housed compared with the control group.

Substance Use

Repeated-measures analyses showed no significant differences in either alcohol or drug use between the 2 groups by time condition ($F_{4,136}=1.1$, $P=.35$ for alcohol use; $F_{4,136}=.98$, $P=.42$ for drug use).

Substance Abuse Treatment Utilization

Five *t* tests were conducted with an adjusted α level of .025. As can be seen from Figure 4, these univariate analyses showed significant differences at 6, 18, and 24 months ($P<.025$) and at 12 months ($P<.05$), with the Continuum group reporting significantly higher use of substance abuse treatment programs than the Housing First group. In addition, a decrease in service use occurred among the Housing First group and an increase occurred among the Continuum group over time.

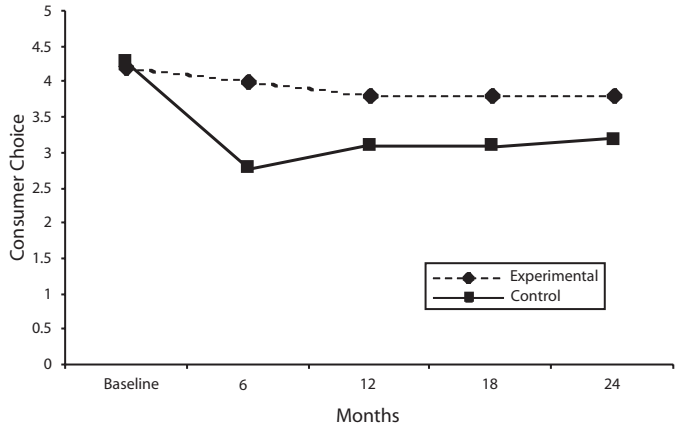
Psychiatric Symptoms

Repeated-measures analyses showed no significant differences psychiatric symptoms between the 2 groups by time condition ($F_{4,137}=.348$, $P=.85$).

DISCUSSION

Our results attest to the effectiveness of using the Housing First approach in engaging, housing, and keeping housed individuals who are chronically homeless and dually diagnosed. The Housing First program sustained an approximately 80% housing retention rate, a rate that presents a profound challenge to clinical assumptions held by many Continuum of Care supportive housing providers who regard the chronically homeless as "not housing ready." More important, the residential stability achieved by the experimental group challenges long-held (but previously untested) clinical assumptions regarding the correlation between mental illness and the ability to maintain an apartment of one's own. Given that all study participants had been diagnosed with a serious mental illness, the residential stability demonstrated by residents in the Housing First program—which has one of the highest independent housing rates for any formerly homeless population—indicates that a person's psychiatric diagnosis is not related to his or her ability to obtain or to maintain independent housing. Thus, there is no empirical support for the practice of requiring individuals to participate in psychiatric treatment or attain sobriety before being housed.

Participants' ratings of perceived choice—one of the fidelity dimensions of the Housing First program—show that tenants at Pathways experience significantly higher levels of control and autonomy in the program. This experience may contribute to their success in maintaining housing and to most consumers' choice to participate in treatment offered by the ACT team



Note. At baseline, participants were asked how much choice they would *like to* have. Subsequent time-points assess how much choice participants *actually* have.

FIGURE 1—Consumer choice in housing: baseline–24 months.

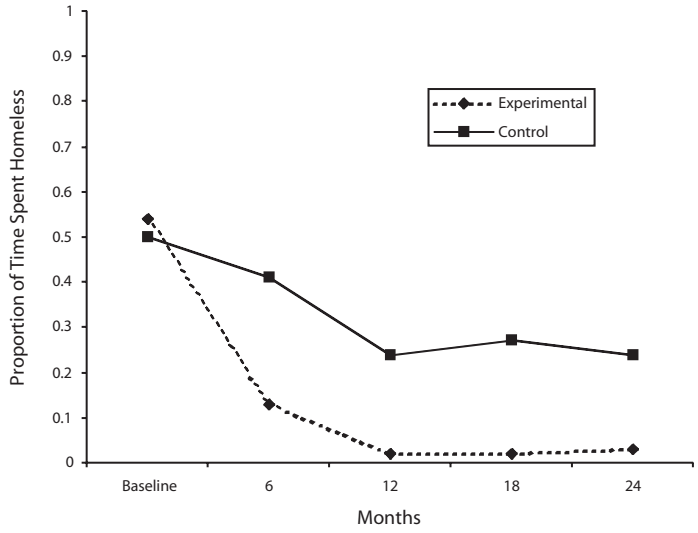


FIGURE 2—Proportion of time spent homeless: baseline–24 months.

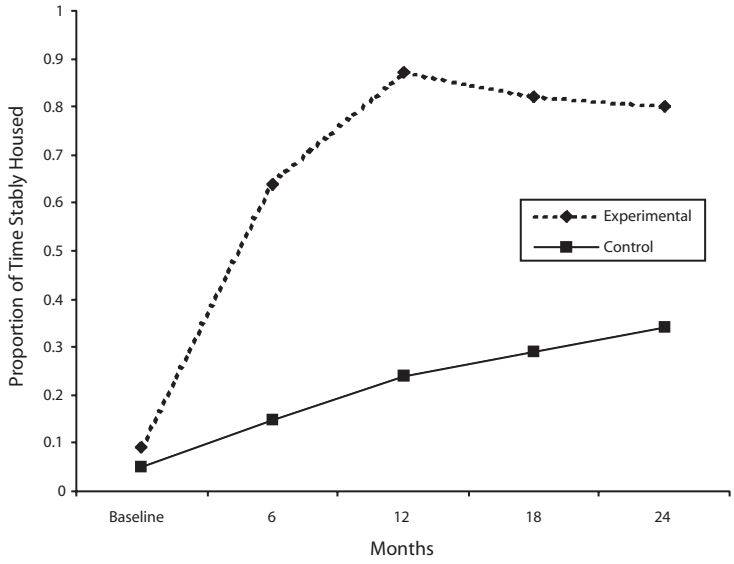


FIGURE 3—Proportion of time stably housed: baseline–24 months.

after they were housed. In addition, contrary to the fears of many providers and policymakers, housing consumers without requiring sobriety as a precondition did not increase the use of alcohol or drugs among the experimental group compared with the control group. Providing housing first may motivate consumers to address their addictions to keep their housing, so that providing housing before treatment, may better initiate and sustain the recovery process.

Our findings indicate that ACT programs that combine a consumer-driven philosophy with integrated dual diagnosis treatment based on a harm-reduction approach positively affect residential stability and do not increase substance use or psychiatric symptoms. In addition, because the ACT teams were providing services directly, substance abuse treatment services use was significantly lower for Housing First residents than for Continuum of Care residents. Because treatment for substance abuse is required, along with sobriety, by the Continuum of Care model, it is not surprising that individuals in the control group show greater use of treatment services. However, despite the control group's higher use of services, their levels of alcohol or drug use were not different from those of the experimental group. This disconnect between drug treatment services use and levels of drug use suggests that the control group may be using treatment facilities as short-term housing.

One limitation of the study is that self-reports of the use of alcohol and drugs and treatment services can be susceptible to reporting bias. Several studies have shown that among people who are homeless and dually diagnosed, there is a high rate of discrepancy between self-reports and client observation for substance use and for utilization of substance abuse treatment services.[33,34] Memory error, nondisclosure, social desirability concerns, and intentional misrepresentation can lead to reporting errors. Powerful systemic reasons for underreporting also exist. For example, participants enrolled in Continuum of Care residential programs, for which sobriety is mandatory, may be inclined to underreport the amount of drugs and alcohol consumed out of fear that such information may reach a caseworker or staff member and lead to the loss of their housing. Errors in self-reporting could be reduced if other measures (e.g., case manager's reports, laboratory drug tests) could be incorporated into a multiple-measure data report.

In conclusion, the outcomes achieved provide grounds for the rejection of the erroneous assumptions underlying the ubiquitous Continuum of Care model, the elimination of treatment requirements as a precondition for housing, and the support of initiatives adopting a Housing First approach to end homelessness and increase integration into the community for individuals with psychiatric disabilities living on our streets. ■

About the Authors

The authors are with Pathways to Housing, Inc, New York, NY.

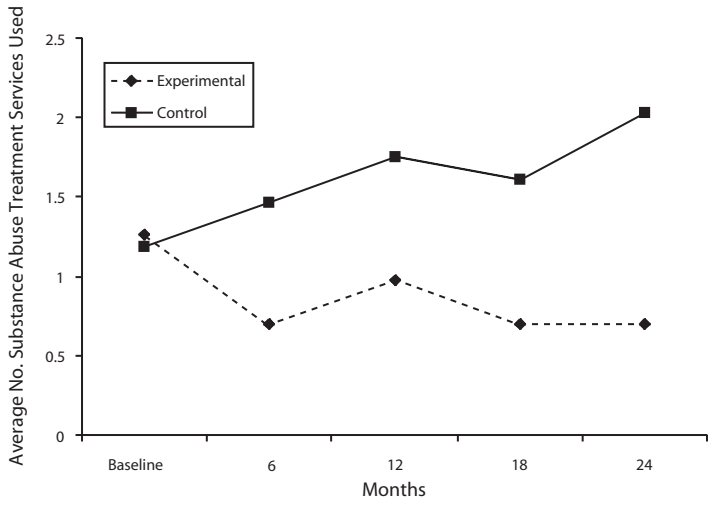


FIGURE 4—Average number of substance abuse treatment services used: baseline–24 months.

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Contributors

S. Tsemberis oversaw all aspects of the study and preparation of the article. L. Gulchur completed data collection and the statistical analysis. M. Nakae assisted with data analysis and literature review.

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Human Participant Protection

The protocol was approved by the institutional review boards of Pathways to Housing, Inc. and New York University. Informed consent was obtained from all participants.

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Housing Characteristics and Children's Respiratory Health in the Russian Federation

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Objectives. We studied housing characteristics, parental factors, and respiratory health conditions in Russian children.

Methods. We studied a population of 5951 children from 9 Russian cities, whose parents answered a questionnaire on their children's respiratory health, home environment, and housing characteristics. The health outcomes were asthma conditions, current wheeze, dry cough, bronchitis, and respiratory allergy.

Results. Respiratory allergy and dry cough increased in association with the home being adjacent to traffic. Consistent positive associations were observed between some health conditions and maternal smoking during pregnancy, many health conditions and lifetime exposure to environmental tobacco smoke (ETS), and nearly all health conditions and water damage and molds in the home.

Conclusions. Vicinity to traffic, dampness, mold, and ETS are important determinants of children's respiratory health in Russia. (*Am J Public Health.* 2004;94:657–662)

Numerous studies have associated indoor housing factors with increased prevalence of respiratory symptoms in children as well as adults.[1–4] Yet there are few studies from the Russian Federation or the former Soviet Union, where a large percentage of the population live in concrete apartment buildings, in which water and heat are supplied by district heating systems and gas is used for cooking. Furthermore, most Russian families benefit from a state health care system that provides pre- and postnatal care.[5] It therefore behooves us to examine housing factors such as smoking, moisture, indoor combustion sources (e.g., gas cooking, tobacco use), and ventilation on the health of school-aged children living in contemporary Russian housing.

METHODS

Study Population

The study population comprised 5951 8- to 12-year-old children in 9 Russian cities. Eight cities of the Sverdlovsk Oblast region and the city of Cherepovets in the Vologda Oblast participated in the study. Cities were selected to participate in a cross-sectional study of air pollution and children's health. In 4 cities, 2 areas were selected—1 to represent a more polluted area and 1 a less polluted area; in 5 cities, only 1 area was included.

Within each area, 1 or 2 elementary schools were selected for participation. The principals of the selected schools were informed about the study and agreed to participate. Teachers were given verbal and written instructions, questionnaires, envelopes, and forms to record questionnaire distribution and collection. Parents were invited to a parents' night where teachers explained the study and the conditions of consent. Teachers were instructed not to urge parents to fill out the questionnaire, as compliance was strictly voluntary. Parents who wished to participate completed the questionnaire either in the classroom or at home, and returned it (via the child) to the teacher in a sealed envelope. There was a 98% response rate.

The questionnaires, which were identified by identification number only, were reviewed by the field coordinators for quality assurance and for encoding written replies. The questionnaires were then sent to the Harvard School of Public Health for optical scanning using internal consistency checks to identify questionnaires requiring additional verification.

The questionnaire had been modified from previous European and North American questionnaires, which originated from respiratory health questionnaires of the British Medical Research Council and the American Thoracic Society.[6] The questionnaire was composed of the following: the child's personal characteristics; the child's respiratory health, presence of atopic diseases, and number of infections during the past year; parents' education and job category (as an indicator of socioeconomic status); parents' smoking habits as well as respiratory and allergic diseases; and details of the home environment and building characteristics. Details of health, housing characteristics, and socioeconomic factors were adjusted for the current Russian conditions.

Health Outcomes

Twenty health outcomes were derived from the questionnaire (Table 1). We focused on the children's current symptoms and conditions of allergy and eye irritation. Some of the health outcomes were composite variables derived from multiple questions. Believing that asthma may be underdiagnosed or not clearly remembered by the parent, we defined a composite variable called "asthma-like symptoms" that included wheezing and shortness of breath. Other outcomes examined the consistency of associations. When parents reported hearing their child wheeze for 3 or more consecutive days or using medication for wheezing in the past year, the child was classified as having severe wheezing within the last 12 months. A child was classified as having current wheeze if within the past 12 months their wheezing caused shortness of breath, woke them at night, or occurred with exercise in addition to any of the conditions described above for severe wheezing.

Exposure Assessment

Exposure assessment was based on questionnaire information on housing character-

TABLE 1—Prevalence of Respiratory Symptoms and Other Conditions

Term	Description	Frequency, %
Ever asthma	Ever told by doctor that child has asthma	1.9
Current asthma	Doctor-diagnosed asthma with parental reporting of shortness of breath or wheeze, or use of asthma medication within past 12 months	1.5
Severe asthma	Hospitalization for asthma/regular medication for asthma within past 12 months	0.8
Ever wheeze	Wheezing heard from distance without a cold	3.1
Current wheeze	Wheezing heard from distance with or without a cold, shortness of breath with wheezing, awakening at night by wheezing, wheezing with exercise, or use of medication or hospitalization within past 12 months for wheezing	13.4
Current severe wheeze	Wheezing heard from a distance for ≥3 consecutive days, hospitalization or medication use for asthma within past 12 months	10.1
Current asthma-like symptoms	Asthma symptoms or asthma medication use, awakening by asthma, wheezing upon exercise, or hospital care for wheezing within past 12 months	10.3
Severe asthma-like symptoms	Current asthma-like symptoms with routine medication use within past 12 months	2.2
Ever cough	Usual cough day or night	25.7
Persistent cough	Cough ≥3 consecutive months within past 12 months	5.5
Ever phlegm	Wet cough or phlegm produced without a cold	7.0
Persistent phlegm	Wet cough or phlegm ≥3 consecutive months within past 12 months	1.5
Dry cough	Ever cough without phlegm	17.8
Persistent dry cough	Persistent cough without phlegm for ≥3 consecutive months within past 12 months	3.6
Upper respiratory infection	Acute upper respiratory infection within past 12 months	76.9
Severe upper respiratory infection	Two or more acute upper respiratory infections within past 12 months	24.2
Current bronchitis	Doctor-diagnosed bronchitis within past 12 months	8.3
Any allergy	Doctor-diagnosed allergy, reported hay fever, or pollenosis	33.2
Respiratory allergy	Hay fever or doctor-diagnosed allergies to airborne substances (e.g., dust, animals, molds, pollens, air pollution, environmental tobacco smoke)	8.0
Eye irritation	Eye irritation sometimes	20.8

istics. Questions inquired about the age of the building, the type of construction, and its proximity to traffic. Apartment-related factors included heating and cooking methods, presence of ventilation, and geographic orientation and size. Respondents reported on smoking within the apartment, water damage, presence of mold, and the number of occupants. Ancillary information on cleaning frequency, parental occupational exposures to chemicals, and parental income, as well as variables related to nonrespiratory health outcomes, was collected. Density indicators were derived from information on apartment size and number of occupants.

Covariates Used for Adjustment

Univariate analyses explored several potential confounders. Gender, age, preterm birth, parental atopy, parents' education in a specialty field beyond high school, and smoking variables were used as core adjusting covariates in logistic regression unless the variable of interest was smoking itself. Additional models that included income, presence of furry pets, and sharing a bedroom as adjusting variables were explored but not presented because our basic findings were not altered.

Statistical Methods

The odds ratio (OR) was used as a measure of effect between the outcome and exposure. We calculated crude ORs and 95% confidence intervals (CIs) based on the Mantel-Haenszel test statistics. We estimated the adjusted ORs in logistic regression analysis. The ORs were adjusted for the covariates described above. The results from the adjusted logistic regression analyses are reported in this paper.

RESULTS

The response rate across schools varied from 96% to 99% and averaged 98% overall. Half of the buildings/homes were constructed within the past 20 years, and 70% of the buildings were concrete high-rises. Eighty-five percent of the respondents lived in single-family apartments, of which 50% were smaller than 40 m^2. Seventy-four percent of the children shared a bedroom. District heating plants provided heat and hot water requirements for 95% of the apartments/homes. Only 5% had a combustion heat source within their home. Gas was the cooking fuel for 80% of the homes, and 73% had no mechanical means of venting exhaust. Only 5% of the housing units had gas water heaters. Sixty percent of the respondents reported that their apartments did not face roadways.

Almost 60% of the families reported having a furry pet at home. Toxic substance exposures of parents at the workplace were reported for 21.7% of the children, and 1.9% had parents with the potential for bringing toxic material home as they did not change their clothes at their workplace. Cleaning of homes was infrequent; nearly 80% said they cleaned less than once per month, with only 3.2% cleaning weekly. Water damage was reported for 22.4% of the living units, and 10.4% reported water damage within the past 12 months. The appearance or detection of molds within the past 12 months occurred in 10% of the homes. A small percentage of mothers (4.2%) admitted smoking during pregnancy. Environmental tobacco smoke (ETS) exposure at home at various stages of the child's life—less than 2 years of age, 2 to 6 years of age, and currently—occurred for 45%, 51%, and 46% of the children, respectively.

Additional variables were derived from smoking responses, occupancy, and size of

TABLE 2—Adjusted ORs for Respiratory Symptoms and Housing Conditions

	Building Age (> 40 y vs ≤ 10 y) OR (95% CI)	Building Material (Concrete vs Wood) OR (95% CI)	Density of Children (4th vs 1st Quartile) OR (95% CI)	Area of Residence (> 60 m^2 vs < 25 m^2) OR (95% CI)	Traffic Near Apartment (Medium vs None) OR (95% CI)	Cooking Fuel (Gas vs No Gas) OR (95% CI)
Current doctor-diagnosed asthma	1.59 (0.75, 3.33)	1.28 (0.46, 3.62)	1.40 (0.50, 3.86)	0.60 (0.24, 1.48)	0.89 (0.41, 1.91)	2.28 (1.04, 5.01)
Current wheeze	1.37 (1.04, 1.80)	1.00 (0.72, 1.38)	1.34 (0.92, 1.96)	0.99 (0.71, 1.37)	1.22 (0.94, 1.57)	1.06 (0.86, 1.31)
Current asthma-like symptoms	1.41 (1.04, 1.92)	1.08 (0.74, 1.56)	1.31 (0.82, 2.09)	1.16 (0.86, 1.69)	1.28 (0.96, 1.69)	1.19 (0.94, 1.52)
Persistent cough	1.78 (1.12, 2.60)	1.24 (0.73, 2.10)	2.55 (1.44, 4.51)	0.48 (0.30, 0.79)	2.27 (1.64, 3.14)	1.19 (0.85, 1.65)
Persistent phlegm	1.83 (0.83, 4.06)	0.69 (0.30, 1.57)	3.49 (1.29, 9.47)	0.27 (0.11, 0.72)	2.21 (1.20, 4.06)	0.85 (0.48, 1.53)
Current dry cough	1.16 (0.91, 1.48)	1.13 (0.83, 1.54)	1.84 (1.31, 2.57	0.54 (0.40, 0.72)	1.30 (1.04, 1.63)	1.04 (0.86, 1.25)
Persistent dry cough	1.65 (0.98, 2.78)	1.40 (0.70, 2.79)	2.08 (1.03, 4.17)	0.66 (0.36, 1.19)	2.46 (1.66, 3.62)	1.31 (0.86, 1.99)
Current upper respiratory infection	1.02 (0.82, 1.27)	0.94 (0.72, 1.23)	1.28 (0.95, 1.72)	0.90 (0.68, 1.17)	1.02 (0.83, 1.25)	0.91 (0.77, 1.08)
Current bronchitis	1.24 (0.90, 1.73)	0.98 (0.66, 1.45)	1.31 (0.82, 2.08)	0.93 (0.60, 1.44)	1.19 (0.87, 1.61)	1.13 (0.87, 1.47)
Any allergy	0.89 (0.74, 1.08)	1.34 (1.05, 1.72)	0.87 (0.66, 1.14)	1.04 (0.81, 1.32)	1.09 (0.91, 1.32)	1.06 (0.92, 1.23)
Respiratory allergy	0.97 (0.69, 1.36)	2.61 (1.45, 4.72)	1.23 (0.78, 1.93)	0.99 (0.65, 1.52)	1.41 (1.04, 1.90)	1.08 (0.83, 1.40)
Eye irritation	0.93 (0.74, 1.16)	1.20 (0.91, 1.60)	0.91 (0.67, 1.25)	0.76 (0.58, 0.99)	1.31 (1.06, 1.61)	0.98 (0.83, 1.16)

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

the living unit. Exposure to ETS sometime during the child's life occurred for 63% of the children. We hypothesized that internal sources of air pollution, including airborne pathogens, might result in higher concentrations that vary inversely to volume of the residence or directly with crowding factors, based on occupant density. The area of the residence was separated by quartiles as a proxy for volume. The number of children and total number of occupants were divided by reported floor area and divided into quartiles to create 2 indicators of crowding.

The ORs for housing conditions are shown in Table 2. The results suggest that living in apartments more than 40 years old might increase the risk of wheeze. Similar results were seen for ever phlegm, ever cough, and persistent cough. The ORs for buildings aged 10 to 20 and 20 to 40 years, compared with buildings less than 10 years old, showed no evidence for a trend by age of residence. Only allergy (any or respiratory) showed an association with concrete versus wooden houses or apartments. Reporting of cough and phlegm conditions was significantly higher for more crowded housing and significantly less for larger residences. Findings are consistent when density is calculated by total number of occupants or just total children per area of the home. There were no observable trends over the 4 quartiles, and higher prevalences were observed for the quartile of most densely crowded residences. A protective effect of a larger apartment/home was seen only for cough and phlegm symptoms and was more pronounced for the larger-area apartments (>60 m^2) versus the smaller units (<25 m^2).

Those reporting a self-defined medium exposure to traffic outside their residence had higher prevalence of both respiratory allergy and eye irritation (nonsignificant). However, the cough and phlegm symptoms showed a significant positive association with traffic, with an apparent trend from light to medium traffic.

Health outcomes were examined for internal heating, gas cooking, gas water heaters, and whether or not exhaust ventilation made any difference in response rates. Only 12 families had unvented gas water heaters, so these results were not reported. Although gas cooking and a combustion heating device were positively associated with increased symptoms, none reached significance. Having some form of exhaust ventilation reduced the risk for respiratory allergy and dry cough, but only the latter was significant (OR=0.77 [95% CI=0.64, 0.93]). For completeness, we examined other symptoms for the influence of combustion and exhaust ventilation and found that doctor-diagnosed asthma and current asthma had a significant positive association with gas cooking. The adjusted ORs were 2.28 (95% CI=1.04, 5.01) for current asthma and 2.12 (95% CI=1.09, 4.11) for doctor-diagnosed asthma. Although the severity of asthma and the various wheeze-related outcomes all had positive adjusted ORs, none were significant at the 95% CI.

In examining all smoking variables, we found for the most part that all adjusted ORs across all outcomes showed positive associations with smoking exposure variables. Current dry cough showed significant associations, as did ever cough, persistent cough, and persistent dry cough. Experiencing a respiratory tract infection within the past year was associated with ETS exposure sometime in the child's life but not necessarily with current smoking in the home. Doctor-diagnosed bronchitis was strongly associated with lifetime ETS exposure (OR=1.26 [95% CI=1.10, 1.44]), but not for bronchitis within the past year. Table 3 presents the adjusted ORs and 95% CIs for 12 of the health variables and 2 of the smoking variables (smoking during pregnancy and the composite variable of any ETS exposure during the child's life). Other smoking variables similar to smoking during pregnancy showed few statistically positive associations, unlike the composite variable of ever being exposed to ETS.

The housing conditions with the strongest and most consistent associations with health outcomes were reported moisture (water damage) and the presence of molds on surfaces. Table 4 presents the adjusted ORs for reported water and mold conditions within

TABLE 3—Adjusted ORs for Respiratory Symptoms and Tobacco Smoke Exposure During Child's Lifetime

	Tobacco Smoke Exposure	
	In Utero OR (95% CI)	Lifetime Exposure OR (95% CI)
Current doctor-diagnosed asthma	2.07 (0.85, 5.03)	1.22 (0.75, 2.03)
Current wheeze	1.41 (0.97, 2.06)	1.12 (0.94, 1.34)
Current asthma-like symptoms	1.44 (0.95, 2.16)	1.15 (0.95, 1.40)
Persistent cough	0.95 (0.51, 1.75)	1.34 (1.02, 1.75)
Persistent phlegm	0.54 (0.13, 2.29)	1.31 (0.78, 2.19)
Current dry cough	1.24 (0.86, 1.78)	1.35 (1.15, 1.58)
Persistent dry cough	1.17 (0.58, 2.38)	1.53 (1.08, 2.16)
Current upper respiratory infection	0.72 (0.52, 1.00)	1.21 (1.06, 1.38)
Current bronchitis	1.57 (1.00, 2.45)	1.05 (0.85, 1.29)
Any allergy	0.90 (0.66, 1.23)	1.18 (1.04, 1.33)
Respiratory allergy	1.10 (0.66, 1.83)	1.04 (0.84, 1.28)
Eye irritation	1.09 (0.77, 1.54)	1.22 (1.06, 1.41)

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

TABLE 4—Adjusted ORs for Respiratory Symptoms and Water Damage and Presence of Molds Within the Past 12 Months

	Water Damage OR (95% CI)	Presence of Molds OR (95% CI)
Current doctor-diagnosed asthma	1.37 (0.69, 2.70)	2.82 (1.63, 4.88)
Current wheeze	1.53 (1.19, 1.95)	1.52 (1.19, 1.94)
Current asthma-like symptoms	1.77 (1.36, 2.30)	1.98 (1.53, 2.55)
Persistent cough	1.51 (1.06, 2.16)	1.88 (1.35, 2.63)
Persistent phlegm	2.15 (1.18, 3.93)	2.46 (1.38, 4.38)
Current dry cough	1.35 (1.08, 1.69)	1.40 (1.12, 1.76)
Persistent dry cough	1.33 (0.85, 2.09)	1.53 (0.99, 2.35)
Current upper respiratory infection	1.23 (0.98, 1.55)	1.74 (1.35, 2.25)
Current bronchitis	1.52 (1.14, 2.03)	1.70 (1.28, 2.27)
Any allergy	1.26 (1.05, 1.52)	1.51 (1.25, 1.82)
Respiratory allergy	1.30 (0.95, 1.77)	1.50 (1.11, 2.02)
Eye irritation	1.21 (0.98, 1.50)	1.42 (1.15, 1.76)

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

the last 12 months. Prevalence of symptoms increased from 35% to almost 100% when mold was present in the home. The association was slightly stronger for mold conditions than for just water damage. All health outcomes were more strongly associated with reported mold and water damage within the past 12 months compared with ever having water damage or molds in the living unit. The association was weaker for molds being reported in the child's bedroom.

Having any furry pet was strongly protective for respiratory allergy (OR=0.61 [95% CI=0.50, 0.74]) but less so for severe wheezing (OR=0.84 [95% CI=0.74, 1.01]) and current bronchitis (OR=0.86 [95% CI=0.67, 1.00]). Having a furry pet was strongly negatively associated with current asthma (OR=0.40 [95% CI=0.25, 0.64]), whereas having a cat was specifically associated with higher rates of doctor-diagnosed asthma (OR=3.29 [95% CI=1.01, 10.72]) but not with asthma symptoms (OR=1.06 [95% CI=0.78, 1.44]).

Examining the relationship between parental exposures to toxic material at work and their children's symptoms yielded interesting results. Even though only about 2% of the responding parents had workplace exposures, there were significant associations with dry cough (OR=2.35 [95% CI=1.54, 3.59]), persistent dry cough (OR=2.18 [95% CI=1.05, 4.55], and severe wheezing (OR=1.76 [95% CI=1.03, 3.02]). Reported frequency of house cleaning revealed no consistent or significant relationships.

DISCUSSION

Consistent with similar health surveys conducted in the United States and Europe, conditions of mold and dampness in living areas are strongly associated with increased respiratory symptoms. In an examination of all published literature, a Nordic scientific review panel concluded that the presence of dampness in a home increased the reporting of cough, wheeze, and respiratory symptoms by 40% over a reference population.[3] The risk appears to be similar for Russian housing. However, the prevalence of moisture and mold in the housing stock is approximately half of the prevalence reported for surveys done in the United States and Canada.[7,8] Jacob et al.[9] showed that high counts of *Cladosporium* and *Aspergillus* spores in house dust were associated with increased risk of allergic sensitization. Their results suggest that higher spore counts, particularly in the winter, are likely to increase the prevalence of allergic symptoms in children.

Cook and Strachan[10] conducted pooled analysis of ORs for parental smoking on asthma, wheeze, chronic cough, chronic phlegm, and shortness of breath symptoms in children exposed to ETS. Our Russian results for ever cough, persistent cough, dry cough, and persistent dry cough are similar to the pooled ORs for cough (OR=1.35 [95% CI=1.13, 1.62]). Also, our findings for phlegm were similar to the pooled ORs of 1.31 (95% CI=1.08, 1.59). Asthma and wheeze, although both significantly associated with parental smoking in the pooled analysis, were not significantly associated with any measure of ETS exposure over the child's life.

Gilliland et al.[11] reported that in utero exposure to maternal smoking without subsequent postnatal ETS exposure significantly increased the association with doctor-diagnosed asthma, asthma symptoms, and asthma severity later in a child's life, as well as most of the wheezing outcomes. Our ORs for asthma and wheeze outcomes were all positively associated with smoking during pregnancy (approximately 2.0) but did not reach $P<0.05$ significance. Given the lack of specificity to the smoking questions asked in this survey, it is not possible to ascertain the separate influence of maternal versus paternal smoking or even age-related responses seen in other studies. Gilliland et al.[11] similarly showed that current and previous ETS exposure was not associated with asthma prevalence but was consistently associated with various wheezing variables.

Apelberg et al.[12] performed a meta-analysis of the studies on the effect of early exposure to household pets on the development of asthma and asthma-related symptoms. Inappropriate time sequence of the exposure and outcome information, typical for cross-sectional studies, was an important source of heterogeneity and an indication of potential selection bias. In studies ensuring a meaningful temporal relation between exposure and outcome, the pooled risk estimates for both asthma (fixed-effects OR=1.11 [95% CI=0.98, 1.25]; $P=0.04$) and wheeze (OR=1.19 [95% CI=1.05, 1.35]; $P=0.03$) indicated a small effect. However, the effect was limited to studies with a median study population age greater than 6 years. In younger children, the effect appeared protective for wheezing (OR=0.80 [95% CI=0.59, 1.08]; $P=0.38$). The authors concluded that the observed lower risk among exposed compared to unexposed young children is consistent with a protective effect in this age group, but could also be explained by selection bias.

In a prospective study of asthma incidence in adolescents, McConnell et al.[13] reported a relative risk of 1.6 (95% CI=1.0, 2.5) for having a furry pet at home. The present study was cross-sectional and did not inquire precisely when the pet had been present. Therefore, the negative associations between the presence of pets and the risk of asthma and allergies could be a result of either avoidance or removal of pets in families with children allergic to respiratory allergens and with asthma problems.

Exposure to nitrogen dioxide (NO_2) from gas cooking is a common experience for the majority of children in this survey. From studies that measured NO_2 indoors, it can be inferred that concentrations will be higher in the absence of exhaust vents. Yet examining possible interactive effects for cooking fuel and ventilation offered no evidence for increased association with the inferred exposure gradient (e.g., not using gas but having ventilation versus having gas but not having ventilation). Garrett et al.[14] reported that gas stoves increased the risk of respiratory symptoms in children (OR=2.3 [95% CI=1.0, 5.2]), whereas the association with direct measures of NO_2 was marginal. Shima and Adachi[15] reported that the prevalence of bronchitis wheeze and asthma significantly increased with indoor NO_2 exposures among girls but not among boys. They also showed that wheeze and asthma incidence were associated with outdoor NO_2 but not indoor NO_2. Examining the effects of NO_2 from gas heaters in school rooms, Pilotto et al.[16] found significant increases in sore throats, colds, and absences from school when hourly peak exposures exceeded 80 ppb compared with background levels of 20 ppb. In a large study of respiratory infections among 1000 infants in Albuquerque, New Mexico, Samet et al.[17] found no associations for either gas stove or NO_2 levels measured in the kitchen or the child's bedroom.

This study of Russian schoolchildren and housing factors poses some interesting observations for further investigation. Only 40% of the respondents reported either light or medium traffic outside their homes/apartments. Persistent cough, phlegm, and dry cough, as well as the prevalence of severe upper respiratory infection in children, were positively associated with medium traffic loadings compared with not living along any roadways. Clustered apartment complexes removed from roadways are common in many Russian cities; it is a situation unlike that of any study reporting associations with asthma and respiratory symptoms for children living close to heavily traveled roads. In US, Western European, and Japanese studies, the reference group always has approximate exposure to some road traffic.[15,18–24] In this Russian study, none of the asthma or wheeze variables showed an association with subjective reporting of traffic exposure. Nevertheless, the possible association of vehicle exhaust on chronic cough and phlegm cannot be dismissed.

Just 1.9% of the children had 1 or both parents reporting occupational contact with potentially toxic substances and not leaving their work clothes at the job site. Another 19.75% had parents who might be exposed but leave their clothing at work. Consistently positive associations were found for wheezing (severe and current) and most of the coughs (persistent dry cough, dry cough, usual cough) as well as asthma-like symptoms and general atopy. These observations suggest that compounds may be carried home on clothing or absorbed in fibers of clothing, leading to children being exposed at home. Many metallurgical and chemical production facilities are located in Cherepovets and throughout Sverdlovsk Oblast. It is likely that some parents are heavily exposed to potentially irritating or toxic materials at work.

Although doctor-diagnosed asthma rates for Russian children were substantially lower than rates children are currently experiencing in the West, the rates are consistent with reports from former Soviet Bloc countries. Jedrychowski et al.[25] reported doctor-diagnosed asthma among 1129 9-year-old children living in Krakow, Poland, as 1.9% for girls and 2.4% for boys in 1995. The International Study of Asthma and Allergies in Childhood showed that the variation in the prevalence of asthma and self-reported asthma symptoms between different countries is striking.[26] In these comparisons the prevalences of all the studied indices of asthma were lower in Eastern than in Western Europe. Corresponding figures for asthma symptoms from video questionnaires were 2% in Russia and between 12% and 20% in the United Kingdom, the United States, Canada, New Zealand, and Australia. Differences in access to health care, diagnostic practice, and environmental and dietary factors are plausible explanations for the large variation in the prevalences of asthma between Russia and Western Europe/North America.

Rates for other conditions and symptoms in Russian children are comparable to rates reported from studies conducted in the United States. In the Harvard 24 Cities Study of air pollution and children's health in 24 North American towns,[27] 33% of the children re-

ported some atopy. The reporting of current asthma symptoms within the past year ranged from 3% to 11% of the children across 24 communities, with persistent wheeze ranging from 4% to 12%. For Russian children, asthma-like symptoms were 10%, with a higher rate of current wheeze (13.4%). Chronic cough in the 24 Cities Study ranged from 4% to 9%, which is consistent with the 5.5% noted in our study. Parents reported chronic bronchitis in the past year at rates of 3% to 10% across the 24 Cities Study, and a rate of 8.3% for our Russian children. The respiratory symptoms in children associated with ETS exposure, water damage, and presence of molds in the Russian housing study were consistent with reports on housing conditions in many other countries.

Russian housing is characterized by large, concrete high-rise structures. Apartments are similar in layout and size, and are served by district hot water for heating and gas for cooking; mechanical ventilation and air conditioning is a rarity. Our study reporting prevalences of conditions and associated health symptoms provides important insights that are applicable to millions of children living in similar housing. ■

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Contributors

J.D. Spengler designed the study and led the analysis and the writing of the article. J.J.K. Jaakkola helped design and oversee the study and analysis, and participated in the writing of the article. H. Parise and A.A. Kosheleva analyzed the data. B.A. Katsnelson and L.I. Privalova helped design and conduct the study, managed staff, checked records, and participated in data analysis.

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Human Participant Protection

Parents were informed that participation was voluntary. No personal identifiers were used in our data files, and all questionnaires have been destroyed. Data were not collected from children.

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Metropolitan-Area Estimates of Binge Drinking in the United States

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Objectives. We estimated adult binge drinking prevalence in US metropolitan areas.

Methods. We analyzed 1997 and 1999 Behavioral Risk Factor Surveillance System data for 120 metropolitan areas in 48 states and the District of Columbia.

Results. The prevalence of binge drinking varied substantially across metropolitan areas, from 4.1% in Chattanooga, Tenn, to 23.9% in San Antonio, Tex, (median=14.5%). Seventeen of the 20 metropolitan areas with the highest estimates were located in the upper Midwest, Texas, and Nevada. In 13 of these areas, at least one third of persons aged 18 to 34 years were binge drinkers. There were significant intrastate differences for binge drinking among metropolitan areas in New York, Tennessee, and Utah.

Conclusions. Metropolitan-area estimates can be used to guide local efforts to reduce binge drinking. (*Am J Public Health.* 2004;94:663–671)

Alcohol use results in approximately 100 000 deaths each year in the United States, which makes it the third leading cause of preventable death in the country.[1] Binge drinking is an especially hazardous pattern of alcohol consumption that causes a substantial proportion of alcohol-related deaths.[2–5] Adverse effects of binge drinking include intentional injuries (e.g., interpersonal violence and suicide), unintentional injuries (e.g., motor vehicle crashes and drownings), fetal alcohol syndrome/effect, and unintended pregnancy.[6–8]

Binge drinking most commonly occurs among males, younger persons, and persons residing in urban or suburban areas,[9,10] and the occurrence is generally higher in the Midwest, the Northeast, and the West.[9–11] Recent studies have shown that binge drinking and its related health consequences have increased in the past few years,[12–14] as binge drinking increased in 19 states while it declined in only 3 states from 1991 through 1999.[13]

Although national and state-based surveys obtain data on alcohol measures on a regular basis, there are only limited data about binge drinking at the local level. Independent surveys of adults in Los Angeles, Calif,[15,16] and Harlem, NY,[17] obtained selected data on alcohol use, and a few states have used the Behavioral Risk Factor Surveillance System (BRFSS) to generate health district or city estimates.[18–21] The lack of local data is unfortunate. Local data can empower communities to address public health issues, to track progress toward *Healthy People 2010* alcohol-related goals,[22,23] and to improve planning and evaluation efforts to prevent alcohol abuse. However, because conducting surveys is time-consuming and expensive, most local health departments lack the resources to collect or analyze survey data.

We used reweighted BRFSS data[24] to examine current alcohol use and binge drinking in 120 US metropolitan areas. The purposes of our study were to (1) estimate the prevalence of binge drinking in metropolitan areas, (2) determine if there were differences between metropolitan and statewide estimates of binge drinking, (3) assess intrastate differences in binge drinking for states with data from 2 or more metropolitan areas, (4) estimate the proportion of current drinkers who were binge drinkers by metropolitan area, and (5) identify the demographic subgroups within metropolitan areas that have the highest overall binge-drinking estimates and are at greatest risk for experiencing adverse effects from this type of alcohol use.

METHODS

Data were obtained from the BRFSS, a state-based system of adult health surveys coordinated by the Centers for Disease Control and Prevention.[11,25] Health risk factor and preventive health services data were obtained through telephone surveys of randomly selected adults aged 18 years and older. Data were collected annually for some measures and biennially for others; questions on alcohol use were included in odd-numbered-year surveys during the 1990s.[25] Current drinking was defined as consuming 1 or more alcoholic beverages in the past month; binge drinking was defined as consuming 5 or more alcoholic beverages on at least 1 occasion in the past month, which was determined by the question, "Considering all types of alcoholic beverages, how many times during the past month did you have 5 or more drinks on an occasion?"

We analyzed data from 1997 and 1999, the most recent years with available metropolitan-level data on binge drinking. The overall sample size was 133 048 in 1997 and 159 921 in 1999; median state sample sizes were 2340 in 1997 (range=1505 to 4923) and 2939 in 1999 (range=1248 to 7543). Median state response rates, on the basis of persons actually reached by telephone, were 76.8% in 1997 and 68.4% in 1999, with individual state response rates ranging from 44.5% to 95.1%.

Self-reported county of residence was used to classify respondents as residents of metropolitan areas in accordance with standard census definitions.[26] Response rates by metropolitan area were unavailable, as all rates were calculated by state. Metropolitan-level estimates are composed of groups of counties, oftentimes encompassing counties in more than one state. Metropolitan-level data were not merged with state data because the state data were weighted to state populations and metropolitan-level data were weighted to county populations. See Table 1 for metropolitan city designations.

All analyses were conducted with SAS[27] and SUDAAN[28] software. To increase sample sizes and to improve the precision of estimates, we pooled 1997 and 1999 data and

TABLE 1—Binge-Drinking Prevalence and 95% Confidence Intervals for US Metropolitan Areas, by Region: 1997 and 1999

Metropolitan Area and Region	n	Binge Drinking, %
Northeast		
Bergen–Passaic, NJ	820	10.0 (7.5, 12.5)
Boston–Worcester–Lawrence–Lowell–Brockton, Mass–NH	6682	17.6 (16.2, 19.0)
Burlington, Vt	1891	19.6 (17.1, 22.1)
Hartford, Conn	1765	14.3 (12.2, 16.4)
Middlesex–Somerset–Hunterdon, NJ	682	12.5 (9.2, 15.8)
Monmouth–Ocean, NJ	796	14.1 (10.9, 17.3)
Nassau–Suffolk, NY	867	11.6 (9.0, 14.2)
New Haven–Bridgeport–Stamford–Waterbury–Danbury, Conn	2542	14.2 (12.3, 16.1)
New York, NY	2588	9.3 (8.0, 10.6)
Newark, NJ	1261	11.2 (9.0, 13.4)
Philadelphia, Pa–NJ	2657	14.6 (12.9, 16.3)
Pittsburgh, Pa	1463	16.3 (14.0, 18.6)
Portland, Me	708	15.3 (12.0, 18.6)
Providence–Warwick–Pawtucket, RI	5186	14.6 (13.3, 15.9)
Rochester, NY	425	15.5 (11.5, 19.5)
Springfield, Mass	819	20.1 (15.3, 24.9)
Median (range)		14.5 (9.3, 20.1)
Midwest		
Bismarck, ND	543	18.6 (15.2, 22.0)
Cedar Rapids, Iowa	404	20.6 (15.7, 25.5)
Chicago, Ill	1793	16.8 (14.8, 18.8)
Cincinnati, Ohio–Ky–Ind	1784	12.5 (10.2, 14.8)
Cleveland–Lorain–Elyria, Ohio	986	8.8 (6.4, 11.2)
Columbus, Ohio	881	11.0 (7.8, 14.2)
Davenport–Moline–Rock Island, Iowa–Ill	421	21.1 (14.8, 27.4)
Des Moines, Iowa	1088	18.2 (15.5, 20.9)
Detroit, Mich	1960	20.0 (17.9, 22.1)
Duluth–Superior, Minn–Wis	495	20.3 (16.0, 24.6)
Evansville–Henderson, Ind–Ky	656	13.6 (8.9, 18.3)
Fargo–Moorhead, ND–Minn	814	17.7 (14.6, 20.8)
Fort Wayne, Ind	589	14.1 (10.4, 17.8)
Gary, Ind	582	16.3 (11.8, 20.8)
Grand Forks, ND–Minn	453	23.4 (17.9, 28.9)
Grand Rapids–Muskegon–Holland, Mich	550	18.1 (14.6, 21.6)
Indianapolis, Ind	1123	15.1 (12.5, 17.7)
Kansas City, Mo–Kan	2475	15.6 (13.5, 17.7)
Lincoln, Neb	756	20.1 (15.7, 24.5)
Milwaukee–Waukesha, Wis	1112	22.7 (19.4, 26.0)
Minneapolis–St Paul, Minn–Wis	5971	16.2 (15.0, 17.4)
Omaha, Neb–Iowa	2346	17.2 (15.2, 19.2)
Rapid City, SD	771	16.9 (13.7, 20.1)
Sioux Falls, SD	1314	21.4 (18.8, 24.0)
South Bend, Ind	510	14.5 (10.6, 18.4)
St Louis, MO–IL	1371	17.2 (14.8, 19.6)
Wichita, Kan	1098	11.0 (8.7, 13.3)
Median (range)		17.2 (8.8, 23.4)

Continued

limited analyses to the 120 metropolitan areas with 400 or more respondents for both alcohol measures; sample sizes ranged from 404 to 6682. We used intercensal estimates to reweight data by the age, gender, and racial/ethnic distributions for each metropolitan area. Missing or unknown data were excluded from all calculations. Data were not age standardized, because we wanted to provide actual binge-drinking estimates for each area.

Metropolitan-level data were available from metropolitan areas in 48 states and the District of Columbia (data not available for Alaska and New Hampshire, where there were no metropolitan areas with sufficient sample sizes). To provide additional context, we pooled state estimates of binge drinking from 1997 and 1999. State estimates were based on data from all state respondents, including those living within metropolitan areas.

We grouped metropolitan areas by census region (Northeast, Midwest, South, and West) and by state, and we calculated regional and national median and range values. To determine the relationship between estimates of current drinking and binge drinking, we calculated the proportion of current drinkers who were binge drinkers for each metropolitan area. For the 20 metropolitan areas with the highest levels of binge drinking, we conducted analyses of binge-drinking estimates stratified by age (18–34 years and ≥35 years), gender, race/ethnicity (White, Black, Hispanic, other), education level (≤high school, >high school), and income (<$25 000, $25 000–$49 999, and ≥$50,000). To improve precision, these analyses were conducted only for subpopulations with at least 50 respondents.

We used 2-sample *t* tests[29] to assess whether differences between statewide and metropolitan estimates and differences between intrastate metropolitan-area estimates were statistically significant. For the 20 areas with the highest levels of binge drinking, we also used 2-sample *t* tests to examine differences in estimates by demographic groups within each metropolitan area. Because of the large number of comparisons, differences were considered statistically significant only when 99% confidence inter-

TABLE 1—*Continued*

South		
Atlanta, Ga	2176	10.8 (9.2, 12.4)
Augusta-Aiken, Ga-SC	429	9.2 (4.7, 13.7)
Austin-San Marcos, Tex	510	22.6 (17.8, 27.4)
Baltimore, Md	2969	11.9 (10.4, 13.4)
Baton Rouge, La	466	16.3 (12.8, 19.8)
Biloxi-Gulfport-Pascagoula, Miss	469	13.5 (9.6, 17.4)
Birmingham, Ala	937	12.9 (10.4, 15.4)
Charleston, WVa	758	8.7 (6.4, 11.0)
Charleston-North Charleston, SC	719	13.7 (10.4, 17.0)
Charlotte-Gastonia-Rock Hill, NC-SC	1153	11.8 (9.3, 14.3)
Chattanooga, Tenn	449	4.4 (2.4, 6.4)
Columbia, SC	732	12.8 (10.0, 15.6)
Dallas, Tex	1123	15.6 (12.7, 18.5)
Daytona Beach, Fla	407	14.7 (10.4, 19.0)
Dover, Del	1446	13.0 (11.0, 15.0)
Fayetteville-Springdale-Rogers, Ark	454	9.9 (6.9, 12.9)
Ft Lauderdale, Fla	850	11.8 (9.3, 14.3)
Ft Worth-Arlington, Tex	535	19.4 (15.1, 23.7)
Greensboro-Winston-Salem-High Point, NC	977	9.6 (7.1, 12.1)
Greenville-Spartanburg-Anderson, SC	1223	8.8 (6.9, 10.7)
Hagerstown, Md	422	8.8 (5.3, 12.3)
Houston, Tex	1221	17.5 (14.7, 20.3)
Huntington-Ashland, WVa-Ky-Ohio	986	6.5 (4.2, 8.8)
Jackson, Miss	626	11.1 (8.1, 14.1)
Jacksonville, Fla	769	12.6 (9.7, 15.5)
Johnson City-Kingsport-Bristol, Tenn-Va	573	6.9 (4.1, 9.7)
Knoxville, Tenn	698	7.2 (5.1, 9.3)
Lexington, Ky	743	10.0 (7.3, 12.7)
Little Rock-North Little Rock, Ark	996	11.5 (9.1, 13.9)
Louisville, Ky-Ind	1490	12.4 (10.3, 14.5)
Memphis, Tenn-Ark-Miss	1271	9.4 (7.4, 11.4)
Miami, Fla	974	10.0 (7.6, 12.4)
Mobile, Ala	468	13.2 (9.7, 16.7)
Nashville, Tenn	1240	10.4 (8.3, 12.5)
New Orleans, La	1002	16.4 (13.6, 19.2)
Norfolk-Virginia Beach-Newport News, Va-NC	1275	14.1 (11.5, 16.7)
Oklahoma City, Okla	1557	9.6 (7.7, 11.5)
Orlando, Fla	715	14.6 (11.6, 17.6)
Raleigh-Durham-Chapel Hill, NC	835	11.4 (8.9, 13.9)
Richmond-Petersburg, Va	932	15.9 (12.8, 19.0)
San Antonio, Tex	496	23.9 (18.8, 29.0)
Tampa-St Petersburg-Clearwater, Fla	1112	12.8 (10.5, 15.1)
Tulsa, Okla	1130	7.4 (5.4, 9.4)
Washington, DC-Md-Va-WVa	7276	11.5 (10.1, 12.9)
West Palm Beach-Boca Raton, Fla	746	12.9 (10.1, 15.7)
Wilmington-Newark, Del-Md	2545	16.3 (13.9, 18.7)
Median (range)		11.9 (4.4, 23.9)

Continued

vals (CI) for differences excluded the null value.

We used logistic regression analyses to examine the independent association of binge drinking with age, gender, education, or race/ethnicity (on the basis of odds ratios [OR] and 95% CI) for the 20 areas with the highest levels of binge drinking. Income was not included in these models because of collinearity with education.

We mapped binge-drinking estimates with ArcGIS 8.2 software (Environmental Research Systems, Inc, Redlands, Calif) for all 120 metropolitan areas, as well as by state, to better understand regional patterns.[30] Cut-points for metropolitan and state estimates were based on quartile ranges.

RESULTS

Considerable variability was found in overall binge-drinking estimates by metropolitan area. The median metropolitan estimate was 14.5%, which ranged from 4.4% in Chattanooga Tenn, to 23.9% in San Antonio, Tex (Table 1). Estimates were generally highest in the Midwest, intermediate in the West and the Northeast, and lowest in the South (Table 1 and Figure 1). However, substantial variation in binge drinking was found in all 4 regions that ranged from a 2-fold difference in the Northeast to a 5-fold difference in the South.

State binge-drinking estimates ranged from 7.4% in Tennessee to 25.2% in Wisconsin (median = 14.8%) (Figure 2) and were significantly different from metropolitan estimates in Montana, Tennessee, and Utah (data not shown). Three of 40 states—New York, Tennessee, and Utah—had statistically significant intrastate differences in binge drinking among metropolitan areas.

Nationally, the proportion of current drinkers who were binge drinkers was 26.7%, which ranged from 16.9% in Chattanooga, Tenn, to 42.2% in Grand Forks, ND–Minn; regional median estimates ranged from a low of 23.6% in the Northeast to a high of 30.4% in the Midwest (data not shown). The geographic pattern for this measure was generally similar to the overall pattern for binge-drinking prevalence. However, Provo–Orem, Utah; Salt Lake City, Utah; and Johnson City–Kingsport–Bristol, Tenn–Va were in the lowest quartile for

TABLE 1—*Continued*

West		
Albuquerque, NM	2064	15.2 (13.1, 17.3)
Billings, Mont	490	10.9 (7.7, 14.4)
Boise City, Idaho	2150	17.0 (14.8, 19.2)
Casper, Wyo	667	15.1 (12.0, 18.2)
Cheyenne, Wyo	759	14.5 (11.7, 17.3)
Colorado Springs, Colo	433	13.7 (10.0, 17.4)
Denver, Colo	1664	16.5 (14.5, 18.5)
Eugene–Springfield, Ore	448	14.7 (9.8, 19.6)
Honolulu, Hawaii	2390	14.0 (12.4, 15.6)
Las Cruces, NM	493	16.9 (13.0, 20.8)
Las Vegas, Nev–Ariz	1893	18.7 (15.9, 21.5)
Los Angeles–Long Beach, Calif	2061	15.3 (13.4, 17.2)
Oakland, Calif	612	14.8 (11.4, 18.2)
Orange County, Calif	642	16.5 (13.2, 19.8)
Phoenix–Mesa, Ariz	1235	9.3 (7.2, 11.4)
Pocatello, Idaho	671	14.4 (11.5, 17.3)
Portland–Vancouver, Ore–Wash	2768	14.9 (13.3, 16.5)
Provo–Orem, Utah	527	4.6 (0.1, 9.1)
Reno, Nev	1531	9.2 (4.7, 13.7)
Riverside–San Bernardino, Calif	724	9.2 (4.7, 13.7)
Sacramento, Calif	420	17.0 (12.5, 21.5)
Salem, Ore	451	9.6 (6.3, 12.9)
Salt Lake City–Ogden, Utah	2398	10.4 (9.0, 11.8)
San Diego, Calif	739	14.9 (12.0, 17.8)
San Francisco, Calif	431	17.3 (13.0, 21.6)
San Jose, Calif	409	16.0 (11.8, 20.2)
Santa Fe, NM	419	15.9 (11.2, 20.6)
Seattle–Bellevue–Everett, Wash	2887	14.9 (13.2, 16.6)
Spokane, Wash	529	19.6 (15.5, 23.7)
Tacoma, Wash	715	13.5 (10.6, 16.4)
Tucson, Ariz	827	8.6 (6.3, 10.9)
Median (range)		14.9 (4.6, 20.0)
National median (range)		14.5 (4.4, 23.9)

Note. CI = confidence interval.

binge drinking and were in the highest quartile for the proportion of current drinkers who also were binge drinkers.

The 20 metropolitan areas with the highest binge-drinking estimates are shown in Table 2 and Figure 2. Twelve areas were in 7 states in the upper Midwest (Iowa, Michigan, Minnesota, Nebraska, North Dakota, South Dakota, and Wisconsin), 3 were in Texas, and 2 were in Nevada. Not surprisingly, there was a strong correlation between high statewide estimates and metropolitan areas with the highest levels of binge drinking.

Binge drinking was significantly more common among persons aged 18 to 34 years than among those aged 35 years and older in 18 of the 20 metropolitan areas with the highest binge-drinking estimates. Among persons aged 18 to 34 years, estimates ranged from 28.8% in Fargo–Moorhead, Minn–ND, to 44.2% in Springfield, Mass (Table 2); in 13 areas, at least one third of persons in this age group were binge drinkers. Among adults aged 35 years and older, binge-drinking estimates ranged from 9.5% in Springfield, Mass, to 18.3% in San Antonio, Tex. Men were statistically more likely to report binge drinking than were women in 18 of the 20 metropolitan areas with the highest overall estimates (Table 2). More than one third of men were binge drinkers in San Antonio, Tex; Milwaukee–Waukesha, Wis; and Grand Forks, ND–Minn.

Comparisons of binge drinking by race/ethnicity were limited, because sample sizes were sufficient in only 4 metropolitan areas for Blacks and in only 7 areas for Hispanics. We found no significant differences in the prevalence of binge drinking by race/ethnicity in these areas (data not shown). Similarly, analyses of binge-drinking prevalence by education level for all 20 areas with the highest estimates revealed no significant differences (data not shown). With the exception of Burlington, Vt, where binge drinking was higher among persons who had income levels below $25 000 compared with those who had incomes above $50 000, there were no other significant differences in binge drinking by income level (data not shown).

Logistic regression models confirmed the strong association between age and gender with binge drinking for nearly all metropolitan areas (Table 3), with a median OR for persons aged 18 to 34 years of 3.76 (range = 1.80–8.21) relative to persons aged 35 years and older and with a median OR for men of 3.72 (range = 2.50–8.11) relative to women. Significantly higher odds ratios for binge drinking among younger adults were found in Springfield, Mass, and Spokane, Wash, compared with Austin–San Marcos, Tex. There were no significant differences in OR between metropolitan areas for binge drinking among men.

In San Antonio, Tex; Sioux Falls, SD; Davenport–Moline–Rock Island, Iowa–Ill; and Spokane, Wash, there was some evidence that lower levels of education were associated with binge drinking, although 95% CIs were close to 1.00 in each area. No association was found between levels of education and binge drinking in the remaining areas. Logistic models confirmed the absence of an independent association between race/ethnicity and binge drinking in areas with 50 or more Black or Hispanic respondents (data not shown), because a significant difference was found only among blacks in Las Vegas, Nev (OR = 1.78; 95% CI = 1.03, 3.08).

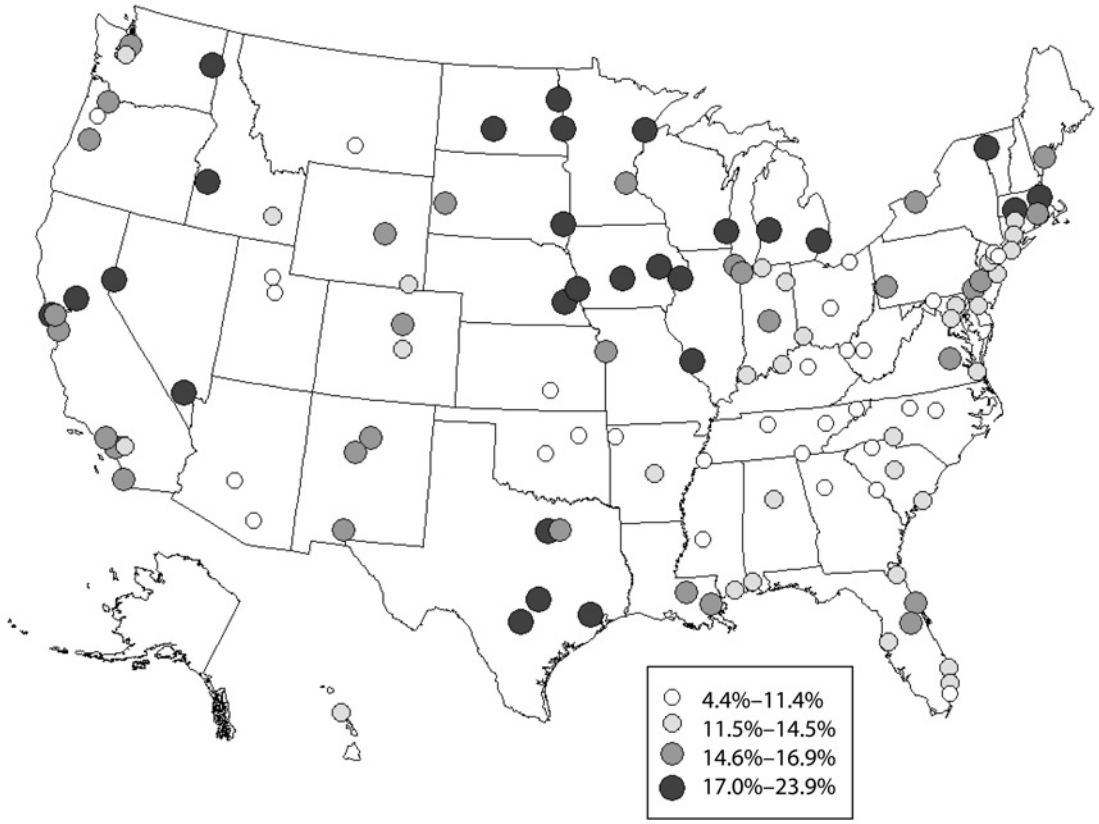

FIGURE 1—Geographic distribution of binge-drinking prevalence estimates for US metropolitan areas: 1997 and 1999.

DISCUSSION

To our knowledge, this is the first study that comprehensively reports binge-drinking estimates for US metropolitan areas. We found that binge drinking was especially common in metropolitan areas located in the upper Midwest, Texas, and Nevada. The proportion of current drinkers who reported binge drinking exceeded 30% in many US metropolitan areas. Analyses of areas with the highest binge-drinking estimates showed that at least one third of persons aged 18 to 34 years in 13 metropolitan areas were binge drinkers, as were more than one third of men in San Antonio, Tex; Grand Forks, ND–Minn; and Milwaukee–Waukesha, Wis. In the 20 areas with the highest binge-drinking estimates, we found that age and gender were strongly and independently associated with binge drinking.

Our results indicate that metropolitan binge-drinking estimates vary across regions, within regions, and within individual states. Although there were few differences when we compared metropolitan estimates with their corresponding state estimates, our ability to detect such differences was limited, because state estimates were based on information provided by all respondents within states, which included persons within metropolitan areas. Future studies are needed that compare metropolitan and nonmetropolitan binge-drinking estimates within states.

Our results were consistent with nationally representative adult data from the National Household Survey on Drug Abuse (NHSDA), which demonstrated higher binge-drinking estimates among young adults, men, and residents in the upper Midwest, as well as the general lack of differences by educational level.[9,10] In contrast to our findings, NHSDA data demonstrated that adult binge-drinking estimates were higher for Whites and Hispanics than for Blacks. Although we did not find similar racial/ethnic differences, we used only a limited number of metropolitan areas for such comparisons. Our metropolitan estimates for binge drinking were generally similar to previous BRFSS-developed estimates for metropolitan areas in Wisconsin, Idaho, and Massachusetts.[18–21]

There are several possible explanations for the substantial variation in binge drinking across metropolitan areas. Because binge drinking among adults varies inversely with age,[6,9,10] metropolitan areas with younger populations are likely to have higher estimates. For example, several metropolitan areas with high binge-drinking estimates, such as Grand Forks, ND–Minn; Austin–San Marcos, Tex; Lincoln, Neb; and Burlington, Vt, have major state universities. However, the OR for binge

TABLE 2—Binge Drinking Prevalence (%), by Age and Gender, and 95% Confidence Intervals for the 20 Metropolitan Areas With the Highest Levels of Binge Drinking: 1997 and 1999

Metropolitan Area	N	Region	Overall	Age Group, y		Sex	
				18–34	≥35	Men	Women
1. San Antonio, Tex	496	South	23.9 (18.8, 29.0)	35.3 (26.6, 44.0)[a]	18.1 (11.6, 24.6)	34.2 (26.6, 41.8)[b]	14.1 (7.3, 20.9)
2. Grand Forks, ND–Minn	453	Midwest	23.4 (17.9, 28.9)	38.1 (25.3, 39.9)[a]	13.2 (8.2, 18.6)	34.5 (25.3, 43.7)[b]	11.8 (6.9, 17.7)
3. Milwaukee–Waukesha, Wis	1112	Midwest	22.7 (19.4, 26.0)	32.6 (25.3, 39.9)[a]	18.3 (14.8, 21.8)	36.5 (30.9, 42.1)[b]	10.2 (7.6, 12.8)
4. Austin–San Marcos, Tex	510	South	22.6 (17.8, 27.4)	29.9 (21.0, 38.8)	18.2 (12.8, 23.6)	32.5 (24.8, 40.2)[b]	13.4 (7.4, 19.4)
5. Sioux Falls, SD	1314	Midwest	21.4 (18.8, 24.0)	35.9 (30.3, 41.5)[a]	14.2 (11.7, 16.7)	30.9 (26.5, 35.3)[b]	12.9 (10.0, 15.8)
6. Davenport–Moline–Rock Island, Iowa–Ill	421	Midwest	21.1 (14.8, 27.4)	28.4 (18.5, 38.3)	17.9 (9.7, 26.1)	29.4 (19.1, 39.7)	13.5 (6.8, 20.2)
7. Cedar Rapids, Iowa	404	Midwest	20.6 (15.7, 25.5)	36.2 (26.1, 46.3)[a]	13.8 (8.9, 18.7)	30.5 (22.3, 38.7)[b]	10.6 (6.5, 14.7)
8. Duluth–Superior, Minn–Wis	495	Midwest	20.3 (16.0, 24.6)	36.9 (26.7, 47.1)[a]	13.1 (9.5, 16.7)	28.9 (21.6, 36.2)[b]	13.6 (8.6, 18.6)
9. Lincoln, Neb	756	Midwest	20.1 (15.7, 24.5)	35.1 (27.9, 42.3)[a]	11.9 (6.3, 17.5)	29.4 (21.9, 36.9)[b]	11.0 (7.4, 14.6)
10. Springfield, Mass	819	Northeast	20.1 (15.3, 24.9)	44.2 (33.4, 55.0)[a]	9.5 (6.0, 13.0)	27.0 (19.1, 34.9)	14.2 (8.3, 20.1)
11. Detroit, Mich	1960	Midwest	20.0 (17.9, 22.1)	33.3 (28.8, 37.8)[a]	14.1 (11.9, 16.3)	31.0 (27.3, 34.7)[b]	10.2 (8.3, 12.1)
12. Reno, Nev	1531	West	20.0 (17.2, 22.8)	33.4 (27.2, 39.6)[a]	14.1 (11.2, 17.0)	29.1 (18.2, 40.0)[b]	10.9 (8.0, 13.8)
13. Spokane, Wash	529	West	19.6 (15.5, 23.7)	38.9 (30.0, 47.8)[a]	10.0 (6.6, 13.4)	32.8 (25.8, 29.8)[b]	7.5 (4.1, 10.9)
14. Burlington, Vt	1891	Northeast	19.6 (17.1,22.1)	35.2 (29.9, 40.5)[a]	10.6 (8.6, 12.6)	29.2 (25.2, 33.2)[b]	10.8 (7.9, 13.7)
15. Ft Worth–Arlington, Tex	535	South	19.4 (15.1, 23.7)	33.1 (24.2, 42.0)[a]	12.0 (8.0, 16.0)	31.1 (23.8, 38.4)[b]	8.0 (4.7, 11.3)
16. Las Vegas, Nev–Ariz	1893	West	18.7 (15.9, 21.5)	30.6 (24.0, 37.2)[a]	13.7 (11.2, 16.2)	27.6 (22.9, 32.3)[b]	9.8 (7.3, 12.3)
17. Bismarck, ND	543	Midwest	18.6 (15.2, 22.0)	29.6 (22.2, 37.0)[a]	13.9 (10.2, 17.6)	25.9 (20.1, 31.7)[b]	12.0 (7.8, 16.2)
18. Des Moines, Iowa	1088	Midwest	18.2 (15.5, 20.9)	33.0 (27.1, 38.9)[a]	11.5 (8.9, 15.1)	28.9 (24.1, 33.7)[b]	8.8 (6.2, 11.4)
19. Grand Rapids–Muskegon–Holland, Mich	550	Midwest	18.1 (14.6, 21.6)	30.4 (23.0, 37.8)[a]	11.9 (8.2, 15.6)	28.3 (22.2, 34.4)[b]	8.7 (5.4, 12.0)
20. Fargo–Moorhead, ND–Minn	814	Midwest	17.7 (14.6, 20.8)	28.8 (22.7, 34.9)[a]	11.4 (8.3, 14.5)	29.2 (23.4, 35.0)[b]	7.7 (5.0, 10.4)

[a]Difference of estimate for persons aged 18–34 years was statistically significant ($P < 0.01$) compared with persons aged 35 years and older.
[b]Estimate for men was statistically significantly different ($P < 0.01$) from estimate for women.

drinking in Austin–San Marcos, Tex, was the lowest among all 20 areas in the 18- to 34-year age group, which suggests that factors besides age distribution may account for higher estimates in this metropolitan area.

Other factors also influence local alcohol estimates. Sociocultural norms, such as religious beliefs, are likely to be influential.[6] For example, alcohol use is proscribed for Mormons, and many members of Southern Baptist churches abstain from alcohol. This may help to explain low binge-drinking estimates for metropolitan areas in Utah and in certain parts of the South. Country of origin, level of acculturation, alcohol availability, price, alcohol outlet density, and type and extent of alcohol-related legislation and level of enforcement (e.g., beverage service practices, drinking and driving laws) also may contribute to local variation of binge-drinking estimates.

Previous research suggests that there may be a distinct drinking culture, especially among males, in parts of the United States, where abstention rates are high and where alcohol is less widely available (so-called "dry" areas, e.g., parts of the South and the Rocky Mountain States) such that the proportion of current drinkers who binge drink in these areas may still be quite high.[6,31,32] We found some evidence of this, especially in Tennessee and Utah metropolitan areas, but even so, the proportion of alcohol users who binge drink was typically highest in areas with the highest prevalence of current alcohol use.

Overall, the BRFSS provides an efficient way to perform alcohol-related surveillance at the metropolitan level, and the local variation found in our study demonstrates the importance of conducting local analyses on an ongoing basis. The BRFSS uses a standardized methodology, relies on an existing infrastructure, and requires no new data collection, which results in cost savings for local health departments. Furthermore, BRFSS data on alcohol consumption, including binge drinking, has been collected annually (rather than in odd years only) since 2001. The increasing focus on binge drinking as an important public health problem, coupled with the growing demand for state and local surveillance data, underscores the need to further develop the BRFSS as a vital component of the public health infrastructure in the United States.

Our study has several limitations. The results probably underestimate the extent of binge drinking[6,33,34] with social desirability[35] and possibly with noncoverage (younger persons are less likely to have household telephones[36,37] and are more likely to drink alcohol[9,10]), which may have had some effect on our estimates.[35] Survey interview mode effects can affect estimates, although a study that compared BRFSS binge-drinking estimates with household survey estimates in Michigan found little difference.[38] Estimates for women may be conservative, because others have used "4 alcoholic beverages or more on 1 occasion in the past month" to define binge drinking for women.[2]

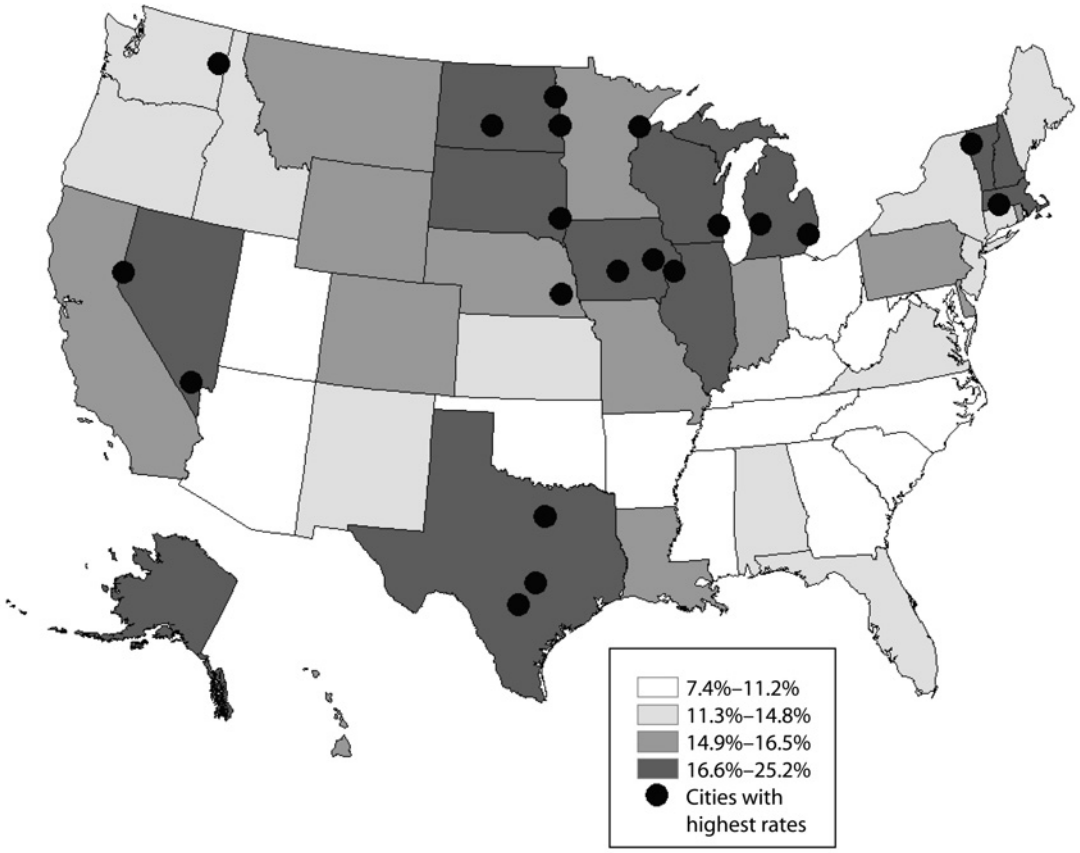

FIGURE 2—State binge-drinking estimates and the 20 US metropolitan areas with the highest binge-drinking levels: 1997 and 1999.

Differences by metropolitan area may be the result of variations in demographics other than age, race/ethnicity, and education, such as employment and social class. Furthermore, our estimates were for entire metropolitan areas, but binge drinking is likely to vary within individual areas as well (e.g., central cities vs suburbs). We were unable to assess response rates by metropolitan area, as all rates were calculated by state. Typical of other telephone surveys in the late 1990s, response rates declined over the study period, and the effect of this decline on our estimates is unknown.[39,40] Because we pooled data, we could not examine trends between 1997 and 1999; nevertheless, in spite of pooling, the number of respondents was small for certain areas and subpopulations, which reduced the precision of some estimates.

CONCLUSIONS

The adoption of public health measures is needed to address the problem of binge drinking. Effective measures include increasing alcohol excise taxes, enforcing the minimum drinking age, and developing comprehensive community-based programs that include education, enforcement, and community mobilization.[41,42] In addition, clinicians should screen all adult and adolescent patients for alcohol abuse in accordance with recommendations by the US Preventive Services Task Force.[43] Screening and brief intervention strategies for alcohol abuse, including binge drinking, are effective and can reduce costs in primary care settings.[44,45] Through this combination of interventions, binge drinking and its attendant health and social consequences can be prevented. ■

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Contributors

D.E. Nelson, T.S. Naimi, R.D. Brewer, and J. Bolen conceived and designed the study, interpretated the data, and wrote the article. H.E. Wells conducted the data analyses.

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TABLE 3—Odds Ratios and 95% Confidence Intervals for Binge Drinking, by Age, Sex, and Level of Education, for the 20 Metropolitan Areas With the Highest Levels of Binge Drinking[a]: 1997 and 1999

Metropolitan Area	Age Group, y 18–34[b]	Sex Men[c]	Level of Education ≤High School[d]
1. San Antonio, Tex	2.29 (1.23, 4.26)	3.31 (1.71, 6.43)	1.82 (1.00, 3.30)
2. Grand Forks, ND–Minn	4.02 (2.21, 7.34)	3.89 (2.08, 7.26)	1.01 (0.52, 1.97)
3. Milwaukee–Waukesha, Wis	2.44 (1.56, 3.81)	5.15 (3.51, 7.54)	0.87 (0.57, 1.32)
4. Austin–San Marcos, Tex	1.80 (0.97, 3.34)	3.15 (1.70, 5.84)	0.90 (0.48, 1.71)
5. Sioux Falls, SD	3.80 (2.69, 5.37)	3.07 (2.17, 4.34)	1.59 (1.12, 2.24)
6. Davenport–Moline–Rock Island, Iowa–Ill	2.19 (1.11, 4.34)	2.81 (1.31, 6.03)	2.18 (1.03, 4.61)
7. Cedar Rapids, Iowa	3.89 (2.08, 7.30)	4.04 (2.17, 7.53)	0.74 (0.41, 1.36)
8. Duluth–Superior, Minn–Wis	3.73 (2.12, 6.53)	2.65 (1.47, 4.78)	0.88 (0.49, 1.56)
9. Lincoln, Neb	4.38 (2.28, 8.40)	3.74 (2.21, 6.32)	1.12 (0.59, 2.10)
10. Springfield, Mass	8.21 (4.34, 15.51)	2.50 (1.34, 4.67)	0.80 (0.43, 1.50)
11. Detroit, Mich	3.21 (2.41, 4.27)	4.03 (3.06, 5.31)	0.98 (0.73, 1.31)
12. Reno, Nev	3.33 (2.24, 4.93)	3.42 (2.32, 5.03)	1.11 (0.76, 1.63)
13. Spokane, Wash	7.80 (4.32, 14.11)	8.11 (4.22, 15.58)	1.78 (1.02, 3.12)
14. Burlington, Vt	4.98 (3.66, 6.78)	3.61 (2.54, 5.12)	0.92 (0.65, 1.31)
15. Ft Worth–Arlington, Tex	3.18 (1.72, 5.89)	5.14 (2.83, 9.34)	1.30 (0.73, 2.29)
16. Las Vegas, Nev–Ariz	2.82 (1.91, 4.18)	3.69 (2.53, 5.39)	1.06 (0.73, 1.55)
17. Bismarck, ND	2.63 (1.61, 4.27)	2.69 (1.61, 4.50)	0.73 (0.44, 1.21)
18. Des Moines, Iowa	4.43 (3.00, 6.54)	4.81 (3.16, 7.32)	0.90 (0.60, 1.35)
19. Grand Rapids–Muskegon–Holland, Mich	3.99 (2.36, 6.75)	4.94 (2.88, 8.48)	1.14 (0.66, 1.96)
20. Fargo–Moorhead, ND–Minn	3.78 (2.41, 5.91)	5.22 (3.22, 8.48)	1.16 (0.71, 1.89)

[a]On the basis of logistic regression models that included age, sex, education level, and race/ethnicity as independent variables.
[b]Reference group = people 35 years old and older.
[c]Reference group = women.
[d]Reference group = more than high school.

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Human Participant Protection

No protocol or institutional review board approval was needed for this study, because data were collected anonymously (no individual identifiers) from a public health surveillance system in which adults voluntarily consented to telephone interviews.

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