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SCIENCE

Zero—What Does It Mean?

Pollution of the environment, additives in food, exposure to radiation, hazards in the workplace, and identification of carcinogens are resulting in calls for zero discharge of pollutants, zero contamination, zero radiation, and zero risk. Congress, state and local governments, and their respective administrative agencies have responded with a stream of new laws and regulations. Some citizens' organizations and legislators are seeking the elimination of all pollution or of what are considered contaminants.

This desire to obtain zero is causing consternation in the minds of many engineers and scientists. When people speak of zero they apparently mean different things. And even in the case of scientific analysis, zero has changed. A few years ago analytical methods might have indicated the absence of a particular chemical in a test sample. Today, with better analytical methods, that same sample would show the particular chemical present; we no longer have the zero we had a few years ago.

Analytical methods are now measuring such minute quantities of chemicals (parts per billion and even parts per trillion) that supposedly identical samples of water from the same effluent, when analyzed, show different concentrations of pollutants. The problem may be one of humans not being skilled enough to get reproducible results with sophisticated equipment (assuming the equipment is not at fault), or it may be like the four blind men trying to identify the elephant—by touching different parts of the elephant, they come up with different conclusions about what an elephant is.

Concern about radioactivity's effect on health is resulting in calls for zero radiation, particularly where nuclear power plants are concerned. And yet there is naturally occurring radiation from space, from rocks, and even from our own bodies and food. Such radioactivity varies from place to place by as much as 400 to 500 percent. Knowing, then, that nowhere on earth is there zero radiation, are we talking about zero based on a particular location—and if so, which location? Or are we talking about a permissible level that is believed to be safe for human beings?

With the advent of new machines, new materials and chemicals, and new modes of living and with a greater knowledge of the things around us, we have suddenly become aware of new risks or risks we were not previously aware of. Through the years society has become used to and accepted certain risks. People learned to control fire, to build homes away from flooding rivers and volcanoes, to control the internal combustion engine. All of these things involve risks. Society has been able to reduce risks in many instances, but where natural forces are involved, risks are always present.

What do we mean when we ask for zero risk? Does zero mean a standard, a limit, or perhaps a goal for each kind of risk? Will we accept (and call zero) 50,000 deaths a year from automobiles, 100 deaths from airline traffic, or 25 to 60 deaths attributable to producing electricity from coal, but refuse to accept any deaths from nuclear power plants producing electricity because we don't want to risk a possible unknown?

Webster's dictionary defines zero (other than the numeral) as (i) a state of total absence or neutrality; (ii) the lowest point, nadir; and (iii) something arbitrarily or conveniently designated zero. In calling for zero, people may be asking for a state of total absence. However, just as it is impossible to stop killer hurricanes and to keep people from falling out of bed, we know we are going to have accidents if we use fire to heat our homes and cook our meals or use other new things that improve the quality of life and lengthen our life-span.

Knowing this, we may have to accept "something arbitrarily or conveniently designated zero" for pollutants, radiation, and risk. The costs of guessing at zero are enormous; laws and regulations must come to grips with this problem. Until they do, costs to society can only continue to go up. Everyone's checkbook will feel it—and there we all know what zero means.—MITCHELL H. BRADLEY, *Washington Office Director, American Society of Mechanical Engineers, Washington, D.C. 20006*

"ENVIRONMENTALLY INDUCED CANCER...SEPARATING TRUTH FROM MYTH"

A Talk By

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to the

Synthetic Organic Chemical Manufacturers Association, Inc.

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I appreciate the opportunity to come here and address you. I welcome it very much. It is probably appropriate that this meeting is being held in New Jersey because I think many problems that have beset industry with respect to regulations are founded in some of the misconceived problems that have been painted in New Jersey.

When I came here in 1979, on a leave of absence, the Federal government had just released the National Cancer Institute (N.C.I.) mortality study by county that was conducted by looking at death certificates from 1950 to 1969; several N.C.I. epidemiologists, as well as members of the New Jersey Department of Environmental Protection, and Department of Health, concluded that after adding up the age-corrected, race- and sex-corrected death data, that New Jersey led all the other states in cancer deaths for white males, expressed per 100,000. Now, that was, indeed, a true statement. That was not a lie. The cancer rates in New Jersey were 205 white male cancer deaths per 100,000 versus the U.S. national average of 174 per 100,000. That created a big hullabaloo. It gave this state the label of "Cancer Alley". Some of the people who were in the New Jersey Department of Environmental Protection at that time have subsequently moved to Washington and have continued to amplify this attitude.

I must admit when I came to New Jersey, I shared the view that most New York City dwellers have, that New Jersey is a terrible place, it's horribly polluted. As a matter of fact, on February 16th of this year the New York Times published a map which was sort of the ultimate view that New York City dwellers have. It showed lung cancer rates in various parts of New York City, Staten Island, on the west side of Manhattan, and parts of Brooklyn. The New York City Department of Health blamed polluted air wafting over the river into the city as the cause of these high cancer rates. The report ignored the fact, easily seen on the map, that there (were) vast "skip" areas, and other cancer "hot spots" that were not in the path of such air flows. The New York City Department of Health report ignored rudimentary epidemiologic methods such as correcting all the data for sex and race. Further, no attempt was made to study cigarette consumption patterns.

Ignored in all of these pronouncements and maps at the federal, state and city levels was the old scientific dictum of controls. This is what differentiates science from politics. When you look back at the cancer rates that were published and resulted in New Jersey being labelled "Cancer Alley", nobody mentioned the fact that Rhode Island was number two, with 203 cancer deaths per 100,000. No one mentioned that other heavily industrialized states did not have high cancer rates.

When industry was being blamed in New Jersey, nobody looked at the cancer rates in Pennsylvania and Ohio which are remarkably similar to New Jersey, in terms of the proportion of workers who work in "dangerous" occupations and the number of people who live in communities that are in proximity to major industrial plants that can be viewed by some as polluting. Pennsylvania, Ohio and New Jersey have several things in common. About 40 percent of the workforce is engaged in what you would call heavy, "dirty" industry. Forty percent of the population in those states lives in proximity to so-called "dirty" plants. When you look at the cancer rates, however, Pennsylvania and Ohio have cancer rates that are like the U.S. national average. Yet, only New Jersey had higher cancer rates.

The answer as to what is different about New Jersey is found very simply in the U.S. census. New Jersey has the most urbanized population; 90 percent of the population is urbanized and it also has the most dense urbanization. Only 70 percent of Ohio and Pennsylvania are urbanized. That 20 percent differ-

ence explains the difference in the cancer rates among the three states. Before it became politically popular to blame industry, the National Cancer Institute epidemiologists showed unequivocally that urban crowding was a predominate factor in cancer causation. It is clearly a surrogate for something else. New Jersey has a rather small land mass and over 80 percent of the people are crowded into a rather small area in the northeast part of the state. It is, therefore, like a large city and if you compare it to similar demographic areas, with equivalent land and population density, and look at it in a controlled way, it is shocking to see that many other cities and counties and regions that do not have any industry have higher cancer rates than New Jersey. For example, San Francisco; Washington, D.C.; Nassau County, which is an eastern suburb of New York City; Westchester County; these demographic areas have rates that are equal to or greater than New Jersey's, and those areas are devoid of heavy, polluting industry.

If you look at other questions beyond urbanization and ask what are the surrogate factors, the ecologists would say, well, it's urban air pollution, and it's still ultimately being derived from industry. Well, we tested that hypothesis. The hypothesis was that pollutants in the air and in the water are responsible for increasing the cancer rates by at least 25 to 30 percent. In order to test that hypothesis, we simply looked at the National Cancer Institutes own Third National Cancer Survey which surveyed 20 million people in this country, back in 1969, -70, and -71. Now, that was incidence data and as I said, it covered 20 million people. Our studies incidentally were conducted with the sole support of a grant from the National Cancer Institute when I served as the Director of the Cancer Institute of New Jersey in 1976.

The diagnoses were far more accurate than the death certificates in the N.C.I.'s mortality study because these were surgical pathology specimens. The slides were all read by Board Certified pathologists. In that Third Cancer Survey, there was a wealth of information and it was not originally designed to answer this hypothesis, but it came out that way. There were seven cities in that survey. Four of them were what we would call "clean" cities; San Francisco, Dallas, Minneapolis and Atlanta. They are urban centres without any kind of heavy industry. In that survey also were Detroit, Pittsburgh and Birmingham, the "Pittsburgh of the South". Those seven cities encompassed 16 million people. We did not massage the data; we simply lifted it out of the tables and put the data next to each other and if the hypothesis was correct that industrial air pollution or urban air pollution were a major factor in cancer causation, we would have expected to have seen that (the) four "clean" cities had lower cancer rates than the three "dirty" cities.

We looked at overall cancer rates for white males as well as for black males, age-corrected, and to our surprise, the three "dirty" cities had an overall 8 percent lower cancer rate than the four "clean" cities. I don't think the 8 percent is statistically significent, however, it invalidates the hypothesis. Furthermore, we looked at specific kinds of cancer that Blott, who is an N.C.I. epidemiologist, and others have said are industry-types of cancers, like lung, larnyx, nasopharnyx, stomach, lymphomas, leukemias, urinary bladder, liver and skin. These are all supposed to be industry-associated types of cancer. The Third National Cancer Survey gave us site specific cancer diagnosis, i.e., by atomic site. Those supposed industry-associated cancers were not higher or greater in incidence in the three "dirty" cities compared to the four "clean" cities. In Detroit, Pittsburgh and Birmingham, 45 percent of the occupational workforce is engaged, and has been engaged for several decades, in industry that most people would say is worrisome. Further, these decades of exposure were without controls. And yet, we did not see greater cancer rates in

these cities. A 45 percent workforce engaged in "dirty" industry translates into something like about 22 or 23 percent of the general population, which is a large enough percentage of the population to skew the data up higher if, in fact, these workers were being exposed to significent levels of carcinogenic hazards.

This is the N.C.I.'s own data. We didn't manipulate it. And, curiously, in the OSHA hearings and in any discussions I have ever held with-people on the Federal level, nobody ever looks at the Third National Cancer Survey. It's almost like their unwanted child because if you examine the data, within it is contained certain answers to questions that bedevil society, government, labor and industry. For example, there are questions raised about low-dose exposures. Let's not worry just about the workers, what about the people in the communities? They are exposed to low-dose emissions. What about the question of synergism, which is almost unanswerable in an experimental sense. The answers, again, to these questions, are found in the Third National Cancer Survey and the comparison of the three "dirty" cities and the four "clean" ones. Detroit, Pittsburgh and Birmingham have virtually every kind of pollutant chemical that you can imagine in the air and in the water. If synergism and chronic low-dose exposures were real hazards, and if, in fact, there were no thresholds, we should have seen higher rates in those three "dirty" cities. They have been in "business" for several decades, and the chances existed.

Now, there has to be a reason why we didn't see more of any type of cancer and the reason is as follows: <u>There are thresholds</u>. There is a threshold for virtually every substance that is noxious to mankind. There are thresholds for deadly viruses. There are thresholds for bacteria, fungi, anything that is noxious and harmful to humans generally has a threshold, and this includes carcinogens.

Now, clearly the exposures, although seemingly horrendous in the three "dirty" cities, were well below the threshold levels because there was no effect. Recall, the Third National Canacer Survey showed 8% <u>less</u> cancer in the "dirty" cities.

Another thing to keep in mind is there is a real biological reason to explain thresholds. It is not a magical thing. We have DNA repair mechanisms that are very efficient. Furthermore, most carcinogens and many toxic substances act by what we term free radical chemical mechanisms. There are endogenous anti-oxidants that we have evolved with over the eons that protect us against free radical pathology. These anti-oxidants can be overwhelmed with excessive doses of a carcinogen, such as occurs in laboratory animals, but human exposures are not at these doses, and we have not been overwhelmed, not even in Detroit, Pittsburgh, or Birmingham. It should be noted that the Third National Cancer Survey was conducted from 1969-1971. The cancers diagnosed at that time were starting to develop during the 1940's and 1950's. In those years, air/water pollutants were not controlled as they are now. We have therefore gone through "worst-case" conditions in our industrialized-urban centers, and nothing happened. Those who beat the drums of doom, as in recent books written by non-scientists, are clearly leading a march down the wrong path in cancer prevention. Abundant controls exist now, and most of them are needed; some are not, however.

Another thing that proves that there are thresholds is the experience with cigarette smoke. Cigarettes of the high-tar variety are perhaps the most carcinogenic substance that mankind deals with in a routine manner on a mass basis. Yet, it has been well proven through smoking dog studies; that thresholds exist; dogs, incidentially, love to smoke. Once they are trained, they jump right into the box and puff like mad. The threshold studies were

conducted through smoking dog studies, as well as through careful autopsies on individuals where smoking history was well recorded, and where the bronchi were serially sectioned and examined. If you smoke 10 to 15 of the 1 mg. tar cigarettes, there are no carcinogenic hazards associated with that. We don't even see the premalignant changes in the epithelium; so that here is the most carcinogenic substance that is massly used and there is, decidedly, a threshold.

Similarly, there are thresholds with alcohol. Alcohol and cigarette smoke are synergistic and together if you smoke the high-tar cigarettes and drink excessive quantities of alcohol, you find that you can account for 35 percent of the cancer deaths. This type of synergism is rare. Most of the cancers of the mouth, larnyx, esophagus and lung are caused by these two factors and they are synergistic. But, again, it has to be with excessive quantities of distilled liquor. It's the alcohol itself, not the congeners or other substances that are found in the drink.

Now, the misconceptions that have been brewing, first in New Jersey, and later in Washington, have aroused not only the ire of you fellows in industry but the ire of University medical scientists who are in the comprehensive and specialized cancer centers such as MIT, Harvard, NYU, Columbia, University of Pennsylvania, McArdle Cancer Center, and others. We are upset at what the Federal government has been doing. Our worry stems from the fact that if society is to listen to the Federal government, it will be led down yet another primrose path, blaming industry, air and water pollution for most of the cancer burden, whereas, in fact, the answers to cancer lie elsewhere and have been very well defined through a great deal of epidemologic research and extensive laboratory research. Whoever attempts to lead the nation towards less cancer had better be correct, because the selection of the wrong path is the equivalent of leading millions of Americans to certain death.

There were two symposia that were held. One of February 28th, March 1st, and March 2nd, and another one, June 6th, 7th, and 8th in New York City. These two symposia were held by the independent university scientists from the comprehensive and specialized cancer centers in this country. The symposia were under the aegis of the New York Academy of Sciences, and the American Health Foundation, with the cooperation of the American Cancer Society, as well as the World Health Organization's International Agency for Research on Cancer.

We came up with the following predominant causes of cancer, and the word, "predominant", is important. An analogous situation is that tuberculosis is predominantly caused by the tubercle bacillus. While there are associated factors in causing tuberculosis, for example, poverty, crowding, and malnutrition, the predominant cause is still the tubercle bacillus, and if you want to eliminate that disease, you control the tubercle bacillus. Similarly, in cancer, there are predominant causes. The University scientists decided to sponsor and attend these two symposia, and to continue putting on such symposia as often as is humanly possible, in order to turn around the propaganda machine that has been launched by the Federal government. This machine is leading the country down the wrong path in cancer prevention and is killing many Americans through misinformation.

The University scientists just went through a 15-year period of going up a blind alley looking for cancer viruses that weren't there. That cost a billion dollars. We cannot afford, not in monetary terms or in human terms, to spend another ten years and another billion dollars or more chasing the wrong culprit in cancer causation. There will be too many lives lost, especially when we know most of the answers for prevention at the present time. While we are still ignorant about how exactly a normal cell becomes malignant,

we do know how to avoid situations that have such a transformation as the end result.

The answers to cancer prevention are as follows. And it's easy to keep the scorecard in mind. There are about 1,000 people a day that die of cancer in this country. 350 of them, or 35 percent, are going to die today from having smoked high-tar cigarettes and having consumed excessive quantities of distilled liquor and they will die of cancer of the mouth, larnyx, esophagus and lung.

Another 45 percent are attributable to diet, and this figure was courageously stated by Dr. Arthur Upton, the Director of the National Cancer Institute on Tuesday, before a Subcommittee on Nutrition. He has confirmed what the University scientists have been saying for the past few years. No other government scientist has had the integrity to state what Dr. Upton stated; he further and strongly recommended major decreases in fat consumption, with increases in fresh fruit and vegetable consumption. 45 percent of cancer deaths are related to disordered nutrition. Under the category of disordered nutrition, there are four subcategories: excess calories; excess fat ingestion; obesity, carrying around an extra 30 or 40 pounds; and nutritional deficiencies such as fiber deficiency and Vitamin A deficiency.

Now, with Vitamin A you have to be careful. There is an optimum dose which happens to be the Recommended Daily Allowance. Too much Vitamin A will also cause cancer in addition to being toxic. Too little Vitamin A will also cause cancer.

There is evidence that the 5 percent of cancer deaths that are due to occupational exposures may have plateaued and may be on the way down. If you look at hemangio sarcomas that are caused by vinyl chloride monomer exposure, the latency periods and the ages of the patients are longer and older, respectively. It means they are developing at slower rates. Similarly, we did a study at NYU showing that mesotheliomas, which are caused by asbestos exposures, are at a plateau. We do not see an increasing incidence of mesotheliomas, despite the fact that most of the people we see at the NYU Cancer Center, where we treat 4,000 new cancer patients a year, are drawn from places like the Veteran's Administration and Bellevue Hospital, and includes a fair number of construction workers, and Brooklyn Navy Shipyard workers. Our Cancer Center is a general one, and does not have a specialized referral component for mesothiomes, as at Mt. Sinai Hospital; therefore, our rates are indictative of the general rates. We have not been seeing an increasing incidence in mesothelioma over the past 12 to 15 years. It's at a plateau. So that I think if controls are kept over the very dangerous chemicals and physical processes that you use in industry, I think we can continue to see what looks like a decline in occupational cancer, which we think is currently around 5 percent. Sir Richard Doll and others think it's much lower, but I would caution you that you cannot relax. The chemicals that you deal with in ever increasing quantities to meet consumer demands and to try to remain competitive in a ferocious world market are dangerous, and I think there are elements of truth to what the government regulators say; we can see a major explosion in industrially caused cancer if we do not have sensible controls. The question is, how rigid should the controls be? I think that we can be less rigid than what OSHA and EPA have proposed recently since there is clear evidence that there is no need for that kind of rigidity. Occupational carcinogens are under control, and new chemicals are receiving close scrutiny; air and water pollution are blameless in carcinogenesis.

A few other points that I would like to make in closing are that in addition to determining that cigarettes and alcohol, disordered nutrition, and occupational exposures cause 85 percent of cancer deaths, there is another 3

percent that are caused by exposure to radiation which is largely background, not nuclear plants, but background radiation. We have enough radioactive potassium in our bodies, for example, to cause 4,000 radioactive decays per minute. That adds up.

Another 2 percent of the cancer deaths are caused by pre-existing medical disorders, like chronic ulcerative colitis, chronic gastritis and things like that. And about 1 percent are caused by prescription drugs for the treatment of serious medical disorders.

The symposium held in June also determined, and this was brought forth by Dr. Cuyler Hammond of the American Cancer Society, that almost zero percent of cancer deaths, it's something like .00001 percent, are caused by air pollution. Water pollution is in the same category of zero percent. Other studies have shown that nuclear power plant accidents, asbestos in hair dryers, asbestos in school rooms, and saccharin/cyclamates can not cause cancer as presently used. And yet, those things that are listed as being zero percent are uppermost in the public's mind. This is due to the propaganda machines. We are treated to outrageous remarks by individuals like Wolfe from Ralph Nader's group that asbestos is the second leading cause of cancer deaths in males in this country. It's an absolutely unfounded statement that, by no stretch of even Sellikoff's imagination, could that be true. And yet, individuals like this are placed politically on the National Cancer Advisory Board and govern, and help to govern the National Cancer Institute's policy. It is terrifying because our nation can be led down the wrong path in prevention.

So you have an uphill fight in terms of public relations but I think that, if controls are kept, truth and reason are on your side and I think that you can win this battle that you are in. Thank you.

QUESTION: I assume that the reason for the higher cancer rates in the cities is due to the factors you describe, more smoking, and so on.

DR. DEMOPOULOS: And they may have far more disordered nutrition. The urban crowding is a surrogate for all of the factors that we know to cause cancer and it is proven too, with animal studies. The reason is ultimately the kinds of stress that you get when you start crowding any living organisms together. This has been well-defined with rat behavioral studies where if you take a room of a given size and keep putting more and more rats in there, even though you provide them with enough food and water, and places to sleep and rest, you will see back biting, tail biting, homosexuality, fighting and all the other things we see in urban settings. In medical school we are taught that certain diseases are urban diseases, i.e., they occur more frequently in urban areas. For example. obesity, hypertension, chronic ulcerative diseases of the GI tract (the gastrointestinal tract), heart disease, suicide, homicide, drug addiction, are all far more common in urban settings. This includes the cities and their surrounding suburbs. Now, I think to that list should be added cancer. And I don't think it's stress per se, but rather it's the way humans choose to relieve their stress, through gorging themselves, drinking themselves into oblivion, and surrounding themselves with smoke, that cause cancer.

QUESTION: I understood that in other parts of the world that different types of cancers occur.

DR. DEMOPOULOS: Well, there are some interesting reports that have come from China recently. There are regions of China that have extraordinary rates of esophageal cancer, for example. Even their chickens have esophageal cancer.

But, that has been traced to the particular kind of food that these individuals make. They apparently take various kinds of vegetables and peel them and let them sit around for months and months and finally they eat them and their chickens eat the same scraps. There are other situations, where there has been a good deal of epidemiologic research done on the nutritional habits of the people in correlating it with diseases, and it keeps coming back to what we eat that causes cancer. And, this is true also for atherosclerosis as well as premature aging. For example, there are tribes in Africa that make a habit of eating raw meat and blood together with milk and this is all they eat. They don't eat any fruits and vegetables. Their life expectancy is about 30 to 35 years of age. They die of heart disease, strokes, hypertension and cancer. And it's the worldwide studies that have been the strongest implicators of diet as a predominant cause of cancer.

QUESTION: In your listing of causes, it seems to me that you came up with about 91 percent. Where is the other 9 percent?

DR. DEMOPOULOS: God. I think that we are not immortal and that we have to accept certain things. I think it's part of the aging process. Almost every male over the age of 85, at autopsy, has cancer of the prostate gland, if the prostate gland is adequately examined. These cancers are microscopic and produce no symptoms. Similarly, urinary bladder cancer is a cancer of the aged. It keeps climbing as you get into the 80 and 90 year old brackets. I think it's an accompaniment to age and Dr. Handler, President of the National Academy of Sciences, has offered the hypothesis that oxygen, one of the most toxic gases known is responsible. Oxygen happens to be diradical. It oxidizes everything, including our lipids and I am sort of surprised, as a free radical pathologist, that we can exist with oxygen. I wouldn't have chosen it. Methane would have been better.

But, Dr. Handler has proposed that simply existing in an aerobic atmosphere is cause enough to give us cancer, and were it not for the major antioxidants that we have, we would have even more diseases. The decline in cancer of the stomach is appropriate to mention at this point in that regard. Cancer of the stomach used to be the commonest cancer killer in this country 40 and 50 years ago. Nowadays, it's a rare disease. In medical centers, we keep these cases extra days as teaching cases because medical students will never see one like it in their career. The reason for the decline in stomach cancer is the increased consumption of fresh fruits and vegetables and the antioxidants therein. BHT has probably helped. Dr. Arthur Upton concurs in this view, as do many University scientists.

QUESTION: Does a significent segment of the scientific community, people working in cancer causation, feel the same way you do about the problem? From your point od view, why is it that these scientific arguments about what industry is doing are presented by people like Wolfe, Epstein and Barry Commoner? Why don't we ever hear from those people who have spent their lives actually doing research?

DR. DEMOPOULOS: A good reason is that University medical scientists do not have a PR or propaganda machine, nor do they have a policy that they are trying to advocate. It's one of the defects of science, in general, that unless there is an organized effort to get the truth out of the medical journals wherein it is buried, you are going to have misconceptions, as we have now. I think the reasons for the origins of the misconceptions lie in the fact that as we prove the lack of validity of the virus hypothesis, there was a void. Everybody asked, well, if it's not viruses, what is it? Unfortunately, the term "environmental cancer" came into being. That was seized as meaning the general community environment rather than the personal environ-

ment created by our habits, diet and lifestyle. There was a void, a gap, before the nutritional data was gathered epidemiologically and from animal studies. We now know what the predominant causes of cancer are but, at the time the time the virus theory was shown to be invalid, the "greenies" and the ecology people came into the picture and stole center stage and they are still at it and they are holding onto it come hell or high water. They seem oblivious to the consequences of leading a march of cancer prevention down the wrong path.

QUESTION: As a taxpayer who was able to pay for tearing the asbestos out of all the schools and other public buildings, I am gratified that you don't seem to think that's a terribly important factor.

DR. DEMOPOULOS: I think it's a tragic misconception and it's the height of hypocracy and stupidity to see classrooms being torn apart. This recently happened in New York City where pretty close to half a million dollars were spent redoing the whole school because of the asbestos scare. Somebody had gone in and sampled the air only in the classrooms and found asbestos there. The conclusion was that it was coming from the ceiling. They tore everything down and they went back and they sampled the air again. They found just as much asbestos. Then, and only then, did they go out and do what is called the control. They went to the outside air and sampled it several blocks away and they found even more asbestos in the outside air. The point is, asbestos is a natural product, it's all over the place. We use it extensively in urban settings; brake linings, construction, everything. The point is that the doses we get in school rooms and office buildings are far below any kind of danger level, yet within these classrooms we have smoking lounges. As the NYU study has shown, we are not seeing an epidemic of mesotheliomas.

If you go into the cafeterias and watch what the kids eat, it's garbage, as far as I am concerned. Loaded with fat. No fresh fruits and vegetables, and these are wrong. It's as stupid as when a cigarette company used to advertise high-tar cigarettes as being good for your "T-zone".

QUESTION: Is there any really definitive data on the effect of the so-called social drugs like marijuana?

DR. DEMOPOULOS: They haven't been used long enough nor in any good epidemiologic studies but just looking at tetrahydrocannabinol, that's a toxic substance. That will damage cell membranes and will damage DNA, so will LSD; so will most of these so-called recreational drugs. I think they are fearsome and they really should be controlled for a variety of reasons, including their carcinogenic and mutagenic potential as well as the fact that they absolutely destroy nerve cells. The reason for producing a "high" is a lot of nerve cells are just getting disconnected. The synapses are missing so that you no longer have the major inhibitions that are inherent in the electrical circuits in the brain that keep us under control. I don't mean moral inhibitions, I mean the ability to focus on one thing at a time, to concentrate. The brain functions by blotting out everything else except what you are supposed to pay attention to. The reason you get a "high" and start seeing visions with LSD amd mescaline is that a lot of circuits are disconnected and you can no longer focus on any one thing; virtually every circuit in your brain has been turned on and is short circuiting.

These drugs are very dangerous. Whenever you start damaging cell membranes like that, you damage a lot of cell control mechanisms and open the way to birth defects, cancer and every other chronic disease, including aging.

QUESTION: In our community we have had some questions about asbestos cement water pipes. Is there any problem?

DR. DEMOPOULOS: I think in specific instances, you would have to sample water like that. You would have to look at it. I don't doubt that in occasional circumstances it is quite possible to carry carcinogenic loads of a chemical substance through the air and through the water, but I think they are unusual; where there are questions, I think you should take a look and see what the concentration is. The dose is all important, for sure. If it's a low dose, I wouldn't worry about it.

QUESTION: There are numerous problems that you have mentioned in your discussion relative to society's view of the cancer situation. Number one, you have the terror factor, the fact that people are more afraid of cancer than they are of heart disease. What, in your opinion, can communities, can we as concerned people, many of us, do to offset that view?

DR. DEMOPOULOS: Well, I think you have already done one thing as industry groups, and that is, I think, coordinated programs as AIHC has carried out on behalf of industry are very important. You must be unswerving in the pursuit of truth, it's good business.

I was shocked and amazed back in 1976 when I came here to direct the Cancer Institute in New Jersey. The press and the Department of Environmental Protection in Trenton were literally tearing industry apart. I always thought that you fellows were really hot stuff and would have answered back and I was surprised that industry was just laying back being beaten up. There was no response. I couldn't understand it. I think AIHC is one way to respond. I think that if you tap the angry potential that I assure you is there in large numbers in the academic university community, you will have a very firm ally provided you seek to serve truth and society. We have had no problems getting topnotch university scientists to stand up and be counted and speak the truth even at the risk of angering the National Cancer Institute from whence their grants come. Our peers judge our grant application; thank God the politicians and "greenies" do not.

"The Commission will notify all parties whether the case will proceed under simplified or conventional rules. Prior to the conference-hearing, the parties will get together to determine what is being disputed and the issues to be resolved. The Judge will then schedule a conference and a hearing. At the Conference the Judge will list in the record all agreements reached and defenses raised. The parties and the Judge will then attempt to resolve the disputed issues.

"If any issue remains in dispute, the Judge will hold a hearing. At the end of the hearing each party can present oral argument. If any party wishes to file a written argument, the Judge should be informed so that a date for filing can be set. A transcript will be taken of the hearing. The Judge will then file a written decision with the Commission. It shall become final 30 days thereafter unless it is called for review by any of the Commission members. The Commission will then issue an order affirming, modifying or vacating the Judge's decision."

DR. WEILL'S SUMMARY REFLECTS VALUE OF LYON MEETING ON MINERAL FIBERS:

Dr. Hans Weill of the pulmonary diseases section of Tulane University's Department of Medicine was charged with summarizing the Symposium on Biological Effects of Mineral Fibers sponsored by the International Agency for Research on Cancer Sept. 25-27 in Lyon, France. His report discussed what was new, what was controversial, where additional research was needed and, in general, reflected the previously reported view of other participants that the meeting was valuable (*OCCUPATIONAL HEALTH & SAFETY LETTER*, Oct. 22, 1979).

He said that considerable progress has been made in the identification and quantification of asbestos fibers in tissue, although some reservations were expressed about differences in tissue mineral fiber counts between labs.

"Questions remained concerning the effect of high temperatures on the physical integrity of asbestos fibers, with a prominent example being the fate of asbestos in brake-lining during use," Dr. Weill's summary said. "Is there consequent altered crystalline structure with the production of unstable fibers? The definitive answer seemed not yet available."

There seemed to be agreement among conference participants that carcinogenic potential of asbestos is importantly related to fiber length, he said. Asbestos exposure in the asbestos-cement industry was considered by some to differ from other sources of asbestos exposure, perhaps related in part to altered surface properties, or possibly because asbestos fibers may be coated with small calcium-containing particles.

"There appeared to be little new information on the relationship of fibrogenesis and carcinogenesis associated with asbestos exposure," Dr. Weill's report said. "Animal investigations seem not to have been helpful in resolving this important question. Some participants continue to ask whether an excess carcinogenic risk (lung cancer) is associated with asbestos exposure in the absence of pulmonary fibrosis (asbestosis).

"It was agreed that we will probably never be able to make the determination in an individual case, but preliminary epidemiologic data suggest that the carcinogenic and fibrogenic dose in some aspects of the industry may be similar or indeed that the fibrogenic dose could even be lower.

"On the relationships between asbestos exposure and smoking in determining the relative lung cancer risk, it should be pointed out that non-smokers have been shown to be at greater risk than non-smoking workers who have not been exposed to asbestos. Data from New York and Quebec suggest that asbestos exposure alone (without cigarette smoking) carries such an increased risk, although the number of excess cases is far smaller than for smoking asbestos workers.

"I sensed disappointment (which I share) in the expression by some conference participants of continuing difficulty in diagnosing mesothelioma, in spite of the use of electron microscopic and histochemical techniques. There did not seem to be general agreement that all mesothelioma panels have uniformly helped to increase the precision with which the diagnosis of mesothelioma can be established. Some panels appeared to be more successful than others.

"It was suggested that perhaps pathologists should borrow some principles and approaches from the developers of the ILO U/C Radiographic Classification, with greater emphasis on standardization, quantitation of inter- and intra-observer variability and similar considerations. Discussants generally felt that there still were inadequate data on dose-response relationships for mesothelioma but the important contribution of Dr. Whitwell was noted as a convincing demonstration of dose relatedness for this tumor."

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On the key question of dose-response relationship, Dr. Weill's summary stated:

"In regard to carcinogenic effects, there appeared to be a consensus that the shape of the curve is linear and that no threshold exists. While in no way refuting these hypotheses, I would only raise the question as to whether we have come to this consensus by genuine agreement or submission. There can be little doubt that cancer risks at low doses over a working lifetime have not to date been estimated by observations at these low levels of exposure but rather by extrapolation via the mechanism of statistical modeling.

"In the absence of observations, this is certainly the correct approach to risk assessment. However, the advantages of obtaining biological response data at long-term low-level exposure with adequate follow-up of exposed working populations are undeniable.

"Additional but incomplete data have emerged in recent years suggesting the probability that dose response curves differ for various phases of the industry, but how and whether this should influence public policy decisions is certainly not clear. Further, as regards the dose relatedness of these diseases, one might ask if Mr. Peto has surrendered on his 'no dose relationship' position. If he is right, what are the control implications? They seem both obvious and dismal.

"Happily, the great majority of the participants of this conference are convinced by the evidence which leads to the conclusion that the lower the dose of asbestos exposure, the lesser the risk of malignant and non-malignant disease.

"Finally, since the validity of dose-response relationships depends critically on measurement of exposure, it is appropriate to ask: are we now measuring airborne asbestos dust with adequate precision and sensitivity? Alternatives to the now widely applied optical microscopic counting of asbestos fibers are EM techniques and mass measurements. There seemed little discussion on this point by the participants of this symposium, which may suggest that these alternatives are not practical for wide use or that their biologic validity or relevance has not been established."

SHELL STUDY SAYS CHEMICAL WORKERS ARE LESS WORRIED THAN PUBLIC:

Workers in the chemical industry are less concerned about workplace exposures than is the general public, according to a study by Rene D. Zentner, manager of opinion research at Shell Oil Co., and presented to the American Institute of Chemical Engineers meeting in San Francisco.

"Despite public apprehension over exposure to carcinogens in the workplace, statistics show that chemical workers, the group most directly affected by it, are less concerned," he said. The study also used data from Cambridge Reports, Inc., whose partner is Pat Caddell, President Carter's pollster.

"For many years, the U.S. chemical industry has enjoyed a good reputation," the report said. "Recently, because of a number of well-publicized industry bad practices, public confidence in it has declined. The public increasingly sees the chemical industry as contributing to air and water pollution, and are beginning to blame workplace exposure to chemicals as a key cause of cancer. Nevertheless, there is general agreement that the safety and health of the workplace have been increasing recently. Chemical workers are significantly more supportive of industry practices than is the general public."

Drawing on a series of tables from the Cambridge Reports national public opinion surveys to support his conclusion that chemical industry workers often support positions taken by industry management in regard to such matters as zero-risk, costs, etc., Zentner said that several trends and conclusions can be drawn:

"The first trend is the well-established national apprehension over the disease cancer. The second trend is the increasing identification by Americans with the American chemical industry as a source of carcinogenic substances. So long as these trends continue to converge, it can reasonably be expected that the public will support legislative policies restricting exposure to chemicals, both of consumers and of workers in the chemical industry.

"It should be kept in mind that the apprehension over widespread exposure to carcinogenic chemicals is a perception not necessarily exact. Indeed, the federal government agency responsible for national environmental policy, the Council on Environmental Quality, stresses the small proportion of chemicals that may cause cancer. . .

"Whatever the underlying phenomena, however, the public increasingly is concerned about its exposure to carcinogenic chemicals, both on and off the job.

"Despite public apprehension over exposure to carcinogens in the workplace, however, chemical

are probably transported by macrophages to the muco-ciliary escalator of the respiratory passages and cleared from the lung to be subsequently swallowed. The fate of the remaining fibres is thought to depend upon diameter, length and possibly straightness. Animal experiments show that, for reasons not fully understood, much less chrysotile than amphibole fibre remains in lung tissue after inhalation has ceased.

65 Fibres of a wide range of chemical structures (including substances as diverse as glass and aluminium oxide as well as asbestos) have been shown to produce mesothelioma after injection into the pleural or peritoneal cavities in animals if the configuration of the fibres falls within a certain range. Experiments showed that for asbestos, glass fibre and aluminium oxide the degree of carcinogenicity is related to the proportion of fibres with diameters between 0.5 and 2.5μm and lengths 10-80μm. In later experiments using glass fibre only the highest incidence of tumours occurred with fibres up to 1.5μm in diameter, including very thin fibres of diameter less than 0.25μm and of length greater than 8μm. It is important to point out that 0.5μm represents the approximate limit of light microscopy in respect of fibre diameter.

66 So although the doses administered were very large indeed and the results of intrapleural implantation of fibres in rats cannot be used as a reliable predictor of risk to man through inhalation, these experiments strongly suggest with the current optical methods the total number of respirable fibres will have been under-estimated but to a similar degree in different types of exposure, so that our conclusions about relative risks are not significantly affected. However there must remain uncertainty about this. The development of methods which measure the whole range of respirable fibres is urgently needed.

Asbestos-related disease, portals of entry and types of exposure

67 *Asbestosis*, which may be defined as fibrosis of the lungs caused by asbestos dusts which may or may not be associated with fibrosis of the parietal (outer) or pulmonary (inner) layer of the pleura, lung cancer (carcinoma of the bronchus), and mesothelioma of the pleura and peritoneum are universally accepted as diseases with a causal relationship to the inhalation of asbestos fibre. Benign pleural effusion (fluid developing in the space between lung and chest wall) may occur acutely in association with other asbestos-related lung diseases or as the only or most prominent disease process. It may be discovered by accident or associated with pain, fever and malaise. Thickening of the pleura may occur, alone or in association with fibrosis of the lung, and may be symptomless or associated with chest discomfort and restriction of breathing.

68 Asbestosis, which by definition is specifically related to asbestos, is difficult to diagnose because its onset is gradual, and because the symptoms and signs may occur in other diseases and are often difficult to detect. Minor changes in x-ray pictures attributable to asbestos may exist for many years without symptoms or progression.

69 *Lung cancer* is easier to diagnose than asbestosis but the tumours which occur in relation to asbestos have no special diagnostic feature. As tobacco-related lung cancer is common and the effects of tobacco and asbestos are known to interact synergistically in smokers exposed to asbestos, it may be impossible to determine in a particular case whether or to what extent a bronchial tumour is related to asbestos.

70 In the case of *mesothelioma*, although there are also diagnostic problems, the position at present is that the

Table 10 (see Vol 2 Table 3a) Cancer of the gastro-intestinal tract and peritoneal mesothelioma in 8 surveys (including 2 studying two different sub-populations) in which an excess of the former has been reported.

Reference	*Population*	*Cases Observed (O)*	*Cases Expected (E)*	*O/E*	*O–E*	*Peri.mes.*	*Fibre* Type*
Selikoff[3]	Insulators	43	13.6	3.15	29.4(S)‡	25	C & A
Elmes & Simpson[5]	Shipyard workers	15	5.2	2.91	9.8(S)	0	Cr, C, A
Selikoff[4]	Amosite workers	26	12.5	2.07	13.5(S)	6	A
Enterline[6]	Maintenance workers	22	13.3	1.65	8.7(S)	0†	C, A & Cr
Selikoff[4]	Insulators	61	37.8	1.61	23.2(S)	63	C & A
Newhouse[8]	Insulation manufacturers	31	20.4	1.52	10.6(S)	15	C, A & Cr
Mancuso[7]	Production workers	16	10.6	1.51	5.4(NS)	6?	?
McDonald et al[9]	Thetford miners and millers	165	144.8	1.14	20.2(NS)	0	C
Enterline[6]	Production workers	37	32.5	1.14	4.5(NS)	0	C
McDonald et al[9]	Asbestos miners and millers	125	150.4	0.83	—25.4(NS)	0	C
Peto et al[9]	Textile workers	16	15.7	1.03	0.4(NS)	0	C & ? Cr

*A = Amosite; C = Chrysotile; Cr = Crocidolite.
†It is known tht many cases of mesothelioma occurred in younger men in this factory.
‡(S) = significant excess (P<0.05) (NS) = no significant excess (P>0.05).

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phyllite alone (Table 15). However, as important gaps in our knowledge of this aspect of the field remain unanswered, the weight which can be attached to this conclusion at present is limited. The high relative risks of lung cancer reported in a single study of workers making insulation material containing amosite even after very short exposure is worrying in view of the increase in utilisation of this material in the United Kingdom since the War (Table 16). There is no reliable information from South African amosite miners to help us on this point. We conclude that a firm judgment on the question whether amosite has been more dangerous than chrysotile in respect of lung cancer is not possible at present and that further work should be commissioned urgently.

ASBESTOSIS IN MAN

80 Quantitative evidence about impairment of lung function in man in relation to fibre type is limited to one study which suggests that crocidolite may have been more harmful than chrysotile. Information from the same amosite insulation manufacturers as were mentioned in connection with lung cancer, and their families, shows that radiological changes suggestive of asbestosis occurred after relatively short exposures. Evidence for a difference in risk of asbestosis with fibre type is at present slight.

MESOTHELIOMA IN MAN

81 As far as mesothelioma is concerned, evidence from miners; from process workers exposed to a single fibre type; from the distribution of neighbourhood and domestic cases; and from the geography of mesothelioma, when combined, presents a powerful case from four different sources that crocidolite has been more dangerous than chrysotile and anthophyllite. The position of amosite may be intermediate between crocidolite and chrysotile. There is no doubt that some crocidolite was used at Rochdale, but the significance of this as a cause of the mesothelioma cases there is not certain. It can be concluded that exposure to chrysotile alone has rarely been shown to cause mesothelioma.

82 The death rate from mesothelioma as certified on death certificates in Britain, although small in absolute terms, has doubled in the decade 1967-76, (Table 14 and Fig 3). Although this trend may be affected by a recent tendency to diagnose mesothelioma more readily, the evidence concerning occupational dust exposure conditions in earlier years (to which deaths occurring at present are mainly due) suggests that the number of deaths from mesothelioma may be expected to increase further before the peak is reached.

CANCER OF THE GASTRO-INTESTINAL TRACT IN MAN

83 Significant excesses of cancer ascribed to these sites have occurred in most populations of industrial workers heavily exposed in the past to mixtures of amphiboles with chrysotile and in one population thought to be exposed exclusively to amosite. The evidence that exposure to chrysotile alone has increased the risk of gastrointestinal tract cancer is less consistent (see Table 10).

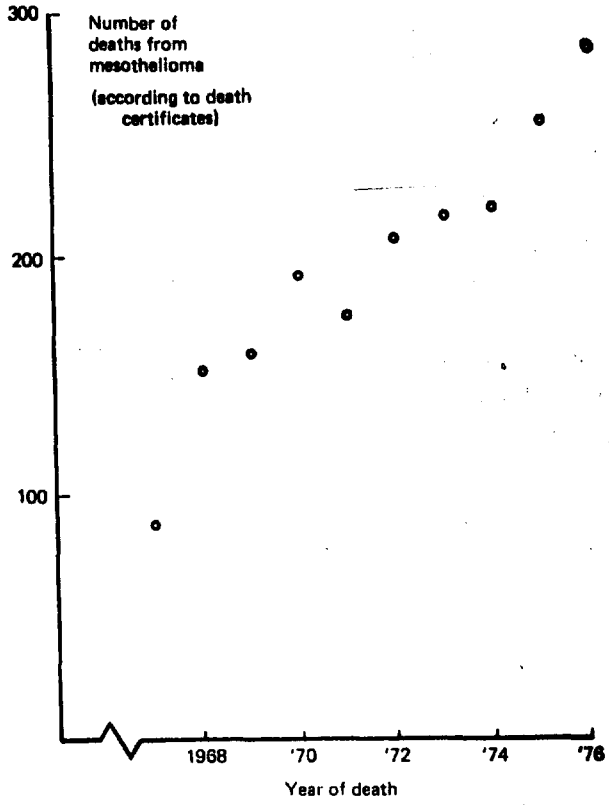

Fig 3 (see Vol 2 Fig 17) Death certificates mentioning mesothelioma. Great Britain, 1967–76 by year.

CHANGES IN INDUSTRIAL PRACTICE

84 Inhalation experiments in animals show that all fibre types of asbestos have the potential to produce similar amounts of the main types of asbestos related disease if the physical configuration of the fibres in the dust is appropriate. Intrapleural injection experiments also show that finely divided chrysotile can be prepared in such a way that it produces a similar number of mesotheliomas to crocidolite. It follows that any change in industrial practice in the direction of the production of more finely divided chrysotile fibre is likely to increase the health risk.

Dose-response relationships

85 The incidence and severity of the pathological response to increasing doses of asbestos in man is crucial to framing a rational policy about an acceptable level of exposure. Thus, if there were a level of dose below which no risk of sickness or death is incurred (ie a threshold) at least such a level would be acceptable. In contrast, if it could be shown that the largest increments in risk per unit of exposure occurred at the lower end of the range of dose, a policy of very strict control might be indicated.

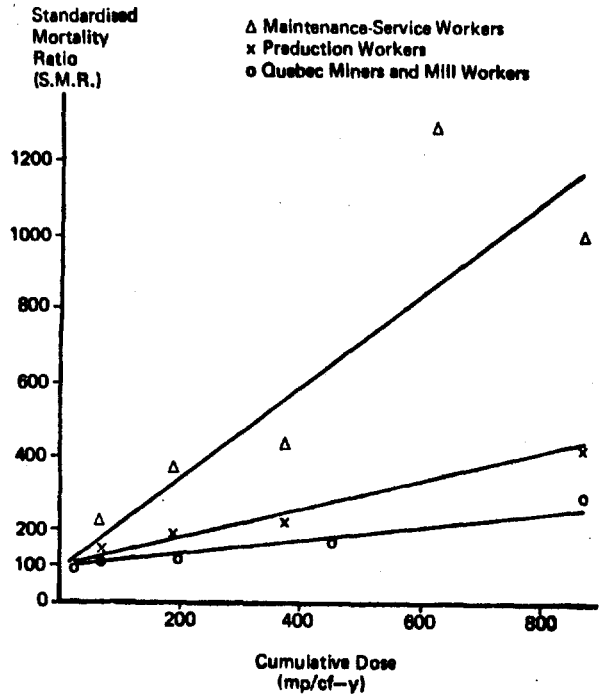

Fig 6 (see Vol 2 Fig 11) Dose-responses for lung cancer with freehand lines drawn.

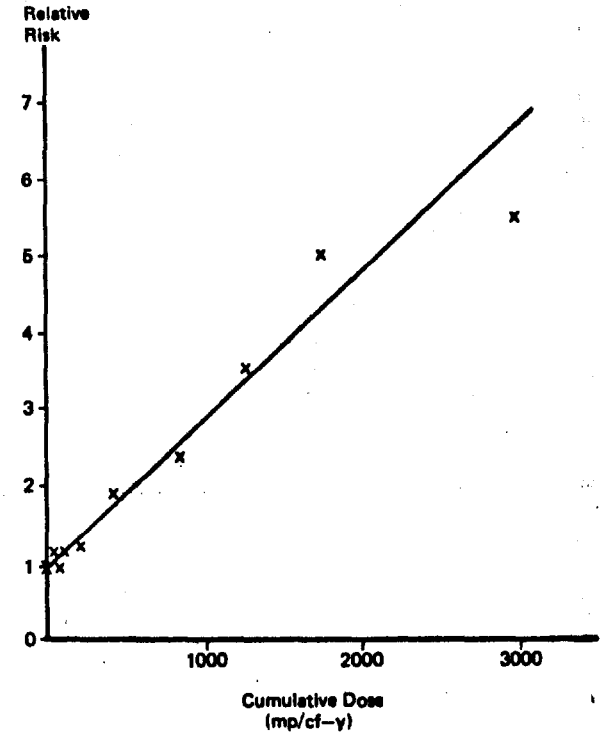

Fig 7 (see Vol 2 Fig 12) Lung cancer dose-response relationship for Quebec miners and millers.
Internal comparison: relative risks calculated relative to the low exposure group.[89]

Table 18 (see Vol 2 Table 31) Mesothelioma deaths in asbestos factory workers in Barking.

Type of exposure to asbestos	*Duration of employment*	*Deaths*	*Rate per 100 000 subject years**
Males			
Low to moderate	Under 2 years	3	31
	More than 2 years	5	83
Severe	Under 2 years	10	77
	More than 2 years	13	195
Total (males)		31	88
Females			
Low to moderate	Under 2 years	0	—
	More than 2 years	0	—
Severe	Under 2 years	9	106
	More than 2 years	5	126
Total (females)		14	97

*Can be regarded as 'rate per 100 000 per annum'
Source: Abstracted from Newhouse and Berry (1976)[26]

the exact relationship is uncertain and may be less than multiplicative. One consequence of this synergism is that any future diminution in the tobacco habit in persons exposed to asbestos will have a greater beneficial effect than in persons not so exposed.

MESOTHELIOMA

99 As far as mesothelioma is concerned the data so far available in man strongly suggest that the risk of this tumour increases with increasing dose. This is suggested by a study of 10 000 workers at a Barking factory by Newhouse and Berry[26] (Table 18); laggers were found to fare worse than process workers. The occurrence of cases due to domestic contact with people occupationally exposed and in the neighbourhood of factories and mines also suggests that relatively low doses may be followed by the development of cancer. However, information about the precise relationship between dose and response is lacking at present.

OTHER ASBESTOS RELATED DISEASE

100 As far as cancer of the gastro-intestinal tract is concerned, information about response to dose is limited to two studies reporting small excesses of these cancers, and the relationships are on the whole irregular and weak compared with lung cancer (Figs 9 and 10). It is worth noting, however, that a significant relationship between increasing dose and increasing response in statistical terms is present for cancer of the gastro-intestinal tract for maintenance workers (exposed to chrysotile and amphiboles) but not for factory workers engaged in the manufacture of asbestos textiles, building products and friction materials (exposed to chrysotile). Nothing definite can be said about the shape of the dose response curve or the existence or otherwise of a threshold.

101 For calcified pleural plaques an irregular increase in prevalence with increasing dose is observed in figures from the single study available (Fig 11) but in Quebec this phenomenon may be due to substances other than asbestos. No information is available on the dose-response relationship of cancer of the larynx.

Extrapolation and the public health

102 The view taken in this section depends upon firstly a number of the conclusions reached in the previous sections (recapitulated in summary form below); secondly certain assumptions about extrapolation of data derived from industry to situations where large numbers of persons are exposed to very low doses; thirdly, the evidence about the size of the population exposed and the severity and duration of the exposure outside the workplace.

103 The conclusions already reached relevant to this section are as follows:

(1) that asbestos fibres, principally comprising chrysotile and amosite, are continuing to accumulate in a wide variety of materials in the UK while, although raw crocidolite imports have ceased, a substantial amount of this material remains;

(2) that amphiboles and mixtures of chrysotile rich in amphiboles are certainly more dangerous than chrysotile alone in respect of mesothelioma and perhaps more dangerous in respect of lung cancer;

(3) that where quantitative data from industrial experience are available there is generally evidence for the existence of an increasing biological response to increasing dose; within the industry we have found no convincing evidence for the existence of a threshold below which no increment of risk takes place for lung cancer or mesothelioma and where sufficient data are available (as in the case of chrysotile and lung cancer, and mixtures of chrysotile and amphiboles and lung cancer) they are consistent with the linear hypothesis. For the other fibres in relation to lung cancer and for mesothelioma in relation to all three fibre types the precise shape of the dose-response curves is unknown. For gastro-intestinal cancer the question of the existence of a threshold is unsettled.

(4) The significance of minor clinical and radiological changes is in so much doubt that extrapolation from industrial experience in respect of asbestosis to the general

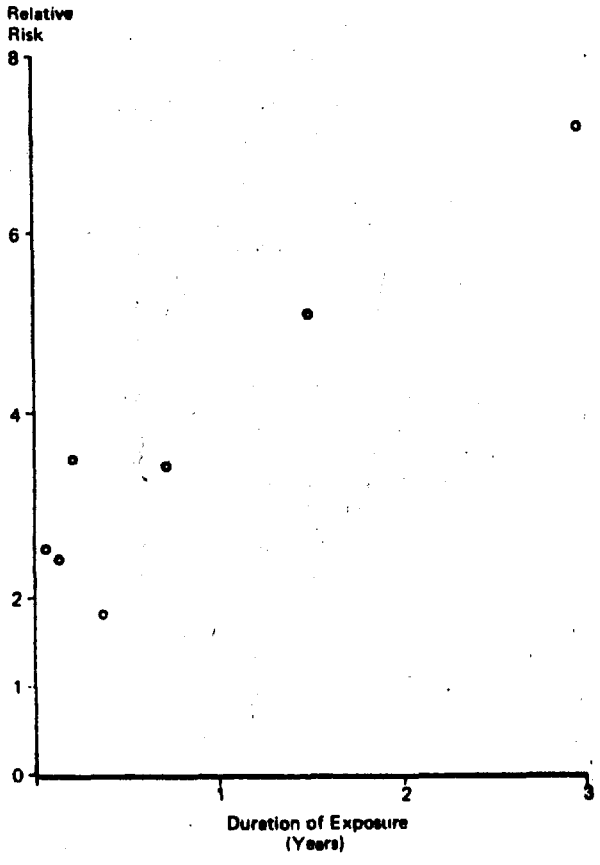

Fig 8 (see Vol 2 Fig 13) Relative risk of death from lung cancer in a group of amosite insulation workers by duration of exposure (After Seidman et al.)[21]

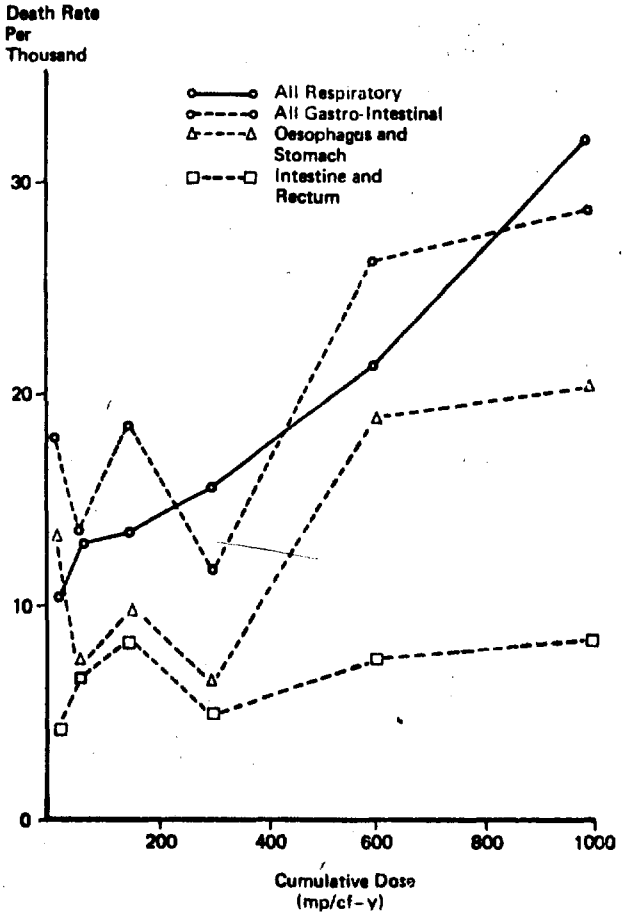

Fig 9 (Vol 2 Fig 15) Mortality from GI cancer and dust in Quebec miners and millers.
Source: McDonald JC and McDonald AD (1977).[88]

Table 19 (see Vol 2 Table 33) Asbestos dust concentrations in UK buildings according to (a) materials used, (b) type of building (Byrom and colleagues, 1969).[88]

	Type of material				*Total*	
Fibres per cm³	*Insulation board*	*Asbestos cement sheeting*	*Sprayed asbestos*	*Other**	*No.*	*%*
0—0.005	17	2	7	8	34	46
>0.005—0.01	8	3	5	0	16	22
>0.01—0.02	6	2	2	0	10	14
>0.02—0.03	2	1	3	0	6	8
>0.03—0.04	2	0	0	0	2	3
>0.04—0.05	2	0	0	0	2	3
>0.05—0.08	2	0	1	0	3	4

*compressed flat sheets, partition board and low density panels.

Fibres per cm³	*Hospital*	*Education*	*Factory or storage*	*Office*	*Shop*	*Place of assembly*	*Residence*	*Misc.*	*Total No.*	*%*
0—0.005	4	15	3	6	1	2	3	0	34	46
>0.005—0.01	1	3	2	2	4	2	1	1	16	22
>0.01—0.02	1	0	4	0	1	1	2	1	10	14
>0.02—0.03	2	0	2	0	0	0	2	0	6	8
>0.03—0.04	0	1	0	1	0	0	0	0	2	3
>0.04—0.05	0	0	0	1	0	0	1	0	2	3
>0.05—0.08	0	0	0	2	0	0	0	1	3	4

1 % of the present standard for chrysotile. We conclude that, in view of the presence in certain buildings of asbestos products, some of which contain crocidolite or amosite which may be subject to abrasion or damage during normal use, larger samples of buildings should be studied, particularly where amphiboles have been sprayed or used in insulation board.

107 Studies of lung tissue taken in samples of persons subjected to autopsy have shown that the prevalence of embedded asbestos fibre may be as high as 50 %, that the prevalence is increasing and that it varies geographically. The component of this prevalence which is occupational, or non-occupational in origin, is not clear from the published work. Many of the fibres found are extremely short.

108 There is no published information about the presence or otherwise of asbestos fibres in water in the UK. There is evidence from abroad which suggests that fibres may be leached from asbestos-containing pressure piping under certain circumstances. Fibres from asbestos deposits in other rocks and ores and from industrial contamination of water supplies have also been found. Following on from the report of the FACC and the results of investigations by the DOE, we believe that it would be advisable to study this phenomenon within the range encountered for the UK of acidity and other chemical characteristics of water supplies for households and the manufacture of food and drink.

The public health risk

AIR

109 We conclude that the presence of chrysotile containing small quantities of amphiboles is unlikely to have produced any material increase in the risk of lung cancer in the general population or any appreciable number of cases of mesothelioma. The same is certainly true of asbestosis. Pleural changes similar to those associated with occupational exposure occur in circumstances where occupationally related exposure is unlikely. The relationship of these to environmental asbestos or other minerals is not proven. Cases of pleural thickening and calcification have been reported in those presumed to have been exposed to asbestos dust non-occupationally in the neighbourhood of mines and factories.

110 As far as the amphiboles crocidolite and amosite and mixtures rich in them are concerned, the position is less certain because of the lack of quantitative data about the relationship of response to dose. Such data as exist suggest that the risk of lung cancer associated with amphiboles may be greater than for chrysotile. An excess lung cancer risk cannot be completely excluded, at

Table 20 A comparison of the number of deaths from cancer and mesothelioma in the UK during 1975

all cancers	139,899	UK
lung cancer	37,152	UK
mesothelioma	256	UK

Source: Annual Abstract of Statistics 1976. Mesothelioma Register

present, in those who have been exposed only to amphiboles in buildings. But the number of cases, if any, is probably very small.

111 While the majority of reported mesotheliomas are attributable to occupational and indirect occupational exposure to amphiboles, some cases have been reported where the exposure has arisen as a result of domestic contact or neighbourhood exposure. Other mesotheliomas cannot be convincingly attributed to asbestos exposure but some of these in other countries appearto have been caused by other fibrous minerals. A marked feature of the asbestos-related mesotheliomas has been the geographical clustering of cases in areas where large quantities of amphiboles were handled. The absolute number of mesotheliomas registered in comparison with other causes of death is low (see Table 20), but despite the improvement of industrial practice in the last twenty years and a number of other favourable points which have been mentioned above which give grounds for optimism, an important area of uncertainty remains.

THE RISK TO CHILDREN

112 Although published instances of asbestos related disease due to the exposure to asbestos of children at home, in schools and in the vicinity of dumps attract attention, they are rare. However, further surveys of school buildings containing asbestos and of dumps in the vicinity of asbestos factories on which children might play are desirable for two reasons. The first is that susceptibility to cancer is known to vary with age, the very young being especially at risk in relation to certain stimuli. The second is that as children can be expected to live longer than adults they have more chance of being affected by carcinogens with long latent periods.

WATER, FOOD AND BEVERAGES

113 The scale of consumption of drinking water supplied through asbestos-containing pipes is such that even a very small increase in risk of abdominal tumours, should it be found to exist, might cause an appreciable number of cases in absolute terms. It would also be extremely difficult to detect. There is however no evidence of the existence of risk in animals associated with the ingestion of asbestos. Industrial experience suggests that any risk in man may be limited to persons exposed to high doses, and the quantities of asbestos fibres in water have been found in other countries to be extremely low. It is understood, however, that further studies on the ingestion of asbestos are being carried out. Following on from the FACC report, we believe that studies of the leaching of asbestos fibres from pressure pipes in the range of acidity encountered in UK public water supplies should be undertaken.

Types of asbestos: implications for public policy

114 Following the lines laid down in the terms of reference of the report each type of asbestos fibre is dealt with separately in this section. However it is important to repeat here a point made in earlier sections. This is that the conclusions reached about the effects of the different fibre types on man relate to past industrial practices, and that any change in industrial practice in the direction of the production of more finely divided fibre is likely to increase the health risk.

CROCIDOLITE

115 There is strong evidence from a number of sources that inhalation of crocidolite can cause mesothelioma, and that although the risk increases with increasing dose it is possible for mesothelioma to occur after relatively brief exposure to this amphibole. A substantial proportion of the deaths from mesothelioma which have occurred and are occurring in Britain have probably been due to the inhalation of amphiboles including crocidolite. Although the available evidence is inconclusive it is consistent with the view that crocidolite may also have been more dangerous than chrysotile as far as the causation of lung cancer is concerned. Crocidolite is one of the amphiboles associated with the excesses of cancers of the gastro-intestinal tract (if these are in reality not misdiagnosed peritoneal mesotheliomas) and may also be concerned in a small number of cases of laryngeal cancer. Evidence from the only study in man in which quantitative comparisons between crocidolite and chrysotile are possible suggests that crocidolite may cause more asbestosis than chrysotile. The animal work on this point is conflicting.

116 We believe that contact between man and crocidolite should be limited to the minimum practicable and stringent regulations for the protection of workers and of the public should continue to be applied in respect of crocidolite-containing materials still present in buildings, vehicles, ships, dumps and other locations.

AMOSITE

117 The importance of reaching a correct judgment about the amphibole amosite is underlined by the import trends to the UK since World War II which show a sevenfold increase in tonnage imported compared to a twofold increase in chrysotile. The tonnage of amosite imported in 1975 (19 200 tonnes) was almost three times the largest import figure for crocidolite since 1945 (6800 tonnes); (see Table 3 of Chapter 1).

118 The principal difficulty about the evidence on the effects of amosite upon health is that very few definable groups of men have been exposed to amosite who have not also been exposed to crocidolite, particularly in the United Kingdom. As a cause of mesothelioma, we take the view that the risk from amosite may be intermediate between chrysotile and crocidolite. For reasons argued

128 In respect of carcinogenicity we conclude that up to the present time chrysotile has rarely caused mesothelioma or cancer of the larynx. These are favourable points which should be taken into account in framing policy. On the other hand it is essential to restate the proven capacity of chrysotile to produce mesotheliomas in animals if the fibre is of the appropriate configuration, and to emphasise once again that chrysotile is probably becoming increasingly finely divided and respirable.

129 In the case of lung cancer there are two studies from North American and one from Britain which provide data which can be used to help us arrive at a standard. In the North American studies (Quebec miners and Enterline's process workers) the doses of dust are recorded in particles per cubic foot. It follows that if use is to be made of them a transformation must be carried out. This at best introduces additional uncertainties. Our approach has been to show the effect of three possible conversion factors. For the Rochdale lung cancer study we show the effect of taking the dust levels as measured and of increasing them by factors of 2 and 5 to take account respectively of the effect of modern instruments, and of the combined effect of modern instruments and personal samplers (see Table 17).

130 The array of figures in Table 21 sets out the excess mortality from lung cancer which would occur to workmen exposed to various chrysotile levels over 50 years from calculations based on the various studies. A range of excesses is allowed for from 2% (i.e. that 2% more of the men exposed at the given level would die of lung

Table 21 (see Vol 2 Table 35) Dust concentrations in fibres/cc that would produce shown excess mortality from lung cancer after 50 years exposure according to linear dose-response models.

Study	*Fibre type**	*Conversion factor†*	*Dust levels for stated excess mortality from lung cancer*			
			0.1%	0.5%	1%	2%
Quebec	C	5	0.5	3	5	11
		2	0.2	1	2	4
		1	0.1	0.5	1	2
Enterline (production workers)	C	5	0.3	1	3	5
		2	0.1	0.5	1	2
		1	0.1	0.3	0.5	1
Enterline (maintenance workers)	C, A, Cr	5	0.1	0.4	0.8	2
		2	0.04	0.2	0.3	0.6
		1	0.02	0.1	0.2	0.3
Rochdale	C, ? Cr	—	0.04	0.2	0.4	0.8
		2	0.08	0.4	0.8	1.6
		5	0.2	1	2	4

*C = Chrysotile, A = Amosite, Cr = Crocidolite.

†For Quebec[27] and Enterline[4] studies figures are ratios of fibres per cc to million particles per cubic foot.

For Rochdale conversion factors are the lowest and highest shown in Table 17 to convert to modern techniques with personal sampling.

Table 22 (see Vol 2 Table 36) Number of excess deaths from lung cancer among one million people that might be caused by the highest observed environmental asbestos dust levels in ambient air and the median or highest levels encountered by Byrom in buildings if they were exposed continuously for 50 years.

Study	*Fibre type†*	*Conversion factor†*	*Number of extra deaths from lung cancer among 10^6 births from stated exposure to asbestos in fibre years per cc*		
			0.01*	0.25††	4**
Quebec	C	5	0.2	5	81
		2	0.5	12.5	203
		1	1.0	25	405
Enterline (production workers)	C	5	0.4	10	162
		2	1.0	25	405
		1	2.0	50	810
Enterline (maintenance workers)	C, A, Cr	5	1.4	35	540
		2	3.4	85	1 350
		1	6.8	170	2 700
Rochdale	C, ? Cr	–	2.7	68	1 080
		2	1.4	34	540
		5	0.5	14	216

† See Table 21.

* 10 nanograms per cubic metre for 50 years taken to be equivalent to 0.01 fibre years per cc.

**0.08 fibres per cc for 50 years is equivalent to 4 fibre years per cc.

†† 0.005 fibres per cc for 50 years is equivalent to 0.25 fibre years per cc.

245 This proposal would be appropriate for the scheduled processes discussed in para 243 above and possibly for some other operations in specified circumstances. We believe, however, that it could not be applied generally. Clearly, any such new requirement on measuring would need to be designed so as to be compatible with local authorities' powers under the Control of Pollution Act. The appropriate Inspectorate in liaison with other parties in HSE and after consultations with the industry, could help in the provision of guidance on the best practicable means of taking and assessing the samples.

Recommendation 33: we recommend that if recommendation 32 is implemented, designated and especially scheduled works which emit asbestos dust into the atmosphere should measure such emissions regularly and systematically and keep appropriate records.

Asbestos in the general atmosphere

246 On the basis of the evidence currently available, we concluded in Chapter 3 that, unless contaminated buildings are very much commoner than seems likely, no appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the UK in the air or in buildings not under active construction or repair. Even so, we recommend in our second report that a programme of work should be prepared to evaluate asbestos exposure in the non-occupational environment. We advised that resources would be most usefully directed initially to places where contamination with asbestos dust was likely to be highest and where information was most quickly needed. We are glad to hear that the Department

Table 25 Current legal and administrative controls for the general public

Relevant legislation etc	*General remarks*	*Summary of the relevant provisions*
Public Health Act 1936 and 1961	Statutory nuisance provisions of Act may apply	1 Local Authorities (LA's) responsible for the control of asbestos emissions. 2 Use of asbestos in the construction of buildings is covered by the Building Regs 1976 made under the Public Health Acts. These Regs apply in whole of England and Wales except inner London where the London Building (Constructional) Bye Laws apply. Similar Regs apply in Scotland and Northern Ireland.
Public Health (Scotland) Act 1897	S16 (General nuisances)	LA's have a responsibility for (1) any accumulation or deposit of mineral refuse which is noxious, or injurious, or dangerous to health; (2) any work etc injurious to the health of the neighbourhood or so conducted as to be injurious or dangerous to health; (3) LA's have a duty to inspect their area from time to time to identify nuisances.
Clean Air Acts 1956 and 1968	Emissions of dust from furnaces covered	Any asbestos in dust from a furnace can be subject to some element of dust control.
Control of Pollution Act 1974	Act provides framework for a systematic and co-ordinated approach to waste collection and disposal	1 LA's have powers to require information from any works about emissions to air at intervals of 3 months or more. 2 A waste disposal site licensing regime now operates.
Merchant Shipping (Dangerous Goods) Rules 1965	The carriage of dangerous goods and substances as cargo on UK registered ships is regulated and controlled under these rules	Specific advice on carriage and packing of individual substances is given in the International Maritime Dangerous Goods Code.
Merchant Shipping Act 1970	Powers are available under S19 to make regulations to secure safe working conditions and practices	1 The Dept of Trade's recommendation made in the 'Code of Safe Working practices for the Safety of Merchant Seamen'. 2 The Department of Trade have issued Merchant Shipping Notices on asbestos.
Deposit of Poisonous Waste Act 1972		1 Disposers of most types of asbestos waste should notify the responsible authority. 2 It is an offence to deposit poisonous noxious or polluting waste on land in such a way that it is liable to give rise to an environmental hazard.
Rivers Prevention of Pollution Acts 1951 and 1961	There is a similar Act in Scotland	Any discharge of liquid effluence to controlled waters from asbestos works requires the consent of the relevant Water Authority in England or Wales or River Purification Board in Scotland.
Consumer Safety Act 1978		There is no legislation dealing specifically with the use of asbestos in consumer products but there is power to introduce such legislation in the Act.
Food Drugs Act 1955 England and Wales		There are similar Acts in Scotland and Northern Ireland; while there are no specific statutory limits laid down in UK for the levels of asbestos in food, this Act will apply to any question of contamination.
Water Act 1973		Water Authorities supply water and LA's should see the water is wholesome.
Health & Safety at Work etc Act 1974		S3 obliges employers and self-employed persons to conduct their undertakings in such a way as to ensure, so far as is reasonably practicable, that persons not in their employment who may be affected thereby are not thereby exposed to risks to their safety.

sample from which this can be ascertained and whether all the precautions taken for large scale work on crocidolite should also apply to minor repair and similar work.

Recommendation 2: we recommend that the term 'containing crocidolite' should be defined and that the definition should specify the technique or techniques of analysis to be used and the criteria for deciding whether crocidolite is present.

145 One approach would be to require analysis of all such materials containing or suspected to contain asbestos to determine the asbestos type. This requirement is already implicit in Regulation 6 of the Asbestos Regulations 1969, inasmuch as notice has to be given to HMFI of all work involving crocidolite. Many responsible building and demolition contractors already carry out bulk sampling before they start work involving materials known or suspected to contain asbestos, and either notify HMFI or not according to whether crocidolite is found. The main practical problem arises with jobs where the quantity of the suspect material is so small that to treat it as containing crocidolite (that is, to use more stringent safety precautions) is cheaper than to have the material sampled and analysed. We understand that some firms already choose the cheaper alternative in such cases: that is, when in doubt, they treat the material as crocidolite and notify HMFI. For smallscale jobs, we consider this approach sensible.

Recommendation 3: we recommend that, if as at present an employer chooses to treat a given substance as containing crocidolite, he should continue to be under no obligation to carry out tests to determine whether crocidolite is in fact present.
On the other hand, we believe that the obligation implicit in Regulation 6 should be strengthened.

Recommendation 4: we recommend that, if an employer does not treat a substance as containing crocidolite, the onus should be on him to show that crocidolite is not present if his action is challenged.
Currently the only satisfactory way of discharging that obligation in respect of old lagging or sprayed coatings is by bulk sampling and laboratory analysis. In future years, when Recommendations 1 and 2 of our First Report are implemented so that asbestos is banned in insulation and sprayed coatings, it may be sufficient for an employer to show proof that the material was installed after the ban came into effect. Meanwhile the Code of Practice also recommended in our First Report will give practical guidance on work in those areas.

Substitution

146 We now turn to those types of asbestos and products containing it for which we consider that the ultimate sanction of prohibition is not justified by the evidence at present. All processes involving the use of these fibres and products at work are subject to the Health and Safety at Work etc Act 1974 and most are subject to the Asbestos Regulations 1969. All would be within the scope of the improvements to the Asbestos Regulations that we are now proposing. Nevertheless we feel that something more is needed than a mere tightening of measures to protect workers exposed to asbestos dust in the workplace, or a more rigorous enforcement of these matters. Where a substance such as asbestos has no clearly defined threshold concentration below which the health risk is non-existent there are obvious advantages in replacing it with an alternative material, provided that alternative is significantly less hazardous.

147 While the evidence so far available indicates that the most commonly used man-made mineral fibres are less harmful to man than asbestos, animal experiments suggest that any ill effects from such fibres are related more to the size and shape of the fibre than its chemical composition. It follows therefore if the size and shape of fibres of materials used as substitutes for asbestos are similar to those of asbestos there may be a potential risk. This is therefore an area in which one should proceed with caution.

148 We have been advised that it is doubtful how far Section 2 of the HSW Act can be taken to require an employer to use a less hazardous substance in place of asbestos even if it is reasonably practicable for him to do so. Because of this doubt, we concentrate here on the principles of possible requirements on substitution without considering in detail how they might be fitted into the framework of existing health and safety legislation or, indeed, to what extent they are already implicit in that legislation. We urge, however, that our recommendations should be implemented in such a way as not to weaken any such implicit requirement. We also emphasise that none of our recommendations should be implemented in such a way as to require the precipitate substitution of asbestos or products containing it by alternative materials whose safety is itself unknown or in serious doubt.

149 We have considered carefully two possible approaches, which are not mutually exclusive. The first approach would be to adopt a system of gradual compulsory substitution. This would mean that the whole range of asbestos products would need to be examined periodically by a central authority (HSE) to identify appropriate candidates for substitution. This examination would involve considering factors such as the health risk associated with each product in both manufacture and subsequent use, its economic importance, the availability of substitutes, their relative performance and cost and the health risk associated with them. Such a scheme would, in effect, amount to a gradual extension of prohibition. It would be substantially similar to the programme for substitution suggested in the TUC's written evidence to the committee and would in our view only be acceptable where the criteria set out in para 142 above applied.

150 The second approach would be to require manufacturers, specifiers and users of products containing asbestos to weigh carefully the risks incurred