

Identification of Sentinel Health Events As Indicators of Environmental Contamination *

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The consensus process was applied to addressing a public health topic; this was a novel endeavor. The following question was addressed: What role, if any, should sentinel health events play in the decision-making process for identifying the effects of environmental exposure? The panel developed three levels of sentinel health events lists: those that are clearly identifiable, those that are potential signs, and those that are indicators of body burdens. Additionally, the panel developed several salient statements regarding the principles of environmental health surveillance and, especially, recommendations for future research.

Introduction

During October 30–November 1, 1989, a panel of individuals skilled in a variety of environmental health fields met in Asheville, North Carolina. Each of the panelists has a professional interest in preventing the adverse human health effects that result from environmental contamination. The objective of this meeting was to examine critically the use of sentinel health events as an approach for improving the capability for public health response. This conference was designed as a sequel to the 10th Biraud-MacJannet Seminar in Veyrier-du-Lac, France (May 4–6, 1988), which revealed among other things the inadequacies of traditional health reporting systems.

The Panel was asked to address four questions and believes that this paper is generally responsive to those questions. The questions were:

a) What role, if any, should sentinel health events (SHEs) play in the decision-making process for identifying the effects of environmental exposure? Response: A well-defined of environmentally related SHEs should provide decision-makers with a set of human health indices. These events could signal the need to initiate actions to prevent health impairment from related environmental exposures.

b) What criteria should be used in identifying such events? Response: Criteria are spelled out in "Methods."

c) How should the occurrence of sentinel health events be brought to the attention of the public health system? Specifically,

how should these events be listed? How should these events be reported? Response: A response directly to how events should be listed is given in "Conclusions." For how to report events, the Panel recommends that a follow-up conference be held to address the specific use of the SHEs proposed in this paper. The Panel further recommends that these conclusions be brought to the attention of industrial managers, public health agency staff and directors, affected or potentially affected citizens, advocacy groups, political leaders, professional societies, and educators.

d) What direction should future research take to improve current knowledge in this area? Response: Research is needed to improve our ability to define specific preclinical illnesses, early biological effects, and exposure doses. Specific biomarkers that identify exposure dose and early effects would greatly advance the field, as would an ability to discriminate between degrees of host susceptibility, perhaps by means of genetic markers. Finally, demonstration projects could help define the effectiveness of various types of action for specific SHEs.

Methods

In 3 days of deliberations, the Panel addressed the following issues: a) the role of sentinel health events or some other measure of exposure or health outcome that would improve the decision-making process for identifying the human health effects that may result from environmental contamination, and b) how such a list of events or measures can be collected and used by the public health community.

The Panel accepted a definition of a sentinel health event: a preventable disease, disability, or untimely death whose occurrence serves as a warning signal. The occurrence of an SHE is to be considered an indication that an environmentally induced health effect may have occurred and implies that some action should be considered. That action may consist of further investigation or analysis, or it may involve a reduction or cessation

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of exposures, depending on the circumstances.

The Panel derived its formulation of sentinel health events from concepts articulated by Rutstein et al. (1). That work, however, focused exclusively on occupational exposures and on individual cases with specific and preventable etiologies. In adapting these concepts to general environmental settings, the Panel emphasized nonoccupational exposures and extended the idea of single-case sentinel health events to include certain patterns of health effects and chemical body burdens within populations that may suggest environmental origins.

The Panel decided *a*) not to include occupationally related sentinel health events that are reported elsewhere; *b*) not to consider indices of contamination of the ambient environment as such. This exclusion applies to effects found in animals, fish, and vegetation; *c*) to provide a list of events or measures that is not exhaustive but may be suggestive of criteria that can be used to select such measures; *d*) to include events or possible reactions regardless of whether data systems are currently available to collect such information; and *e*) not to address the role of smoking, drinking, drug use, or radiation, either alone or as factors that aggravate the effects of environmental exposures. These considerations were considered to be beyond the scope of the Panel's deliberations.

The Panel was concerned with the extent to which environmental contamination may cause adverse health effects in humans. Answering this question is greatly complicated by *a*) our present ability to define potential exposure precisely or to measure doses accurately; *b*) the long and variable latency periods for effects associated with most environmental exposures; *c*) the relative rarity of most environmentally related diseases; *d*) the nonspecificity of most potential outcomes. Only rarely can one attribute delayed health events to a particular exposure strictly on the basis of clinical features; *e*) the possibility that multiple factors may interact; *f*) and inadequate health data systems.

In the face of these uncertainties an urgent need exists for systematic guidance by which an association between environmental exposures and health outcomes can be evaluated. This guidance can be served by surveillance of SHEs with the objective of avoiding unnecessary disease, disability, and premature death consequent to environmental exposures. Ideally, SHEs would be specific for particular environmental exposures; occur shortly after exposure; identified through simple, inexpensive, and well-standardized methods; identified through existing health surveillance mechanisms such as vital records, hospital discharge data, disease registries; and capable of leading to preventive action.

The first category of indicators identified by the Panel refers to individual events that may occur in only one person but may also affect many exposed individuals. The latter categories reflect events that may affect whole populations.

Conclusions

Category I. Clearly Identifiable, Environmentally Caused SHEs

The Panel considered a wide range of potential SHEs, none of which fit the entire set of ideal criteria for this category (specificity, short latency, ease of identification). Examples that most

closely fit the criteria are described below and include several acute environmental exposures and two cancers.

Poisoning (pesticides, toxic gases, heavy metals, solvents, chemical spills). Poisonings produce short latency effects from high-dose exposures, but some have long-term effects. In community settings, they may signal wider exposures that require public health action. They are identifiable through medical record sources (hospitals, emergency rooms, poison control centers, physician notifications).

Methemoglobinemia. Methemoglobinemia is subacute or chronic condition, usually manifest in infants, resulting from excessive nitrate levels in drinking water. Public health identification requires access to medical records in hospitals or through physicians.

Mesothelioma. Mesothelioma is a rare form of cancer of the lining membranes of the thoracic or abdominal cavity and is largely specific for asbestos exposure, independent of cigarette smoking, and sometimes arises in settings of nonoccupational exposure where the degree of exposure may not be extensive. The tumor can be identified through existing tumor registries and medical records, although not very reliably through death certificates. Latency usually exceeds 20 years.

Hepatic angiosarcoma. Hepatic angiosarcoma is a rare liver cancer in humans that is attributable to a considerable degree to vinyl chloride monomer exposure. No cases of angiosarcoma have yet been attributed unequivocally to nonoccupational, environmental contamination with vinyl chloride. However, any case of angiosarcoma should alert public health officials and health care providers to a possible environmental source, which may include arsenic and thorium in addition to vinyl chloride. Angiosarcoma can be identified with some reliability in tumor registries and through medical record systems but not in vital records. Latency is long (10–30 years).

The Panel also discussed SHEs in a broader environmental context. This extended beyond chemical contaminants and encompassed physical environmental stresses such as noise and extreme weather conditions, as well as certain infectious diseases, commonly food or waterborne. Although such considerations were beyond the specific focus of the Panel, the continuing need for public health departments to address environmental concerns over a wide spectrum of etiologies should be recognized.

Category II. Potential Signs of Population Exposure to Environmental Contaminants

The adverse health events listed below are not necessarily attributable to environmental contamination because they occur at some background rate or level in the general population among persons with little exposure to environmental pollutants. However, when these events occur in a population at a higher rate than expected, the excess should serve as a warning sign that environmental exposures may account for some of the excess. To serve as warning signs, therefore, the rates of these events must be compared in both exposed and nonexposed groups or in a population before and after the introduction of an environmental contaminant.

The occurrence of one of these events in an individual is not an SHE. But an excess frequency of one of these events in a population may be a useful indicator of undue exposure to environmental contamination.

These events may be divided into three groups based on the accessibility of the data (Table 1). Responses identified in lists B and C of Table 1 include preclinical indicators of adverse health effects, and as such, may be among the most sensitive and early responses to environmental contamination. They may be particularly useful in studies that compare baseline or pre-exposure, conditions with postexposure changes, and in studies that seek to determine whether subtle effects have been induced in a population by the introduction of a new source of environmental contamination, such as a hazardous waste treatment facility. They require special surveys because such data are not routinely obtained in clinical settings. It must be remembered that measurements of biologic function require standardized methods.

Category III. Indicators of Body Burdens Potentially Due to Environmental Exposures

The Panel identified several conditions for which measurements demonstrate that environmental exposures have occurred, whether or not these could lead to explicit illnesses. In general, these conditions imply some biochemical handicap and have been designated by the term "body burden." These will not

Table 1. Potential signs of population exposure to environmental contaminants.

Disease	Source ^a
A. Diseases identifiable through existing health reporting systems	
Low birth weight	Vital statistics
Birth defects	Vital statistics, hospital discharges, birth defect registries
Spontaneous abortions	Hospital discharges
Chronic respiratory disease in children	Hospital discharges
Active leukemia in children	Cancer registries, vital statistics, hospital discharges
Acute granulocytic leukemia in adults	Cancer registries, vital statistics, hospital discharges
Aplastic anemia	Hospital discharges, vital statistics
Asthma in children	Hospital discharges
Dermatitis and dermatoses	Hospital discharges
Skin cancer	Cancer registries, hospital discharges
Malignant melanoma	Cancer registries, hospital discharges, vital statistics
Lung cancer in nonsmokers	Cancer registries
Bladder cancer in nonsmokers	Cancer registries
Primary liver cancer in nondrinkers	Cancer registries
B. Diseases/defects not usually identifiable through existing health reporting systems	
Acute sensory irritation (eye, respiratory, olfactory)	
Developmental defects	
Hearing loss in children	
Chromosome defects	
C. Deviation from normal biological functions requiring special surveys to detect	
Neurological function	
Immunological function	
Renal function	
Cardiac function	
Hematologic function	
Respiratory function	
Reproductive function	
Liver function	
Auditory function	

^aExpected sources for information about the diseases; sources may not exist in all communities and may not contain desired information.

Table 2. Indicators of body burdens potentially due to environmental exposures.

Blood lead (ZPT) ^a
Heavy metals in blood, urine, hair, nails
Carboxyhemoglobin
Organophosphates (cholinesterase) ^b
PCBs and PBBs (polychlorinated and polybrominated biphenyls)
Other pesticides
Adducts ^c

^aZPT, zinc protoporphyrin, an easily measured metabolite involved with the structural materials of hemoglobin. Lead impairs the use of ZPT and hence elevated ZPT in screening tests indicates a likelihood of a lead body burden.

^bCholinesterase is an enzyme that is specifically blocked by organophosphate pesticides, and a decrease in cholinesterase may reflect a relatively recent exposure to such pesticides.

^cAdducts are combinations of pollutants with one or more molecules in the body, such as DNA or hemoglobin, which tend to persist and can be detected at very low concentrations.

usually be available from ordinary health data sources but will require special surveys directed at specific hypotheses (Table 2). An example might be a person with a blood lead level of 25 $\mu\text{g}/100\text{ g}$, when unexposed persons have 12 $\mu\text{g}/100\text{ g}$. The exposed person is said to have a body burden of lead. This third category is used to reflect the occurrence of an environmental exposure and the absorption of a detectable dose by an individual. A single occurrence could serve as an SHE.

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