decent living conditions for people. It is reasonable to ask whether we can afford *not* to mount this effort.

In 1963, when the executive secretary of the World Health Organization was approached with a plan to eradicate smallpox, he scoffed. Because he believed such an attempt would be doomed to failure, he requested that an American be asked to direct the project and thus take the heat for its failure. Dr. Donald Henderson and CDC accepted the challenge, and today smallpox is a disease for the history books. Lead poisoning, this silent, relentless destroyer of brain cells, can have the same fate if we have the same kind of vision. \Box

Herbert L. Needleman, MD

References

1. Weisel C, Demak M, Marcus S, Goldstein BD. Soft plastic bread packaging: lead content and reuse by families. Am J Public Health 1991; 81:756–758.

- Needleman HL, Gatsonis C. Low-level lead exposure and the IQ of children: A meta-analysis of modern studies. JAMA 1990; 263:673–678.
- Brown RS, Hingerty BE, Dewan JC, Klug A. Pb(II)-catalyzed cleavage of the sugarphosphate backbone of yeast tRNA(Phe)implications for lead toxicity and self-splicing RNA. Nature 1983; 303:543–546.
- 4. Bailey C, Kitchen I. Ontogenesis of proenkephalin products in rat striatum and the inhibitory effects of low-level lead exposure. Dev Brain Res 1985; 22:75–79.
- Marcovac J, Goldstein GW. Picomolar concentrations of lead stimulate brain protein kinase G. Nature 1988; 334:71–73.
- Rice D, Gilbert SG. Low-level lifetime lead exposure produces behavioral toxicity (spatial discrimination reversal) in adult monkeys. Toxicol. Appl Pharmacol 91:484–490.
- US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. *The nature and extent of lead poisoning in children in the United States: A report to Congress.* Washington, DC: Govt Printing Office, 1988.

- 8. Centers for Disease Control. *Strategic Plan for the Elimination of Childhood Lead Poisoning.* Department of Health and Human Services, February 1991.
- Rosner D, Markowitz G. A "gift of God"?: The public health controversy over leaded gasoline during the 1920's. Am J Public Health 1985; 75:344–352.
- Rabin R. Warnings unheeded: A history of child lead poisoning. Am J Public Health 1989; 79:1668–1674.
- 11. United States General Accounting Office. *HUD not fulfilling responsibility to eliminate lead-based paint hazard in Federal housing.* Report by the Comptroller General. Pub. No. CED-81-31. GAO, 1980 Dec 16.
- "Administration planning only modest spending to curb lead poisoning" by Philip J. Hilts. *The New York Times*; p. A.12, February 21, 1991.

Requests for reprints should be sent to Herbert L. Needleman, MD, University of Pittsburgh, School of Medicine, Suite 305, Iroquois Building, 3600 Forbes Avenue, Pittsburgh, PA 15213.

Can Stress Cause Cancer?

In this issue of the journal Hatch and her colleagues1 report on an examination of the time and space relationship between local excess cancer rates and the highly publicized episode of radiation release during the Three Mile Island nuclear accident of 1979. The ecological correlation between the accident that caused environmental release of what appears to have been a small amount of radiation into the atmosphere and a subsequent rise in nearby cancer rates is important in several direct and indirect ways. The difficulty that the investigators faced in developing the explanation for the increase in cancer rates is typical and illustrative of the problems frequently faced by epidemiologists and their collaborators. The problem has become larger during what might be termed the "environmental era" of the last few decades.

The scientific problems posed by this era of heightened concern about the environment are far-reaching. The issues involve sociology, psychology, biology, physics, and medicine as well as principles of politics and economics. Our inadequate understanding of the etiology of cancer specifically and of the relationship between environmental change and human and biological well-being in general suggests that it would be unwise to expect a simple or quick alleviation of our environmental anxieties. I believe that the important article by Hatch et al. illustrates this point very well.

Some realities about contemporary environmental problems and disasters are becoming apparent. It is essential to consider these realities so that a modern concept of environmental studies for public health practitioners can be developed. The large size of the human population on earth makes it impossible to simply revert to a time we may think of as cleaner and less threatening to our health. Admittedly, we may have reason to wonder whether there ever was a time when environmental circumstances were, overall, better than they are now for human health. There can be no doubt, however, that the scientific advances that have provided the opportunity for better human health are not distributed efficiently and equitably among human beings. Nor can anyone deny that the advances of civilization, for example, better sanitation, higher food productivity, artificial heat and light, transportation, and communication, have been achieved at a significant environmental cost. The important question that we have not yet adequately addressed concerns the optimum balance point where we get the greatest benefit at the least cost.

It is often thought that the beginning of the contemporary environmental era was signaled by Rachel Carson's book *Si*-

lent Spring. Major events since that time have become milestones of environmental concern (Love Canal, Times Beach, Missouri, Three Mile Island, and Chernobyl). More recently, attention has focused on the issue of induced changes in the earth's atmospheric envelope, which appear to be leading to global warming thus creating the potential for massive environmental change. Any one of these issues, as well as the others not mentioned, provide the questions and scientific challenges that concern the future of public health in the United States and around the earth. At present, despite noteworthy advances during the last few decades, public health sciences and epidemiology have not been able to provide answers that are satisfactory to the public. Are we now going to be faced with yet another new problem, namely, does the publicity surrounding environmental problems constitute a public health threat because of the stress it can produce? This possibility is worrisome because there are many who think that strong publicity is one way to help prevent similar problems from happening in the future.

There are several other important implications of the report by Hatch et al. The increase in cancer rates in the vicinity of the Three Mile Island accidents meets the American Public Health Association's definition of an epidemic,² "the occur-

Editorials

rence in the community . . .¹ of a group of illnesses of similar nature, clearly in excess of normal expectation." The attribution of this particular epidemic to the adverse effect of postaccident stress provides new and potentially important insights into the etiology of cancer and into the circumstances that may eventually need to be managed to better protect the public health. The idea that stress may be an environmental culprit could change many aspects of the way we handle acute environmental problems like the Three Mile Island accident. The way such environmental events are publicized and managed may need to be reconsidered if further evidence supports the conclusion that stress can cause or promote a disease like cancer. Studies of cancer incidence at the Love Canal before the problem was publicized in 1978 provided no clear evidence of increased cancer risks even though chronic exposure to environmental toxins had taken place over years and decades.3 However, since the entire neighborhood surrounding the Love Canal was relocated at the time the problem of toxic exposure was publicized in 1978, it has not been possible to determine whether the stress associated with publicity surrounding that event might have lead to increased cancer rates in the years after 1978.

It will be important for public health officials to devise expanded research strategies for past and future environmental episodes that consider every plausible hypothesis for disease etiology. Latency periods prior to clinically apparent cancer may be very long. The promotion of already initiated cancer cells, through a stress-induced neuroendocrine mechanism, is not a far-fetched idea. Parenthetically, we should remember that only three or four decades ago many doubted that tobacco smoke could cause lung cancer and that the environmental pollution during an intense London fog in 1952 could have been responsible for some 4,000 deaths. As we consider the plausibility of the stress explanation in the report by Hatch et al., it is important to observe that a temporary promotion of cancer should lead to a short period of increased risk, followed by a compensatory period of decreased risk. The decreased risk occurs because new cancers are not initiated by a promotional event. Promotion can only hasten the progression of an existing cancer to the stage when the disease becomes clinically apparent. Perhaps mortality rates for cancer will need to be added to such studies.

The theories of initiation and progression of cancer are complex, but in general, a promotional event will not produce new cancers, although it may appear to do so in the short run. The data presented by the authors are consistent with such a pattern. As the authors clearly recognize, it is also difficult to rule out the possibility that increased clinical surveillance, perhaps stimulated by the same anxiety that produced the stress, could produce a shortterm increase followed by a compensatory decrease in cancer cases diagnosed among the study population. The fact that the initial increase did not occur until three years after the nuclear accident lends more support to the promotion hypothesis than the increase-in-clinical-surveillance hypothesis as the explanation for this phenomenon. Whatever the final explanation for this cancer cluster following the Three Mile Island nuclear accident, the cluster underscores the need for more epidemiologically driven studies of major and minor environmental accidents. The investigators who conduct such studies should keep an open mind to the potential etiological mechanisms by which the circumstances around us may cause human diseases, including cancer.

Dwight T. Janerich, DDS, MPH

References

- Hatch MC, Wallenstein S, Beyea J, Nieves JW, Susser, M. Cancer rates after the Three Mile Island nuclear accident and proximity of residence to the plant. Am J Public Health 1991; 81:719–724.
- American Public Health Association. The Control of Communicable Diseases in Man (11th ed.). New York: American Public Health Association, 1970. p. 289.
- Janerich DT, Burnett WS, Feck G, Hoff M, Nasca P, Polednak AP, Greenwald P, Vianna N. Cancer Incidence in the Love Canal Area. Science 1981; 212(June 19):1404– 1407.

Address reprint requests to: Dwight T. Janerich, DDS, MPH, Professor of Epidemiology, Department of Epidemiology and Public Health, Yale University School of Medicine, 60 College Street, New Haven, CT 06510.

Erratum

In: Kahn HS, Williamson DF, Stevens JA: Race and weight change in US women: the roles of socioeconomic and marital status. Am J Public Health 1991;81:319–323.

The abstract was improperly copy edited by staff, resulting in the deletion of some important information in the Results section. The Results section of the abstract should read as follows:

"After multiple adjustments, Black race, education below college level, and becoming married during the follow-up interval were each independently associated with an increased mean weight change. Using multivariate logistic analyses, Black race was not independently associated with an increased risk of major weight gain (change $\geq +13$ kg), but it was associated with a reduced likelihood of major weight loss (change ≤ -7 kg) (odds ratio = 0.64 [95% CI = 0.41, 0.97]). Very low family income was independently associated with the likelihood of both major weight gain (OR = 1.71 [95% CI = 1.15, 2.55]) and major weight loss (OR = 1.86 [95% CI = 1.18, 2.95])."

The Journal regrets the error.