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DEPARTMENT OF HEALTH & HUMAN SERVICES

Public Health Service

Centers for Disease Control
Atlanta GA 30333

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THE PERCEPTION OF RISK

Vernon N. Houk, M.D.
Director
Center for Environmental Health
Centers for Disease Control
and
Assistant Administrator
Agency for Toxic Substances and Disease Registry
Public Health Service
Department of Health and Human Services
Atlanta, Georgia 30333

National Conference on Environmental Public Health
Ramada Inn Central
Atlanta, Georgia

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INTRODUCTION

Risk may be defined in various ways. Risk can be considered the possibility of loss, injury, disadvantage, destruction, contingency, or danger. Risk can also be considered to be equivalent to threat. A risk can be considered to be someone or something that creates or suggests a hazard or an adverse chance, a dangerous element or factor.

In recent years, risk assessments and risk analyses have become increasingly sophisticated. This is an extremely difficult area which is not based on objective science but primarily on judgment and our concepts of risk.

The most important issue is how we perceive risks and what we, as a society, feel are acceptable. The United States has emerged from an era where the main concern of society was survival to an era where our main preoccupation seems to rest on extending longevity and improving the quality of life. This philosophy is in conflict with our increasing suicide rate among young people; our continued abuse of recreational drugs, alcohol consumption, and cigarette smoking; and our motor vehicle injury and death rates.

What are the risks or hazards that we are concerned about, and how do we perceive them? In order to analyze differences in our perception of risk, it first needs to be determined what the risks are that we encounter in our daily lives, what the frequency of injury from such risk is, and finally, how we consider and deal with these risks. These various risks are listed in tables which I will now show (Numbers 1 - 5).

--#1

ESTIMATED FATALITIES PER YEAR IN THE UNITED STATES

(TAKEN FROM 1979 VITAL STATISTICS)

1.	TOTAL DEATH/YEAR	1,914,000
2.	MAJOR CARDIOVASCULAR DISEASES	958,000
3.	MALIGNANT NEOPLASMS	403,000
4.	HOMICIDE	52,000
5.	SUICIDE	27,000
6.	MOTOR VEHICLE FATALITIES	50,000
7.	OTHER CAUSES	824,000

--#2

WHAT ARE THE CHANCES (BASED ON DEATH RATES)?

1. A 1 OUT OF 2 CHANCE OF DYING FROM A MAJOR CARDIOVASCULAR DISEASE.
2. A 1 OUT OF 4 OR 5 CHANCE OF DYING FROM CANCER.
3. A 1 OUT OF 40 CHANCE OF GETTING MURDERED.
4. A 1 OUT OF 40 CHANCE OF GETTING KILLED IN AN AUTOMOBILE CRASH.
5. A 1 OUT OF 70 CHANCE OF COMMITTING SUICIDE.

--#3

WHAT ARE THE CHANCES PER YEAR FOR THE U.S. POPULATION (220 MILLION)?

0.9% (9/1,000) OF THE POPULATION DIE OF ALL CAUSES.

0.44% (4/1,000) DIE OF MAJOR CARDIOVASCULAR DISEASES.

0.18% (2/1,000) DIE OF CANCER.

0.02% (2.4/10,000) DIE OF HOMICIDE.

0.012% (1.2/10,000) DIE AS THE RESULT OF SUICIDE.

0.02% (2.2/10,000) DIE AS THE RESULT OF MOTOR VEHICLE CRASHES.

--#4

MOTOR VEHICLES - HAZARDS AND PREVENTIVE MEASURES

* ONE INJURY IN 50 - 60 PEOPLE PER YEAR.
* 50,000 FATALITIES PER YEAR.
* ONLY 11% OF CAR OCCUPANTS WEAR SEAT BELTS.
* IF 75% OF THE POPULATION WORE SEAT BELTS, 14,000 LIVES PER YEAR COULD BE SAVED.
* AIRBAGS WOULD RESULT IN A 50% REDUCTION IN FATALITIES, SAVING 25,000 LIVES PER YEAR.

--#5

FACTS ABOUT CIGARETTE SMOKING

1. THE SMOKE FROM $\frac{1}{200}$ CIGARETTE/DAY IS A VIRTUALLY SAFE DOSE FOR THE NONSMOKER.
2. TOBACCO'S CONTRIBUTION TO CANCER DEATHS IS ESTIMATED TO BE 30%.
3. CIGARETTE SMOKERS HAVE TOTAL CANCER DEATH RATES TWICE THAT OF NONSMOKERS.
4. HEAVY SMOKERS (MORE THAN 1 PACK A DAY) HAVE A 3-4 TIMES GREATER EXCESS RISK OF CANCER MORTALITY.
5. 85% OF LUNG CANCER CASES ARE DUE TO CIGARETTE SMOKING.
6. LUNG CANCER DEATHS IN THE UNITED STATES INCREASED FROM 18,313 IN 1950 TO 90,828 IN 1977.

Faced with these statistics and with a limited amount of resources, we should examine whether it is necessary to reduce the risk from exposure to suspect carcinogens as it is now suggested by some to a theoretical incidence of one excess cancer in a population of 1,000,000 or one in 100,000.

Before we examine that question, I would briefly like to show a slide giving some examples of human carcinogens. (Slide #6)

--#6

EXAMPLES OF HUMAN CARCINOGENS AND LIFESTYLE FACTORS INFLUENCING THE INCIDENCE OF CANCER

(66 - 88% OF CANCERS ARE ENVIRONMENTALLY INDUCED)

AFLATOXIN	HEMATITE	VINYL CHLORIDE
4-AMINOBIPHENYL	MUSTARD GAS	SOOT AND TARS
ARSENIC	2-NAPHTHYLAMINE	IONIZING RADIATION
ASBESTOS	NICKEL	ULTRAVIOLET LIGHT
BENZENE	STILBESTROL	TOBACCO
CADMIUM OXIDE	ALCOHOL	DIET
CHROMIUM	INFECTION	

As this slide shows, it has been estimated that 66 to 88 percent of all cancers are environmentally induced. To this, I would like to add that there is, to some extent, a

genetic predisposition to the development of cancer. In talking about environmentally induced cancers, this does not mean that these cancers are necessarily induced by synthetic chemicals, that is chemicals made by industry. Lifestyle influences the incidence of cancer more than the exposure to industrial chemicals in "low" concentrations. In addition, smoking is a major cause of cancer, and we should not forget infectious diseases as has recently been demonstrated again very dramatically with the association between Acquired Immunodeficiency Syndrome (AIDS) and Kaposi sarcoma.

That lifestyles affect the incidence of cancer is vividly demonstrated by the changing incidence of specific types of cancer in migrating populations. For instance, the incidence of cancer of the stomach in Japanese living in Japan is much higher than in Japanese and their descendants who have immigrated to the United States. In contrast, cancer of the breast in females in Japan is much lower than in Japanese females who have immigrated to Hawaii or to the United States and in their descendants. Both Japan and the United States are highly industrialized countries so that exposure to industrial chemicals in these two populations would most likely be similar; however, the lifestyles and eating habits of these two groups are different.

Having examined these facts, let us now look at how risk assessments have been conducted in the last few years in the

United States and what impact they may or may not have on preventing disease. There are a number of industrial chemicals now which have been shown to cause cancer in rodents when rodents are exposed to these chemicals at high doses through gastric lavage, inhalation, or direct skin application. The doses at which these chemicals cause cancer in small groups of rodents varies. This may somehow be related to their degree of toxicity although such relationships have not been examined in detail. Some believe that any amount of a carcinogen, be it ever so small, is capable of inducing cancer in the host if the host receives any dose of such a chemical. Others do not believe this.

For example, a very basic concept in toxicology is that for each chemical, there is a dose-response relationship. This was first pointed out by Paracelsus, who stated it in a negative way, "Dosis sola facit, vt venenum non fit," which in English means "The doses alone makes a thing not poison." In other words, reducing the amount given will render the chemical harmless. Commonly, the aphorism is seen as "Dosis sola facit venenum" (Dose alone determines poisoning).

In most instances, animal studies have used high dosage levels of the chemical to be tested, and the results of such studies have then been extrapolated to lower untested dosage levels in the animals. With such extrapolations estimates have been made

to determine at which point only one excess cancer in a population of a million or one cancer in population of 100,000 would occur. For these extrapolations, most recently, multistage models have been used, and it has been assumed that the dose-response curve would be more or less linear for all carcinogens.

Very few studies have examined the dose-response relationship of carcinogenesis more extensively. Most studies have been conducted with rather high doses. In the United States only one study (Staffa and Mehlman, 1979) was conducted in mice where large numbers of animals (24,000 mice) and several dosage levels of N-2-Fluorenylacetamide 2-FAA (30, 35, 45, 60, 75, 100, and 150 ppm) were employed. The results of this study suggest that perhaps the linear quantal non threshold model is inconsistent with the data. This leaves many questions about "risk assessments" as they are usually modeled.

Most likely, at very low dosage levels, antioxidants, such as vitamin E, vitamin C, selenium, and unsaturated fatty acids, would have a protective effect against naturally occurring and man-made carcinogens which cause lipid peroxidation. The contribution to cancer that very low doses of carcinogens would make might well be overriden by lifestyle in general and would be modified by dietary intake of natural antoxidants and thus may not greatly contribute to the risk of cancer or other

chronic diseases. In other instances where the proximate carcinogen is a metabolite, insufficient amount of such a carcinogen may be produced at low dosage levels because of alternate metabolic pathways. Furthermore, repair mechanisms within the cell may have a protective effect against carcinogens at very low dosage levels.

For most of the chemicals which have been shown to cause cancer in animals, it is not known whether they actually cause cancer in humans and if they caused cancer at what dosage levels they would do so above background levels. Since this information is not available, extrapolations have been made from rodent populations to human populations. It is not at all clear at this point, whether that is justified. It can be stated that all of the carcinogens that have caused cancer in humans also cause cancer in animals. The reverse is not true. As more carcinogens are identified, it has also become evident that species variations exist. For some chemicals it has been shown that cancer can be produced in mice but not in rats, or cancer can be produced in mice and rats but not in hamsters and subhuman primates. This depends to some extent on differences in metabolism in different species. Many of the parent chemicals are indirect carcinogens and have to be modified in the body to the proximate carcinogen. The metabolism of chemicals may employ several pathways. It is possible, for instance, that at low dosage levels, only one of those pathways

is used, and at high dosage levels, this one pathway is saturated and other pathways come into play. In such situations, the proximate carcinogen may only be formed at high dosage levels. This has never been extensively examined.

Furthermore, many chemicals that are classified as carcinogens act as promoters of carcinogenesis. It is not at all clear what their effect would be at very low dosage levels. Thus, from rather insufficient animal data, extrapolationsd are made to establish acceptable levels of risk for human populations. The methods used appear to err on the conservative side, are unsubstantiated by scientific data, and may grossly overestimate risk.

In addition to all the uncertainties inherent in such risk assessments, there are also many uncertainties about exposure of populations to chemicals that are in the environment. It has not at all been determined with certainty, whether people receive a dose at all from chemicals that are in soil and what that dose is. We have made some estimates; however, we are still in the process of conducting experiments to determine whether such estimates are justified. Thus, the scientific data base is really not there to make these types of risk assessments and to spend billions of dollars to reduce exposures to such low levels that there would only be a theoretical chance of getting one additional cancer in a million population lifetime exposure.

What needs to be done is to develop the scientific data base so that more meaningful hazard evaluations can be made if humans are exposed to chemicals which are suspect carcinogens. Such hazard evaluations should only be made in the context of preventing disease in the future. They should not be used to establish a "cause-effect" relationship between nonspecific cancer in a given individual and past exposure to low doses of a suspect carcinogen. In individual cases, such cause-effect relationships cannot be established from animal data or from epidemiological studies because of competing factors that come into play at very low dosage levels.

The afore does not mean that we should not use "risk assessment" in making decisions; however, we should recognize that the process and the results are not as precise as some would lead us to believe. In doing risk assessments, we emphasize carcinogenesis because of our concern that only a small number of molecules may still cause cancer, while other toxic effects would only occur at higher doses of the chemical. However, such risk assessments are only one part of our armamentarium to estimate potential hazards resulting from exposure to synthesized or naturally occurring chemicals. For example, foodstuffs such as peanut butter and cornmeal may contain aflatoxin at concentrations which according to risk assessment calculations would suggest that we should have an epidemic of liver cancer in the United States, particularly in

the Southeast, where these foods are consumed in large amounts. In reality, human liver cancer in the United States is a rare event and is not increased in the Southeast. Similarly, risks estimated for ethylene dibromide (EDB) according to one calculation would suggest that if water containing 1 ppb (mg/k) of EDB were consumed, this would result in one additional cancer in each population of 1,500. The number of cancers would be even higher if humans were exposed to higher concentrations. It is curious that no data at all suggesting a higher incidence of cancer in populations occupationally exposed exists.

Until we do have greater precision using the risk assessment instrument, I would suggest the following approach as reasonable. This approach would divide the process into several factors.

1. Substance hazard evaluation. Is the substance known to be hazardous to animals and humans if human data exist? What are the data? What is the mechanism of action and how have the dose response curves been calculated?

2. If the substance is hazardous in animals, what is the theoretical hazard to humans? Through what avenues of exposure? Or through what mechanism of action?

3. Populations. Are there populations exposed to this material, are the theoretical avenues of exposure possible, and what is the likely degree of exposure?

4. Pilot studies. Is there evidence in the population exposed for continuous exposure? Can such exposure be quantified if the chemical is persistent in humans or can historical data be used to assess this? Based upon the action of the chemicals in those most likely most highly exposed, are there any biologic tests which could be used as an indicator of exposure, or which could be used as an early indicator of adverse health effects?

5. Health studies in the general population. From analysis of appropriately done epidemiologic studies, is there evidence of harm to humans?

The public health role is one of prevention. Even in the absence of conclusive evidence that harm to humans has occurred, if the data and evidence from laboratory animal studies is sufficiently strong that harm may occur, then preventing exposure to future populations is a reasonable action. Here the policymaker must address what the potential harm is, what is the potential remedial action, what is the cost of that remedial action, and is it warranted? In determining whether or not it is warranted, it should be borne

in mind there are resource needs for other areas of known beneficial intervention, such as providing prenatal care to mothers-to-be, vaccination of children, improving the nutrition of the general population, and trying to reduce the incidence of homicide, automobile injuries and deaths, and improving our quality of life, which might reduce the suicide rate and the increased use of substances of abuse.

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Staffa, J.A. and Mehlman, M.A. 1979. Inovations in cancer risk assessment (ED_1 study). Pathotox Publishers, Inc., Park Forest, South Illinois.