

ENVIRONMENTAL HEALTH INDICATORS

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ABSTRACT

The issue of environmental health began more than a century ago, and the problem has continued to worsen over the years, in part because of the chemicals that have been introduced by modern technology. Studying environmental health, however, is a difficult procedure because of the complications involved with accurately measuring an individual's exposure to a certain contaminant. For example, the toxicity of a substance is influenced by absorption, distribution, elimination, and accumulation within the body, the pathway of entry, the stability of the chemical, and synergistic interactions with other substances. Monitoring environmental health effects is also complicated by the long latency period involved with many diseases, multiple causes of the same effect, and the cost of conducting large studies.

Environmental health indicators, measures that provide information about patterns or trends of specific diseases, can be an effective means of assessing environmental health, depending on the availability of and feasibility of collecting pertinent information. Data already collected by the Rhode Island Department of Health, such as birth and death records, lead screening data, and hospital discharge data, in combination with additional data that can be collected, would provide a sound basis for the development of a number of environmental health indicators.

After investigating the health data collected in Rhode Island and researching the background data of a group of selected diseases, I made recommendations regarding what additional data would be useful, and how this information could be gathered.

The State data regarding the incidence of asthma, a lung disease characterized by shortness of breath resulting from hyperresponsiveness to a variety of stimuli, are based on hospital discharge records. Additional information about less severe cases could be gathered from physicians by defining asthma as a reportable disease, or by implementing a sentinel or electronic reporting system. Another possibility would be to monitor the sale of asthma medication either through the number of prescriptions filled at pharmacies or through inventories taken at various drug companies.

Data on melanomas, the most severe type of skin cancer, are collected in the State Cancer Registry. In addition to knowing the rate of the disease, it would be useful to evaluate the efficacy of prevention programs and educational efforts. This could be done by monitoring the sale of sunscreen through inventories at drug stores or by assessing its use by incorporating questions into health surveys.

Data regarding lung disease, a term that encompasses a variety of respiratory problems, are obtained from hospital discharge records and data regarding lung cancer are obtained from the Cancer Registry. More specific information regarding the various causes of these diseases can be obtained in a variety of ways. Examination of chest X-rays can be used to evaluate asbestos exposure; the rate of sales of radon testing kits can indicate the success of prevention and public education; and the level of cotinine, a chemical associated with second hand smoke, in donated blood can be used to estimate exposure to environmental tobacco smoke.

The state maintains a lead screening database that contains information about blood lead levels in children. In addition to this, the concentration of lead in dust samples taken from homes could be one means of evaluating exposure.

X-ray fluorescence could be used to evaluate long-term accumulation in the bones.

The state does not conduct regular screening for other metals, such as mercury, cadmium, and arsenic. Information regarding exposure to these metals is compiled from mandatory reports made by physicians when they diagnose a health effect caused by these metals or determine that exposure has taken place. The body burden of heavy metal can be assessed through blood tests or tissue tests, and ambient concentrations from water samples can be an indicator of exposure.

Carbon monoxide poisoning results when CO binds to hemoglobin and causes an oxygen deficiency in the brain. Low level exposure to this colorless, odorless gas can be evaluated from blood tests taken from samples donated to the blood bank.

Miscarriages and birth defects are noted on medical records and birth records, but some do not manifest themselves until later in life. These health problems can be tracked over time by establishing registries. The valuable information compiled in these registries could serve as indicators that link outcomes with exposure.

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PREFACE

When I first began thinking about a thesis topic I knew I wanted to focus on environmental health, but I was not sure how to approach the subject. In the Spring of 1997 I took a class with Professor Caroline Karp titled Environmental Indicators (ES192), which introduced me to indicators-measurements used to depict trends regarding various aspects of the environment. After being inspired by this class and my advisor, Professor Harold Ward, I decided to combine my interest in environmental health with the up-and-coming field of indicators. The focus of my thesis then became clear. Rather than limiting myself to studying one disease, I decided to take an overview of environmental health and develop indicators for a sub-set of environmental health effects.

After doing preliminary research regarding environmental health, indicators, and health data collected in Rhode Island, I chose a group of specific health effects on which to focus my attention. I based my selection on several criteria, including: whether people in Rhode Island suffered from the health effect, whether the State collected data regarding the disease, and whether a feasible means of measuring or evaluating the number of cases of the disease over time could be developed.

For example, I did not choose to investigate gastroenteritis, an illness that results from consumption of contaminated shellfish, because

the majority of the cases go unreported. This health effect usually presents itself as stomach pain and intestinal problems. People demonstrating these symptoms usually treat themselves with over-the-counter drugs to relieve the discomfort, and return to a state of health within a few days. As a result, most people fail to seek medical attention, resulting in an underestimation of the actual incidence of the disease in the population. The limited documentation of the disease, as well as the difficulty involved with accurately monitoring gastroenteritis, would make the development of an indicator rather difficult.

The sub-set of health effects that I did select includes asthma, melanomas and other types of skin cancer, lung disease (other than asthma) and lung cancer, lead poisoning, heavy metal poisoning (other than lead), carbon monoxide poisoning, miscarriages, and birth defects. For each of these health effects, basic information about the diseases and strong evidence linking the diseases to environmental exposures are readily available. In addition, the nature and treatment of these health effects increase the feasibility of successfully monitoring the disease over time, which in turn, leads to the development of possible indicators.

After making my selection, I researched the background of each health effect, such as the cause, the symptoms, and the best treatment. Then, I investigated what data about each health effect were being collected in Rhode Island, and whether the incidence was being reported. I

combined my findings in these two areas, determined what additional information would be useful for developing indicators, and made recommendations regarding different means of collecting these data.

My first chapter introduces environmental health and discusses the difficulties involved with research in this area. I then define indicators, and summarize the data collected by the Rhode Island Department of Health in chapters two and three. Each of the following chapters is dedicated to a specific health effect in which I explain the background of the disease, briefly reiterate what data are collected in Rhode Island, and state my recommendations for indicators. All of my findings and my recommendations are summarized in the concluding chapter.

CHAPTER 1 : HISTORY AND BACKGROUND

Environmental health discussion began in England in the mid-nineteenth century with concerns about environmental sanitation. This idea, defined as man's effort to control all those factors which exercise or may exercise a deleterious effect on his physical, mental or social well-being,¹ prompted Edwin Chadwick to establish the first sanitary commission, followed by the Health of Towns Association, organized by Dr. Southwood Smith.²

Environmental problems related to health began to attract attention in the United States at this time as well. Lemuel Shattuck, a Boston publisher, wrote The Report of the Sanitary Commission in Massachusetts in 1850.³ This document, which came to be known as the Shattuck Report, laid the groundwork for a new era of public health work in the United States.⁴ Stephen Smith, a New Yorker, also brought a great deal of attention to health abominations such as pigsties adjacent to school yards, at this time.⁵ Later, in the 1890s, a professor at the Massachusetts Institute of Technology, William Thompson Sedgwick, became known as the apostle of the sanitary awakening in America because of his extensive work in the field.⁶

¹ Schifferes, Justus J. Healthier Living. John Wiley and Sons Inc. New York: 1970, p6.

² Ibid, p6.

³ Ibid, p7.

⁴ Ibid, p7.

⁵ Ibid, p8.

⁶ Ibid, p8.

Environmental health continues to be an issue of concern in today's society. Modern technology and scientific advances have simplified difficult tasks and have improved the quality of life over the years; however, they have degraded the environment and introduced new hazards in the process. The term environmental sanitation has been replaced with the more modern term environmental health; not only the phrase, but the definition itself, has evolved with the changing times. Defined in the broadest sense, environmental health is a subfield of public health concerned with assessing and controlling impacts of people on their environment (vegetation, animals, natural or historic landmarks) and the impact of the environment on them.⁷ This statement encompasses the entire field of environmental studies without providing a clear explanation of environmental health. A more precise definition of environmental health, according to the Dictionary of Environmental Health and Occupational Safety, is the art and science of the protection of good health, the promotion of aesthetic values, the prevention of disease and injury through the control of positive environmental factors, and the reduction of potential physical, biological, chemical, and radiological hazards.⁸

The overall goal is to protect human health by reducing the risk of disease caused by exposure to toxic environmental factors. However, it is not simply exposure to these chemicals that produces adverse health effects in humans. In

⁷ Moeller, Dade W. Environmental Health. Harvard University Press, Cambridge: 1992, p1.

⁸ Koren, Herman. Illustrated Dictionary of Environmental Health and Occupational Safety. CRC Press, New York: 1996.

order for exposure to a substance to cause toxic manifestations, a sufficient concentration of the agent or its biotransformation products must reach the appropriate sites in the body for a certain period of time.⁹ Therefore, in order to evaluate the potential hazard of a specific toxic agent in an individual fully, it is necessary to identify the type of effect, the dose required to produce the effect, the duration and frequency of the exposure, and the susceptibility of the exposed individual.¹⁰

The range of information needed to evaluate environmental health effects makes this a complicated field of study. A greater understanding of the relationship between human health and the environment has the potential to result in benefits for both people and the environment. This, however, is a challenging field of study.

BIOLOGICAL PATHWAY OF TOXINS

After exposure to a substance has occurred, the toxicity of the agent depends on the various processes of absorption, distribution, biotransformation, and elimination or accumulation that take place in the body. The pathway for each chemical is determined by a number of factors that will be explained below.

⁹ Biomarkers and Risk Assessment: Concepts and Principles. World Health Organization, Geneva: 1993, p25.

¹⁰ Ibid, p25.

Absorption

The primary routes of absorption are the skin, lungs, and gastrointestinal tract. Once inside the body, absorption depends on the polarity of the substance. Chemicals that exist in a polar state are water-soluble, while chemicals that exist in a non-polar state are lipid soluble.¹¹ As a result, factors that influence the lipophilicity of the chemical indirectly influence its absorption; some large molecules require a certain amount of lipid solubility in order to cross cell membranes.¹²

Absorption also depends on the acidity of the substance. Weakly acidic drugs exist in a non-polar state in the presence of an acidic medium, and in a polar state when in the presence of an alkaline medium. For weak bases, the reverse is true: weak bases exist in a polar state in an acidic medium and in a non-polar state in an alkaline medium.¹³ These characteristics influence where along the gastrointestinal tract the chemical will be absorbed. The stomach and the upper small bowel are acidic, so acidic chemicals are absorbed here; the lower bowel and the upper colon are more alkaline, so alkaline chemicals are absorbed there.¹⁴

Some chemicals are not absorbed from the gastrointestinal tract at all and therefore produce no systemic toxicity. For example, barium is used as a contrast

¹¹ Philp, Richard B. Environmental Hazards and Human Health. CRC Press, Inc. New York: 1995, p5.

¹² Ibid, p4.

¹³ Ibid, p5.

¹⁴ Ibid, p5.

medium for X-rays, and passes through the body unabsorbed. However, barium exposure by other means can be toxic.¹⁵ Absorption differs for each chemical and depends on the pathway of exposure.

Distribution

Once the substance has been absorbed, it is distributed to various parts of the body. Initially, distribution is based on the rate of blood flow to different tissues, so toxins are most likely to accumulate first in highly vascular organs. Then, as equilibrium states are reached, the toxins are redistributed on the basis of solubility.¹⁶

Many chemicals that are highly lipid soluble, such as DDT, are sequestered in body fat. This accumulation is rather stable, but the toxins may be released during periods of fat loss such as starvation or excess dieting, lactation, or illness. If the released chemicals reach a high concentration at target sites they may cause a toxic response.¹⁷

Elimination

The final step in the biological path is elimination of the chemical from the body. The most significant means of excretion are urine, feces, and bile; additional means include saliva, sweat, milk, tears, and mucus.¹⁸ The kidneys are the primary source of eliminating increased levels of toxic waste metabolites.

¹⁵ Ibid, p4.

¹⁶ Ibid, p6.

¹⁷ Ibid, p6.

They are a highly effective blood filter because they receive twenty-four percent of the total cardiac output.¹⁹ However, some of the small, water soluble molecules pass through the glomerulus, a tightly bound bundle of blood vessels that passively filters out molecules in the kidneys.²⁰

A secondary source of excretion is the lung, which eliminates volatile substances such as solvents, alcohol, and gaseous anesthetics.²¹ If the chemical substance enters the small intestines, it can be excreted in the feces or reabsorbed into the bloodstream.²²

Cumulative Effects

If the substance is not eliminated from the body through one of the various methods, or if the exposure has been continuous, toxicity may occur. If the rate of intake exceeds the rate of detoxification, the chemical will accumulate slowly.²³

The half-life of a substance is the amount of time needed for the chemical concentration to decrease by fifty percent. If the exposure intervals greatly exceed the half-life, accumulation is unlikely; if exposure intervals are less than or equal to the half life and the dose is repeated frequently, the chemicals tend to

¹⁸ Ibid, p10.

¹⁹ Ibid, p10.

²⁰ Ibid, p10.

²¹ Ibid, p11.

²² Ibid, p11.

²³ Ibid, p19.

be stored in various organs and tissues.²⁴ Accumulation of chemicals in the body, depending on toxicity, tends to lead to environmental health effects.

DIFFICULTIES IN MEASURING HEALTH EFFECTS

Various factors involved with environmental health effects make them difficult to monitor. First, controlled experiments cannot be done because it is unethical to expose human subjects to potentially hazardous substances, and if a drug is available, everyone in the study must receive the best possible treatment. As a result, health effects must be studied from cases that occur in the population. Since there is no control or monitoring that goes on in everyday life, multiple pathways of exposure make the source difficult to pinpoint. In addition to this, the chemical stability and xenobiotic interaction of some substances influence monitoring techniques. The large populations needed for significant studies, as well as the cost and technology of conducting these studies, are all factors to consider when examining environmental health effects.

Multiple Pathways of Exposure

One problem that complicates the process of measuring environmental health effects is multiple pathways in which the toxin or pollutant can enter the body. The most common pathways of exposure include air, water, and food.²⁵ Identifying the critical pathway, the major contributor of exposure, is the most

²⁴ Ibid, p20.

²⁵ DeKoning, H.W. Setting Environmental Standards: Guidelines for Decision-Making. World Health Organization, Geneva: 1987, p1.

efficient and economic way to monitor and control health effects.²⁶ The greatest hindrance in this process is the lack of information about the quantity and quality of total exposure that results from multiple pathways.²⁷ As a result, efficient steps to limit or eliminate exposure cannot always be taken either because the origin of the exposure is unknown or the multiple pathways cannot be controlled.

Toxicity of many substances varies depending on the route by which they enter the body; therefore, methods of evaluating the potency of the chemical may not be reliable for different pathways of exposure.²⁸

Information about the chemical cannot be applied to the total chemical exposure because the absorption of a chemical, as well as the biotransformation that takes place, is influenced by the pathway of exposure.²⁹ For instance, cadmium is known to be carcinogenic if inhaled, but not if it is ingested or absorbed through the skin.³⁰

Even if there is only one pathway of exposure, the area of contact may influence the uptake of the chemical. For substances that enter the body dermally, the rate of absorption at the forehead is twenty times greater than the rate of absorption if the chemical comes in contact with the forearm.³¹ Transit time for ingested material to pass through the intestinal tract can range from ten

²⁶ Ibid, p1.

²⁷ Ibid,p3.

²⁸ Philp, p46.

²⁹ Ibid, p46.

³⁰ Ibid, p46.

to eighty hours depending on several factors, including age and diet. This, in turn, influences absorption time and ultimately influences the toxicity of the chemical. The majority of risk assessments focus on the critical pathway of exposure or examine only one route of entry, which may lead to inaccurate or incomplete conclusions about the toxicity of the chemical.³²

An example that illustrates several of the complications described here is dichlorodiphenyltrichloroethane, commonly known as DDT. This chemical is an insecticide formerly used in the United States which has been banned by the EPA primarily because of its adverse effects on the environment, as well as the great controversy over the risk posed to human health.³³ This chemical enters the body through inhalation, absorption, ingestion, or contact, and is hazardous to the central nervous system, the kidneys, the liver, the skin, and the peripheral nervous system.³⁴ The effects listed here are associated with exposure to DDT, but more specific knowledge regarding the direct cause of these effects is limited by the complications introduced by the multiple pathways of exposure.

Chemical Stability

Another difficulty in monitoring human health is the stability of some pollutants. Certain chemicals degrade inside the body within a few hours of

³¹ Ibid, p46.

³² Ibid, p46.

³³ Beatty, Rita Gray. The DDT Myth: Triumph of the Amateurs. The John Day Company, New York: 1973, p3.

³⁴ Koren, Illustrated Dictionary of Environmental Health and Occupational Safety.

exposure.³⁵ As a result, these toxins cannot be detected in the body. However, this does not mean that potential toxic exposure has not occurred.³⁶ Benzidine, a known human carcinogen, aflatoxin, and vinyl chloride, are toxic chemicals which degrade quickly in the body, but still have the potential to cause harm.³⁷

Multiple Causes of Effect

Often times it is difficult to attribute a toxic effect to one certain exposure because various chemicals can produce the same or similar reactions in the body.³⁸ For instance, inhibition of enzymes in the heme synthesis may be caused by either exposure to lead or to a deficiency of iron.³⁹ Identifying the cause with great certainty usually occurs when the outcome is rare and known to be caused by exposure to a specific toxin. An example of this is angiosarcoma resulting from exposure to vinyl chloride.⁴⁰ However, this clarity does not exist for the majority of environmental pollutants.

Xenobiotic Interactions

Xenobiotics are defined as substances foreign to living organisms, either natural, anthropogenic, therapeutic, or toxic.⁴¹ The body reacts to these substances individually, as well as to the interaction of two or more. Some

³⁵ Monitoring Human Tissue for Toxic Substances. National Academy Press, Washington D.C.: 1991, p29.

³⁶ *Ibid*, p30.

³⁷ *Ibid*, p30.

³⁸ Philp, p54.

³⁹ Biomarkers and Risk Assessment: Concepts and Principles, p37.

⁴⁰ Philp, p54

agents which may not be extremely harmful, or are non-toxic at low levels, may become toxic if exposure occurs in the presence of other chemicals.⁴² This is referred to as a synergistic effect, in which the total response of the combined effect of two substances is greater than the sum of their individual effects (additive effect). For example, the risk of developing lung cancer increases dramatically when a smoker is exposed to asbestos. Synergistic effects occur most commonly with regard to carcinogens. The presence of a certain substance may induce enzyme production by interfering with metabolism; this may then increase the concentration of carcinogens from other sources.⁴³ Hundreds of industrial chemicals, such as benzopyrene and 3-methylcholanthrene, as well as many pesticides, such as DDT and lindane, are known to be enzyme inducers.⁴⁴ Natural enzyme inducers also occur in food, a-solatin in potatoes, and tomatin in tomatoes, for instance.⁴⁵

This type of synergistic reaction may occur in reverse as well. Rather than an increase in the toxicity of a chemical, an antagonistic effect diminishes the toxicity or prevents the effect of a certain drug in the presence of others.⁴⁶ An antagonistic effect, as well as a synergistic effect, may result from a variety of different factors, including altered excretion, altered biotransformation, or

⁴¹ Ibid, p3.

⁴² Ibid, p26.

⁴³ Ibid, p26.

⁴⁴ Ibid, p26.

⁴⁵ Ibid, p27.

⁴⁶ Ibid, p26.

competition for receptors.⁴⁷ These types of interactions complicate monitoring of environmental health effects.

Population Size for Study Purposes

The size of the population in studies of environmental health effects is also a limiting factor. In order to obtain results that are statistically significant, it is necessary to have a large sample population that is representative of the general public or a subgroup to which the results can be generalized. In order to detect the incidence of one health effect among one hundred people, it is necessary to test or study three hundred subjects. To detect the incidence of one in one thousand, three thousand subjects are needed.⁴⁸ Studies become even larger and more difficult when the outcome of interest is rare, or when the toxic reactions are independent of dose.⁴⁹

As a result of this, researchers sometimes use animal studies and extrapolate the results to humans. The reliability of these data is continually under dispute not only because of the difference between animals and humans, but also because many chemicals are toxic at the extremely high doses administered to the animals under study, are not toxic at lower levels to which people would be exposed.⁵⁰ The cytotoxicity of the high doses used in testing induces the rate of cell proliferation for repair, increasing the chances of

⁴⁷ Ibid, p26.

⁴⁸ Ibid, p61.

⁴⁹ Ibid, p61.

⁵⁰ Ibid, 49.

developing malignant mutations.⁵¹ This, as well as general differences between humans and animals causes uncertainty when extrapolating data from animal studies.⁵²

Long Latency Period

The long latency period involved with many environmental health effects also complicates research. If exposure takes place twenty, thirty, or even forty years before the disease manifests itself, identifying the source and extent of exposure is often challenging. A different approach would be to conduct a prospective study to monitor the development of a disease after a known exposure has taken place. This, however, is very costly and difficult because of the extended time period over which the subjects need to be monitored. Losing participation in the study for various reasons, such as death or relocation, is another factor that sometimes impedes the success of studies focused on health effects with long latency periods. In addition to this, the long waiting period necessary to get results limits the action that can be taken in the short term to protect people from the substance under suspicion.

Cost and Technology

The high cost of research and technology are other factors that make studies of environmental health indicators difficult. Large studies necessary to

⁵¹ Ibid, 49.

⁵² Ibid, p2.

yield significant findings are often expensive to conduct. Some research areas do not often get the funding they need because there is not enough evidence that the results will provide benefits that will make the research worthwhile. In addition to this, pharmaceutical companies finance a great deal of the research in the health field; as a result, funding is often limited to studies that have a strong market potential.

Great technological progress has been made in the last few decades which allow chemicals to be detected at lower levels or health effects to be diagnosed at earlier stages of development. However, sometimes studies are hindered because it is not feasible or practical to use the present technology on a large scale. The high cost, as well as the availability and accuracy of the technology necessary to conduct the research or make the diagnosis should be considered when studying environmental health effects.

CHAPTER 2 : INDICATORS

EPA FRAMEWORK

The United States Environmental Protection Agency has created a conceptual framework for an indicator model which can be used to monitor environmental effects, and to evaluate programs designed to control, prevent, or improve these effects. This Pressure, State, Response, Effect model describes the connections between human activities and the state of the environment, however it does not attempt to specify one to one links between environmental stressors or environmental changes, and societal responses.⁵³

The model states that human activities exert **pressures**, such as pollution or changes in land use, on the environment. Direct pressures are the actual biophysical inputs or outputs that put stress on the ecosystem, such as the release of anthropogenic pollutants, resource harvesting, or species introduction. Social structure, technology changes, and human population growth, several factors that motivate human activity, can be considered **underlying social pressures** that ultimately contribute to the direct pressures on the environment. It is not only these pressures of purely human origin that prompt the direct pressures, but also **indirect pressures** which result from the interaction of human activities and natural processes, such as agriculture, the nutrient cycle, and flooding.⁵⁴

⁵³ *Conceptual Framework to Support Development and Use of Environmental Information in Decision Making*: USEPA Document #239-R-95-012, Washington, DC: April 1995.

<http://www.epa.gov/indicators/defne.html>,

⁵⁴ Ibid.

These pressures cause changes in the **state** of the environment, including differences in air quality, habitat diversity, and water flow. This broad category of the model focuses on ambient physical, chemical, biological, and ecological conditions, as well as, human health, and environmentally related welfare.⁵⁵

Society then **responds** to these changes by taking action to address any undesirable observed or predicted change in the ecosystem, human health, or welfare. The response may be voluntary, legally-mandated, or incentive-driven, and can focus on mitigation, restoration, prevention, or adaptation.⁵⁶ Action can be taken on the government level through new policies and regulations, in the private sector through changes in technology and waste disposal, or on an individual level through changes in consumption patterns and recycling efforts. Cooperative efforts such as research, education, and land use planning are also examples of possible responses.⁵⁷

Effect demonstrates the relationships among the other variables in this framework. These relationships are derived from models and analyses that provide substantial evidence linking problems, potential causes, and/or solutions. Effects are indicators of attributed relationships between two or more pressures, states, and/or responses. The two variables can be within the same

⁵⁵ Ibid

⁵⁶ Ibid.

⁵⁷ Ibid.

category, a direct and an indirect pressure, for instance, or from different categories, such as a pressure and a response.⁵⁸

Although the other categories of measure described above are sometimes used as environmental indicators,⁵⁹ effect indicators contribute the most useful information to this framework by providing the most appropriate criteria for making environmental decisions. Effective management responses to environmental concern are based on relationships between variables rather than on information about the individual variables in isolation. For example, a change in the state of the environment alone does not necessarily signify a problem. If the change is a problem but the pressure causing the change is unknown, then it is difficult to decide on an appropriate response. However, an effect indicator would demonstrate the relationship between the state and the pressure, providing more informative data on which to base a response.⁶⁰

INDICATORS

The United States Environmental Protection Agency defines environmental indicators as parameters, measured or observed properties, that provide significant information about patterns or trends in the state of the environment, in human activities that are affected by the environment, or in the relationship among such variables.⁶¹ More specifically, environmental indicators

⁵⁸ Ibid.

⁵⁹ Ibid.

⁶⁰ Ibid.

⁶¹ Ibid.

are numerical measurements that, ideally, are easily obtainable at low cost from information that is currently being gathered. Indicators that are intended to be used outside of the agency that defines them as well as within, should be defined in a manner that is comprehensible by the general public and easily presentable in the media.⁶²

Indicators integrate information about environmental management at all different scales, not just information that high-level policy-makers can understand and use.⁶³ Indicators are a way to organize and present aggregate statistics into a more usable form. The information can be presented quantitatively and qualitatively in the form of graphs, diagrams, maps, or photographs.⁶⁴

In the most general sense of the term, “indicator” can be described as any measure that provides information regarding some variable of interest. As a result of this broad definition, indicators exist in various fields to measure a variety of different concerns. With regard to environmental health, biological indicators are the dominant area of interest.

Biological indicators are organisms, or populations of organisms, whose occurrence, vitality, and responses change under the impact of various

⁶² *Sustainable San Francisco*. <http://www.ci.sf.ca.us/environment/sustain/intro.html>.

⁶³ *Conceptual Framework to Support Development and Use of Environmental Information in Decision Making*.

⁶⁴ *Ibid.*

environmental conditions.⁶⁵ Absorption of a stimulus causes a reaction within the organism that provides information about changes in the level of the stimulus in the environment.⁶⁶ When referring to environmental health indicators, the organisms of interest are people, and the stimuli to which they are responding are the various environmental toxins or pollutants that affect the human body in some way.

Since the field of environmental health is so broad, the definition of the indicator must be customized for the audience it will serve. For example, several indicators exist for the incidence of asthma over a certain period of time. How the indicator is defined is based on the ability to make the most direct measure as well as personal interest of the party gathering the data. For example, for a pharmaceutical company, an indicator of asthma rates can be reflected in the number of prescriptions filled for asthma medication, while for health-care providers, a good indicator could be the number of asthma cases presenting in the emergency room during a given period of time. Although both of these measures are indicators of asthma, they are not the same measure, and they serve the needs of different groups of people. As a result of their diversity, indicators are applicable and informative in a variety of fields.

⁶⁵ Kovacs, Margit. Biological Indicators in Environmental Protection. Ellis Horwood limited, New York: 1992, p7.

⁶⁶ Ibid, p7.

Indicators of any type are only informative if the data can be compared to some standard, referred to as a baseline measurement.⁶⁷ In theory, the standard should be measures taken from an unexposed population; however, a completely unexposed population is almost non-existent, so the baseline should be as close to unexposed as possible.⁶⁸ Once a baseline is established, the goal is to compare the past and present states in order to help visualize the rate and direction of environmental change, and make predictions about the future.⁶⁹ If no baseline is obtainable or available, repeated measures could be made in order to evaluate the trend over time, and the first data set could be used as a baseline. The data obtained from these comparisons can be used to evaluate the effectiveness of policies or regulations that have been implemented as measures to protect human health.

The most useful indicators are those that measure most directly the subject under study. In the case of environmental health, the goal is to protect human health; therefore, the most applicable indicator is a measure of some health-end effect. However, it is often difficult to measure the health-end effect itself, or its measurement is complicated by other factors that interfere with the information that the indicator is trying to obtain. In this case, it is necessary to use a different type of indicator to gain similar information.

⁶⁷ Ibid, p12.

⁶⁸ Ibid,p12.

⁶⁹ Ibid, p11-12.

One step away from measuring the health effect itself, is measuring something in the body that signifies the presence or development of the disease of interest. One approach is to investigate biomarkers, some alteration within the body that occurs at a biochemical, cellular, or molecular level that indicates that exposure to a potentially toxic substance has taken place.⁷⁰ Biomarkers may also be changes that are preliminary developments of a disease. For example, the presence of cotinine, the metabolite of nicotine, is a biomarker of exposure to secondhand smoke. Another option is to measure body burden, the level of a certain substance that has accumulated in body tissue, such as the amount of lead in one's blood or bones.⁷¹ Indicators such as these, that measure changes within the body, provide data that can be used to estimate the degree of environmental impact and potential danger of the substance on human health.⁷²

If measurements within the body are impossible, an exposure indicator can be developed. This type of indicator measures the level of pollutant in the environment to which the individual or population is exposed. Exposure indicators are an indirect measure of human health, and therefore not as informative as other types of indicators previously described. Physical and chemical measurements of the pollutants provide only quantitative data on the presence and levels of pollutants in the environment; measurements of changes

⁷⁰ Monitoring Human Tissues for Toxic Substances, p49.

⁷¹ Human Health and the Environment: Some Research Needs, US Department of Health and Human Services: 1984, p278.

⁷² Kovacs, p7.

within the body provide more precise information on the extent of pollution reaching and effecting the population being studied.⁷³

Health-end-effect

The health end-effect measure examines the proportion of people within a defined population who are afflicted by a certain health outcome associated with a causal environmental factor. Health-end-effect indicators are the most desirable type because they are a direct measure of human health, exactly what needs to be protected. This type of indicator is useful when a specific exposure is known to cause a certain health effect, especially when the level of exposure cannot be precisely measured because of multiple pathways of exposure or inadequate measuring capabilities in the environment.

The desired trend among health-end-effects is a declining proportion of a given population suffering from the specific disease or illness. The cause of the decreasing trend of this indicator over time can be attributed to several possibilities, all of which are positive outcomes. It may signify a reduction of the contaminant in the environment, or a decrease in exposure to the contaminant as a result of protective measures that are being taken to protect the population. Another feasible explanation, although not an indication of an improvement in environmental health, but rather advanced technologies, may be improved capabilities in the medical field that detect signs of the disease before it manifests

⁷³ Ibid, p7.

itself; this leads to preventative treatment of the disease before it progresses to the health-end-effect that is being measured.

There are also several reasons why one might observe an increasing trend in health-end effects. It may mean that higher levels of the contaminant exist in the environment, or that the health-end effect has a relatively long latency period and although exposure to the toxin has been reduced, the individual is now becoming symptomatic. The long latency period also makes it difficult to determine the source and extent of exposure. In addition to this, it may be too late to initiate protective measures because damage from the exposure has already been done. Another possibility may again be improved detection capabilities, but this time the improved technologies may identify cases that existed in the past but were never diagnosed. Since the goal is to protect human health, an increasing trend of health-end effects is not the outcome of choice. It may indicate a growing public health problem, or an area that warrants attention from researchers and policy makers. For example, the protective measures currently in place may not be successfully preventing the disease. Perhaps the critical pathway of exposure is not being addressed, or the primary cause has been misidentified.

Biomarkers

Technology over the last decade has developed methods of detecting exposure to various chemicals and pollutants at low concentrations, and

assessing their behavior and effect on a cellular and molecular level.⁷⁴ Data regarding these exposures and changes are gained by studying biomarkers, generally defined as any measure reflecting an interaction, chemical, physiological, or biological, between the biological system and a potential hazard.⁷⁵ With regard to environmental health, biomarkers are more precisely defined as alterations in the human body that occur at a biochemical, cellular, or molecular level on a continuum between exposure and disease.⁷⁶ Theoretically, a biomarker can be reflected in each step in the chain of events from exposure through disease manifestation.⁷⁷ However, actual measurements of such biomarkers depend on technology and understanding of the developmental process of the disease.

Tests for biomarkers are often done on various human tissues, secretions, excretions, or exhaled air.⁷⁸ The most common substances tested for biomarkers include blood and urine, although breast milk, semen, and adipose tissue are used as well.⁷⁹ Analyses of these body fluids and tissues detect metabolites of chemicals, enzymes, and other biochemical substances that document, not simply the presence of a toxin, but the interaction of the toxin with another

⁷⁴ Monitoring Human Tissues for Toxic Substances, p49.

⁷⁵ Biomarkers and Risk Assessment: Concepts and Principles, p11.

⁷⁶ Monitoring Human Tissues for Toxic Substances, p49.

⁷⁷ *Ibid*, p50.

⁷⁸ Lee, Lester W. et al. *Human Tissue Monitoring and Specimen Banking: Opportunities for Exposure Assessment, Risk Assessment, and Epidemiologic Research*. Environmental Health Perspective, 103 (supplemental 3) 3-8 (1995).

⁷⁹ Monitoring Human Tissues for Toxic Substances, p50.

substance in the body.⁸⁰ The measurements taken from body fluids determine not only whether exposure has occurred, but also the extent of the exposure if indeed it did occur. Periodic measures of toxic substances or metabolites could detect the occurrence of toxic exposure at an early stage, perhaps before it reaches the stage of adverse health effects.⁸¹

Biomarkers are also often used to assess exposure to chemicals that exist in the environment in low doses. The low concentration in the environment, as well as the multiple pathways of exposure, makes it difficult to assess the risk of certain chemicals.⁸² Many of these chemicals, such as organochlorine pesticides, become concentrated through the links of the food chain. This process known as biomagnification, results in higher predatory species, such as humans, containing the highest levels of the chemical.⁸³ Biomarkers indicate cumulative internal doses of toxins over time from all pathways and means of exposure.⁸⁴

Biomarkers of Exposure

Biomarkers of exposure, the first of three sub-categories of biomarkers, provide qualitative information as to whether an individual has been exposed to a certain substance.⁸⁵ It is defined as the measure of a biological response, such as cytogenetic or reversible physiological changes, in an individual exposed to

⁸⁰ Biomarkers and Risk Assessment: Concepts and Principles, p11.

⁸¹ Lee.

⁸² Ibid.

⁸³ Philp, p72.

⁸⁴ Lee.

⁸⁵ Monitoring Human Tissues for Toxic Substances, p50.

some toxic agent.⁸⁶ The measurements reflect the level of a metabolite of some exogenous substance, or the product of the interaction between the exogenous substance and some molecule or cell.⁸⁷ For example, heme metabolism is disrupted by exposure to lead, so an appropriate biomarker for this toxin is the measurement of the concentration of various intermediates of heme metabolites.⁸⁸

The levels of the biomarkers in the body are assessed through the analysis of biological samples such as urine, feces, blood, and exhaled air.⁸⁹ Exposure biomarkers provide the most direct evidence of human exposure to a given agent because the measure is taken from within the body, rather than from concentrations of the substance in food, water, and air to which the individual is exposed.⁹⁰ They are used extensively in the surveillance of people exposed to metals such as lead, cadmium, mercury, nickel, chromium, and arsenic, and organic chemicals such as aniline, benzene, carbon disulfide, styrene, chlorobenzene, and chlorinated aliphatic hydrocarbons.⁹¹

Biomarkers can be used to estimate quantitatively the dose to which the individual has been exposed. Knowledge about the formation and the removal of the biomarkers, in conjunction with the biomarkers themselves, can create a

⁸⁶ Biomarkers and Risk Assessment: Concepts and Principles, p25.

⁸⁷ Ibid, p13.

⁸⁸ Monitoring Human Tissues for Toxic Substances, p51.

⁸⁹ Biomarkers and Risk Assessment: Concepts and Principles, p26.

⁹⁰ Ibid, p25.

⁹¹ Ibid, p31.

link between the ambient exposure and the internal dose, the actual amount of the chemical that is reaching the body.⁹²

The most informative studies are those that involve multiple biomarkers from samples taken at the same time from the same individual.⁹³ The analysis of these biomarkers, assuming that they have different half-lives, can provide a great deal of information regarding exposure history. If an individual has undergone a single exposure in the recent past, the levels of the biomarkers with short half-lives will be elevated relative to those with longer half-lives.⁹⁴ Simultaneous elevation of the levels of biomarkers with long half-lives and short half-lives signifies that continuous exposure to the toxic agent has taken place, while distant exposure will be represented by the presence of only biomarkers with long half-lives.⁹⁵

Additional information about biomarkers is obtained through models. They are used to show quantitative relationships between biomarkers under various exposure conditions or at different times after exposure has taken place.⁹⁶ Mathematical models are used to describe the kinetic formation and elimination of various biomarkers of exposure.⁹⁷ These models also define the quantitative relationship between biomarkers taken from available samples such as blood, and less readily available samples such as tissue DNA, which in turn, may

⁹² Ibid, p26.

⁹³ Ibid, p30.

⁹⁴ Ibid, p30.

⁹⁵ Ibid, p30.

⁹⁶ Ibid, p31.

provide data that are more directly related to the health effect under investigation.⁹⁸

While important data can be gained from biomarkers of exposure, this method has some disadvantages. For example, biomarkers of exposure reflect some reaction, but not necessarily a critical interaction with macromolecules in target cells.⁹⁹ Another limitation is that lab methods to test for the biomarker might be sensitive, but if there is no bioaccumulation, only recent exposure can be assessed.¹⁰⁰ As a result, biomarkers of exposure should supplement rather than replace, measures of ambient exposure to toxins and other means of investigating environmental health effects.¹⁰¹

Biomarkers of Effect

Biomarkers of effect, another type of biomarker, measure changes at the tissue, cellular, or molecular level in response to exposure to an environmental chemical.¹⁰² These changes are not necessarily indicators of impaired functioning, they are indicators of a biological reaction following exposure to a chemical.¹⁰³ The effect measure is based on biochemical indicators such as enzyme activity measured in target organs or body fluids.¹⁰⁴ The effect of the

⁹⁷ Ibid, p30.

⁹⁸ Ibid, p30.

⁹⁹ Monitoring Human Tissues for Toxic Substances, p51.

¹⁰⁰ Ibid, p50.

¹⁰¹ Biomarkers and Risk Assessment: Concepts and Principles, p31.

¹⁰² Monitoring Human Tissues for Toxic Substances, p51.

¹⁰³ Ibid, p51.

¹⁰⁴ Ibid, p51.

biological reaction depends on the quality, strength, onset, and duration of exposure.¹⁰⁵

Sometime biomarkers of effect are not mechanistically related to chemically induced lesions, but rather represent concomitant independent changes.¹⁰⁶ A limited range of tissues is available for biomarker testing, so the more accessible tissues are often used as surrogates for known or putative target tissues.¹⁰⁷ Ultimately, this type of biomarker measures the effect of some reaction that is taking place in the body, but the information gained from the analysis is actually being used to assess exposure.¹⁰⁸

Biomarkers of effect have several different uses. They may be used directly in hazard identification and dose-response assessments which are important aspects of risk assessment.¹⁰⁹ In terms of preventive measures, ideal effect markers are changes that are still reversible; however, non-reversible effect biomarkers are still useful in diagnoses, epidemiological studies, and clinical interventions.¹¹⁰ One disadvantage of biomarkers of effect is that a reaction in response to a chemical agent may be detected, but it is not always possible to relate the effect to one specific causative agent.¹¹¹ For example, an effect marker of lead is the inhibition of enzymes in the heme synthesis pathway. These

¹⁰⁵ Ibid, p51.

¹⁰⁶ Biomarkers and Risk Assessment: Concepts and Principles, p37.

¹⁰⁷ Ibid, p37.

¹⁰⁸ Ibid, p27.

¹⁰⁹ Ibid, p37.

¹¹⁰ Ibid, p37.

¹¹¹ Ibid, p37.

changes are indicators of lead, but not exclusively lead; they are also caused by an iron deficiency.¹¹² As a result, more comprehensive studies are needed before conclusions can be drawn based on biomarkers of effect alone.

¹¹² Ibid, p37.

Biomarkers of Susceptibility

Biomarkers of susceptibility, the last sub-category of biomarkers, are indicators of an inherent or acquired ability of an organism to respond to the challenge of exposure to a specific xenobiotic substance.¹¹³ They are factors that may increase or decrease an individual's risk of toxic response. For example, enzyme activity controls the activation and detoxification of xenobiotics within the body. Differing rates of these enzymes influence a person's susceptibility by altering the biologically-effective dose of the environmental agent.¹¹⁴

Different responses from two individuals subjected to the same environmental exposures can be explained by susceptibility. Genetic differences in metabolism may result in substantially different internal doses at the target site, and therefore a markedly different reaction in the body.¹¹⁵ Major factors such as age, sex, diet, as well as health status, and previous exposure can also influence the susceptibility of an individual exposed to a certain chemical.¹¹⁶

Age is one of the major factors influencing susceptibility because many biological functions are less efficient during the extremes of life. For instance, enzymes required for metabolizing drugs are not fully developed until six to twelve months of age, causing a higher toxicity among infants. The renal function of children is also underdeveloped, which leads to a longer half-life of

¹¹³ Ibid, p13.

¹¹⁴ Ibid, p59.

¹¹⁵ Ibid, p59.

¹¹⁶ Ibid, p59.

certain substances. After age seventy-five, systems slow down and return to a functioning level similar to newborns, increasing the susceptibility of the elderly as well.¹¹⁷

The differences in body composition between adults and young children are other reasons for increased susceptibility early in life. Infants have a greater percentage of water and extracellular fluid, resulting in a greater volume in which to dilute water soluble drugs.¹¹⁸ The higher toxic concentrations in the smaller bodies of the babies increase the risk of experiencing damage from toxins. Other factors include a slower basal metabolic rate among children, greater permeability of the skin, and prolonged gastric emptying which leads to increased absorption of heavy metals.¹¹⁹

Susceptibility based on sex results primarily from differences in body size, percentage of fat, and basal metabolic rate between men and women. Pregnancy also influences changes in metabolism, body composition, and fluid content.¹²⁰

Diet is another factor that influences susceptibility. Aside from a balanced diet providing general nutrition and overall well-being, certain components may adsorb (collect on the surface) a toxicant, which would limit the amount that is available for absorption. For example, calcium reduces lead toxicity because it competes for the same absorption site on the intestinal mucosa; high-fiber diets

¹¹⁷ Philp, p21.

¹¹⁸ Ibid, p21.

¹¹⁹ Ibid, p21.

¹²⁰ Ibid, p22.

shorten transit time of substances in the gastrointestinal tract, which lessens the time available for absorption.¹²¹

Certain nutrients also influence susceptibility. Nutritional status influences the rate of absorption of a toxicant and the quantity that reaches the critical target tissue. In a person with high nutritional status, the body sequesters toxic agents in body depots such as adipose tissue or bone mineral, instead of distributing the substance more evenly throughout the body. Low nutritional status allows the toxins to spread to vital organs such as the brain, liver, and kidneys where the substance has more dramatic adverse effects.¹²²

Body Burdens

Another indicator that can be used instead of a health-end-effect is the measure of body burden. This is a measurable level of a specific compound in the body tissue.¹²³ For example, the measure of the amount of lead that accumulates in bones is a body burden, as well as the measure of DDT or PCBs that is stored in adipose tissue. Body burdens are not as informative as biomarkers because they measure the amount of the substance itself, rather than a change, but they provide more useful information than evaluations of ambient concentration.

¹²¹ Ibid, p27.

¹²² Monitoring Human Tissues for Toxic Substance, p55.

¹²³ Human Health and the Environment: Some Research Needs, p278.

Body burdens measure the internal dose of exposure, defined as the total amount of a chemical that is absorbed by the body over a period of time.¹²⁴ The external dose is defined as the amount of a chemical agent in the environment with which an individual has contact.¹²⁵ This is determined by taking measurements of the level of the chemical that exists outside the body through methods such as area or personal monitoring.¹²⁶ The evaluation of the dose based on measures taken from inside the body provides more informative data because it is more directly related to human health, the most important focus.

The course of accumulation and reduction of the body burden differs on an individual level for each chemical. The amount of contaminant that exists in the body depends on the pattern of exposure, the solubility of the substance in body tissue, and the chemical affinities that the substance has within the body.¹²⁷ Metabolic reactions, as well as the mechanism and rate of excretion from the body, also influence the body burden of each compound.¹²⁸

The significance of a body burden must be interpreted on an individual basis because of the variability among substances. The presence of an elevated body burden does not necessarily mean that the person is in danger of developing a disease, and the absence of a body burden does not mean that exposure has not taken place. For example, the build up and reduction of carbon

¹²⁴ Biomarkers and Risk Assessment, p25.

¹²⁵ *Ibid*, p25.

¹²⁶ *Ibid*, p25.

¹²⁷ Human health and the Environment: Some Research Needs, p278.

¹²⁸ *Ibid*, p278.

monoxide can result in a body burden of carboxyhemoglobin just minutes or hours after exposure has taken place.¹²⁹ However, this body burden can be reduced to a normal level within a few hours after the exposure is terminated.¹³⁰ In this case, although no body burden may exist when the person is tested, it does not mean that carbon monoxide poisoning, or adverse effects from carbon monoxide exposure, did not occur.

On the other hand, DDT and its metabolites DDE and DDD, are stored in adipose tissue over time; this serves as a relatively easy measure of body burden.¹³¹ Almost everyone has a body burden of DDT or its metabolites so it is a good indicator in terms of evaluating level of exposure; however, it does not indicate higher risk for any specific disease.¹³²

The toxicity of different chemicals depends on a variety of factors and cannot be evaluated on body burden alone. Testing for body burdens increases the opportunity for detecting early biologic changes before clinical manifestations of the disease occur, but the usefulness of measuring body burdens increases if they accompany studies of biomarkers.¹³³

In order to estimate the risk of developing a disease, it is necessary to understand the internal dose-response relationship. This requires biokinetic research in addition to knowledge of body burdens. However, if the incidence of

¹²⁹ Ibid, p278.

¹³⁰ Ibid, p278.

¹³¹ Monitoring Human Tissues for Toxic Substances, p29.

¹³² Ibid, p29.

¹³³ Human health and the Environment: Some Research Needs, p279.

disease is proportional to the internal dose, body burdens alone are useful for estimating relative risks, such as the risk of disease between males and females or between urban and rural residents.¹³⁴

Exposure

The last category of environmental health indicators is exposure. Exposure assessment on an individual level evaluates characteristics of the person, such as age and gender, in combination with his or her specific interactions with the contaminated environment, such as duration and critical pathway of exposure. It measures how much of a contaminant is available to the body through a certain route of entry. The logistics of this type of exposure assessment, however, makes it difficult to conduct on a large scale for the general population.

Exposure assessment for a community or defined population can be evaluated through ambient monitoring, measuring the amount of the contaminant in the water or the air to which an individual is exposed.¹³⁵ This is the most indirect of the four methods of assessing human health because it is essentially a measure of the state of the environment; estimates of the level of the toxin that the body actually encounters are made from these ambient measures.¹³⁶ This is not the most accurate evaluating exposure because of the many factors that influence absorption of the chemical in the body, such as age, duration of

¹³⁴ Monitoring Human Tissue for Toxic Substances, p52.

exposure, and the critical pathway of entry, to name a few. However, ambient measurements are a crucial aspect of many prevention programs that stress avoiding the initial exposure. For example, measures of the concentration of lead dust on the floor of a toddler's home are a good indicator of whether or not it is a safe environment for the child.

Another use for an exposure measure obtained from monitoring chemicals from the point of emission, such as drains or incinerators, is tracking industrial compliance with legislative regulations or setting new standards.¹³⁷ Although not the best indicator of environmental health because of its remoteness to humans, measures of ambient concentration can still provide useful information. If there is a correlation or a causal link between some exposure and a certain health effect, a decrease in ambient levels will reduce the risk of exposure, and will ultimately lower the risk of developing the health effect, despite the related uncertainties. Until new methods of measuring the impact of environmental exposure on the body are developed, many indicators will fall into this category. However, many prevention programs root themselves in avoiding exposure.

¹³⁵ Philp, p51.

¹³⁶ Ibid, p51.

¹³⁷ Ibid, p51.

CHAPTER 3 : RHODE ISLAND DATA

RHODE ISLAND DEPARTMENT OF HEALTH DATA

The Rhode Island Department of Health is in charge of the majority of public health concerns that require attention from the government. The responsibilities of the Department cover a wide range of areas, including collecting data on a great number of health issues. A portion of these data is related to environmental health, either directly through data pertaining to a specific environmental health problem, or indirectly through data such as birth and death records which can be used when studying various health effects. The specific data that are collected for each topic vary among databases.

Birth Records

One of the largest databases maintained by the Department of Health is birth records. This database covers all people born in the State of Rhode Island and all Rhode Island residents born out of state, from 1897 to the present. The more recent records are computerized but the majority of these data are still hard-copy records. They are supplemented by archives of birth records which are available for all births in the state between 1853 and 1897. The data are collected at the hospital using the electronic birth certificate system. If the birth

takes place at home, the parents are responsible for reporting their child's data to the Department of Health.¹³⁸

The database contains more than a list of names and birth dates. Each record contains the weight and length of the baby at birth, as well as the score he or she received on the Apgar Test,¹³⁹ a number used to express the condition of a newborn infant sixty seconds after birth. The score is the sum of points received on assessment of the heart rate, respiratory effort, muscle tone, reflex irritability, and color. The test was named after an American anesthesiologist, Virginia Apgar (1909-1974).¹⁴⁰ The recovery score, which is also recorded, is based on the same assessment tests as the Apgar score, however the condition of the infant is evaluated at various intervals greater than one minute after birth.¹⁴¹ In addition to these scores, the estimated date of conception, as well as a clinical estimate of gestational age and developmental age, are included on the birth record.¹⁴²

Birth defects and injuries are also reported on the birth records; however, they are thought to be under-reported because each individual case is not verified. Information that may be relevant in evaluating some of the birth defects, such as smoking and drinking habits of the mother, is recorded on

¹³⁸ *Health Data Inventory, A Compendium of Databases Maintained by the Rhode Island Department of Health (Draft)*. Rhode Island Department of Health, August 1997.

¹³⁹ Jay Buechner, Ph.D. Rhode Island Department of Health. October 31, 1997.

¹⁴⁰ Dorland's Illustrated Medical Dictionary. W.B. Saunders Company, Philadelphia: 1994.

¹⁴¹ Dorland's Illustrated Medical Dictionary.

¹⁴² Buechner.

hospital medical records, but this information is not compiled in the Department of Health database.¹⁴³

Information from the birth records without names or any other identifying information (to protect confidentiality) can be made available to researchers. It is often used in retrospective studies and surveys linked to hospital records.

Researchers who request that names be linked to the birth records must participate in a confidentiality procedure prior to being granted access to these data.¹⁴⁴

Death Records

The database containing death records is similar to that of birth records. It contains information on all people who died in the State of Rhode Island and all Rhode Island residents who died out of state from 1948 to the present. The archives contain records dating back to 1853.¹⁴⁵ Funeral directors are responsible for obtaining personal information about the deceased, as well as obtaining the cause of death from the physician.¹⁴⁶ The death records include the age of the individual, the occupation and/or industry in which the deceased was involved, and the cause of death. Although the occupation/industry of the deceased is recorded as “lifetime,” suggesting that this information contains a complete listing of occupations over the years, a detailed history of the person’s life is not

¹⁴³ Buechner.

¹⁴⁴ Buechner.

¹⁴⁵ Buechner.

¹⁴⁶ *Health Data Inventory*.

recorded. The data reflect where the individual worked for the majority of his or her life, or his or her most recent occupation. This information, despite the imperfections, is often used to identify certain predominant environmental or occupational health risks.¹⁴⁷

The cause of death recorded on the death certificate provides more specific data than the occupational/industrial experience because multiple causes of death are listed. The immediate cause is listed first, followed by any underlying or contributing factors that may have influenced the death of the individual. For example, if someone died in an automobile accident, the immediate cause would be a head injury, the underlying cause of death would be a car accident, and the contributing causes would be multiple trauma. The detail recorded here is helpful in finding environmentally related diseases, as well in linking other risk factors to disease and death.¹⁴⁸

Fetal Death Records

The Department of Health also compiles fetal death records in a separate database similar to the database for the general death records. Whether the death was spontaneous or an induced abortion is recorded, as well as the gestational age of the fetus at the time of death.¹⁴⁹ This information is collected primarily from health care institutions and every once in a while from funeral directors.¹⁵⁰

¹⁴⁷ Buechner.

¹⁴⁸ Buechner.

¹⁴⁹ Buechner

¹⁵⁰ *Health Data Inventory*.

The database contains information since mid-June of 1973, when mandatory reporting of all fetal deaths, regardless of length of gestation, was initiated. Prior to this time, deaths occurring before twenty weeks of development were not recorded.¹⁵¹

Linked Birth-Infant Death Records

Since 1970, the Department of Health has been combining the data from birth records and death records to form the Linked Birth-Infant Death Records.¹⁵² An infant death is defined as any resident of the state of Rhode Island who dies prior to his or her first birthday. The information is obtained from the Vital Records that the department already collects, so the details of the data are consistent with that described previously.¹⁵³ An infant death prior to one year of age is often associated with some risk factor that was present at birth, such as low birth weight. Linking these records helps define the cause of death and identify any risk factors that may have been present.

The Cancer Registry

The Cancer Registry is one of the databases that deals with a specific disease that has multiple causes, some of which may be environmental. Health care agencies and providers report these data to the Department of Health on a regular basis. This Registry covers all Rhode Island residents as well as all

¹⁵¹ Bill Smith, RI Department of Health: April 1, 1998.

¹⁵² Ibid.

¹⁵³ *Health Data Inventory*.

cancers diagnosed in the State of Rhode Island.¹⁵⁴ The cancers are organized by type of cancer, based on primary tumors. If two primary tumors exist, a second record exists that is linked with the first by common demographics.¹⁵⁵ All metastasized tumors are not listed as separate records, but are described as different stages of the disease on the primary record.

The registry includes detailed information about the tumor site, the stage at diagnosis, and the type of cancer cell.¹⁵⁶ More information about occupation and exposure are collected with certain types of cancer. For example, a more detailed patient history is taken if the individual is diagnosed with mesothelioma, a type of lung cancer strongly associated with exposure to environmental asbestos.¹⁵⁷

This registry is comprehensive, but does not include in-situ cancers, cancers that will not spread to other parts of the body through the lymph nodes. Squamous cell and basal cell carcinomas, two types of skin cancer, fall into this category. Data for these types of cancer are not reported because these cases are easily curable with simple treatment, and treatment is usually administered in a physician's office rather than in a hospital. Collecting data from private practices is often difficult, and therefore rarely required by the Health Department.¹⁵⁸

¹⁵⁴ *Health Data Inventory*.

¹⁵⁵ Buechner.

¹⁵⁶ John Fulton, Ph.D. Rhode Island Department of Health: December 5, 1997.

¹⁵⁷ *Ibid.*

¹⁵⁸ *Ibid.*

Hospital Discharge Data

The State also collects Hospital Discharge Data on any in-patient discharged from an acute-care general hospital. All in-patients are included in this database, regardless of whether they are residents of Rhode Island. Hospital staff abstract the data from hospital medical records after the patient has been discharged and report them to the State.¹⁵⁹

The discharge diagnosis for each patient is coded according to the *International Classification of Diseases, 9th Revision, Clinical Modification* (ICD-9-CM). This is the same system used to classify procedures and causes of death. There is one principal diagnosis and six additional sub-diagnoses. This coding allows for a more accurate and more comprehensive diagnosis, as well as simply more detailed information on the discharge record. This database contains information about procedures and treatments, but it does not contain patient identification or patient history. History and identification information are recorded on personal medical records, but since 1989 it has not been compiled in the database that is maintained by the Department of Health.¹⁶⁰

Lead Screening Data

The Lead Screening Data compiled by the state of Rhode Island consists of blood lead levels for all children being screened in Rhode Island, as well as children who are residents of Rhode Island who are screened out of state. The

¹⁵⁹ Buechner.

law requires that all children between six months and six years of age have an annual screening for lead, regardless of whether they demonstrate any symptoms. Screening may be discontinued if a child, who has remained in the same environment, has two tests prior to age three that both yield results $<10\mu\text{g}/\text{dl}$. Lead data are essentially complete because physicians are required to send the all blood sample, with the exception of diagnostic or confirmatory samples, to the state laboratories to be tested. If a physician uses an on-site analytical device to screen for lead, he or she must agree to report the results to the Department of Health electronically. Testing done outside the state laboratories and reported electronically to the Department of Health is limited to Rhode Island Hospital and the lead clinic at St. Joseph's Hospital.¹⁶¹

If the test results show a level of lead in the blood above, a letter is sent to the physician regarding a follow-up test; the physician then notifies the family and may provide parents with educational materials about lead. If the child's blood lead level exceeds $20\mu\text{g}/\text{dl}$, the family is eligible for home inspection and visits from an in-home nurse who helps with education. The inspector's report is sent to parents, treating physicians, and property owners if the family is living in a rented home. The owner of the property is then required to make the house safe; however, the changes need to be conducted by a certified abatement person

¹⁶⁰ Ibid.

¹⁶¹ Susan Feeley, MPH, Rhode Island Department of Health: December 9, 1997.

before being implemented in order to ensure that the hazards are completely and safely abated.¹⁶²

The cut-off point for high levels of blood lead in Rhode Island used to be 25µg/dl. However, recent pressure from the Centers for Disease Control and Prevention to take action in response to blood lead levels lower than 25µg/dl, has prompted the state to change its action level to 20µg/dl.¹⁶³

Each record in the blood lead database represents a lab test, and contains the basic demographic information, as well as the birth date of the individual, the results of the test, and the type of test, finger stick or venous. The results from the finger stick are not as accurate as the venous test because dust from the child's finger may contaminate the blood. The false positive rate for this type of test ranges from 20-50%; therefore, the venous test is used in high-risk populations, and there is a high follow-up rate for finger stick tests that yield positive results.¹⁶⁴

The database maintained by the Department of Health has records beginning in 1990; hard copy records from prior to this time are archived. Prior to 1993, an indirect test called the EP test was done to assess blood lead levels rather than measuring them directly. Increased levels of EP resulted from a disruption of the heme synthesis that occurs when blood lead levels exceed 35µg/dl. This identified children with increased blood lead levels who were

¹⁶² Ibid.

¹⁶³ Ibid.

then given a test that measured lead directly. The EP test overlooked children with elevated levels that were below the cutoff point. New testing procedures were implemented in 1993; as a result, the data from the last five years are much more accurate.¹⁶⁵

Access to this information in aggregate form, or by census tract without identifiers is easy to obtain. Researchers may be granted privileges to these data with identifiers if they meet the requirements to receive approval from the institution's review board.¹⁶⁶

The Health Interview Survey

The Department of Health also maintains a database that compiles information from The Health Interview Survey. This survey is conducted about every five years (1985, 1990, 1996) in order to collect information on health status, health behaviors, health insurance coverage, access to health care and other selected topics that are relevant at the time the survey is being administered.¹⁶⁷ Since one section of the survey addresses current health issues, it is a good opportunity to gather preliminary data on a topic that has yet to be studied in detail. Data regarding injuries, emergency room visits, and treatment by an independent physician are also covered by some of the questions in this

¹⁶⁴ Ibid.

¹⁶⁵ Ibid.

¹⁶⁶ Ibid.

¹⁶⁷ Buechner.

survey.¹⁶⁸ The information for this database is collected through telephone interviews of 2500-2600 randomly selected Rhode Island households.¹⁶⁹

Therefore, the accuracy of the data is based on self-report by the people who are being interviewed.

Demographics for Various Diseases

The majority of the demographic information contained in these databases is consistent among databases with a few small differences. The basic format is composed of street address, city, state, zip code, county, and census tract. The hospital discharge data do not contain a street address, and The Health Interview Survey only contains information about city, state, county, and primary care area.¹⁷⁰

All of the databases record information about race. The databases that are considered vital statistics, specifically birth records, death records, and fetal death records, define “Hispanic” more precisely according to country of origin, rather than as a general group like the other databases.¹⁷¹ This demographic information is an important factor when doing studies that investigate trends of disease or the incidence of disease within a certain sub-population.

¹⁶⁸ Ibid.

¹⁶⁹ *Health Data Inventory*.

¹⁷⁰ Ibid.

¹⁷¹ Buechner.

UTILIZATION OF RHODE ISLAND HOSPITALS

Detailed information about the Hospital Discharge data are summarized in the report titled Utilization of Rhode Island Hospitals. This report compiles information regarding the in-patient care for people admitted to non-Federal acute care general hospitals within the state. These data are collected from all eligible hospitals through a continuous reporting system called the Rhode Island Uniform Hospital Discharge Data System (UHDDS). Rhode Island General Laws 23-17-10 requires hospitals to comply with this reporting system as of October 1, 1989. Participating hospitals include: Kent County, Landmark, Memorial, Miriam, Newport, Rhode Island, Roger Williams, St. Joseph's, South County, Westerly, and Womens and Infants.

The Department of Health summarizes these data in a report entitled Utilization of Rhode Island Hospitals. The first of these reports encompasses hospitalization use between October 1, 1994 and September 30, 1995. The Department of Health is currently working on another report that covers more recent data.

Utilization of Rhode Island Hospitals organizes the information from in-patient records in a variety of ways. It presents data according to hospital, patient age, patient sex, diagnostic code groupings, procedure code groupings, and grouped charges. It also includes tables on specific topics such as obstetrical utilization and external cause of injury. The analysis and presentation of the data have been done in a manner as similar as possible to that of the annual summary

reports from the National Hospital Discharge Survey. In accordance with the national format, the Rhode Island report excludes all data pertaining to discharges of newborn infants.¹⁷²

Conditions diagnosed and procedures performed recorded on hospital records are coded according to *The International Classification of Diseases, 9th Revision, Clinical Modification* (ICD-9-CM). These diagnoses are divided into 18 major groups, and 39 specific categories within these groups. The procedures are divided into 16 major groups and 43 specific categories. Each discharge report lists the first seven diagnoses and the first ten procedures. The Department of Health chose to present in this report several specific categories within these diagnostic and procedure groups that were of special interest or of high frequency.¹⁷³

Utilization data regarding discharges, patient days and average length of stay are organized by age group and sex. Age is divided into four categories: under 15 years, between 15-44 years, between 45-64 years, and over 65 years. All of the information presented here is also presented as a rate per 1,000 population, based on population estimates obtained from the Bureau of the Census, United States Department of Commerce for the state of Rhode Island as of July 1, 1995. This is the source of population information used for all of rate calculations throughout this document.

¹⁷² Casey, David B, MBA, Buechner, Jay S, Ph.D. *Utilization of Rhode Island Hospitals: October 1, 1994- September 30, 1995*. Office of Health Statistics, Rhode Island Department of Health: October 1997.

The report presents discharges based on first-listed diagnosis group in several different tables. The analysis in this section does not take into account any patient with an unknown diagnosis. One table displays discharge data based on sex and first-listed diagnosis group; another displays length of stay based on sex and first listed diagnosis group. The discharge rate per 10,000 population by first-listed diagnosis is calculated and incorporated into a table with the rate for the United States which allows for easy comparison between Rhode Island and national data. Discharge information is also presented in a table listing the leading reasons for hospital admission based on sex and first-listed diagnosis.

Information is organized not only by diagnosis, but also by reported procedures being performed during the inpatient stay. The data here exclude any discharge that does not have a listed procedure. Similarly to data described previously, the raw numbers are reported according to sex and procedure group, as well as the rate of each procedure per 100,000 population in Rhode Island as compared with national statistics for procedures. A smaller table also presents a listing of the most commonly performed procedures by sex and procedure category.

Another section of the *Utilization of Rhode Island Hospital* report is dedicated to obstetrical utilization. These data are presented by hospital and delivery. The delivery category is further sub-divided into more specific

¹⁷³ Buechner.

classifications: total vaginal deliveries, vaginal deliveries after a previous cesarean delivery, total cesarean delivery, primary cesarean delivery, and cesarean after previous cesarean delivery. The rates for total and primary cesarean deliveries, as well as the rate of vaginal deliveries after previous cesarean, are also included. Another table presents the average length of stay by type of delivery. This average is calculated after identifying women who were admitted and discharged on the same day as having a length of stay equal to one day.

The final category by which hospital discharge data are organized is by external cause of injury. The percent of total discharges is calculated for each cause of injury. Data included here represent discharges that list injury as the first-listed diagnosis. The same data are also presented according to age groups.

The organization of the data in this report is not based on specific environmental health problems. However, certain health effects related to the environment, such as asthma and cancers, are incorporated into some of the tables, which may be helpful in evaluating environmental health data and developing environmental health indicators.

Now that indicators have been explained and the health data collected in Rhode Island have been summarized, each of the following chapters will focus on one of the health effects investigated in this thesis. They include: asthma, melanomas and other types of skin cancer, lung disease and lung cancer, lead

poisoning, heavy metal poisoning, carbon monoxide poisoning, miscarriages,
and birth defects.

CHAPTER 4 : ASTHMA

ABOUT THE DISEASE

Asthma is an environmental health problem that has become more prevalent over the years. Asthma is a lung disease, characterized by airway obstruction (reversible either spontaneously or with treatment), airway inflammation, and airway hyperresponsiveness to various stimuli.¹⁷⁴

Airway obstruction causes the clinical manifestations of asthma, such as wheezing, dyspnea, and cough, and is thought to be initiated by inflammatory events in the airways.¹⁷⁵ The narrowing of the airways results from the contraction of the smooth muscles of the airway passage, secretion of mucus, and inflammation of the airways.¹⁷⁶

Several different inflammatory cells infiltrate the airways of asthma patients, which then cause complex interactions that result in epithelial disruption and mucosal edema.¹⁷⁷ Epithelial injury can increase permeability and sensitivity to inhaled allergens, irritants, and inflammatory mediators.¹⁷⁸

Airway hyperresponsiveness is an extreme bronchoconstrictor response to any number of physical, chemical, and pharmacological agents, such as

¹⁷⁴ Colorado HealthNet, 1997. http://bcn.boulder.co.us/health/chn/site/idx_asthma.html.

¹⁷⁵ Ibid.

¹⁷⁶ Beggs, Paul John and Curson, Peter Hayden. *An integrated Environmental Asthma Model*. Archives of Environmental Health, vol 50 (no 2): 1995, p 87-94.

¹⁷⁷ Colorado HealthNet, 1997.

¹⁷⁸ Ibid.

environmental irritants, viral respiratory infections, cold air, or exercise.¹⁷⁹

Airway inflammation is a key factor in hyperresponsiveness, and the level of hyperresponsiveness tends to correlate with the clinical severity of asthma.

Asthma can be subdivided into three different categories: extrinsic asthma, when sensitization to a certain allergen can be specified;¹⁸⁰ intrinsic asthma, when no allergen can be identified and a third type, exercise-induced, when the cause is not allergic or pollution related, but rather increased physical activity.¹⁸¹

The cause of asthma is controversial. There is evidence that a genetic link is a major predisposing factor in the development of asthma.¹⁸² Asthma is 26% more prevalent among African-American children than among white children, and African Americans with asthma experience more severe disability and have more frequent hospitalizations than white children.¹⁸³ Despite these statistics the majority of evidence suggests that environmental factors are more important than racial factors in the onset and persistence of asthma.¹⁸⁴ Studies indicate that viral respiratory infections and high allergen exposure during infancy increase the risk of developing asthma.¹⁸⁵ The association between the seasonal peaks of

¹⁷⁹ Ibid.

¹⁸⁰ Koren, HS. *Environmental Risk Factors in Atopic Asthma*. International Archives of Allergy and Immunology. 1997 May-July; 113 (1-3): 65-68.

¹⁸¹ Ibid, p67.

¹⁸² Beggs and Curson.

¹⁸³ National Institute of Allergy and Infectious Diseases: National Institute of Health, June 1996. www.niaid.nih.gov/factsheets/allergystat.htm

¹⁸⁴ Ibid.

¹⁸⁵ Beggs and Curson.

respiratory tract infections and the exacerbation of asthma suggests that these viral infections related to climate can provoke acute asthma.¹⁸⁶

Ozone, a prime ingredient in smog, is one atmospheric pollutant that causes respiratory problems.¹⁸⁷ Ozone in the stratosphere absorbs ultraviolet radiation and prevents the harmful rays from penetrating into the earth's atmosphere.¹⁸⁸ However, tropospheric or ground-level ozone can have a harmful effect on human health. Ozone (O₃), a highly reactive gaseous form of oxygen results from the interaction of sunlight and volatile organic compounds and nitrogen oxides.¹⁸⁹ Volatile organic compounds are emitted from motor vehicles, chemical plants, refineries, factories, and other industrial sources. Nitric oxides result from motor vehicles, power plants, and other forms of combustion.¹⁹⁰ Ozone levels are higher between May and September as a result of higher temperatures, increased sunlight, and stagnant atmospheric conditions.¹⁹¹

Exposure to ground-level ozone aggravates a variety of respiratory problems, including asthma, among people already suffering from such diseases. This powerful respiratory irritant may cause shortness of breath, chest pain when inhaling, wheezing, and coughing.¹⁹² People with pre-existing respiratory

¹⁸⁶ Ibid.

¹⁸⁷ *Health and Environmental Effects of Ground-Level Ozone Fact Sheet*. USEPA Office of Air and Radiation, Office of Air Quality Planning and Standards, July 17, 1997.
<http://ttn.www.rtpnc.epa.gov/naaqsfm/o3health.htm>

¹⁸⁸ Philp, p99.

¹⁸⁹ *Health and Environmental Effects of Ground-Level Ozone Fact Sheet*.

¹⁹⁰ Ibid.

¹⁹¹ *American Lung Association Fact Sheet- Ozone Air Pollution: 1997*.

<http://www.lungusa.org/global/news/report/>

¹⁹² Ibid.

conditions such as asthma, chronic bronchitis, or emphysema are more sensitive to the effects of exposure to ground-level ozone. The National Ambient Air Quality Standard (NAAQS) for ozone was 0.12 parts per million averaged over one hour until 1997 when it was reduced to 0.08ppm. This level has not been in place long enough to determine whether the change will have a significant impact on human health; however, studies involving healthy adults and children undergoing moderate exercise while exposed to levels lower than this standard demonstrate decreased functioning of the lungs and respiratory systems.¹⁹³

One study conducted by Weisel et al. reported that asthmatics and average individuals had similar reactions when exposed to ozone, but the asthmatics had a greater response than the non-asthmatics when later exposed to allergens.¹⁹⁴ This study also reported that there was a significant increase in the number of emergency department visits for asthma on days when there was a high level of smog and tropospheric ozone for all five years over which the study was conducted.¹⁹⁵ There were an increased number of visits on days when ozone levels were greater than 0.06 ppm. This increase is even greater when the emergency department is located in a city. For instance, one year on a high-

¹⁹³ Ibid.

¹⁹⁴ Weisal, CP et.al. *Relationship Between Summertime Ambient Ozone Levels and Emergency Department Visits for Asthma in Central New Jersey*. Environmental Health Perspective. 1995 March; 103 supplemental 2: 97-102.

¹⁹⁵ Ibid.

ozone day, the rate of visits for asthma in Atlanta was 38% higher than the rate on an average ozone-level day.¹⁹⁶

Children are particularly sensitive to exposure to ground-level ozone for several reasons. Children inhale more air per pound of body weight than adults, and since their respiratory system is not fully developed, they are more susceptible to environmental pollutants.¹⁹⁷ Children are also more often exposed to ozone because they tend to play and exercise outdoors during the summer months when ground-level ozone is at its peak.¹⁹⁸

Children are more susceptible to asthma. Although children comprise twenty five percent of the population, they account for forty percent of the asthma cases that require treatment.¹⁹⁹ Ozone can aggravate asthma, not only by increasing the number of attacks that an individual may suffer, but also by increasing the severity of the attack as well.²⁰⁰ Epidemiological studies show that hospital admissions for asthma implicate ozone as a contributing factor to the exacerbation of this respiratory disease.²⁰¹

Evidence suggests that tropospheric ozone has contributed to the growing rate of respiratory problems over the years. Ozone exposure does not cause the health effects, but rather aggravates the problem among those who are

¹⁹⁶ Ibid.

¹⁹⁷ *Health and Environmental Effects of Ground-Level Ozone Fact Sheet.*

¹⁹⁸ *American Lung Association Fact Sheet- Children and Ozone Air Pollution: 1997.*

<http://www.lungusa.org/global/news/report/>

¹⁹⁹ *Health and Environmental Effects of Ground-Level Ozone Fact Sheet.*

²⁰⁰ *American Lung Association Fact Sheet- Ozone Air Pollution: 1997.*

²⁰¹ Koren, HS. *Environmental Risk Factors in Atopic Asthma.*

already afflicted. From 1990-1994, the number of people with self-reported asthma in the United States has increased from 10.4 million to 14.6 million.²⁰² The increased number of attacks leads to increased use of medication, more medical treatment, and more visits to the emergency room.²⁰³ All of these factors reflect an increasing health problem associated with asthma.

RHODE ISLAND DATA

The State of Rhode Island collects relatively limited data about the incidence of asthma. It appears in the hospital discharge data that are organized by first-listed diagnosis coded according to the *International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)*.²⁰⁴ The discharge data are presented in two tables: one tabulates the number of asthma cases by sex; the other presents the information as a rate per 10,000 population for Rhode Island and compares this to national rates.

1839 patients with a first-listed diagnosis of asthma and an average stay of 3.7 days were discharged from Rhode Island hospitals between October 1, 1994 and September 30, 1995. Of these cases, 731 were male and had an average length of stay of 3.1 days, and 1108 were female with an average length of stay of 4.1 days. This rate of asthma-related hospitalizations (based on first-listed diagnosis) per 10,000 population in the state of Rhode Island is 18.6, slightly lower than the national rate of 19.5.

²⁰² National Institute of Allergy and Infectious Diseases.

The Department of Health also has information regarding the mortality rate from asthma. These data are not compiled in an individual database, but the number of deaths caused by asthma can be tabulated from the cause of death listed on the death certificates. The cause of death is coded according to the ICD-9-CM, the same system used to code hospital discharge data. Although this data is not readily available, it can easily be obtained.

INDICATORS

There are many different aspects of asthma that can be evaluated; therefore the most desirable information must be determined in order to develop the most appropriate indicator. Evaluations of the burden of this disease based on mortality rather than morbidity would greatly underestimate the importance of asthma because the number of deaths from asthma is relatively low in comparison to the number of people who suffer from the disease.²⁰⁵ Ideally, the desired goal is to prevent the disease from occurring. However, since the cause of asthma is unknown, it is impossible to prevent. The next best thing is to decrease the rate of attack among people who have the disease. In order to gather this information, an indicator should not focus on the number of people in the population who suffer from the disease, but rather on the rate of attack among those people afflicted with the disease.

²⁰³ *Health and Environmental Effects of Ground-Level Ozone Fact Sheet.*

²⁰⁴ Casey and Buechner.

²⁰⁵ Beggs and Curson.

The information collected by the state regarding hospital discharge data and mortality rates is helpful in determining trends of asthma, but the data are limited to the most severe cases of asthma. Additional information about the less severe cases, such as those cases that are treated in doctors' offices or in the emergency room, rather than requiring hospital admission, would be useful.

One concern might be that an indicator based on medical records would underestimate the incidence of asthma because certain subgroups of the population, such as those who cannot afford or chose not to seek medical care, would be excluded from the data. For example, if fewer people from the lower socioeconomic class seek medical treatment for asthma because of financial reasons, but suffer from more frequent attacks because of increased exposure to pollution and environmental contaminants in their homes, because their homes are located in the inner cities and low-income communities, then the incidence of asthma based on mortality, hospitalizations, and doctor visits would be underestimated. The valuable characteristic of an indicator, however, is that it does not have to be directly representative of incidence in order to be informative. Despite the pockets of missing data, the trend determined by the information that is collected would indicate whether environmental health is improving or deteriorating overall.

The difficulty arises however, in collecting this information from private practices and other health care facilities across the state. The greatest limiting

factors involved with collecting these data are funding to implement a reporting system, and compliance among physicians to actually report.

One possible way to collect data regarding less severe cases of asthma would be to add asthma to the list of reportable diseases. Currently, the state of Rhode Island requires several communicable, environmental, and occupational diseases to be reported to the state, either electronically, by phone, or by mail. Since asthma is not a dangerous communicable disease, reporting by mail would be the most appropriate method.

The state could supply physicians and health care facilities with cards on which to record information about each case of asthma that they treat. Aside from basic demographics, circumstances surrounding the asthma attack, such as exercise or occupational exposure at the time of the attack, could be recorded on these cards as well. A coding system similar to the discharge diagnosis used by hospitals could be developed to characterize the circumstances associated with or known to cause an asthma attack. The simple code, rather than a short description could then be recorded on the card. Another possibility would be a listing of common circumstances surrounding an attack that physicians would just check off, rather than learning new codes. These are some data that would be useful to collect.

These cards could then be mailed to the state at the end of each week or the end of each month, and the data could be compiled by the Department of Health. If the cards are well organized and simple to complete, they would

require a minimal effort to fill out and mail. Realistically, however, the compliance rate for this type of reporting would be low; physicians have so much paper work as it is, that many of them may not be willing to complete additional forms.

Rather than requiring reporting of asthma across the state, a more feasible option would be implementing a sentinel system. In this case, a select group of physicians and health care facilities are chosen as representative of the general population; these select few, rather than everyone across the state, are required to report. Compliance rates may be higher and the possibility of an incentive to report may be a realistic option when working on a smaller scale. Doctors that volunteer to participate may not be representative of the average physician, however, that does not mean that his or her patients are not representative of the average population. This type of system would result in an accumulation of valuable data as long as an evaluation of the patients was done to ensure that they were a representative sample of the population.

Another possibility which may be feasible in the future when better technology is available, would be an electronic reporting system similar to the system that is currently used to collect data on blood lead levels. As of 1993, the Center for Disease Control began conducting a surveillance of lead poisoning in five states, and the program has been expanding throughout the United States.²⁰⁶

²⁰⁶ Bullard, Robert. Confronting Environmental Racism: Voices From the Grassroots. South End Press, Boston: 1993, p88.

It is a laboratory-based reporting system which allows labs to report their blood lead analysis to the CDC using a standard software package. The CDC then compiles the data and calculates the statewide incidence and prevalence.²⁰⁷

In the state of Rhode Island, lead data are reported electronically to the Department of Health either through a modem or by diskette.²⁰⁸ This system works because there are only a few labs across the state that measure blood lead levels; however, it would be difficult to implement for physicians because of the much larger number of private practices that exist and the variety of software packages that are in use. Improved technology and adequate funding could surpass these limitation and electronic reporting may be a realistic option in the future. Another possibility could be a reporting system over the internet. Rather than attempting to implement the same or similar software packages in all doctors' offices, the state could assist physicians in getting networked. The Health Department could then put the reporting forms on a web page. Physicians could then fill them out and forward them directly to the state. This would create consistency while by-passing some paperwork. Implementing this type of system would be a worthwhile effort because access to the internet would provide capabilities beyond that of simply reporting data to the state.

When developing indicators for asthma, informative data are not limited to medical records. For instance, monitoring the sale of prescription drugs for

²⁰⁷Ibid, p88.

²⁰⁸ Feeley, March 9, 1997

asthma or the sale of inhalers, in addition to the rate of asthma attacks could provide useful information. An increased use of medication may reflect additional treatment in response to an increased rate of asthma attacks, or it may suggest an increase in preventative treatment leading to a decrease in the rate of asthma attacks. These data, in combination with the rate of attack, could determine whether environmental health is improving. Evaluating the sale of preventative asthma medication over time is also one way to evaluate educational and preventative programs, or pollution standards that have been implemented to try to reduce the rate of asthma. Increased sales, accompanied by decreased rates of asthma attacks, would suggest that these efforts have been successful. Increased sales, accompanied by increased rates of asthma attacks, would suggest that these programs are not helping to improve the state of health among asthmatics, and that a stronger effort or a different approach may be necessary to more successfully address the problem .

Using data from medication sales is an informative indicator, but it also has some limitations. Similar to collecting data from doctors, collecting data from pharmacists depends on compliance to report the sale of the drug. Collecting these data directly from the manufacturer or supplier rather than the pharmacists may avoid the issue of compliance, however valuable information regarding where the drugs are being distributed may be lost. Another consideration to keep in mind is that the sale of the prescription drugs does not

ensure that the patient is actually taking the medication. Despite this factor, the overall trend in the sales is a good indicator of treatment.

CHAPTER 5 : MELANOMAS AND OTHER SKIN CANCERS

ABOUT THE DISEASE

Skin cancer, the most common type of cancer in the US, affects an average of 600,000 Americans annually.²⁰⁹ It is most common among light-skinned individuals who are exposed to ultraviolet rays from natural or reflected sunlight, or tanning beds for prolonged periods of time, or who have undergone a considerable number of X-rays.²¹⁰ Skin cancer tends to occur on places on the body that are consistently exposed to sunlight, such as the face, neck, hands, and arms.²¹¹ Farmers, fishermen, lifeguards, construction workers, and other outdoor laborers have an increased risk of developing skin cancer because of their constant exposure to the sun.²¹²

There are three main types of skin cells that can be affected by cancer: flat, scaly squamous cells; round basal cells; and melanocytes, cells that give skin its color.²¹³ Basal cell carcinoma is the most prevalent non-melanoma and has a very good prognosis.²¹⁴ It is usually a small raised bump with a smooth pearly appearance that is firm to the touch. It may sometimes resemble a scar.²¹⁵ This

²⁰⁹ CancerNet from the National Cancer Institute. www.gretmar.com/cancernet/304724.txt

²¹⁰ Sabatini, MM. *Dermatology Nursing*. 7(1):45-50, Feb.95.

<http://chid.aerie.com/simple/simple.html>

²¹¹ Ibid.

²¹² Ibid.

²¹³ CancerNet from the National Cancer Institute.

²¹⁴ Ibid.

²¹⁵ Ibid.

type of cancer may spread to other tissues surrounding the cancerous cells, but it does not spread to other parts of the body.²¹⁶

Squamous cell carcinoma appears on areas of the skin that have been exposed to a great deal of sunlight, but also in areas that have been burned, exposed to chemicals, or treated with X-ray therapy.²¹⁷ This type of cancer usually looks like a firm red bump, which may sometimes feel scaly, bleed, or crust. Squamous cell carcinoma also has a good prognosis, but unlike basal cell, it may spread to the lymph nodes.

Basal cell and squamous cell carcinomas are most prevalent in Florida, Hawaii, California, Texas, and Arizona. The incidence of these cancers is correlated with states that are closest to the equator and areas with increased ultraviolet radiation.²¹⁸

These two types of skin cancer are easily detected and easily treated in a doctor's office. Surgery, used in 90% of the cases, cuts the lesion from the skin along with some healthy cells through an excision.²¹⁹ Micrographic surgery removes the cancerous cells, and as little normal tissue as possible.²²⁰ Several non-surgical treatment options exist as well: electrodesiccation uses heat to destroy the affected tissue, cryosurgery destroys the lesion by freezing, and laser therapy, used only if the skin cancer is detected early, burns the cancerous cells

²¹⁶ Ibid.

²¹⁷ Ibid.

²¹⁸ Sabatini.

²¹⁹ *Cancer Facts and Figures-1997*. American Cancer Society.
www.cancer.org/statistics/97cff/97facts.html

with a narrow beam of light.²²¹ These treatments are usually very effective; however, people with skin cancer are at high risk for developing additional skin cancers.²²²

The third and most serious type of skin cancer, melanoma, initially affects the melanocytes, the cells that give skin its color. This cancer, also known as cutaneous melanoma or malignant melanoma, is the most infrequently encountered but most serious type of skin cancer, accounting for 75% of all deaths from skin cancer.²²³ It can metastasize quickly to other parts of the body through the lymph system or through the blood.²²⁴

The exact cause of melanomas is unknown, but exposure to ultraviolet radiation is most commonly associated with this disease. Rather than resulting from chronic overexposure to UV rays like nonmelanoma skin cancers, melanomas are caused by intense intermittent exposure to UV rays, especially during early childhood and teenage years.²²⁵

Early signs of melanomas can be detected as changes in the size, shape, and color of existing moles. Precautions should also be taken if moles become hard, itchy, lumpy, swollen, or tender. Melanomas may also present themselves as new moles. They most commonly appear on the trunk, head, and neck of

²²⁰ Ibid.

²²¹ Ibid.

²²² CancerNet from the National Cancer Institute.

²²³ Ibid.

²²⁴ Ibid.

²²⁵ *Cancer Rates and Risks, 4th Edition*. National Institute of Health, National Cancer Institute: 1996, p164.

men, and on the arms and legs of women.²²⁶ Individuals with a history of melanoma are more likely to develop a second one.²²⁷

The primary treatment for melanomas is surgery. An operation known as a conservative re-excision takes out any cancer and a small amount of healthy skin around it, and a wide surgical excision removes any cancer and more of the healthy skin surrounding the tumor to ensure that all the malignant cells have been removed.²²⁸ Chemotherapy to kill any remaining cancerous cells may follow the surgery depending on the individual needs of each patient.

The most effective way to prevent skin cancer is to avoid exposure to ultraviolet light by limiting sun exposure, using sunscreen, and wearing wide brimmed hats and other protective clothing.²²⁹ It is also suggested that adults practice skin self-exams once a month in order to detect possible lesions early.²³⁰

These protective measures have been promoted for years, but recent studies state that although the use of sunscreen has increased over the last 25 years, so has the rate of skin cancer.²³¹ The mortality rate from melanomas has increased at a rate of approximately 4% per year in recent years.²³² In 1997, about 40,300 people were expected to develop melanomas and 7,300 were expected to

²²⁶ Ibid.

²²⁷ CancerNet from the National Cancer Institute.

²²⁸ Ibid.

²²⁹ Ibid.

²³⁰ *Cancer Facts and Figures-1997*.

²³¹ *Studies Doubt Sunscreens Stop a Cancer*. The New York Times, Wed Feb 18, 1998. pA17.

²³² CancerNet from the National Cancer Institute.

die.²³³ Some studies suggest that the risk of melanomas may actually be increased by using sunscreen because it allows the most susceptible individuals, those with a light complexion, to spend more time in the sun.²³⁴ Despite this evidence, other doctors believe that, because of the long latency period, the exposure that caused the most recent cases of melanomas took place prior to the increased use of sunscreen; therefore, it is too early to declare that sunscreen provides no protection against skin cancer.²³⁵

RHODE ISLAND DATA

The Rhode Island Department of Health collects information about melanomas in the cancer registry. The records are organized according to case, so they can be sorted by type of cancer and then analyzed.²³⁶ Since each newly diagnosed case of cancer is required to be reported to the state, the database contains comprehensive records about the incidence of melanomas in Rhode Island. Each record contains information regarding demographics, tumor site, stage at diagnosis, treatment information, and data about any other cancer in the body.²³⁷

Data regarding basal cell and squamous cell carcinomas are not collected at the state level for several reasons. First, these types of skin cancers are very common and easily treatable. People with squamous cell or basal cell

²³³ Ibid.

²³⁴ *Studies Doubt Sunscreens Stop a Cancer*. The New York Times, Wed Feb 18, 1998. pA17.

²³⁵ Ibid.

²³⁶ John Fulton, Dec 5, 1997.

carcinomas also tend to have multiple small lesions; this does not lend itself to being recorded in the type of database that is currently maintained in which each record is a case of cancer rather than a patient.²³⁸ In addition, the lesions are usually treated in a physician's office and do not require hospitalization. This presents the dilemma involved with collecting medical records from private practices, a very difficult, costly, and complicated task.²³⁹

Data regarding the mortality rates of melanomas are also available from the state. This information on melanomas is not maintained in a separate database from all other cancers, but it can easily be obtained by examining the cause of death listed on the death records.

INDICATORS

Extracting the records of melanomas from the cancer registry and interpreting the trend over time is a useful indicator. It provides a count of the number of cases of melanomas that develop in the course of the year, which, when examined over time, demonstrates the trend that the health effect is taking. Since melanomas have a long latency period, it is necessary to examine long-term results; the effects of education and preventative measures initiated today would not be apparent for thirty to forty years. The information obtained from the direction of the curve established by the rate of melanomas, in addition to the trend depicted by the mortality rates from this disease, can be used to determine

²³⁷ Ibid.

whether or not early detection programs are working, and whether education and prevention efforts have been successful. This knowledge can influence decisions of officials regarding where to focus more attention and funding. These indicators may also provide additional knowledge about the disease, and my help answer questions, such as whether or not sunscreen actually provides protection against the harmful ultraviolet rays that cause melanomas.

The data currently collected in Rhode Island act as a good health-end effect indicator. This is the most direct measure of human health and therefore, very informative. For example, it is a much more precise indicator than an estimate of exposure to ultraviolet rays because of the variability that exists among individuals in terms of exposure as well as sensitivity to exposure. No biomarker or body burden exists that could act as a measure of melanomas at the stage in between exposure and effect. Consequently, the best indicator is based on the data that are currently being collected by the state.

Additional information about the rates of basal cell and squamous cell carcinomas could be useful; however, since exposure to ultraviolet rays are the main cause of all three types of skin cancers, it is likely that the trends depicted by basal cell and squamous cell carcinomas would be similar to those of melanomas. Keeping this in mind, as well as the cost and difficulties involved with collecting these data from doctors' offices, compiling this information at the

²³⁸ Ibid.

²³⁹ Ibid.

state level may not be cost-effective. Perhaps in the future when better technology is available to implement an electronic reporting system, such as the ones described for asthma, as well as additional funding, gathering data on basal cell and squamous cell carcinomas may be worthwhile.

In the meantime, however, a more feasible indicator may be tracking the sales or use of sunscreen. Monitoring sales of sunscreen either at the store level or from the distributor would provide us with limited, but informative data. For example, the purchase of sunscreen does not guarantee its use, but it does provide information about what portion of the population are aware of the dangers and are attempting to protect themselves from damaging ultraviolet rays.

The actual use of the sunscreen would be more accurately recorded through self-reports of personal behaviors regarding sun exposure and protective measures. One way to obtain this information would be to incorporate several questions about sunscreen use and sun exposure into the Health Interview Survey that is conducted by the state about every five years.²⁴⁰ This is a survey taken from a random sample of 2500-2600 Rhode Island households with a phone, where an adult agrees to report for all members of the household.²⁴¹ Examining the results of this survey over time will provide information as to whether the use of sunscreen is increasing or decreasing.

²⁴⁰ *Health Data Inventory*.

²⁴¹ *Ibid.*

However, the possibility of recall bias must be considered when evaluating these data. The information gained from this process, in addition to the rate of melanomas, would be helpful in future studies that try to determine whether sunscreen protects against the development of melanomas.

CHAPTER 6 : LUNG DISEASE AND LUNG CANCER

ABOUT THE DISEASES

The number of deaths from lung disease (including asthma) in America is topped only by cancer and heart disease.²⁴² This life-threatening disease encompasses a variety of conditions that impair or disrupt the normal functioning of the lungs and inhibit the supply of oxygen to the body.²⁴³ Lung disease may be caused by a number of different factors, such as air pollutants that irritate the airways, carcinogens that promote tumor growth, tobacco smoke, and infectious agents that interfere with the biological responsibilities of the lungs.²⁴⁴

The symptoms that characterize lung disease vary as much as the causes of the disease. Some conditions involve wheezing, chest pains, or shortness of breath while exercising; other disorders may involve a persistent cough, sometimes accompanied by sputum or blood. Lung disease caused by an infectious agent may result in fever or chills. Patients may demonstrate any number or combination of these symptoms.²⁴⁵

Lung cancer, although diagnosed separately from lung disease, is also a leading cause of death in America. It is the most common fatal malignancy in the

²⁴² *Lung Disease Data 1996*. American Lung Association, p1.

²⁴³ *Ibid*, p2.

²⁴⁴ *Ibid*, p3.

²⁴⁵ *Ibid*, p3.

United States.²⁴⁶ The prognosis for most cases of lung cancer is generally grim, but somewhat more positive if the disease is diagnosed early, before the tumor cells have had the opportunity to spread to other parts of the body. The difficulty arises, however, in early detection because the beginning stages of the disease are usually asymptomatic; patients show no sign of coughing, pain, or shortness of breath.²⁴⁷

Cigarette smoke is the most common cause of lung cancer, responsible for 87% of all cases²⁴⁸. Some of the other substances that cause lung disease, however, are also responsible for causing or exacerbating lung cancer.

One of the major causes of concern for lung disease, as well as lung cancer, is air pollution. The most dangerous components of indoor air pollution are asbestos, radon, and environmental tobacco smoke. Outdoors, particulate matter is also of great concern. Each of these causes is discussed in more detail below.

ASBESTOS

Asbestos is the name given to a variety of fibrous mineral silicates. They occur naturally in the form of strong, flexible fibers that can be separated into thin threads and woven.²⁴⁹ This material is used in a variety of industries because of its tensile strength and flexibility, as well as its resistance to heat,

²⁴⁶ Ibid, p5.

²⁴⁷ Ibid, p7.

²⁴⁸ Ibid, p7.

chemicals, acids, and friction.²⁵⁰ Some of its many uses include insulation, fireproofing, sound absorption, and strengthening material for cement and plastics. It is commonly found in the brake linings and clutch pads of automobiles, as well as in many fire-resistant fabrics, paints, and heat-protective materials.²⁵¹

Four types of asbestos are commonly used, and are often differentiated by their color. The most common type, used in 90% of asbestos industry, is chrysotile, or white asbestos, which exists as curly, flexible fibers.²⁵² Anthophyllite is also white, but these fibers are straight and brittle.²⁵³ Amosite is light gray to pale brown in color and is composed of straight, brittle fibers.²⁵⁴ Crocidolite, also known as blue asbestos, is straight blue fibers.²⁵⁵ All of the different types of fibers tend to break easily into tiny particles of dust which can float and stick to clothing. Asbestos that is sealed into finished products such as walls, tiles, and pipes does not pose a threat to human health; however, damaging the material by drilling or sawing, for example, releases the fibers into the air where they become a hazard.²⁵⁶ Serious health problems occur when

²⁴⁹*Cancer Facts: Questions and Answers About Asbestos Exposure.* National Cancer Institute, National Institute of Health. May 1994, p1.

²⁵⁰ Waldbott, George L. *Health Effects of Environmental Pollutants.* The CV Mosby Company, St. Louis: 1978, p 211.

²⁵¹ *Cancer Facts: Questions and Answers About Asbestos Exposure.*, p2.

²⁵² *Ibid*, p1.

²⁵³ *Ibid*, p1.

²⁵⁴ *Ibid*, p1.

²⁵⁵ *Ibid*, p1.

²⁵⁶ *Ibid*, p3.

these fibers are inhaled or swallowed.²⁵⁷ Once these almost indestructible fibers reach the lungs, they cannot be expelled or dissolved.²⁵⁸

Asbestos exposure greatly increases an individual's risk of developing several different lung diseases. Asbestosis, also known as "white lung syndrome," is a form of fibrotic pneumoconiosis²⁵⁹ which develops slowly and usually manifests itself 20-30 years after the exposure.²⁶⁰ It is a chronic lung ailment characterized by shortness of breath and lung damage. Asbestosis is also known to increase the risk of developing other dangerous lung infections.²⁶¹

Another adverse effect is mesothelioma, a special type of cancer linked specifically to asbestos exposure. It affects the thin membranes that line the chest (pleura) and the abdomen, and it metastasizes quickly to other parts of the abdominal cavity.²⁶² This disease also has a long latency period and usually occurs 30-40 years after the first exposure.²⁶³

Asbestos also causes several other cancers, including cancers of the lung, larynx, and gastrointestinal tract.²⁶⁴ Within the population of people exposed to asbestos, the risk of developing lung cancer (not mesothelioma), increases tremendously among those who smoke. Recent studies indicate that the rate of carcinomas of the lung for exposed smokers is 60x greater than exposed non-

²⁵⁷ Ibid, p1.

²⁵⁸ Waldbott, p 211.

²⁵⁹ Philp, p96.

²⁶⁰ Waldbott, p211.

²⁶¹ *Cancer Facts: Questions and Answers About Asbestos Exposure.*, p2.

²⁶² Philp, p96.

²⁶³ Ibid, p96.

smokers,²⁶⁵ and 90x greater than non-exposed non-smokers,²⁶⁶ a significant increase in risk. A synergistic effect between asbestos and cigarette smoke also increases the development of bronchogenic carcinomas.²⁶⁷ The toxicological interaction between the two substances yields altered biological responses that are far worse than the predicted additive effects of the two chemicals working independently.²⁶⁸ The two exposures may be simultaneous or sequential, but the combination of the two yields dramatically increased risk. This risk however, can be reduced by half or more if an exposed individual quits smoking for at least five years.²⁶⁹

Signs of asbestos-related illnesses do not usually manifest themselves until many years following the initial exposure. Despite the long latency period, early detection of a disease is important for successful treatment.²⁷⁰ People who have been or may have been exposed to asbestos, particularly through occupational exposure, should be aware of symptoms such as shortness of breath, persistent cough, blood in the sputum, pain in the chest or abdomen, difficulty swallowing, prolonged hoarseness, and significant weight loss.²⁷¹ A physical examination, as well as a possible chest X-ray, are usually

²⁶⁴ *Cancer Facts: Questions and Answers About Asbestos Exposure.*, p2.

²⁶⁵ Philp, p96

²⁶⁶ *Cancer Facts: Questions and Answers About Asbestos Exposure.*, p4.

²⁶⁷ Human Health and the Environment: Some Research Needs, p42.

²⁶⁸ *Ibid* p160.

²⁶⁹ *Cancer Facts: Questions and Answers About Asbestos Exposure.*, p4.

²⁷⁰ *Ibid*, p3,5.

²⁷¹ *Ibid*, p4.

recommended.²⁷² Once a diagnosis has been made, a variety of treatments, such as surgery, anticancer drugs, or radiation, can be administered to meet the specific needs of the patient.²⁷³

RADON

Radon is a colorless, odorless gas that results from the natural decay of uranium in soil and rock.²⁷⁴ Radon breaks down into components known as radon progeny, often referred to as “radon daughters.” These dangerous molecules emit high-energy alpha particles that increases the risk of developing lung cancer.²⁷⁵ Radon is the second leading cause of lung cancer, preceded only by cigarette smoking.²⁷⁶ The combination of smoking and radon exposure greatly increases the risk of developing lung cancer in comparison to non-smokers who are exposed.²⁷⁷

Radon in buildings originates mainly from surrounding uranium-containing soil such as granite, shale, phosphate, and pitchblende. The dangerous gas, undetectable by the human senses alone, enters structures through openings such as cracks in walls, basement floors, and foundations.²⁷⁸ The level at which remedial actions should be taken has been identified as four

²⁷² Ibid, p4.

²⁷³ Ibid, p5.

²⁷⁴ *American Lung Association Fact Sheet-Radon*. American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfact.html>

²⁷⁵ *Lung Disease Data 1996*. American Lung Association.

²⁷⁶ *American Lung Association Fact Sheet-Radon*.

²⁷⁷ Ibid.

²⁷⁸ Ibid.

picocuries per liter of air (pCi/L). One out of every fifteen houses in the United States has radon levels that exceed this standard. Although underlying geology increases the risk of radon in some sections of the country, homes with elevated levels do not necessarily cluster in the same neighborhoods.²⁷⁹ Therefore, the only way to determine levels of radon is through individual testing, which can easily be done using do-it-yourself home test kits.

Once an increased level of radon has been established, several options are available to remediate the situation. In newly developing areas, the soil under the basement could be ventilated before the houses are built. This is a relatively inexpensive and easy method of prevention. The simplest method of decreasing radon levels in an existing building, is to seal cracks in floors and walls from which the gas may be entering into the house.²⁸⁰ Radon gas can also be removed from underneath the concrete foundation through sub-slab depressurization which uses pipes and fans to move the gas above the roof where it can safely disperse.²⁸¹ Another option is soil depressurization which ventilates the soil so that radon is drawn away from the structure before it enters the building.²⁸² These types of repairs should be made by an EPA or state certified contractor.²⁸³

²⁷⁹Ibid.

²⁸⁰Ibid.

²⁸¹Ibid.

²⁸²Ibid.

²⁸³Ibid.

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke, or secondhand smoke, originates from either the end of a burning cigarette, or from the smoke that is exhaled by the smoker.²⁸⁴ Sidestream smoke accounts for almost half of the smoke that is generated from smoking a cigarette.

Not only does secondhand smoke contain almost all of the carcinogenic and toxic substances that have been identified in the mainstream smoke that the smoker inhales, but it contains increased levels of these contaminants.²⁸⁵ Of the 4000 chemicals contained in secondhand smoke, 200 are known poisons, and 43 are known carcinogens.²⁸⁶ Some of these compounds include tar, carbon monoxide, hydrogen cyanide, phenols, ammonia, formaldehyde, benzene, nitrosoamine, and nicotine.²⁸⁷

Exposure to environmental tobacco smoke causes lung cancer among non-smokers, an estimated 3000 cases a year, as well as other health problems particularly in children.²⁸⁸ EPA estimates state that secondhand smoke causes between 150,000 and 300,000 lower respiratory tract infections in children under 18 months of age annually, 7,500 to 15,000 of which require hospitalizations.²⁸⁹ Children also suffer from exacerbated asthma, pneumonia, ear infections,

²⁸⁴ *Cancer Facts:Environmental Tobacco Smoke*. National Cancer Institute, National Institute of Health: February 8, 1995, p1.

²⁸⁵ *Ibid*.

²⁸⁶ *American Lung Association Fact Sheet- Secondhand Smoke*. American Lung Association, 1997. <http://www.lungusa.org/global/news/report/>

²⁸⁷ *Cancer Facts:Environmental Tobacco Smoke*, p1.

²⁸⁸ *American Lung Association Fact Sheet- Secondhand Smoke*.

bronchitis, coughing, wheezing, and increased production of mucus as a result of exposure to environmental tobacco smoke.²⁹⁰ Exposure to this pollutant in utero, as well as during infancy, causes alterations in the lung function and structure, which ultimately increases a child's risk of long-term pulmonary problems and risk of suffering from Sudden Infant Death Syndrome (SIDS).²⁹¹

Among non-smoking adults, environmental tobacco smoke can exacerbate pulmonary symptoms in people already suffering from asthma, chronic bronchitis, and allergies.²⁹² Healthy individuals exposed to secondhand smoke may also suffer from less severe symptoms, such as eye irritation, sore throat, nausea, and hoarseness.²⁹³

PARTICULATE MATTER

Particulate matter is the term used to describe air pollution consisting of complex and varying mixtures of solid particles and liquid droplets suspended in the air.²⁹⁴ They include carbon-based particles such as soot, ashes, dirt, dust, acid aerosols, and plant matter such as pollen.²⁹⁵ The sources of these particles vary from automobile exhaust, industrial smokestacks, mining, and construction,

²⁸⁹ Ibid.

²⁹⁰ Ibid.

²⁹¹ *Cancer Facts: Environmental Tobacco Smoke*, p4.

²⁹² Ibid, p4.

²⁹³ Ibid, p4.

²⁹⁴ *Fact Sheet: Health and Environmental Effects of Particulate Matter*. United States Environmental Protection Agency, Office of Air and Radiation; Office of Air Quality Planning and Standards. <http://ttnwww.rtpnc.epa.gov/naaqsfm/pmhealth.htm>

²⁹⁵ *Lung Disease Data 1996*.

to simpler things such as unpaved roads and extensive wood-burning.²⁹⁶ Once emitted, particulate matter can remain suspended in the air and travel long distances; the effects of the problem, therefore, may burden people in areas distant from the origin of the pollution.²⁹⁷

High levels of particulate matter in the air have been associated with a variety of significant health problems. Among these are persistent coughs, outbreaks of respiratory illness, aggravated asthma, chronic bronchitis, shortness of breath resulting from decreased lung function, and premature death.²⁹⁸

The elderly and people with pre-existing respiratory problems or heart disease are at greatest risk from particulate matter.²⁹⁹ More specifically, those individuals suffering from asthma or chronic obstructive pulmonary disorder (COPD), including emphysema and chronic bronchitis, are sensitive to exposure to particulates.³⁰⁰ Children are also more susceptible to the effects of particulate matter because they breathe 50% more air per pound of body weight than adults, and because their respiratory systems are still developing. Exposure at a young age can result in short term effects, such as coughing and difficulty breathing, as well as long term effects that interfere with the normal development of healthy lungs.³⁰¹

²⁹⁶ Ibid.

²⁹⁷ *Fact Sheet: Health and Environmental Effects of Particulate Matter*

²⁹⁸ Ibid.

²⁹⁹ *American Lung Association Fact Sheet- Particulate Matter Air Pollution*. American Lung Association, 1997. <http://www.lungusa.org/global/news/report/viron/virmaterfac.html>

³⁰⁰ *Lung Disease Data 1996*.

³⁰¹ *Fact Sheet: Health and Environmental Effects of Particulate Matter*.

Fine particles, less than 2.5 microns in diameter (a human hair is 75 microns), are the most dangerous particulate matter. They are easily inhaled deeply into the lungs. Once inside, they can be absorbed into the bloodstream or can remain embedded in the lungs for an extended period of time.³⁰² These tiny particles easily penetrate the alveoli, the smallest air sacs of the lung; since this area of the lung has a slow clearance system, the deposits remain fixed in the lung and continue to cause damage over time.³⁰³ Coarse particles, larger than 2.5 micrometers in diameter, are less harmful because they tend to get trapped in the nose or throat before they have the opportunity to enter the lungs and cause more severe damage.³⁰⁴

RHODE ISLAND DATA

Lung disease data collected by the Rhode Island Department of Health are part of the hospital discharge records organized according to the first listed diagnosis. There is no specific category for “lung disease” because of the array of symptoms and various characteristics involved with the disease. According to the *International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM)* lung disease falls under the diagnostic category titled “Diseases of the respiratory system.”³⁰⁵ This information is broken down by sex, length of stay, and rate per 10,000 population. This category is divided into four sub-categories:

³⁰² *American Lung Association Fact Sheet- Particulate Matter Air Pollution.*

³⁰³ *Lung Disease Data 1996.*

³⁰⁴ *Ibid.*

³⁰⁵ Casey and Buechner.

Acute respiratory infections; Chronic disease of the tonsils and adenoid; Pneumonia; and Asthma.³⁰⁶ Other lung diseases are specified within these four main categories.

Between October 1, 1994 and September 30, 1995, 11,197 people were discharged from Rhode Island hospitals with a first listed diagnosis of “Diseases of the respiratory system.” Of these, 5,331 were male and 5,866 were female. The total rate per 10,000 population is 113.1, 14.2 points lower than the national rate of 127.3.

All of the data regarding lung cancer is compiled in the cancer registry maintained by the state. As mentioned earlier, Rhode Island law mandates that all newly diagnosed cases of cancer be reported to the state, the cancer registry is a complete recording of cancer data. The records are organized according to type of cancer and therefore the cases of lung cancer can be extracted for examination. Each cancer record contains demographic information about the patient, as well as data regarding tumor site, stage at diagnosis, treatment, and any other cancer in the body.³⁰⁷ In addition to the standard information collected about each cancer case, mesothelioma records, because this type of cancer is unique to asbestos exposure, include a more detailed description of the occupational history of the patient, and any other information about the source of exposure.

³⁰⁶ Ibid.

³⁰⁷ John Fulton, Dec 5, 1997.

According to the Rules and Regulations Pertaining to the Reporting of Communicable, Environmental, and Occupational Diseases, mesothelioma, asbestosis, and any other asbestos-related disease with the exception of benign conditions of the pleura, are required to be reported to the Department of Health within six months of diagnosis. These diseases are reported through the use of confidential morbidity cards mailed by the physician directly to the Office of Occupational and Radiological Health, part of the state department of health. The morbidity cards are distributed to doctors across the state by the Office of Occupational and Radiological Health.³⁰⁸

ASBESTOS INDICATORS

All of the preliminary signs of exposure to asbestos described above are not exclusively related to asbestos. Mesothelioma, on the other hand, rarely occurs without exposure to asbestos, and therefore, is an informative indicator of asbestos-related diseases. As stated above, mesothelioma, asbestosis, and other asbestos-related diseases are reported to the state, and cases of mesothelioma are recorded in the cancer registry as well. Analyzing the number of cases reported each year will indicate a trend in the prevalence of asbestos-related diseases. However, this analysis must be done over an extended period of time because the latency period for the development of these diseases is twenty to forty years.

³⁰⁸ *Rules and Regulations Pertaining to the Reporting of Communicable, Environmental, and Occupational Diseases*. State of Rhode Island and Providence Plantations, Department of Health, June 1966; amended April 1996.

As a result, the data gained here could provide prevention information for the future.

Another possible indicator besides the rate of mesothelioma would be chest X-rays that exhibit signs of asbestos exposure. Because of the risks involved with radiation from X-rays, this should not be a procedure implemented to screen the general population for asbestos exposure, but rather a means of determining what percentage of people who have chest X-rays taken because of respiratory problems, have been exposed to asbestos. This information would suggest what proportion of severe lung or respiratory illnesses result from asbestos exposure. Although this indicator would be based on limited data, the trend over time would depict the state of environmental health.

Ideally, an indicator of asbestos exposure, would be developed by extracting a copy of the actual X-ray from the patient's file and having the interpretation done by a specialist who is trained in reading X-rays for asbestos-related diseases. This method, however, would require a separate database for this information, and its success would be limited by the logistics and cost of obtaining the X-rays, as well as hiring the specialist to read the X-rays.

A simpler, but less direct assessment of asbestos exposure would be to develop a means of measuring the ambient concentrations of asbestos fibers. Since this is an exposure measure, it is not as informative as an internal measure,

but does provide useful information when there are limited means of gathering other data.

RADON INDICATOR

Exposure to radon is associated with the development of lung cancer; however, when a doctor diagnoses a patient with lung cancer, he or she cannot distinguish whether the cancer was caused by radon exposure, another carcinogenic agent such as tobacco smoke, or a combination of causative factors. Since this is the case, an indicator of radon exposure cannot be based on the trend in the number of lung cancer cases over time.

There is no biomarker or body burden associated with radon, so the best means of assessing exposure is by estimating ambient levels of radon in one's home or office. Even this becomes difficult to measure because of the variability in radon levels among houses in the same neighborhood. Radon testing can be done on an individual basis, but compiling this information is a challenging task.

Perhaps a better indicator would be monitoring the sales of radon testing kits. This would not provide information regarding the level of radon to which people are being exposed, but it would provide data about the number of people who are aware of the dangers of radon and who are taking steps to determine whether they are at risk. An increase in the number of testing kits sold would indicate that the public is receptive to education programs about radon. Hopefully, increased awareness and detection of high levels of radon would

increase the rate of remediation measures being taken to reduce radon exposure. This cannot be determined by the information gathered from this indicator alone, but rather could be obtained by monitoring the actual rate of remediation.

ENVIRONMENTAL TOBACCO SMOKE INDICATORS

Environmental tobacco smoke is also associated with the development of lung cancer, in particular among non-smokers. Similar to radon, however, a doctor cannot attribute a diagnosed case of lung cancer to secondhand smoke, as opposed to a variety of other factors. As a result, the incidence of lung cancer cannot be relied upon as an accurate indicator of exposure to environmental tobacco smoke.

Although the health-end effect cannot be used as an indicator, exposure to secondhand smoke can be detected through the presence of a biomarker.

Cotinine, the metabolite of nicotine, is present in blood, urine, and saliva after exposure to environmental tobacco smoke, and can be easily measured through a simple test.³⁰⁹ The specificity of cotinine as a marker for cigarette smoke makes it an excellent biomarker, and it makes it the most widely accepted method for assessing the uptake of nicotine from tobacco, both among smokers and non-smokers.³¹⁰ Levels of cotinine can be used to determine if a non-smoking individual has been exposed to secondhand smoke, but it cannot be used to

³⁰⁹ Ecobichon, Donald J. and Wu, Joseph M. Environmental Tobacco Smoke: Proceedings of the International Symposium at McGill University 1989. Lexington Books, Lexington MA: 1990, p70.

estimate risk by comparing values between smokers and non-smokers because of differences in the way nicotine is metabolized in the two populations.³¹¹

Cotinine has a half-life of approximately ten to twenty hours in the blood and in the urine, so it is an appropriate biomarker of short-term exposure to secondhand smoke.³¹² Since it does not represent a cumulative dose, it is not an accurate means of assessing the risk of developing lung cancer. It is a more informative indicator of exposure to environmental tobacco smoke related to childhood illnesses and short term respiratory problems such as asthma, bronchitis, coughing, and wheezing.

Repeated measures of cotinine levels in the body over time would be one way to estimate individual exposure to secondhand smoke. This would be a more direct measure than making estimates based on external measures of environmental tobacco smoke; however, the logistics of repeated testing are problematic.

One way of estimating a trend of overall exposure to secondhand smoke could be to test cotinine levels in a random sample of blood donated to the blood bank. Currently, there is no way to distinguish blood donated by smokers from blood donated by non-smokers. A simple question, "Do you smoke?" added to the list of screening questions would easily identify the smokers. Their donations

³¹⁰ The Health Consequences of Involuntary Smoking, a report of the Surgeon General. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control: 1986, p205.

³¹¹ Ecobichon, Donald J. and Wu, Joseph M, p70.

³¹² Ibid.

could be marked with a sticker identifying them as being a smoker. No other identifying characteristics would be included on the sticker. These donations could then be excluded from the pool from which the random sample is chosen for cotinine testing. Since the testing would only be done on non-smokers, any cotinine present in the blood would be the result of exposure to secondhand smoke.

There is a great deal of variability among individual exposure to environmental tobacco smoke depending on factors such as working or socializing in a smoke-free environment, or living with a family member who smokes. However, if blood samples are chosen randomly from the blood bank, these factors should not interfere with the overall trend of cotinine levels. One consideration to keep in mind is that the population that donates blood may be healthier than the general public, but this is not necessarily a result of lower exposure to secondhand smoke. Blood from the same individuals would not be tested each time, but repeated cotinine testing over time from random samples would depict a trend of exposure to environmental tobacco smoke across the general public. This trend could be used to determine whether no smoking policies and regulations are being enforced and whether overall exposure to secondhand smoke is decreasing.

PARTICULATE MATTER INDICATORS

Exposure to particulate matter is associated with a variety of respiratory problems, including persistent coughs, outbreaks of respiratory illness, aggravated asthma, chronic bronchitis, and shortness of breath resulting from decreased lung function. Patients admitted to the hospital as a result of these symptoms and conditions would most likely be included in the discharge data under the category of "Diseases of the respiratory system." Particulate matter is not responsible for the condition of all of the patients included in this category, but is most likely responsible for some. Although the health effects caused by particulates cannot be evaluated in isolation, examining the trend over time determined by aggregate data for respiratory diseases in general would provide an estimate of whether exposure to particulates is decreasing.

Examining these data in conjunction with ambient measures of particulate matter would also be useful. Tracking the correlation between increased cases of respiratory problems and days with high levels of particulate matter would make this indicator more informative, and possibly identify specific diseases that could also act as good indicators. A decreasing trend in the rate of hospitalization and a lower level of ambient particulates would indicate that fewer people are suffering from health effects because the exposure to damaging particulate matter is decreasing. A decrease in hospitalizations while the level of particulate matter remains constant or increases may suggest that education efforts regarding particulates are successful, and people are implementing

preventative measures to lower their exposure, such as staying indoors and limiting exercise on days when particulate levels are high.

Hospital discharge data accounts for only the most severe cases of respiratory problems; data regarding those cases that were treated in a doctor's office or in the emergency room would also be desirable. However, collection of this data presents the same problems that were discussed with regard to asthma.

CHAPTER 7 : LEAD POISONING

ABOUT THE DISEASE

Lead is one of the heavy metals most commonly encountered in the environment and most thoroughly studied. Epidemiological studies demonstrate that low-level exposure causes adverse effects on neurobehaviorial functioning, intelligence, and general health.³¹³

The majority of damage from elemental lead takes place once the material has been ingested. The body does not absorb it through the skin if dermal contact occurs or through the alveoli of the lungs if inhalation takes place.³¹⁴ The exception is tetraethyl lead, which is absorbed through the skin or the alveoli and easily penetrates the central nervous system. Exposure to tetraethyl lead however, is basically limited to sniffing leaded gasoline.³¹⁵ This has been a small source of exposure since the ban on the use of leaded gasoline.

Once the lead is ingested, absorption occurs in the gastrointestinal tract through the calcium channels. It then appears in the red blood cells and is gradually redistributed to hair, teeth, and bones where it can be stored for an extended period of time. The half-life of lead in the blood is thirty days, but once it accumulates in the bones, the half-life increases to twenty-five years.³¹⁶ 90-95%

³¹³ Hu, Howard, et.al. *Bone Lead Measured by X-Ray Fluorescence: Epidemiologic Methods. Environmental Health Perspective*. 103 (supplemental 1): 105-110 (1995).

³¹⁴ Philp, p133.

³¹⁵ Ibid, p133.

³¹⁶ Ibid, p133.

of the body burden of lead in adults is stored in bones, while in children, this level is 70-80%.³¹⁷ The remaining 20-30% of lead in a young child's or infant's body that does not get stored in bones, circulates in the blood to the brain where it impairs metal functioning, retards speech development, and deteriorates motor skills.³¹⁸

Children are more vulnerable to lead than adults because of increased exposure and absorption, as well as because their neurological systems are still developing and the blood/brain barrier is not fully developed. Children are more likely than adults to come in contact with dirt contaminated with lead that settled out of car exhausts before leaded gasoline was banned, and lead dust from paint that has been removed or that has peeled off the walls of older buildings.³¹⁹ Sources of adult exposure more likely result from lead glazes or lead solder in cans and pipes that is leached by soft water. Occupational exposure among printers, welders, boat repair men, and workers in the jewelry industry also poses a threat to human health.³²⁰

Central nervous system toxicity is rare and only occurs in severe cases of childhood lead poisoning. Symptoms of this health effect include vertigo,

³¹⁷ Hu.

³¹⁸ Philp, p133.

³¹⁹ Ibid, p133.

³²⁰ Ibid, p133.

irritability, progressive delirium, vomiting, and convulsions.³²¹ The mortality rate is 25% if treatment is implemented, and jumps to 65% if left untreated.³²²

Children and adults exposed to lead do not often show symptoms, and when they do, they are typically signs of general ill health. People often experience indigestion, loss of appetite, and constipation.³²³

RHODE ISLAND DATA

Rhode Island requires that all children in the state between six months and six years of age be screened for blood lead. The law also mandates that physicians and other health care providers submit the blood samples to the Department of Health Lab, where the testing is conducted.³²⁴ More efficient testing strategies were implemented in 1993; as a result, the data from 1993 on is particularly comprehensive and accurate. This database is one of the most complete sources of information maintained by the Department of Health.³²⁵

In addition to this, all diagnosed cases of adult lead poisoning related to occupational exposure must be reported to the State under Category VI of reportable diseases, Other Reportable Illnesses.³²⁶ This is accomplished by using confidential morbidity cards. The Department of Health will supply physicians and authorized medical personnel with the morbidity cards; any physician or

³²¹ Ibid, p133.

³²² Ibid, p133.

³²³ Ibid, p132.

³²⁴ *Health Data Inventory*.

³²⁵ Susan Feeley, Dec 8, 1997.

employee of a licensed physician making the diagnosis of adult lead poisoning will then mail one of these cards directly to the Division of Occupational and Radiological Health. The report of lead poisoning must be made within ten business days of the diagnosis.³²⁷

The reporting by laboratories differs from that of private practices. The State requires that labs, with the exception of the State Laboratory, submit all test results for Category VI Diseases, positive and negative, to the Division of Family Health.³²⁸ These reports are submitted electronically according to the standards established by the Rhode Island Department of Health regarding electronic reporting of blood lead results.³²⁹

INDICATORS

The lead database maintained by the state focuses on blood lead levels. This information lends itself to the development of body burden indicators. Direct measures of body burdens of lead in the blood, as well as in the bones, are good markers of exposure as well as preclinical indicators of disease.³³⁰ In addition to these, several biomarkers exist as well. Hematological alterations, changes in the metabolism of vitamin D, and anemia are biomarkers of lead.³³¹ Anemia, however, is the least informative indicator because this condition cannot

³²⁶ *Rules and Regulations Pertaining to the Reporting of Communicable, Environmental and Occupational Diseases.*

³²⁷ *Ibid.*

³²⁸ *Ibid.*

³²⁹ *Ibid.*

³³⁰ Human Health and the Environment: Some Research Needs, p249.

always be attributed to lead; the more common cause of this effect is a deficiency of iron.³³²

Although these other biomarkers exist, the blood lead levels are the most direct measure of lead exposure. It is also logical to take advantage of the data that are currently being collected before proposing to collect additional information. Examining the trends in the data that are compiled in the State database will demonstrate whether childhood exposure to lead is increasing or decreasing. The demographic information collected with each blood sample is also useful for determining specific neighborhoods that have high lead levels or pin-pointing certain communities where educational efforts about prevention should be focused.

One way of estimating exposure in high-risk areas is by measuring the amount of lead that exists in a child's environment. Dust samples taken from the floor of a home, where children are most likely to come into contact with it, could be analyzed to determine the concentration of lead that exists. This measure is not as specific as the amount of lead found in a child's blood, but it is one means of estimating exposure without conducting a blood test.

BONE LEAD MEASURED BY X-RAY FLUORESCENCE

Measuring the accumulation of lead in the bones is a more involved process than testing for lead in the blood; however the short half-life of lead in

³³¹ Ibid, p249.

the blood limits the assessment of exposure. An indicator that can better assess the long-term exposure to lead is the body burden in bones. Lead accumulation in the skeleton has a half-life of twenty-five years so extended exposure can be detected.³³³ A study conducted by Howard Hu et al. examines the technique of X-ray fluorescence (XRF) as a means of measuring lead levels in bone.

X-ray fluorescence is a non-invasive, painless, procedure that involves a minimal amount of radiation. XRF instruments use low level gamma radiation to provoke the emission of fluorescent photons from a specific area of the body.³³⁴ The photons released from the body are detected and counted over a wavelength spectrum. Characteristic emissions of lead are extracted from this investigation in order to determine the amount of lead in the bone being tested.³³⁵

There are two techniques that can be used. L-XRF uses weakly penetrating radiation and focuses on electron emissions from the L-shell. K-XRF uses more deeply penetrating radiation and focuses on the electron emissions from the K-shell.³³⁶ When compared to chemical analysis in cadavers, measurements taken using these techniques yield high accuracy, in part because the measure is unaffected by possible confounders such as the shape, size, and density of the bone.³³⁷

³³² Biomarkers and Risk Assessment, p37.

³³³ Philp, p133.

³³⁴ Hu.

³³⁵ Ibid.

³³⁶ Ibid.

³³⁷ Ibid.

Besides the accuracy of the measurement, other advantages of XRF exist. The software can be run by trained technicians so there is no need for a physicist. The instruments used in this technique can measure levels below the level that can currently be detected through other methods of lead testing. This allows greater analysis of the data where the customary procedure would categorize these minimal levels as zero, eliminating potentially valuable information for epidemiological studies.³³⁸ In addition to this precision, the level of uncertainty can also be determined based on the analysis of characteristic X-rays.³³⁹

Despite the technological capabilities, inconsistency in the measurements may result from the variability of skeletal lead distribution. This can be limited by measuring two or more bone sites, but residual variability cannot be totally eliminated.³⁴⁰

Other factors that must be considered when using X-ray fluorescence are the processes that affect bone metabolism and the liberation of lead from bones.³⁴¹ For example, pregnancy and osteoporosis must be taken into consideration. Blood lead levels have been found to be elevated in postmenopausal women; within this population, higher levels occur among nulliparous women.³⁴² Evidence suggests that lead stores are mobilized during

³³⁸ Ibid.

³³⁹ Ibid.

³⁴⁰ Ibid.

³⁴¹ Ibid.

³⁴² Ibid.

pregnancy, as well as during osteoporosis.³⁴³ Additional epidemiological studies need to be done in order to obtain further data on the relationship between parity, nutrition, and endocrine factors on the outcomes of bone lead. X-ray fluorescence, in addition to blood lead testing, has the potential to make significant contributions to future research involving the health effects of lead exposure in high risk population, such as occupationally exposed individuals.

³⁴³ Ibid.

CHAPTER 8 : HEAVY METAL POISONING

MERCURY POISONING: ABOUT THE DISEASE

Mercury, like lead, is a heavy metal responsible for environmental health problems. The major sources of mercury emissions are the electrical industry, pulp and paper factories, and chlorine production. Half a pound of mercury is emitted for each ton of chlorine produced.³⁴⁴ Mercury is also often used in agriculture to counteract the growth of fungi; it then leaches from the soil into the waterways, which in turn contaminates the fish.³⁴⁵ Mercury present in paint evaporates as the paint dries, resulting in toxic concentrations in the air.³⁴⁶

Mercury exists in three forms: elemental, inorganic compounds, and organic compounds. Elemental mercury is toxic when inhaled as a vapor. The major source of vaporized mercury is natural degassing of the earth's crust which is transported throughout the atmosphere.³⁴⁷ When inhaled, the vapor is absorbed across the alveoli in the lungs, as well as the blood-brain barrier. This leads to acute poisoning within a few hours, characterized by weakness, chills, metallic taste, nausea, vomiting, diarrhea, labored breathing, cough, and tightness in the chest.³⁴⁸ Chronic exposure to mercury vapor can cause central

³⁴⁴ Waldbott, George L. Health Effects of Environmental Pollutants. The C.N. Mosby Company, St. Louis: 1978, p150.

³⁴⁵ Ibid, p151.

³⁴⁶ Ibid, p151.

³⁴⁷ Philp, p135-136.

³⁴⁸ Ibid, p136.

nervous system disturbances such as tremors and behavioral changes including depression, irritability, shyness instability, confusion, and forgetfulness.³⁴⁹

Inorganic compounds of mercury, the most common being mercuric chloride, settle and cause damage in the liver and kidneys, especially in the kidney tubules that reabsorb vital agents from the urine.³⁵⁰ When present in the small intestines, inorganic compounds of mercury cause diarrhea. Vaporized compounds diffuse through the alveolar membrane, travel to the brain, and interfere with coordination.³⁵¹ Severe acute toxicity may result in intense abdominal pain, while chronic exposure often causes acrodynia, an allergic flushing of the skin referred to as “pink disease”³⁵²

The most common and most important environmental organic compound is methyl mercury.³⁵³ It penetrates the biological membranes easily which allows for dispersion throughout the body.³⁵⁴ It binds to red blood cells and circulates in the body before the majority is deposited in the brain and destroys cells responsible for coordination.³⁵⁵ The first symptoms of methyl mercury poisoning are numb fingers, lips, and tongue, slurred speech, and uncoordinated voluntary muscular movements.³⁵⁶

³⁴⁹ Ibid, p136.

³⁵⁰ Waldbott, p155.

³⁵¹ Ibid, p155.

³⁵² Philp, p136.

³⁵³ Ibid, p137.

³⁵⁴ Waldbott, p155.

³⁵⁵ Ibid, p155.

³⁵⁶ Ibid, p155.

Much of the mercury to which people are exposed comes from contaminated food. It can enter the food chain through soil, water, and air.³⁵⁷ Methylmercuris chloride is commonly used as a fungicide. Consumption of the seed grains treated with this chemical is one source of entry into the body.³⁵⁸

Another common pathway of exposure is through the consumption of contaminated fish and mollusks.³⁵⁹ The water may become contaminated by direct discharges of mercury or mercury compounds into the water, or by the action of bacteria present in the waterway. Inorganic mercury from industrial waste settles on the bottom of lakes and rivers because of the heavy specific gravity. In the absence of oxygen, bacterium called methanobacterium amelanskis, anaerobic bacteria discharged into the water with raw sewage,³⁶⁰ convert the inorganic mercury to toxic methyl mercury.³⁶¹

The contaminant then becomes concentrated as it moves through the food chain. Plankton passively absorb the mercury, and then fish ingest the mercury by eating the plankton. Fish also absorb mercury through their gills. The metal continues to accumulate in the body fat over time so older fish have a higher concentration of mercury than younger fish. The contamination is passed on to birds, whales, seals, people, and any other animals that consume the fish.³⁶²

³⁵⁷ Ibid,p157.

³⁵⁸ Philp, p137.

³⁵⁹ Ibid, p137.

³⁶⁰ Philp, p138.

³⁶¹ Waldbott, p152.

³⁶² Ibid, p152.

This chain of events continues even after mercury discharge into the water may have been eliminated because mercury is not biodegradable. The only way mercury ceases to be a threat is when it is buried by uncontaminated sediment and can no longer interact with other organisms in the body of water.³⁶³

Mercury poisoning causes a variety of health effects among afflicted individuals. Some preliminary symptoms which may be attributed to other causes include tiredness, irritability, and headaches.³⁶⁴ As the poisoning progresses, more severe symptoms become evident, such as numb arms and legs, difficulty swallowing, blurred or restricted vision, loss of hearing, loss of muscular coordination, inflamed gums, diarrhea, and a possible metallic taste in one's mouth.³⁶⁵ The effects of the poisoning depend on the extent of the exposure and the sensitivity of the individual at risk.

Exposure to mercury may not always be accompanied by symptoms of mercury poisoning. This is particularly dangerous for pregnant women because although they may not demonstrate symptoms of exposure, they will pass the toxicity on to their fetuses.³⁶⁶ Exposure to mercury in utero results in infants born with mental retardation and deformities.³⁶⁷ The methyl mercury has a strong affinity for the placenta and fetal tissue; this protects the mother from mercury

³⁶³ Philp, 138.

³⁶⁴ Waldbott, p153.

³⁶⁵ Ibid, p153.

³⁶⁶ Ibid, p153.

³⁶⁷ Philp, p137.

poisoning, but exposes the more sensitive fetus.³⁶⁸ An indicator of mercury exposure would therefore not only protect the person being exposed, but also protect the unborn fetus who may also be unknowingly exposed.

CADMIUM POISONING: ABOUT THE DISEASE

Cadmium is another metal that exists naturally in the environment at low levels, but can be toxic if high level exposure occurs. It can form soluble or insoluble organic or inorganic compounds.³⁶⁹ Some cadmium becomes soluble during weathering of rock, but the majority is produced as a byproduct of electrolytic zinc plants. Major environmental emissions are released from smelting operations for copper, lead, and zinc, automobile exhaust, and the manufacturing of pigments and alloys.³⁷⁰ Other common uses include metal plating, plastic stabilizers, photographic chemicals, catalysts, fungicides on golf courses, and the manufacture of nickel-cadmium batteries.³⁷¹

Once cadmium is released into the environment, it is readily absorbed by plants, where it is stored in leaves and seeds. Cadmium is often present in sewage sludge fertilizers, so it is readily absorbed by various crops which are eventually consumed by people and livestock.³⁷² Cadmium is introduced into the human diet from cereal grains, organ meats, such as liver and kidneys, from animals that have been exposed, and shellfish that were harvested from

³⁶⁸ Waldbott, p156.

³⁶⁹ Philp, p140.

³⁷⁰ Ibid, p140.

³⁷¹ Ibid, p140.

contaminated waters.³⁷³ Cadmium travels through and accumulates in the food chain in the same manner as mercury.

After high exposure, cadmium can be found in the red blood cells which then distribute the metal to the liver, pancreas, prostate, and kidneys.³⁷⁴

Nutritional deficiencies, such as a lack of zinc, iron, or calcium, may increase cadmium toxicity by increasing the absorption from the gastrointestinal tract.

Calcium deficiency increases the synthesis of calcium-binding proteins and cadmium absorption.³⁷⁵

Workers exposed to cadmium fumes often develop respiratory problems, which may reach the severity of emphysema or obstructive pulmonary disease if chronic exposure takes place.³⁷⁶ The only treatment currently used to combat cadmium toxicity is the elimination of the exposure, followed by supportive measures.³⁷⁷

ARSENIC POISONING: ABOUT THE DISEASE

There are several sources of environmental arsenic. Some arise naturally from the weathering of rock; other sources include pressure treated wood, emissions from smelting gold, silver, copper, zinc, and lead ore and from combustion of fossil fuels. Arsenic is also used in certain herbicides and

³⁷² Ibid, p140.

³⁷³ Ibid, p140.

³⁷⁴ Ibid, p140.

³⁷⁵ Ibid, p141.

³⁷⁶ Ibid, p141.

³⁷⁷ Ibid, p141.

pesticides.³⁷⁸ The amount of arsenic absorbed by the plant depends on the type of soil in which it grows; fine soil with a high percentage of clay and organic material hinders the uptake of arsenic.³⁷⁹ Contaminated waterways result from runoff or fallout, and contaminated drinking water can be attributed to wells drilled in arsenic containing rock.³⁸⁰

Arsenic has been used historically in pharmaceuticals as an agent to produce rosy cheeks and edema or fullness. It was also used to treat trypanosomiasis and syphilis.³⁸¹ Despite these medicinal benefits, continued exposure can lead to chronic poisoning. People with a high intake of fish in their diet are more prone to arsenic poisoning because of the biomagnification of the metal as it passes through the food chain.³⁸²

Arsenic is absorbed through the gastrointestinal tract and distributed to the lungs, skin, and mucous membranes in the body.³⁸³ It accumulates in the liver, kidneys, heart, and lungs, and creates deposits in bones, teeth, hair, and nails. Measures from these tissues can be used as body burdens and are important for conducting diagnostic tests for arsenic exposure.³⁸⁴

Acute arsenic poisoning, which involves severe abdominal pain, is a rare result of arsenic exposure. Arsine, a gaseous form of arsenic produced from the

³⁷⁸ Ibid, p141.

³⁷⁹ Ibid, p142.

³⁸⁰ Ibid, p142.

³⁸¹ Ibid, p141.

³⁸² Ibid, p142.

³⁸³ Ibid, p142.

³⁸⁴ Ibid, p142.

electrolytic process, is also extremely toxic and causes rapid and often fatal hemolysis.³⁸⁵ The more common health effect is chronic poisoning, characterized by muscle weakness and pain, skin pigmentation, gross edema, gastrointestinal disturbances, kidney and liver damage, peripheral neuritis, and eventually possible paralysis.³⁸⁶ Another indication of exposure is the development of Mee's lines, white lines on the fingernails. These are often used to determine whether exposure has occurred.³⁸⁷

RHODE ISLAND DATA

High exposures to mercury, cadmium, and arsenic are not usually widespread throughout the general public. Diseases related to occupational exposure to these metals must be reported to the Department of Health under Category VII- Occupational Diseases, of the Rules and Regulations Pertaining to the Reporting of Communicable, Environmental and Occupational Diseases.³⁸⁸ The physician or authorized medical personnel who believes that a patient is suffering from any of these diseases must report the incident to the Office of Occupational and Radiological Health, Rhode Island Department of Health within 30 days of examining the patient.³⁸⁹

³⁸⁵ Ibid, p142.

³⁸⁶ Ibid, p142.

³⁸⁷ Ibid, p142.

³⁸⁸ *Rules and Regulations Pertaining to the Reporting of Communicable, Environmental and Occupational Diseases.*

³⁸⁹ Ibid.

The Department of Health provides the physicians and other health care providers with standard cards for reporting necessary information. This information includes the name, address, phone number, and occupation of the patient; the name, address, phone number, and business of the employer; the name and phone number of the reporting medical professional; the nature of the disease; and any other information that the Department of Health may deem necessary.³⁹⁰ The health care providers then send the completed cards to the state. These data can be examined over time and used as an indicator of environmental and occupational exposure to various metals. Investigation of the occupational information and the demographics may show clusters of diseases that reflect a source of exposure.

Although the collection method used by the state attempts to track the number of cases of poisoning from these heavy metals, the information gathered here is limited by compliance among physicians to actually report, as well as by the difficulties diagnosing the diseases. As described above, only the most severe cases demonstrate symptoms clearly attributed to exposure to a specific metal. Therefore, the data compiled by the Department of Health may not represent accurate exposure to mercury, cadmium, and arsenic.

³⁹⁰ Ibid.

INDICATORS

The information regarding the incidence of metal poisoning collected by the state is a measure of the health-end effect, the most direct measure of human health. In this case however, because so many of the symptoms caused by exposure to these metals reflect general ill health and may be attributed to a variety of other causes, the health-end effect is not the most informative measure. If another means of assessing exposure to these metals, such as testing for a biomarker or a body burden, were available, a more complete trend of exposure could be obtained. This information could help identify high-risk populations as well as identify the source of exposure so that preventative measures could be implemented before the disease progressed to the more severe stages of poisoning. Additional monitoring such as this would also incorporate exposures other than occupational, such as consuming fish that were caught in contaminated waters.

MERCURY POISONING INDICATORS

The symptoms, distribution, accumulation, and excretion of mercury depend on whether it is in the elemental state or a molecular compound, and what type of chemical transformation is taking place in the body.³⁹¹ The effects of mercury exposure vary and do not usually express themselves immediately after

³⁹¹ Waldbott, p152.

exposure. As a result of these characteristics, the health end-effects of mercury exposure are not good indicators.

A more informative indicator is the body burden of mercury on various tissues where the metal accumulates. Not only is this a more accurate measure of exposure, but it also can potentially reveal exposure before a great deal of damage has been done. The body burden in various tissues is more valuable than the level of mercury in the blood because the metal disappears from the blood rapidly. However, lack of mercury in the blood or urine does not mean that exposure has not occurred, but rather that the mercury may have degraded quickly.³⁹² The amount of mercury stored in body tissues such as kidneys, hair, and the thymus gland are more informative because mercury tends to accumulate in these areas over time.³⁹³

Testing tissues for body burdens, with the exception of hair and nails, is an invasive procedure that is not conducted on a regular basis. Since tissue testing is not always a simple procedure, general testing of the population would be an inefficient means of assessing mercury exposure. Exposed populations need to be identified first, and then tested for body burdens if symptoms of mercury poisoning arise.

One way of identifying exposed populations would be through external measures of mercury, such as levels in fishable waters or levels in the fish

³⁹² Ibid, p153.

³⁹³ Ibid, p153.

themselves. Exposure measures are the least informative indicators because of the level of uncertainty involved, but they are the most relevant measure when focusing on prevention. If the exposure indicators are used simply as a preliminary means of identifying high-risk populations, and followed up by testing for body burdens or education about prevention, valuable information about the effects of mercury exposure throughout the population could be gained. Ambient measures of exposure should lead to increased monitoring and tests for body burdens especially among pregnant women and other sensitive populations.

In addition to identifying high-risk populations, measures of mercury levels in the water could also be used as an indicator to warn fishermen of potential hazards in an effort to prevent or limit mercury poisoning before it becomes a problem. Ambient measures can also direct attention to the source of exposure, and perhaps implement changes to stop the pollution at its source.

CADMIUM POISONING INDICATORS

Chronic exposure to cadmium causes respiratory problems and in severe cases may result in emphysema or obstructive pulmonary disorder. These conditions are not exclusive to cadmium exposure; the correct cause may never be identified and therefore never be reported to the state. The data collected by the Department of Health are therefore limited by the minimal reporting. Tests for a cadmium body burden in various tissues such as the liver, pancreas,

prostate, and kidneys, would be a more complete measure of cadmium exposure. Identification of a body burden would be an earlier detection of exposure which could lead to preventative measures before the poisoning progresses to causes more damage.

Similar to mercury, however, tissue testing is not done on a regular basis and therefore cannot be used as a screening method for the general population. Once again, high-risk populations must be identified and tests should be done at the earliest sign of exposure in order to initiate medical treatment and prevent further harm by eliminating exposure.

ARSENIC POISONING INDICATORS

Preliminary symptoms of arsenic exposure, such as muscle weakness and pain, loss of appetite, and gastrointestinal disturbances, are common complaints and are not unique to arsenic. As exposure continues, the health effects become more severe and may result in kidney, liver, and nerve damage. The detection of arsenic through a body burden would be not only an indicator of exposure, but also a means of earlier diagnosis.

Once again, tests done on various tissues such as bone, teeth, liver or kidneys, should be done if the patient complaining of the symptoms has had potential exposure to arsenic. Screening of the general population would be too extensive and too invasive a procedure to collect data regarding a disease which is usually limited to certain occupations and specifically exposed communities.

Detection of arsenic exposure through body burdens would provide more complete information regarding the trends of exposure across the population.

CHAPTER 9 : CARBON MONOXIDE POISONING

ABOUT THE DISEASE

Carbon monoxide is a colorless, odorless gas produced by the incomplete burning of carbon-containing fuels.³⁹⁴ Carbon monoxide has an affinity for hemoglobin 250x greater than oxygen; therefore, when ambient exposure occurs, the CO, rather than the oxygen, binds to hemoglobin, reducing the oxygen-carrying capacity of the blood.³⁹⁵ This increase in carboxyhemoglobin (COHb), interrupts the delivery of oxygen to various tissues in the body; therefore, tissues with a high demand for oxygen, such as the brain and heart, are most susceptible to toxicity.³⁹⁶ Effects of exposure to carbon monoxide include fatigue, headaches, dizziness, and weakness. Increased levels of CO exposure may result in nausea, vomiting, confusion, disorientation, loss of consciousness, and even death.³⁹⁷

Prolonged exposure to low levels (10-30 ppm) or short-term exposure to high levels (> 100 ppm) of carbon monoxide results in accumulation in the blood. It takes from three to five hours to clear the contaminant from the blood.³⁹⁸ The

³⁹⁴ *American Lung Association Fact Sheet-Carbon Monoxide*. American Lung Association, 1997.

<http://www.lungusa.org/global/news/report/viron/virmaterfac.html>

³⁹⁵ Penney, David G. Carbon Monoxide. CRC Press: New York, 1996, p94.

³⁹⁶ *Ibid*, 164.

³⁹⁷ *American Lung Association Fact Sheet-Carbon Monoxide*..

³⁹⁸ Folinsbee, Lawrence J. *Human Health Effects of Air Pollution*. Environmental Health Perspective. 100: 45-46, 1992.

rate of the clearing process is decreased if low-level exposure still exists because this reduces the gradient for excretion through the lungs.³⁹⁹

The extent of damage from carbon monoxide poisoning depends on a variety of factors, including age, length and concentration of exposure, and form and timing of treatment.⁴⁰⁰ Mild poisoning does not usually result in long-term effects, but the severity of the neurological damage increases with the severity of the exposure. Two types of disability may follow carbon monoxide poisoning. The patient may regain consciousness but demonstrate obvious neurological disabilities that improve over time.⁴⁰¹ Patients in the second category (10-36% of severe poisonings) appear to have recovered completely after regaining consciousness. This period of normalcy, referred to as the “lucid interval” may last a few days or a few weeks, and is followed by delayed deterioration and long-term disability. A lucid interval lasting ten to forty days may result in dramatic deterioration or even death.⁴⁰²

Thousands of people become ill from exposure and 250 people die annually from carbon monoxide poisoning.⁴⁰³ Exposure usually occurs in garages, tunnels, smoke-filled rooms, and poorly ventilated areas with appliances or other sources of combustion.⁴⁰⁴ Potential sources of CO include

³⁹⁹ Ibid.

⁴⁰⁰ Penney, p 239.

⁴⁰¹ Ibid, p 240.

⁴⁰² Ibid, p 240.

⁴⁰³ *American Lung Association Fact Sheet-Carbon Monoxide..*

⁴⁰⁴ Folinsbee.

furnaces, ovens, water heaters, fireplaces, wood or coal burning stoves, space heaters, automobile exhaust, gas-powered lawn mowers, and cigarette smoke.⁴⁰⁵

High-risk populations include people who spend an extended period of time in traffic, such as commuters and policemen, auto-tunnel workers, and garage attendants. People suffering from angina and cardiovascular disease are also susceptible because their bodies cannot increase the coronary flow to compensate for the CO in the same manner as healthy people.⁴⁰⁶ Fetuses, infants, and people with anemia are also more sensitive than average individuals to carbon monoxide exposure.⁴⁰⁷

The most critical step in treating someone exposed to carbon monoxide is the cessation of tissue hypoxia. This can be accomplished by supplying the patient with 100% oxygen, either through an oxygen mask or endotracheal intubation.⁴⁰⁸ Oxygen therapy is usually administered for two hours, and varies depending on the extent of exposure. The cardiovascular system should be monitored during the oxygen therapy in order to detect any subclinical cardiac ischemia.⁴⁰⁹

RHODE ISLAND DATA

Information regarding cases of carbon monoxide poisoning are collected by the State under Category VII of the reportable diseases, Occupational

⁴⁰⁵ *American Lung Association Fact Sheet-Carbon Monoxide..*

⁴⁰⁶ Folinsbee.

⁴⁰⁷ *American Lung Association Fact Sheet-Carbon Monoxide..*

⁴⁰⁸ Penney, p262.

Diseases. The same regulations described for reporting of poisoning from heavy metals apply to carbon monoxide poisoning: any authorized medical personnel examining a patient believed to be suffering from a disease related to occupational exposure to carbon monoxide is required to report the case to the Department of Health.⁴¹⁰ In the case of carbon monoxide intoxication or asphyxiation, the medical personnel must report the case immediately.

The data collected regarding carbon monoxide poisoning focus specifically on occupational exposure and exclude other sources of contamination. As seen with the other health effects already discussed, this information is limited by the rate of compliance to report among physicians. Occupational exposure, leading to reporting of occupational information, as well as the opinion of the patient, influences the physicians' decision to comply.

CARBON MONOXIDE POISONING INDICATORS

The data currently compiled by the state are a good indicator of occupational exposure to carbon monoxide. The records can be examined on an annual basis to determine whether the number of cases of carbon monoxide poisoning is increasing or decreasing over time. Since the information collected by the State incorporates occupational information, this indicator can also determine whether the exposure is continually occurring in the same workplace environments, and whether conditions have improved after an incident of carbon

⁴⁰⁹ Ibid, p263.

monoxide poisoning. The State can use this information to decide whether follow-up procedures are necessary, and whether education, prevention, and remediation efforts are successful.

Since the data collected by the Health Department focus primarily on occupational exposure, an indicator for other sources of carbon monoxide exposure would also be useful. Many devices have been developed to detect ambient levels of carbon monoxide. Inexpensive wall-mounted instruments are available that sound a bell or a buzzer when carbon monoxide levels become dangerous.⁴¹¹ The level above the ground at which these measurement are made is important in determining human exposure. For instance, the level of CO at ground level where construction combustion equipment was present, was within the acceptable limits; the warm exhaust air rose, however, creating a CO level ranging from 190-320 ppm twenty feet above the floor. Electricians working near the ceiling became ill, while the construction workers on the ground remained unharmed.⁴¹² This is one example of the limitations involved with ambient measures of carbon monoxide.

A more precise indicator of human exposure to carbon monoxide is measuring the level of the biomarker, carboxyhemoglobin, in the body. Since carbon monoxide has such an affinity for hemoglobin, blood is the tissue of

⁴¹⁰ *Rules and Regulations Pertaining to the Reporting of Communicable, Environmental and Occupational Diseases.*

⁴¹¹ Penney, p4.

⁴¹² Ibid, p9.

choice for sampling carbon monoxide exposure.⁴¹³ The most informative measure is the percent concentration of carboxyhemoglobin. This can be assessed by spectrophotometric methods, a procedure that determines the amount of CO without liberating it from the hemoglobin, or through a variety of volumetric methods that require the CO to be released from the hemoglobin and extracted as a gas.⁴¹⁴

Another method of determining the level of carboxyhemoglobin in the body is by measuring the alveolar gas, the gas present in the deep regions of the lungs. This technique is based on the principle that under certain conditions, the gas in the lungs will equilibrate with the gas in the blood.⁴¹⁵ Since the composition of the gas is continuously changing, a method of breath-holding has been developed to achieve equilibrium.⁴¹⁶ The individual being tested must expire to residual volume, inspire maximally, hold his or her breath for twenty seconds, and then expire as far as possible. The first 500 ml of exhaled air are discarded; the remaining gas is collected in an air-tight bag with the aid of a three-way valve. The gas is then analyzed to determine the level of carboxyhemoglobin.⁴¹⁷ This is an efficient method of assessing carbon monoxide without a blood sample, however, it cannot be used on subjects suffering from

⁴¹³ Carbon Monoxide. Committee on Medical and Biological Effects of Environmental Pollutants. National Academy of Sciences, Washington, DC: 1977, p199.

⁴¹⁴ Ibid, p199.

⁴¹⁵ Ibid, p202.

⁴¹⁶ Ibid, p203.

⁴¹⁷ Ibid, p204.

chronic lung disease because the composition of their alveolar gas varies greatly.⁴¹⁸

Each of the techniques described above is a good measure of carbon monoxide exposure on an individual level. Either procedure could be used to measure CO levels among patients with CO poisoning or among people who have undergone extreme exposure but have not yet reached the stage of poisoning. These intense episodes of exposure are the most dangerous situations, but represent only the most severe cases.

Long-term exposure to low levels of carbon monoxide can be detrimental as well. Using these methods to assess exposure to CO in the general population is possible, but more complicated. Routinely taking blood or air samples from a random sample of the population would indicate exposure to elevated background levels of CO, but this approach is not practical. A more feasible option would be the same as that suggested for evaluating exposure to environmental tobacco smoke: selecting and testing a random sample from the blood donated to the local blood bank. Cigarettes are a source of carbon monoxide so the level of carboxyhemoglobin would be higher among smokers. Once again, incorporating the question "Do you smoke?" into the protocol, and marking blood donated by smokers with a small sticker, would eliminate these samples from the pool so they do not skew the results.

⁴¹⁸ Ibid, p204.

Examining the trend in the level of carbon monoxide in the blood over time would indicate whether ambient levels to which people are exposed are increasing or decreasing. The level of carbon monoxide in the environment is not usually easy to decrease. If high levels exist, efforts can be made to educate people about the dangers of exposure to carbon monoxide, monitor levels of CO on a regular basis, and prevent exposure in areas where the levels exceed the standards.

CHAPTER 10 : MISCARRIAGES AND BIRTH DEFECTS

ABOUT MISCARRIAGES AND BIRTH DEFECTS

The term miscarriage is defined as the loss of a pregnancy from the womb prior to the 28th week of gestation.⁴¹⁹ For the purpose of this discussion, “miscarriage” will refer only to spontaneous abortions, and will exclude deliberate termination of pregnancies.

Several factors limit the accuracy with which the actual incidence of miscarriages can be determined. Many miscarriages take place before a woman misses her menstrual cycle, so she is unaware that she is pregnant. Some spontaneous abortions takes place soon after the missed menstrual cycle and before the woman has been examined by a doctor; therefore there is no record of her pregnancy. The reverse could also be possible: a woman could skip a menstrual cycle and then bleed heavily, leading her to believe that she has had a miscarriage, when in fact, she was not really pregnant.⁴²⁰ All of these situations make it difficult to assess the incidence of miscarriages.

The cause of a miscarriage can be the result of a variety of factors; therefore, it is often difficult to determine the actual reason for the spontaneous abortion. Chromosomal and fetal structural abnormalities are the highest risk factors for miscarriages, followed by abnormal development of the placenta called a hydatidiform mole, increasing maternal age, abnormalities of the uterus,

⁴¹⁹ Lachelin, Gillian C.L. Miscarriage: The Facts. Oxford University Press, New York: 1985, p1.

and a variety of maternal diseases.⁴²¹ The mother's decision to use substances such as cigarettes, alcohol, and drugs, also contributes to the risk of miscarriages. In addition to these causes, exposure to a variety of environmental factors is associated with an increased incidence of spontaneous abortions.⁴²²

Human studies, as well as animal studies, have linked environmental exposure to PCBs with an increased risk of miscarriages.⁴²³ The concentration of progesterone, a hormone necessary to maintain pregnancy, must remain high in order to avoid losing the fetus. The presence of PCBs in the body accelerates the breakdown of progesterone by the liver, and thereby increases the risk of having a spontaneous abortion.⁴²⁴

PCBs are one of the environmental exposures that has been linked to an increased rates of miscarriages. Although there are many other chemicals associated with spontaneous abortions, a lack of documentation exists because of the difficulties involved with conducting these studies.⁴²⁵ Exposure assessment usually involves questionnaires and interviews; information obtained through these methods is subject to recall bias and insufficient medical evidence.⁴²⁶ These factors, in addition to the large at-risk and control populations needed for these

⁴²⁰ Ibid, p33.

⁴²¹ Ibid, p37-39.

⁴²² Ibid, p40-42.

⁴²³ Colborn, Theo et al. Our Stolen Future. Plume, New York: 1997, p181.

⁴²⁴ Ibid, p181-182.

⁴²⁵ Lachelin, p42.

⁴²⁶ Ibid, p42.

studies, explain the limited knowledge regarding environmental exposures and miscarriages.

Exposure to environmental toxicants not only increase the risk of miscarriages, but the risk of birth defects as well. The placenta is the structure that attaches the fetus to the wall of the uterus, through which nutritional requirements are transferred from the mother to the fetus, and excretory products are transferred from the fetus to the mother.⁴²⁷ The placenta does not acts as a protective barrier, but rather allows foreign chemicals, in addition to the substances necessary for proper growth, to diffuse into the womb where they may exist in measurable amounts.⁴²⁸

The point at which exposure occurs during the gestational period influences whether or not the chemical will impact the fetus. For example, the presence of a chemical or hormone at a certain time may not affect the fetus, while exposure to this substance a few weeks earlier may have been detrimental to the development of the unborn child.⁴²⁹ This “window period” during which a substance can cause harm is associated with a variety of chemicals, and its length and position within the gestational period are not the same for all agents. This makes it difficult to study the actual effects and potency of environmental

⁴²⁷ Ibid, p76.

⁴²⁸ Wison, James G. Environmental and Birth Defects. Academic Press, New York: 1973, p113.

⁴²⁹ Colborn et al. p51.

exposures on fetuses. Lumping together all exposures to a certain substance without accounting for timing, may mask the magnitude of the impact.⁴³⁰

The exposure to the chemical does not necessarily have to occur during the pregnancy, but could have taken place prior to conception. Persistent chemicals that accumulate in adipose tissue or in bones over time are released during gestation; the mother transfers these contaminants to her child through the placenta during pregnancy, and through breast milk during infancy.⁴³¹

Often times the mother is unaffected by the environmental exposure, while the adverse effects of the substance manifest themselves in the child. For example, a pregnant woman who consumes a considerable portion of seafood contaminated with mercury may not demonstrate symptoms of mercury exposure; her newborn child, however, is likely to exhibit congenital defects, such as neurologic symptoms resembling cerebral palsy.⁴³²

Evidence of a strong link between birth defects and exposure to chlorophenoxy fungicides and herbicides was found in a study conducted in an agricultural region of Minnesota.⁴³³ Researchers found that exposure to endocrine-disrupting compounds, such as the herbicide 2,4-D, are associated with increased rates of urogenital defects not only in the offspring of farmers who apply the chemicals and are therefore directly exposed, but also among

⁴³⁰ Ibid. p62.

⁴³¹ Ibid. p212.

⁴³² Wilson, p78.

⁴³³ Colborn et al. p255

children of families living in the area. Birth defects are most prominent among children conceived in the spring when the herbicides are usually applied.⁴³⁴

Another study conducted in 1980 involving mothers who consumed contaminated fish from the Great Lakes demonstrates the effects of in-utero exposure to PCBs and other chemicals.⁴³⁵ Women defined as fish-eaters were characterized as having consumed contaminated fish two or three times a month during the six years preceding pregnancy. The PCBs from the fish accumulated in the body fat of the women, and were then passed on to their children through the placenta and breast milk. Babies born to fish-eating women had a lower birth weight, a smaller head circumference, and weaker reflexes than babies born to non-fish-eaters.⁴³⁶ The differences between the two groups of children persisted as they developed. At seven months of age, the children of fish-eaters demonstrated signs of impaired cognitive functioning; at age four, they scored lower on verbal and memory tests than children who were not exposed to such high levels of PCBs and other contaminants.⁴³⁷ This is one of many studies that has associated environmental exposure with negative outcomes in newborns. The effects described in this study are not obvious and easily identifiable birth defects; however, these subtle changes have the potential to adversely impact an individual's future.

⁴³⁴ Ibid. p255

⁴³⁵ Ibid. p190.

⁴³⁶ Ibid. p190.

⁴³⁷ Ibid. p190-191.

Substances that harm the fetus but do not effect the mother are sometimes referred to as “hand-me-down poisons” because they cause health problems across generations.⁴³⁸ The health effects are not always apparent at birth, but may become evident later in life. Anomalies often present themselves during puberty if the deformities involve the reproductive system as a result of exposure to endocrine disrupters.⁴³⁹ Estrogenic chemicals found in the environment, such as octylphenol and butyl benzyl phthalate, are associated with a variety of reproductive disorders. Some health problems in women include clear-cell vaginal cancer and under-developed reproductive organs.⁴⁴⁰ In men, the effects of early exposure to estrogenic chemicals include an increased risk of prostate cancer, smaller testicles, and reduced sperm count.⁴⁴¹ Only the congenital defects noticeable at the time of delivery are recorded on medical records; these data are limited because they exclude malformations that present themselves years later. This adds to the difficulty of monitoring and studying health effects caused by in utero exposure to various environmental toxicants.

RHODE ISLAND DATA

The Rhode Island Department of Health compiles information about miscarriages in the fetal death records. Whether the death was spontaneous or an induced abortion is recorded, so the number of miscarriages can be separated

⁴³⁸ Ibid. p53.

⁴³⁹ Ibid. p62.

⁴⁴⁰ Ibid. p54.

⁴⁴¹ Ibid. p255.

from the total number of deaths.⁴⁴² This database includes all fetal deaths in the state. As a result of this, in addition to the miscarriages that take place before the woman is aware that she is pregnant, there is probably an underestimation of the actual number of miscarriages that take place in the population.

Birth defects are recorded on the birth records and hospital discharge records that are maintained by the state. The incidence of various anomalies is also probably under-reported because, as discussed earlier, some birth defects are not physically apparent at the time of birth. Certain abnormalities manifest themselves later in life, or become evident only after further medical investigation has taken place.

The Department of Health also has information linking birth records and infant death records.⁴⁴³ These data do not fall into the category of miscarriages or birth defects, but they are related and could provide useful information. The cause of death among children younger than one year of age is often associated with birth defects or conditions present at birth such as low birth weight. Examining both of these records may help determine the cause of death.

INDICATORS

The fetal death records exclude data about spontaneous abortions that occur very early in the gestational period. However, the information currently

⁴⁴² Buechner.

⁴⁴³ *Health Data Inventory*.

being collected are sufficient to be used as a health-end-effect indicator because there is no feasible way to gather additional data regarding fetal deaths.

The many factors that influence miscarriages make it difficult to determine which factors, or which combination of factors, are responsible for the spontaneous abortion. Despite the fact that exposure to one environmental contaminant cannot be specified as the sole reason for a woman's miscarriage, environmental exposures are among the various causes. Research in this area could provide an estimate of the number of miscarriages that are related to environmental conditions. If this estimate could be obtained, a declining rate of miscarriages could reflect an improvement in environmental conditions.

If the overall goal is to reduce the number of miscarriages, and exposure to environmental toxicants are a causal factor, reducing the level of contaminants to which people are exposed would lower the risk of having a miscarriage. It is important to lower overall exposure, not just exposure during pregnancy, because some chemicals that accumulate in the body over time can harm the fetus.

An indicator for birth defects is somewhat more complicated than an indicator for miscarriages. The listing of birth defects on the birth records compiled by the state could act as a health-end effect indicator, as well, but in a limited sense. Once again, environmental exposures are one of many factors that contributes to birth defects, and these causes cannot be specified for each case. However, as explained above, if an estimate of the number of birth defects

resulting from environmental exposures is made, a declining trend in the number of anomalies recorded annually and a decrease in environmental exposures, would reflect an improvement in environmental health over time.

This indicator would be informative, but it would only cover a portion of the birth defects that are associated with environmental exposures. As described above, a number of contaminants in the environment mimic the effects of hormones in the body, disrupt the endocrine system, and result in adverse effects on the development of the reproductive system of the fetus. Although the child is born with these deformities, they may not be apparent until later in life. As a result, these types of birth defects go undetected, and are therefore not included on the birth records compiled by the state.

One way of collecting these data would be to start a registry focused on birth defects involving reproductive disorders. Some studies have linked certain health outcomes with specific chemicals, while there is still a wide range of effects resulting from a diverse field of chemicals for which there is limited documentation. The registry should contain three groups of reports: defects for which there is causal evidence associated with a specific chemical, defects for which there is anecdotal evidence or an association linking the health effect with a certain exposure, and defects for which there is no causal hypothesis. The compilation of these data over time would be an indicator of the rate of these types of birth defects. In addition to this, the registry could serve as a means of researching the effects of other environmental contaminants on human health.

Since there are many xenoestrogens that have yet to be studied, collecting data now could be a valuable source of information for future studies. New trends in health effects, as well as correlations between exposures and outcomes could be discovered by examining the information contained in this type of database.

CHAPTER 11- CONCLUSIONS

All of the environmental health effects described above have varying characteristics. The information available about each one depends upon the extent of knowledge that exists about the causes, nature, and treatment of the disease, as well as upon the ability to monitor and track its incidence within the population. As a result of these differences, the conclusions made about each of the diseases and the recommendations regarding the collection of data and development of indicators must be made on an individual basis.

The suggestions regarding the development of indicators for each of the disease that have already been discussed are summarized in the following table.

Health Effect	Additional Useful Information	Possible Ways to Collect the Information
Asthma	1. Collect treatment information from physicians and emergency room visits. 2. Monitor medication sales	1a. define as reportable disease 1b. sentinel system for reporting 1c. electronic reporting system 1d. report over the internet 2a. report # of prescription 2b. inventory from drug company
Melanomas and Skin Cancer	1. Sale of sunscreen 2. Use of sunscreen 3. Rates of squamous cell and basal cell carcinomas	1a. inventory from drug stores 1b. inventory from distributor 2. health interview survey 3. same as 1a-1d
Lung Disease and Lung Cancer	1. Chest X-rays for respiratory patients (Asbestos) 2. Rate of sales for radon testing kits (Radon)	1. extract from medical records 2a. inventory from stores 2b. inventory from distributor

	<ul style="list-style-type: none"> 3. Cotinine levels in blood (ETS) 4. Collect respiratory treatment information from physicians and emergency room visits 	<ul style="list-style-type: none"> 3. blood tests from samples donated to the blood bank 4. same as asthma 1a-1d
Lead Poisoning	<ul style="list-style-type: none"> 1. Bone lead levels 2. Concentrations of lead dust in homes 	<ul style="list-style-type: none"> 1. X-ray fluorescence 2. Analyzes samples of dust from home
Heavy Metal Poisoning	<ul style="list-style-type: none"> 1. Levels of metals in the water 2. Body burdens of metals 	<ul style="list-style-type: none"> 1. ambient monitoring of metals 2a. blood tests 2b. tissue testing
Carbon Monoxide Poisoning	<ul style="list-style-type: none"> 1. Carbon monoxide levels in blood 	<ul style="list-style-type: none"> 1. blood tests from samples donated to the blood bank
Miscarriages and Birth Defects	<ul style="list-style-type: none"> 1. Records of birth defects that manifest themselves later in life, such as reproductive disorders 	<ul style="list-style-type: none"> 1. registries

The recommendations made for each of the selected health effects reflect some of the possibilities for collecting additional data in order to advance the development of environmental health indicators. Continued research should focus not only on developing indicators for health effects that have not been examined here, but also on improving the recommendations that have been made. Future technology and medical advancements could simplify the process of gathering important data and should be incorporated into any existing system.

The suggestions made above regarding possible means of collecting data should not be limited to the diseases for which they were recommended. Registries, for example, are not only a means of collecting data about miscarriages and birth defects, but can also be used to collect information regarding a variety of other environmental health effects. They serve as a reliable source for conducting surveillance research, and for determining trends over time within a certain population.⁴⁴⁴ They also provide researchers with a population for conducting studies on specific diseases.

Gathering information from doctors and hospitals is not the only means of collecting data for these registries. Another resource could be health insurance companies. They routinely gather a great deal of patient information that could be an integral part of registries. Compiling these data that are already being collected is an efficient means of developing indicators.

Registries probably supply the most comprehensive data for developing indicators. The basic structure can be applied to almost any disease, and the detailed information that is collected can be altered to reflect the nature of the specific health effect. The development of additional registries would provide insight into the field of environmental health. The long-term benefits gained from these registries would surpass the cost and effort needed to establish and maintain them.

⁴⁴⁴ Lee.

Another means of compiling a range of data in the field of environmental health is through tissue banking, storing biological specimens to be tested for biomarkers and body burdens.⁴⁴⁵ The samples of different tissues could be taken from cadavers (although this may raise some ethical issues), as well as from patients who have undergone surgery or biopsies. Tests could be done on these specimens to monitor chemical or heavy metal accumulations in the body that cannot be tested without invasive procedures, such as measuring the amount of mercury stored in the kidneys. This information could be used to monitor levels of exposure, as well as to correlate exposures with certain diseases. Tissue banking would serve as a means of researching health effects and chemicals which have not yet been explored in depth. These data could then be used in the future to develop indicators for diseases which are not currently monitored.

The success of tissue banking depends on the manner in which the samples are preserved, and the precision with which each sample is documented. Initiating and maintaining a tissue bank is more complicated than organizing a registry, but the potential for learning more about environmental health over time is the same, if not greater.

A more indirect, but useful source of gathering data on various environmental health effects is through the Health Interview Survey that is conducted by the State, as well as nationally, every five or six years. One section of the standardized survey incorporates questions focused on an area of interest

⁴⁴⁵ Lee.

at the time it is administered. The flexibility incorporated in this survey allows for the addition of questions regarding various environmental health effects. Current research needs can be used to determine the issues that the survey will address. This information will fluctuate depending on various research needs, and can potentially be applied to a wide range of health effects.

Registries, tissue banking, and the Health Interview Survey are several ways to gather additional data for a variety of environmental health effects. Efforts focused on establishing more registries and tissue banks could lead to the development of countless indicators for a variety of diseases. In addition to gathering data, a statistical analysis could be done on the records collected by the Department of Health to determine which health effects are most prevalent and deserve the most attention. From there, a cost-benefit analysis could be done to determine if collecting these data would be worthwhile. Future research into these areas, as well as into the recommendations made above, could result in great advancements in the field of environmental health.

BIBLIOGRAPHY

- American Lung Association Fact Sheet-Carbon Monoxide.* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfac.html>
- American Lung Association Fact Sheet-Children and Ozone Air Pollution,* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfact.html>
- American Lung Association Fact Sheet-Ozone Air Pollution.* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfact.html>
- American Lung Association Fact Sheet-Particulate Matter Air Pollution.* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfac.html>
- American Lung Association Fact Sheet-Radon.* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfact.html>
- American Lung Association Fact Sheet-Secondhand Smoke.* American Lung Association, 1997.
<http://www.lungusa.org/global/news/report/viron/virmaterfact.html>
- Beatty, Rita Gray. The DDT Myth: Triumph of the Amateurs. The John Day Company, New York: 1973.
- Beggs, Paul John and Curson, Peter Hayden. *An integrated Environmental Asthma Model.* Archives of Environmental Health, vol 50 (no 2): 1995, p 87-94.
- Biomarkers and Risk Assessment: Concepts and Principles. World Health Organization, Geneva: 1993.
- Bullard, Robert. Confronting Environmental Racism: Voices From the Grassroots. South End Press, Boston: 1993.
- Buechner, Jay Ph.D. Rhode Island Department of Health. October 31, 1997.

- Cancer Facts:Environmental Tobacco Smoke.* National Cancer Institute, National Institute of Health: February 8, 1995.
- Cancer Facts and Figures-1997.* American Cancer Society.
www.cancer.org/statistics/97cff/97facts.html
- Cancer Facts: Questions and Answers About Asbestos Exposure.* National Cancer Institute, National Institute of Health: May 1994.
- Cancer Rates and Risks, 4th Edition.* National Institute of Health, National Cancer Institute: 1996.
- CancerNet from the National Cancer Institute.
www.gretmar.com/cancernet/304724.txt
- Carbon Monoxide. Committee on Medical and Biological Effects of Environmental Pollutants. National Academy of Sciences, Washington, DC: 1977.
- Casey, David B, MBA, Buechner, Jay S, Ph.D. *Utilization of Rhode Island Hospitals: October 1, 1994- September 30, 1995.* Office of Health Statistics, Rhode Island Department of Health, October 1997.
- Colborn, Theo et al. Our Stolen Future. Plume, New York: 1997.
- Colorado HealthNet, 1997.
http://bcn.boulder.co.us/health/chn/site/idx_asthma.html
- Conceptual Framework to Support Development and Use of Environmental Information in Decision Making:*
USEPA Document #239-R-95-012, Washington, DC: April 1995.
<http://www.epa.gov/indicators/defne.html>
- DeKoning, H.W. Setting Environmental Standards: Guidelines for Decision-Making. World Health Organization, Geneva: 1987.
- Dorland's Illustrated Medical Dictionary. W.B. Saunders Company, Philadelphia: 1994.
- Ecobichon, Donald J. and Wu, Joseph M. Environmental Tobacco Smoke: Proceedings of the International Symposium at McGill University 1989. Lexington Books, Lexington, MA: 1990.

Fact Sheet: Health and Environmental Effects of Particulate Matter. United States Environmental Protection Agency, Office of Air and Radiation; Office of Air Quality Planning and Standards.

<http://ttnwww.rtpnc.epa.gov/naaqsfm/pmhealth.htm>

Feeley, Susan MPH, Rhode Island Department of Health, December 9, 1997.

Folinsbee, Lawrence J. *Human Health Effects of Air Pollution.* Environmental Health Perspective. 100: 45-46, 1992.

Fulton, John Ph.D. Rhode Island Department of Health, December 5, 1997.

The Health Consequences of Involuntary Smoking, a report of the Surgeon General. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1986.

Health Data Inventory, A Compendium of Databases Maintained by the Rhode Island Department of Health (Draft). Rhode Island Department of Health, August 1997.

Health and Environmental Effects of Ground-Level Ozone Fact Sheet. USEPA Office of Air and Radiation, Office of Air Quality Planning and Standards, July 17, 1997 <http://ttn.www.rtpnc.epa.gov/naaqsfm/o3health.htm>

Hu, Howard, et.al. *Bone Lead Measured by X-Ray Fluorescence: Epidemiologic Methods.* Environmental Health Perspective. 103 (supplemental 1): 105-110 (1995).

Human Health and the Environment: Some Research Needs, US Department of Health and Human Services: 1984.

Koren, Herman. Handbook of Environmental Health and Safety: Principles and Practices. Pergamon Press, New York: 1980.

Koren, Herman. Illustrated Dictionary of Environmental Health and Occupational Safety. CRC Press, New York: 1996.

Koren, HS. *Environmental Risk Factors in Atopic Asthma.* International Archives of Allergy and Immunology. 1997 May-July; 113 (1-3): 65-68.

Kovacs, Margit. Biological Indicators in Environmental Protection. Ellis Horwood Limited, New York: 1992 p7.

Lachelin, Gillian C.L. Miscarriage: The Facts. Oxford University Press, New York: 1985.

Lee, Lester W. et al. *Human Tissue Monitoring and Specimen Banking: Opportunities for Exposure Assessment, Risk Assessment, and Epidemiologic Research.* Environmental Health Perspective, 103 (supplemental 3) 3-8 (1995).

Lung Disease Data 1996. American Lung Association.

Moeller, Dade W. Environmental Health. Harvard University Press, Cambridge: 1992.

Monitoring Human Tissue for Toxic Substances. National Academy Press, Washington D.C.: 1991.

National Institute of Allergy and Infectious Diseases: National Institute of Health, June 1996.
www.niad.nih.gov/factsheets/allergystat.htm

Penney, David G. Carbon Monoxide. CRC Press: New York, 1996.

Philp, Richard B. Environmental Hazards and Human Health. CRC Press, Inc. New York: 1995.

Rules and Regulations Pertaining to the Reporting of Communicable, Environmental, and Occupational Diseases. State of Rhode Island and Providence Plantations, Department of Health, June 1966: amended April 1996.

Sabatini, MM. Dermatology Nursing. 7(1):45-50, Feb.95.
<http://chid.aerie.com/simple/simple.html>

Schiffes, Justus J. Healthier Living. John Wiley and Sons Inc., New York: 1970.

Schutzer, Steven E. Lyme Disease: Molecular and Immunologic Approaches. Cold Spring Harbor Laboratory Press, Plainview, NY: 1992.

Studies Doubt Sunscreens Stop a Cancer. The New York Times, Wed Feb 18, 1998: pA17.

Sustainable San Francisco.
<http://www.ci.sf.ca.us/environment/sustain/intro.html>

Waldbott, George L. Health Effects of Environmental Pollutants. The CV
Mosby Company, St. Louis: 1978.

Weisal, CP et.al. *Relationship Between Summertime Ambient Ozone Levels and
Emergency Department Visits for Asthma in Central New Jersey*.
Environmental Health Perspective. 1995 March; 103 supplemental 2:
97-102.

Wison, James G. Environmental and Birth Defects. Academic Press, New York:
1973.

