Statement of

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16. Abstract (Limit: 200 words) This testimony before the Subcommittee on Oversight and Investigations provided an overview of the occupational causes of cancer. A critical assessment was made of past approaches to identify and evaluate occupational carcinogens. According to this testimony, even once a substance has been identified, little seems to have been done to institute control measures for the protection of society. Coke oven workers in the steel industry exposed to coal combustion byproducts died of lung cancer at a rate of ten times that of other steel workers. Some occupational groups exposed to arsenic (7440382) died of lung cancer at a rate of two to eight times the national level. As late as 1971 thousands of American workers were still exposed greatly to aromatic amines although the link between these substances and bladder cancer had been known for 80 years. Similarly, uranium workers were still being exposed to radon daughters sufficient to cause lung cancer at three times the normal rate. The spread of mesothelioma to the wives and children of asbestos (1332214) workers through the carrying home of the fibers on their work clothes, the levels of arsenic in the urine and hair of children living near copper smelters, the increased rate of lung cancer among those living in areas where arsenical insecticides were used were noted. The increased incidence of diseases of the central nervous system, the liver, the bones of the fingers, the lungs, and the reproductive system have all been attributed to exposures to vinyl-chloride (75014).

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I am Dr. Joseph K. Wagoner, Chief, Industrywide Studies Branch of the National Institute for Occupational Safety and Health (NIOSH), and President-elect of the Society for Occupational and Environmental Health. I am an epidemiologist, having graduated from the Harvard University School of Public Health. I joined the U.S. Public Health Service in 1960 and served for 11 years with the National Cancer Institute before coming to NIOSH in 1971. At NIOSH, my branch is responsible for conducting most of the industrywide studies on the effects of chronic or low-level exposure to industrial materials, processes, and stresses, particularly studies of industrial carcinogens. My testimony will provide an overview of occupational causes of cancer.

The potential dimensions of environmentally induced disease are staggering. The Chairman of the Environmental Task Force for National Health Policy Planning reported to the Secretary of Health, Education, and Welfare in 1970 that environmentally induced diseases cost society as much as 35 billion dollars a year, which he said constituted a substantial burden tolerated only because it is hidden in an increasing annual bill for medical attention of all kinds. Given the choice between reducing the economic and social burden of medical care by prevention or by therapy, the Chairman concluded, "Prevention of ill health in its broadest sense should be the objective of environmental control."

Among the most serious of these environmentally induced diseases is cancer. The Council on Environmental Quality (CEQ) estimates that 75 to 85 percent of all cancer is related to our environment. Much of this can be traced to the massive and often unregulated increase in the number and volume of synthetic organic chemicals. Indeed, CEQ also estimates that

approximately 700 new chemical substances are introduced into commerce each year. The human impact of these chemicals is now becoming manifest in the form of severe chronic disease, notably cancer. These cancers are first detected in the occupational setting among workers because of their high and prolonged exposures.

To more fully appreciate the need for new approaches to the identification and control of occupational carcinogens it seems appropriate that a critical assessment be made of past approaches and the attending legacy those approaches have left this generation. In the past, the major approach to the identification of occupational carcinogenic hazards has been the fortuitous clinical recognition of cancer clusters. With the replacement of communicable disease by chronic disease as the principal cause of death in industrial countries, epidemiologists have increasingly turned their attention to post hoc studies of environmental and occupational factors. Clearly both of these approaches are possible only after workers have been made sick or have died as a result of involuntary exposure. This has been the case for arsenic, asbestos, benzene, benzidine, beta-napthalamine, chromates, coal combustion by-products, nickel, radon daughters, and other carcinogens of industrial origin.

Of even greater concern is that even when substances have been known to cause cancer, little has been done to institute control measures for the protection of society. Society has made little progress in the identification and control of occupational cancer in the 200 years since Percivall Pott's epic discovery of scrotal cancer among London chimney sweeps. A chronology of this lack of progress was the subject of my

opening address at the Conference on Occupational Carcinogenesis sponsored by the New York Academy of Sciences last year. I would like to review that chronology here and submit the complete paper for the hearing record.

Although it has been known for more than 200 years that coal combustion by-products cause cancer, today thousands of American workers are inhaling the same class of substances. Coke oven workers in the steel industry who are exposed to these materials die of lung cancer at a rate of ten times that of other steel workers. Although it has been known for 130 years that arsenic causes cancer, today over a million and a half American workers are inhaling that same substance, and some occupational groups exposed to arsenic are known to be dying of lung cancer at two to eight times the national rate. Although it has been known for 95 years that uranium workers are subject to lung cancer, as late as 1970, thousands of American miners were still exposed to radon daughters of sufficient concentration to cause three times the normal risk of lung cancer. Although it has been known for over 80 years that aromatic amines (benzidene and beta-napthalamine) cause bladder cancer, as late as 1971 thousands of American workers were still exposed, and in some plants, were literally sloshing in these substances. Although it has been known for nearly a quarter of a century that asbestos causes lung cancer, today over 2 million workers continue to inhale this substance.

It was not until passage of the Occupational Safety and Health Act of 1970 that a Federal program was established to set and enforce occupational health standards. Even today, standards openly recognizing these substances as carcinogens have been established for only two--

aromatic amines and radon daughters. Because of the latency period between exposure to carcinogens and the onset of cancer, even if adequate controls were instituted today for these and many other carcinogens, it would be up to 40 years before we would see a noticeable reduction in human cancer.

The situation in which we find ourselves is all the more tragic for the fact that over the past few years we have begun to document the intrusion of cancerous agents from the workplace into the general community. We now know that the wives, children, and relatives of asbestos workers have died of mesothelioma and others will follow as a result of allowing asbestos to be carried into the home on the workers' clothes or bodies. We now know that people living near asbestos factories have developed mesothelioma. We now know that virtually 100 percent of all urban dwellers coming to autopsy show the presence of asbestos in lung tissue. We now also know that individuals living in areas where arsenical insecticide spraying was common are dying of lung cancer at an increased rate. We now know that people living near copper smelters have an increased risk of lung cancer. Even children living near these same smelters have elevated levels of arsenic in their urine and hair. We now also know that measurable levels of some cancer producing agents, traceable to industrial sources, are present in our water supplies.

Perhaps the most striking example of the need for an alternative method to the identification and control of occupational and environmental carcinogens can be seen through the recent episode involving vinyl chloride. As early as 1930, the first adverse health effects of vinyl chloride were reported. Over the years, observations and studies have indicated a wide range of toxicity attributed to the effects of vinyl chloride on the

central nervous system, the liver, the bones of the fingers, and the lungs. Only recently has the spectrum of known toxicity of vinyl chloride broadened to include carcinogenesis. In early 1971, an Italian investigator, P. L. Viola, first reported the induction of tumors of the skin, lungs, and bones in rats exposed by inhalation to high levels of vinyl chloride. These findings were confirmed by another Italian scientist, Cesare Maltoni, who also noted angiosarcoma of the liver in animals exposed to vinyl chloride, prior to the first report of this rare tumor in vinyl chloride workers. Maltoni also reported adenomas and adeno-carcinomas of the lung, neuroblastoma of the brain, lymphoma, and various other tumors in mice, rats, and hamsters exposed to vinyl chloride by inhalation.

In January 1974, representatives of the B.F. Goodrich Chemical Company informed NIOSH that the deaths of several of their employees from angiosarcoma of the liver may have been related to exposure to vinyl chloride. Subsequent epidemiologic investigations of workers exposed to vinyl chloride by NIOSH demonstrated an excessive number of deaths due to cancer of the same four sites--the liver, lung, and the lymphatic and central nervous systems--that Maltoni had seen in animals. Epidemiologic and histopathologic evidence identified vinyl chloride as the causal agent in the etiology of this excessive cancer risk in humans.

Vinyl chloride likewise serves as a striking example of the problem of mutagenesis related to occupational and environmental exposures. In 1975, laboratory tests with certain strains of bacteria suggested that vinyl chloride was mutagenic. Subsequent reports by investigators in different countries demonstrated that male workers exposed to vinyl chloride had excessive numbers of chromosomal aberrations. Thus, concern

was expressed that vinyl chloride may cause (germinal) cell mutations in humans and subsequently affect future generations. Recently a team of investigators, including NIOSH scientists, reported excess fetal mortality among the wives of male workers occupationally exposed to vinyl chloride as contrasted with controls not so exposed. No such excess was demonstrated prior to the husband's exposure to vinyl chloride.

The observation of the carcinogenicity and mutagenicity of vinyl chloride first in animals and subsequently in man strongly supports the need for conducting laboratory testing prior to the introduction of chemical or other agents into the industrial or community environs. Further, support for the predictive value of such testing is provided by the examples of diethylstilbesterol (DES), bis-chloromethyl either (BCME), and mustard gas, agents first shown to be carcinogenic in animals and subsequently in man.

There are those who seem disturbed by the implication that when a chemical is shown to be carcinogenic or mutagenic in other species, we have to consider it as a potential carcinogen or mutagen in man. Some of them have been quoting out of context Pope's famous phrase, "The proper study of mankind is man." I wish to emphasize in closing that when we as a society are faced with the task of detecting and preventing the causes of insidious diseases characterized by a long period during which the hazard may be unrecognized, man is no longer the proper object of our study. The fact that the proper study of mankind is very often not man himself has been recognized as the basis of all modern experimental medicine, without which much of our progress in the health sciences would have been impossible.

Mr. Chairman, I will be pleased to answer any questions about the NIOSH program that you or members of your Subcommittee may have.