Commentary

*Environmental Health Perspectives*
*Vol. 48, pp. 105-106, 1983*

# Clinical and Epidemiological Evaluation of Health Effects in Potentially Affected Populations

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It is tempting to regard the problem of investigation of health hazards associated with toxic chemical wastes as almost insoluble at present. Exposures are poorly defined, disease patterns are not well identified, the relation of effects in other biological systems to illness in man is not understood. Neither the "dose" nor the response has been established and certainly not quantitatively. Pessimism can be augmented with the proposition that generalizations are inappropriate.

> Although analogies can be drawn among waste sites, chemical, geological, or medical generalizations not based on detailed site-specific analyses should always be considered unreliable (*1*).

It would be highly desirable if it were possible to secure analyses of every dump site and all details and all combinations over time. When the biochemists and the analytical chemists complete their work, we would then call in the hydrologists to provide patterns for us.

That will be an ideal world. But I am reminded, for example, that Japanese clinicians knew something was wrong at Minamata Bay long before we were able to measure methylmercury, and I think that Dr. Epstein in London worked at least a dozen years after Burkett, to find the Epstein-Barr virus and its possible relationship to lymphomas described in East Africa. I am reminded that long before we knew of nickel carcinogenicity a small town practitioner in South Wales identified the hazard. He saw a patient with sinus cancer, and saw a second one three months later. He was struck by the fact that both patients worked in the local nickel smelter. He thought it perhaps too much of a coincidence for such a rare tumor to occur randomly in two nickel smelter workers and proposed an etiological association. We still don't know exactly what it is in nickel smelters that produces sinus or lung cancer, but we know that it occurs. It is only in the last half dozen years that Blumberg and others have begun to clarify the relationship of hepatitis B virus to the extraordinary incidence of liver cancer in China and Hong Kong and South East Asia, East Africa, India.

Thus, epidemiological hunches have turned out to be very useful. However, by and large it has been planned epidemiological studies that have provided the best information concerning environmenl hazards. I am not sure we all appreciate how recent this is. We assume (correctly) that we can investigate the disease potential of environmental contamination, that it should be studied, that exogenous agents can produce disease. But such confidence, such acceptance, is rather new. How recent? Richard Doll writes (*2*):

> In 1952 . . . many people still believed that cancer was a degenerative process inextricably confounded with aging, that most cancers were, in consequence, unavoidable. . . . Sixteen years later . . . it was generally accepted that most cancers were attributable to extrinsic causes and that at least 4 out of 5 were preventable. . . .

Those 16 years were very productive. Epidemiological methods were developed that gave us both valuable data and understanding.

We are now going to have to go beyond that, however. Either we are going to have to change the toxic chemical waste dump sites or we are going to have to change epidemiology. And we can. We can consider approaches such as sero-epidemiology, biochemical epidemiology, epidemiological immunotoxicology. These are areas I believe we will be investigating in the next years. Concomitantly, I am not sure that we have fully utilized the approaches which in those 16 years gave us our understanding. They were in considerable part in two areas, the first, being large-scale prospective population stud-

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ies. The classic ones, of course, were those at the American Cancer Society by Hammond and his colleagues and Doll and his colleagues in Britain; the first of over a million people in the United States and the second of British doctors. These were prospective, with well-defined large populations. The second approach was to study the experience of heavily exposed groups, generally occupationally exposed, to determine what the biological endpoints might be, and to obtain some sense of dose-response relatnships, so that at least an effort at extrapolation could be made. I suspect that exploitation of what those 16 years showed to be effective might now very well be in order.

The first two papers for this session will review how we might apply our proven methodology. I am not sure that the designs can be worked out. I am not sure that it will be done with ease, but at least we can attempt to utilize what has been demonstrated to be effective. The first paper will examine the question whether it is possible to obtain direct epidemiologic evidence of effects of chemical contamination of public water supplies. Dr. Hammond of Mount Sinai and our Chinese colleagues from the Peking Institute of Cancer Research in China (Dr. You and Dr. Wang) have been studying this question for the last year. Dr. Lilis will review the potential of the second approach—study of an occupational group intimately working with toxic chemical wastes. We will approximate the ideal that Dr. Landrigan correctly addressed. The exposures will be uniquely monitored; there will be continuing analysis of all toxic wastes and recurrent biological monitoring of the work force of 400 to 500 people exposed to these known toxic chemicals.

An example of new approaches to biological monitoring will conclude these contributions, as Dr. Thomas Clarkson reviews an advance in the investigation of low-level environmental health effects from cadmium, lead, and mercury.

## REFERENCES

1. Lawrence, W. E. (Ed.). Assessment of Health Effects at Chemical Disposal Sites. Proceedings of a symposium, New York, N.Y., June 1-2, 1981.
2. Doll, R. Avoidable cancer: attribution of risk. S. Afr. Cancer Bull. 25(3): 125-146 (1981).