than 40 per cent greater than it actually was.[14] It seems reasonable to anticipate a reversal in the climbing death rates from other smoking-related chronic diseases before the turn of the century.

Success in shifting patterns of behavior is impressive in itself. Often, health gains have been the result of unstructured responses to sometimes serendipitous developments in the science base. The fact that some of the recent gains have been fostered by carefully designed and managed efforts makes them even more noteworthy. Such progress offers hopeful prospects for managing further health gains for adults as well as children. Undetermined as yet is the extent to which gains have occurred among marginal populations, that is, among those most receptive to the requisite modifications in life style or behavior. In any case, an examination of these efforts to date, especially when coupled with the success of programs against diseases of childhood and infancy, reveals a series of health improvements that, in terms of productivity, would be the envy of any industrial manager.

The health gains for adults also have important demographic implications that should be studied in today's health-policy arenas. Perhaps of greatest urgency is the need to examine estimates of health-related costs. Many projections regarding retirement, Social Security outlays, the use of medical services, the need for nursing-home beds, and the costs of health care are based on assumptions of overstated mortality rates for various age groups. A mortality rate that is declining more rapidly could change these projections substantially.

Nursing-home care provides an illustration. It is currently projected as the health sector that will have the greatest growth in spending over the next quarter century — a projected increase of about 56 per cent, assuming constant mortality rates. But if a declining mortality rate (the rate of the past 10 years, for example) is factored in, the spending increase changes to 109 per cent.[15] Such increased costs might be offset by a narrowing of the period of morbidity until shortly before death,[16] but the need for careful assessment is apparent.

In summary, the proportionately greater gain recently recorded in this country in the life expectancy of adults relative to newborns represents an important reversal of the historical trend. It serves as a tribute to science's ability to identify health problems and develop solutions, but it also offers a challenge for us to apply our national resources effectively to capture the additional gains that are possible and to prepare for the demographic implications of increased life expectancy for adults.

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MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

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EXPOSURE TO LEAD FROM THE MYSTIC RIVER BRIDGE: THE DILEMMA OF DELEADING

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City dwellers in the United States are at risk of exposure to lead from numerous sources, including lead-based house paint,[1] employment in trades using lead,[2] airborne lead from automotive and industrial emissions,[3] contaminated food[4] and drinking water,[5] house

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dust,[6] soil,[7] and occasionally, pottery glazes.[8] Exposure from each of these sources is additive, and increased absorption of lead from any source reduces the already narrow margin between present-day background exposure and lead toxicity.[9]

Another, less well-recognized source of urban exposure to lead is the exterior paint applied to steel structures such as bridges, expressways, and elevated railways.[10,11] Unlike interior paint, which must now contain no more than 0.06 per cent lead by weight,[12] exterior paint may contain as much as 90 per cent lead. As such paint ages and cracks, rust may form beneath it, causing chips with a high lead content to flake to the ground, where they may be ingested by children.

Abatement of exposure to flaking exterior paint presents a public-health dilemma. Deterioration cannot be allowed to progress, and rusted exterior paint cannot simply be sealed in place with inert coatings. Consequently, abatement requires removal of the paint. However, removal substantially increases the risk that lead particles will be dispersed into the environment. These finely divided particulates can create a highly pervasive hazard of community exposure.

Public officials in Massachusetts confronted this dilemma in the case of the Mystic River Bridge, a high suspension bridge that connects the Charlestown section of Boston with Chelsea and passes over densely populated communities. A program for removal of paint from the bridge was undertaken after the discovery that lead-based paint was flaking into subjacent neighborhoods.

This report summarizes the environmental and epidemiologic data that delineate the lead-exposure hazards from the Mystic River Bridge. In anticipation that the need to remove lead-based paint from steel structures will arise elsewhere, we also outline recommendations for the safe removal of lead-based paint from exterior steel.

Background

Environmental studies conducted at the Mystic River Bridge in 1977 for the Massachusetts Port Authority found that surface soil directly beneath the bridge contained 1300 to 4800 parts per million (ppm) of lead[13]; the usual lead content of North American soils is below 100 ppm.[14] Analyses of trace elements indicated that the major source of the lead in soil was deteriorated lead-based paint from the bridge.[13] A concurrent pediatric survey found that 53 of 109 children (49 per cent) living in Chelsea in the blocks nearest the bridge had blood lead levels greater than or equal to 30 μg per deciliter,[15] the level considered by the Centers for Disease Control to indicate increased lead absorption in children[16]; by contrast, 30 of 82 more distantly located children (37 per cent) had elevated blood lead levels.

To reduce lead exposure under the bridge, the Port Authority initiated a program to remove lead-based paint from the structure.[15] The paint on the parts of the bridge that passed over neighborhoods was to be removed by abrasive blasting, and the parts were to be recoated with zinc primer. Deteriorated paint on the center span (over the river) was to be removed by scraping, and this section recoated with lead-based paint. Paint removal began in Charlestown in 1979.

In the abrasive-blasting process, grit was delivered to the bridge through high-pressure hoses from ground reservoirs; workers then sprayed the grit through nozzles at the surfaces to be cleaned. The workers wore positive pressure, air-supplied respirators. To minimize environmental emissions, each worker was enclosed in a movable booth attached to the bridge; the booths were sealed to the bridge with canvas shrouds and maintained under negative air pressure. Spent grit and paint chips were drawn downward through large-diameter hoses and trapped in cyclones and scrubbers.

Breaks in technique were noted during the abrasive blasting. Canvas shrouds were not always tightly sealed and occasionally fell to the ground; suction hoses occasionally developed leaks and became disconnected; at times spent grit and paint chips were allowed to accumulate beneath the bridge. On the center span, workers removed paint with mechanical scrapers and applied fresh lead-based paint with spray guns. Industrial dust masks were the only form of respiratory protection worn by workers on the center span.

Environmental Studies

To evaluate environmental emissions from the abrasive blasting, high-volume air samples were obtained in Charlestown in June 1980 by the Massachusetts Department of Environmental Quality Engineering at a site 27 m from the bridge. The mean lead content in four samples was 5.32 μg per cubic meter of air; the U.S. Environmental Protection Agency's standard for lead in community air is 1.5 μg per cubic meter (expressed as a three-month average).[17] The highest lead concentration in the air (12.9 μg per cubic meter) was measured on a day when a canvas shroud was not in place for two hours of a six-hour sampling period. Air-pollution controls were tightened after that sampling. In July, the mean lead concentration in air 12 m from the bridge was 1.43 μg per cubic meter (peak, 3.54 μg per cubic meter).

Samples of spent grit and of surface soil (the top 1 cm) were obtained in July 1980 in Charlestown within 30 m of the bridge; additional samples of surface soil were collected at 30 to 80 m and at 100 m. Comparison soil samples were obtained within 30 m of the bridge in Chelsea, where no abrasive blasting had occurred, at the Bunker Hill Monument in Charlestown, and at a rural area in Bedford, Mass.

Three samples of spent grit collected from open areas beneath the bridge had a mean lead concentration of 8127 ppm (range, 4080 to 12,900 ppm). In 10

samples of surface soil collected from within 30 m of the bridge in Charlestown, the mean lead content was 3272 ppm. The soil lead concentration decreased with distance to a mean of 457 ppm at 30 to 80 m, and to 197 ppm at 100 m. Soil samples from within 30 m of the bridge in Chelsea had a mean lead concentration of 1003 ppm. In samples from the Bunker Hill Monument, the soil lead concentration was 165 ppm, and in Bedford, 83 ppm.

Occupational Studies

To evaluate occupational lead exposures, the Division of Occupational Hygiene, Massachusetts Department of Labor and Industries, and the Occupational Health Program, Harvard School of Public Health, studied 13 abrasive-blasting workers and 19 workers on the center span.[18] Workers' exposures to airborne lead were measured in breathing-zone samples collected with personal sampling pumps; samples were analyzed for lead content by atomic absorption spectrophotometry. Medical evaluations consisted of an interview, physical examination, determination of blood lead, zinc protoporphyrin, hematocrit, hemoglobin, and blood urea nitrogen levels, and testing of neurobehavioral performance. Blood lead levels were analyzed by anodic stripping voltammetry on samples collected in lead-free tubes.

The highest air lead exposures were found in the vicinity of the abrasive-blasting enclosures (range, 10 to 1090 μg per cubic meter) and among workers performing the scrape-and-prime operation on the center span (24 to 1017 μg per cubic meter). Lower exposures were found in helpers (256 to 386 μg per cubic meter) and a vacuum-truck operator (46 μg per cubic meter) on the abrasive-blasting job and in a foreman (38 μg per cubic meter), a mid-coat painter (30 μg per cubic meter), and final-coat painters (6 to 9 μg per cubic meter) on the center span.

Blood lead levels were highest in workers on the center span (mean, 61.2 μg per deciliter; range, 30 to 96 μg per deciliter); 11 center-span workers (58 per cent) had blood lead concentrations over 60 μg per deciliter — the level considered by the National Institute for Occupational Safety and Health to represent excessive lead absorption in workers.[19] Abrasive-blasting workers had lower blood lead levels (mean, 33.2 μg per deciliter; range, 25 to 47 μg per deciliter).

Symptoms of lead toxicity were common in workers on the center span: tiredness (37 per cent vs. 8 per cent of abrasive-blasting workers), memory problems (26 vs. 15 per cent), and sleep disturbances (21 vs. 8 per cent). Four of the center-span workers (21 per cent) reported two or more symptoms indicative of central-nervous-system dysfunction; no abrasive-blasting workers reported such symptoms. Four workers were identified as having mild lead-related anemia (hemoglobin levels of 12.4 to 13.9 g per deciliter). No kidney disease was identified. No differences were noted in neuropsychological performance between groups with different levels of lead exposure.

Community Studies

To evaluate possible community exposures to lead, fingertip blood samples were obtained by the Boston Childhood Lead Poisoning Prevention Program, City of Boston Department of Health and Hospitals, with assistance from the Centers for Disease Control, from 123 children one to five years old living in Charlestown within 0.3 km of the bridge.[20,21] Samples collected at the Bunker Hill Health Center and by door-to-door visits were analyzed by atomic absorption spectrophotometry. Dust samples were collected during home visits and were also analyzed for lead content by atomic absorption spectrophotometry. Houses of children found to have blood lead levels greater than 30 μg per deciliter were evaluated by x-ray fluorescence for interior sources of lead-based paint.

Four children (3.3 per cent) had blood lead levels above 30 μg per decliter; the highest value was 35 μg per deciliter. All four lived within two blocks of the bridge. Two (who were siblings) were found to have lead-based paint in their home; however, the paint had not deteriorated, and no teeth marks were evident. No lead-based paint was found in the homes of the other two children. Within two blocks of the bridge blood lead levels above 30 μg per deciliter were present in four of 47 children. Beyond that distance, none of 76 children had elevated levels — a significant difference ($P = 0.0192$ by Fisher's exact test). The mean lead content of four house-dust samples from within one block of the bridge was 7580 ppm (range, 4930 to 10,000 ppm); the mean level in five samples from one to three blocks of the bridge was 628 ppm — another significant difference ($P = 0.0002$).

Conclusions

The environmental data collected in this investigation indicate that the air, surface soil, and household dust near the Mystic River Bridge are heavily contaminated with lead. These high concentrations appeared to reflect contributions from abrasive blasting, flaking of deteriorated lead-based paint from the bridge, and automotive exhaust. Although the precise contribution from particulates released by abrasive blasting is not known, blasting appears to account for a considerable portion of the 2000-ppm difference in soil lead concentrations found between the Charlestown and Chelsea samples. In effect, abrasive blasting accelerated the process of environmental dissemination of lead due to paint flaking from the bridge.

The medical data indicate that lead particulates formed during the paint removal and repainting of the Mystic River Bridge caused excessive lead absorption and lead toxicity among workers; the center-span workers were most seriously affected.[18] The data also indicate that significantly greater absorption of lead has occurred in children living closer to the bridge

than in those at greater distances.[20] It appears likely that both automotive emissions and particulates from the abrasive blasting have contributed to the children's lead absorption, but the data do not permit definition of the precise contribution from each source.

Although these data document that the initial, insufficiently cautious removal of lead-based paint from the Mystic River Bridge resulted in serious occupational and environmental exposures to lead, it is noteworthy that reductions in lead concentrations in air were achieved between June and July 1980 by proper application of air-pollution controls. That observation suggests that with proper supervision lead-based paint can be safely removed from the bridge.

Recommendations

On the basis of these findings, the Massachusetts Department of Public Health and the Centers for Disease Control in concert with the National Institute for Occupational Safety and Health recommended that removal of lead-based paint from the Mystic River Bridge continue, since removal would eliminate a major source of lead exposure in the Boston community. However, it was stipulated (with concurrence by the Massachusetts Port Authority) that removal must be conducted with utmost attention to environmental controls. To do otherwise would defeat the purpose of paint removal.

Detailed recommendations were developed for environmental monitoring and control. They included stipulations on air sampling, shrouding, maintenance of equipment, proper storage of spent grit, and daily wet sweeping of areas beneath the bridge. Additional guidelines were developed for worker protection; workers will be afforded the same safeguards against lead absorption and toxicity as those provided to workers in general industry under the Occupational Safety and Health Administration's lead standard.[22] For the protection of community residents, it was stipulated that all persons living within 30 m of the bridge should be warned of the dangers of lead exposure[23] and that they be offered the chance for determination of base-line blood lead and erythrocyte protoporphyrin levels before the start of the next cycle of abrasive blasting. In addition, it was recommended that all children under 17 years of age and all pregnant women living within 30 m of the bridge should have monthly blood lead and zinc protoporphyrin determinations throughout the next cycle of paint removal, that anyone found to have blood lead levels greater than 30 μg per deciliter should be referred immediately for medical evaluation, and that careful consideration should be given to the possibility of removing young children and pregnant women from areas within 30 m of the bridge during future paint-removal operations. Finally, it was determined that removal of paint from the bridge by abrasive blasting be halted if air lead levels adjacent to the process cannot be controlled, or if increases occur in community residents' blood lead levels.

In cities across the United States, there are thousands of bridges and other steel structures coated with lead-based exterior paint. The potential for serious occupational and environmental exposures to lead during future work on these structures is large. Ultimate control of the hazard of lead-based exterior paint will be achieved through the development of safe, efficient alternative coating materials.

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