AMERICAN CHEMICAL SOCIETY, 1981(June); 15(6): 615.

ES&T OUTLOOK

Is cancer environmentally caused?

It depends on the definition of "environment"

"For 30 years, oncologists have tried to prove a link between cancer and air pollution, and in every case it's been negligible." That was what John Higginson, director of the International Agency for Research on Cancer (Lyon, France), told a symposium held at the ACS Atlanta meeting under the auspices of the ACS Committee on Environmental Improvement, and it was the message that other physicians and risk experts also brought. Despite public perceptions to the contrary, industrial chemicals at large in the environment account for only a few percent of all cancers.

The growing support for that conclusion has come hand in hand with a growing recognition that the development of cancer is a complex, multistep process that can be modulated by many factors—especially diet and general "life-style." "The subject of cancer became very simplified in the '60s and '70s," said Higginson, and many believed that single chemicals, in small quantities and by themselves, could simply and diversely cause cancer. "We realize now that we oversimplified the problem."

Some factors, of course, have clearly been shown to cause cancer in man by themselves; these include cigarette smoking, alcohol consumption, and occupational exposure to vinyl chloride and asbestos. But, according to R. K. Boutwell of the McArdle Laboratory for Cancer Research at the University of Wisconsin (Madison), most of the agents in the environment that are considered to be carcinogenic need metabolic activation or promotion by another chemical to make their effects felt. A chemical that is only an initiator may have no effect without subsequent promotion. "Small doses of an initiator do not add up," he said.

Because carcinogenesis is a multistep process, it is difficult—except in such extreme cases as cigarette smoke—to point to a direct "cause." "If I hit somebody on the head with a hammer and a lump appears, everyone knows what it means," said Higginson. "Unfortunately, a great many cancers appear to be associated with what we call 'risk factors' "—which cannot be called carcinogens themselves. Examples include absence of dietary fiber, age at first marriage, and obesity.

A study by Higginson of cancer statistics from communities at different stages of development in southern Africa suggested that 70–80% of cancers can be attributed to environmental factors—where "environmental" refers not only to industrial chemicals but also to diet and behavior. This study and others suggest that over three-quarters of these environmental factors are derived from life-style.

The most persuasive argument for this view is in data on cancer incidence among Mormons, who have strict dietary laws. Mormons, who do not smoke or drink alcohol or anything containing caffeine, have a rate of cancer incidence half that of the average U.S. white population. What is more, no significant differences in cancer incidence are seen between urban and rural Mormons. "These data and others suggest that only a small part of the total cancer burden can be directly related to industrialization in a general sense or to diffuse exposures in the general population," said Higginson.

As for the possibility that hereditary factors might be involved, data on Japanese immigrants to the U.S. show that by the third generation, incidence of colon, rectum, breast, and prostate cancer—all very rare in Japan—have risen to average U.S. levels; stomach cancer—common in Japan—has dropped to the low U.S. level.

According to Richard Wilson, a Harvard nuclear physicist turned risk assessor, less than 1% of cancers are due to general exposure to industrial chemicals; 3–5% are due to occupational exposures. (Higginson added the observation that "most epidemiologists are falling all over themselves to push up the estimate so they won't be accused of selling out to industry"; both he and Wilson apparently feel that these numbers are upper limits.) And Wilson points out that given such a small total risk when all industrial chemicals are combined, the risk of any given chemical to the general population becomes very small and very hard to estimate. "The uncertainty is colossal," he said.

Wilson also said that the issue of a carcinogenic chemical's having a threshold, or "safe" level, is a red herring: "It's really an irrelevant question. It's very hard to find out, and once you do find out, so what?" For example, he said, there is no threshold level in accidents between automobiles and pedestrians—one person and one car in the street implies some finite risk of an accident; yet it is a risk that people are willing to take. "The issue is really whether the risk of a chemical is big or small, and it can be small because exposure is low, or small because its potency is low," he said.

What does all this say about protecting the public from environmentally caused cancers? Higginson puts it this way: "It is a common fallacy when considering multifactorial origin of disease to imply that all factors must be controlled to prevent it. In fact, all stepwise processes have their weak link and all that may be necessary is to break the chain." Both he and Wilson said there was a need to make sure that future chemicals do not add to the problem, but they both had strong words for the current approach. "There's a problem these days," Higginson said. "When you do a study on organic chemical X, the whole focus is on chemical X, ignoring the 100 other parameters."

– Stephen Budiansky

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International Environment & Safety, March/April 1978: 7-8.

Asbestos in the Underground – no cause for alarm

The following report is based on tests, which were carried out over a period of two days, on the respirable fibre concentrations in the London Underground. These results show that the Underground is completely safe as far as the level of asbestos dust is concerned.

In November 1977, claims were made that Underground users were being subjected to dangerous levels of asbestos dust. A pamphlet, called *"Killer Dust on the Tube"*, accused London Transport management of seriously neglecting their duties to employees and the public regarding the dust. The document was produced by the British Society for Social Responsibility in Science and a branch of the National Union of Railwaymen as part of a campaign for urgent action by the authorities.

The pamphlet claimed that the major source of asbestos dust, which can cause lung cancer and a cancer of the chest lining, were Tube train brake shoes, and lagging and insulating materials.

In the light of these claims, *International Environment & Safety* undertook to organise an independent survey of the levels of asbestos dust. We therefore asked Winton Laboratories* who have had wide experience in monitoring asbestos fibre and other airborne contaminants, if they would conduct an independent check on the amounts of asbestos dust in the air within the Underground. When we approached the London Transport Executive about this project they readily gave their permission for the tests to be carried out and offered every assistance to Winton during the testing.

In order to achieve the greatest degree of independence it was decided from the outset that, despite Winton's capability to identify and quantify any asbestos present, a third party laboratory would be used to determine what, if any, asbestos was present in the samples and in what quantity. In this case Environmental Analysis Ltd of Wirral, Cheshire, was chosen because of their considerable experience in this field and the high repeatability which they have been able to show in the past.

TEST METHOD

Test Sites

Commencing at a time before the first train of the day passed, tests continued for almost two days, samples being collected at seven stations within the most heavily trafficked Central London area namely:

Baker Street
Piccadilly Circus
Leicester Square
Camden Town
Highgate
Tottenham Court Road
Embankment

At each test site, there were 10 in total, samples of airborne dust and organic vapour were collected.

Sample Collection Procedure

The solids were collected on a standard 2.5cm pre-weighed 0.8 μm Gelman membrane filter at a flow rate and test duration as recommended in the asbestos fibre sampling procedure contained in the Asbestosis Research Council Technical Note No. 1, recognised by the Factory Inspectorate. After passing through the filter the air sample entered the gas and vapour collection tube containing a suitable chromatograph absorber.

In order to provide as complete as possible a dust level profile at each site, the airborne dust particule size distribution was measured using a light-scattering electronic particle monitor, solid samples were scraped from the tunnel lining for use in the analysis of tunnel dust and secondary airborne dust samples were taken at a higher flow rate (ca. 10 litres/min.) as compared with the dust samples taken for asbestos measurement (ca. 2 litres/minute).

The collected samples were then analysed by Environmental Analysis.

TEST EQUIPMENT

Air Samplers:

Rotheroe & Mitchell personal air samplers Model LC2.

Rotheroe & Mitchell high volume air sampler Model L10B.

Filter heads and absorption tubes from Winton Monitair 378 particulate and gas monitoring unit using pre-conditioned and weighed 2.5cm. 0.8μm millipore membrane filters.

Flow Meters: Laboratory standard GAP guages.

Electronic particle size distribution equipment, Royco self-contained portable Model 218.

LABORATORY ANALYSIS PROCEDURE

After arrival at the laboratory the filter membranes were re-conditioned and re-weighed using the same CAHN electrobalance as was used for the pre-weighing.

The filter membranes were then mounted on microscope slides and rendered transparent with glycerol triacetate. The "reading" was then carried out at a magnification of x450 using phase contrast microscopy. 100 fields were examined in every case, asbestos and similar fibres, as defined in Technical Note No. 1 (more than 5 microns long, having a length to breadth ratio equal to or greater than 3), being counted and the fibre concentration being calculated in terms of fibres per millilitre of air sampled. Any other particulate matter observed in the inspection of each 100 fields was also noted.

Three of the gas and vapour absorption tubes were analysed using the laboratory's Gas Chromatograph-Mass Spectrometer-Digital Equipment PDP/11 computer set-up.

RESULTS

The general findings were that the respirable fibre concentrations varied from a high of 0.32 fibres/millilitre at Piccadilly Circus to a low of 0.022 fibres/millilitre at Tottenham Court Road. The other identifiable materials generally found to be present were glass and other synthetic fibres up to 200 microns in length, cellulose and natural textile fibres, hair, seeds and pollen, general dust and carbonaceous particulates up to 25 microns.

TABLE 1
Asbestos and Similar Fibre Concentrations

LOCATION	*Fibre concentration (fibres/millilitre)*
Baker Street Station, Bakerloo Line, Northbound.	0.15
Baker Street Station, Bakerloo Line, Southbound.	0.19
Piccadilly Circus, Piccadilly Line, Westbound.	0.23
Piccadilly Circus, Piccadilly Line, Eastbound.	0.32
Leicester Square, Northern Line, Southbound.	0.10
Leicester Square, Northern Line, Northbound.	0.21
Camden Town, Northern Line, Southbound.	0.11
Highgate, Northern Line, Southbound.	0.21
Tottenham Court Road, Central Line, Eastbound.	0.022
Embankment, District/Circle Line, Westbound.	0.15

**Winton Laboratories Ltd, McMillan House, 54-56 Cheam Common Road, Worcester Park, Surrey. The company offer a complete environmental testing and consultancy service to industry.*

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TABLE 2
Other Particulates in Airborne Suspension Quantitative Analysis

	Concentration (mg/m³)
Baker Street Station, Bakerloo Line, Northbound	4.84
Baker Street Station, Bakerloo Line, Sothbound	0.61
Piccadilly Circus, Piccadilly Line, Westbound	0.019
Piccadilly Circus, Piccadilly Line, Eastbound	0.470
Leicester Square, Northern Line, Southbound	0.41
Leicester Square, Northern Line, Northbound	1.00
Camden Town, Northern Line, Southbound	5.76
Highgate, Northern Line, Southbound	0.69
Tottenham Court Road, Central Line, Eastbound	1.20
Embankment, District/Circle Line, Westbound	0.12

Qualitative Analysis

In addition to the asbestos fibres, other natural and synthetic fibres were present – glass fibres, textile and cellulose. However the bulk of the mass (up to 90% by volume) was made up of general dust and carbonaceous particulates in the size range 0.5-25 μm.

Summary

The survey shows that the total amount of respirable fibre in airborne suspension was, at no point greater than one-sixth of the Threshold Limit Value (TLV) for asbestos fibre – the quantity to which present regulations allow continuous exposure. This figure varies from those measured in 1974 in tests carried out by the Asbestosis Research Council and the TUC Centenary Institute of Occupational Health, which indicated that the asbestos levels were between 1000 and 10,000 times below the statutory limit. The differences in results are due to the fact that the method employed by Winton, though the one provided by statute, is not the same as the one employed in the 1974 tests. The statutory method assumes that all observed fibres are asbestos, regardless of their real composition. This system works well for an environment where asbestos could be expected to account for a high proportion of airborne dust - for example in an asbestos factory. However, problems of interpretation could arise in an environment where asbestos forms only part of a large number of constituents as has been detected in our survey of the Underground.

Subsequent work using an analytical transmission electron microscope was carried out on behalf of the LTE by Dr. F. D. Pooley of University College, Cardiff. Results showed only 1-2% of the total respirable fibre in Underground dust samples, to be asbestos.

Conclusions

Winton Laboratory concluded that in terms specifically of asbestos fibre, dust-levels in the Underground are very safe compared with the asbestos TLV. The one reservation that has to be made is that some proportion of the non-asbestos fibres present may exhibit similar physical properties to asbestos fibres and as such should not be ignored. However, even taking the most pessimistic view would not materially alter the conclusion that conditions are very safe.

We feel that this exercise has been a very useful one in terms of asbestos monitoring and co-operation from all parties concerned. Passengers can certainly have no fear whatsoever of travelling on the Underground with such levels of safety margins as has been found by recognised scientific tests.

On the IE&S survey the London Transport comments:

London Transport, using the statutory method, recently obtained results similar to those of Winton but – and this is where the difference emerges – when the airborne dust samples were analysed by Dr F D Pooley of Cardiff University (who is a recognised authority on fibre counting and identification) he quoted only about two per cent of the fibres as being asbestos.

Thus only 1/50th of the fibres were asbestos and as Winton's measurements showed on average a level of only 1/10th of the statutory limit, the consequent multiplication gives a content only 1/500th of the statutory limit, for asbestos fibres. This is in reasonably close agreement with the tests carried out in 1974, which used a method measuring the mass of the asbestos rather than a count of the fibres, as in the more recent tests. Differences in technique, location and the fact that the results are separated by some years could account for the factor of two between 1/50th and 1/100th. ■

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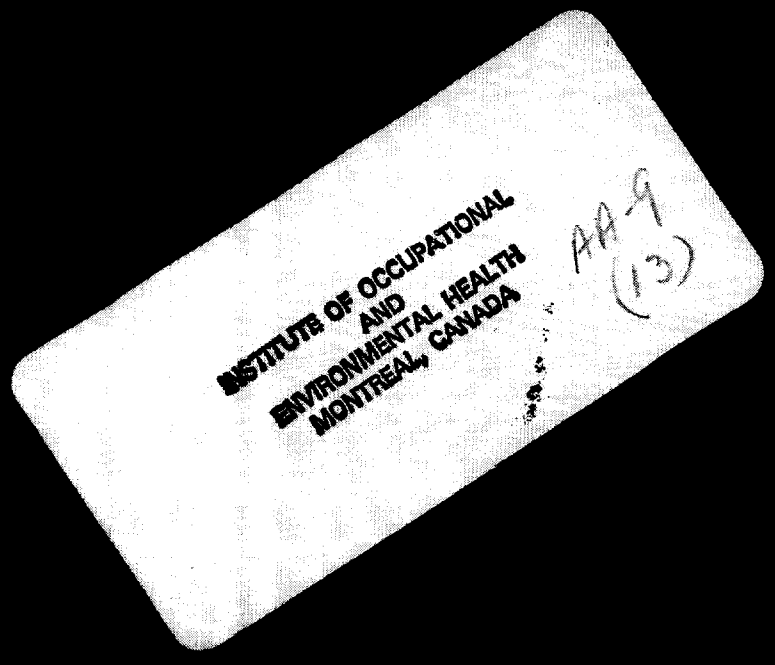
CANCER IN MODERN MORTALITY



CANCER IN MODERN MORTALITY

Uncritical acceptance by many people of the notion that there is an 'epidemic of cancer', due to exposure to modern chemicals in the general environment and the workplace, has prompted the Chemical Industries Association to commission a paper analysing the actual position in England and Wales, based on published official mortality data and the best available scientific analysis.

It is hoped that this review, by N E J Wells, a health economist, will form a constructive addition to the debate on cancer in the workplace.

Available at: Chemical Industries Association
Alembic House
93 Albert Embankment
London SE1 7TU - ENGLAND

Telephone: 01-735-3001

CANCER IN MODERN MORTALITY

INTRODUCTION

Extravagant claims concerning the role of cancer in modern mortality are both misleading from a factual point of view and dangerous because they are likely to divert attention away from more urgent health problems. The purpose of this brief paper is, therefore, to examine trends in mortality in England and Wales and to identify accurately the significance of cancer and particularly those cancers which may be linked to occupations.

TRENDS IN MORTALITY

Since 1921 the crude death rate from all causes has hardly varied (Table 1). This levelling off reflects the rapidly changing age and sex structure of the population rather than any sudden halt to the downward trend in mortality which had occurred between 1870 and the first 20 or 30 years of this century. (In 1931 those aged 65 or more, amongst whom death rates are at a peak, accounted for 7.4 per cent of the total population; today the proportion is nearly 15 per cent.)

Table 1. Death rates per million population 1921-75, England and Wales

	All Ages		
Males		**Females**	
1921-1925	12946	1921-1925	11418
1926-1930	12891	1926-1930	11369
1931-1935	12743	1931-1935	11378
1936-1940*	13495	1936-1940	11630
1941-1945*	15158	1941-1945*	11086
1946-1950*	12735	1946-1950*	10881
1951-1955	12502	1951-1955	10874
1956-1960	12346	1956-1960	10884
1961-1965	12448	1961-1965	11185
1966-1970	12360	1966-1970	11174
1971-1975	12353	1971-1975	11421

*Civilian

In order to understand more fully current trends in mortality it is necessary to study age specific death rates. To eliminate short-term fluctuations, caused for example by flu epidemics, the rates are calculated over 5 year periods (Figures 1 and 2).

At the older ages (55 or more for men and 65 or more for women) rates have declined steadily throughout the period 1921-74. For women the rate of decline has been steeper than for men; for example at ages 65-69 the net decrease over the 50 years was 13 per cent for men and 44 per cent for women. But for individuals below these ages the Office of Population Censuses and Surveys identifies three distinct periods:

(a) 1921-5 to 1941-5. In this period there was a steady decrease in mortality at almost all ages; it was steeper for females than males and for younger than older people. In World War II mortality statistics were based on the artificially selected less fit civilian population and it is this, rather than war casualities, which accounts for the increase in male mortality at some ages in 1941-5.

(b) 1941-5 to 1956-60. There was a downturn in mortality rates, particularly at the younger ages. For the under 20s rates were cut by a half or more in just 15 years.

(c) 1956-60 to 1971-75. Mortality rates, particularly among the groups where the rate of decline had been steepest in the previous period, began to flatten out or even to increase.

Figure 1. Deaths by age, 1921-75

a Males

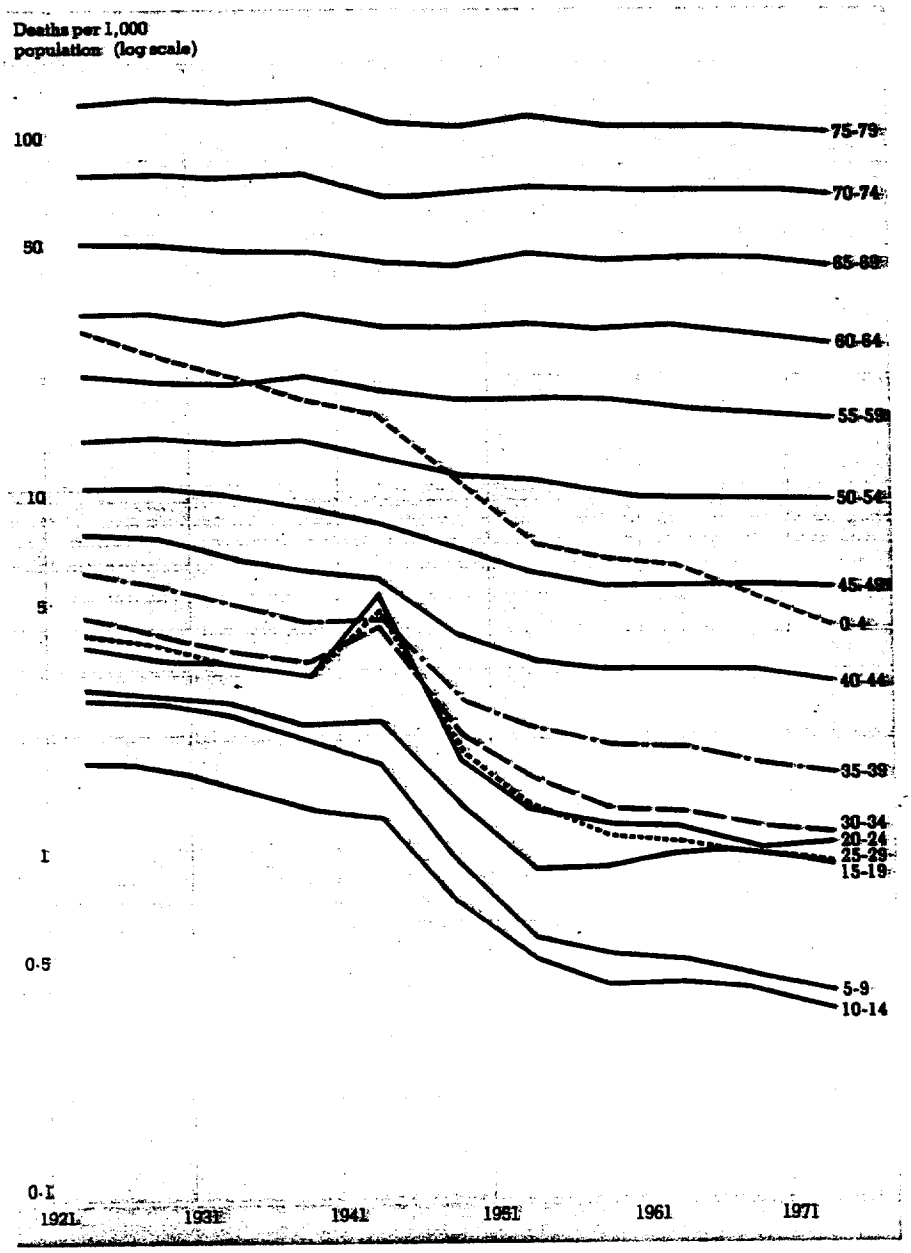
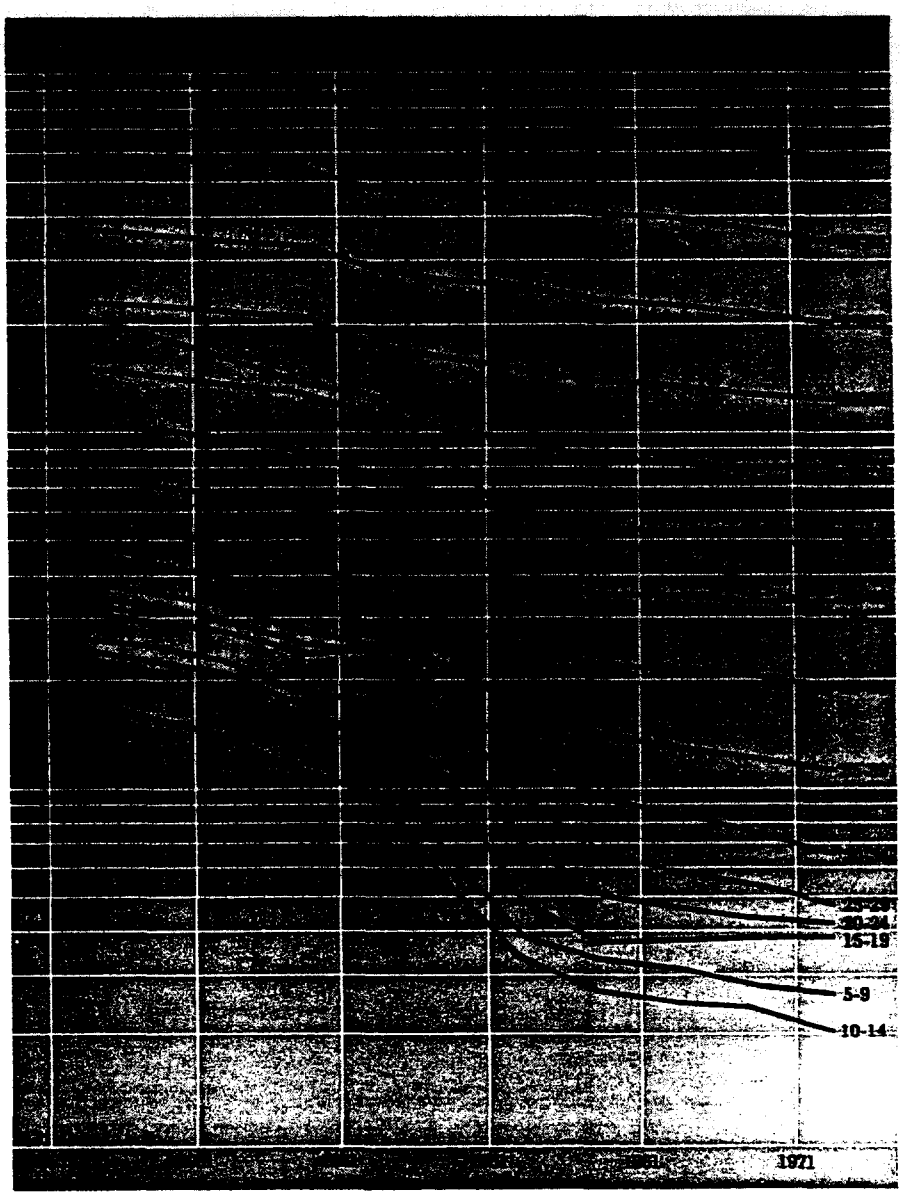


Figure 2. Deaths by age, 1921-75

b Females



CAUSE OF DEATH

Trends in mortality rates ascribed to particular diseases may reflect changes of medical philosophy, and in clerical and statistical routine as much as in the real incidence and prevalence of the diseases themselves. In order to minimise such sources of error, long-term comparisons are sometimes best confined to broad and well-recognised disease groups.

Figure 3 shows the relative importance of nine main disease groups as cause of death in 1931 and 1977. The most striking feature of the comparison is the virtual disappearance of mortality due to infective diseases (of which the most important was, of course, tuberculosis) and to maternal causes, together with the reduction in the relative frequency of deaths due to respiratory, digestive and genito-urinary disease. To complement these decreases there have been relative increases in mortality due to three cause-groups, circulatory disease, neoplasms and accidents; between them these causes now account for 75 per cent of all deaths.

CANCER

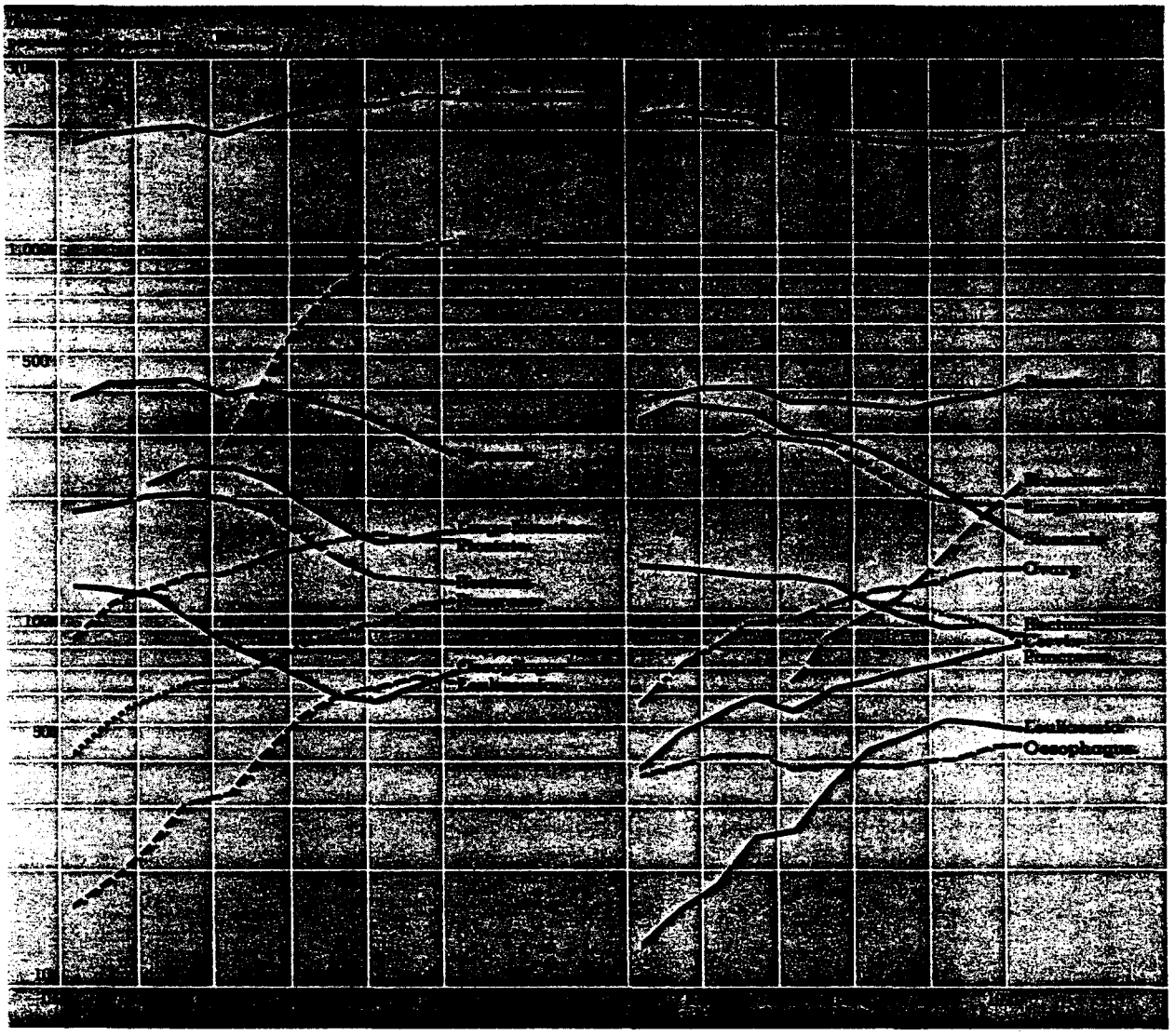
Cancer is not usually thought of as a single disease. The problems of aetiology, diagnosis and prognosis vary so much from site to site that it is customary to analyse the mortality statistics of each site separately. It is, nonetheless, of interest to study the picture of malignant disease as a whole: to look at the trends of total cancer mortality and to see the contribution of individual sites and types of cancer.

Figure 4 shows how the steady trends for total cancer mortality conceal a complicated pattern as the rates for individual types have risen and fallen. For males the death rate for all types of cancer has been increasing at about one per cent per year for the last 25 years while for females the very slow rate of decline amounting to about seven per cent over the 20-year period 1943-63 now appears to have been reversed. *In both sexes the most striking trend is that ascribed to cancer of the lung:* over the past 25 years there has been a more than three-fold increase in the male and a slightly less than three-fold increase in the female death rate. The increase in the total male cancer death rate appears to be almost entirely due to the 'explosion' of lung cancer which now accounts for about 40 per cent of male cancer deaths. *At every age the total male death rate for all cancers other than of the lung is decreasing.* Although lung cancer accounts for a much smaller proportion of all female cancer mortality (about ten per cent) the increase in mortality due to this cause appears to be largely responsible for the recent slight upturn in total female cancer mortality.

It can hardly be doubted that the increase in lung cancer mortality reflects a true increase in disease incidence associated with cigarette smoking, but there is no other site where we can be so certain in interpreting the observed trends in mortality. In some types (for example, cancer of the pancreas and leukaemia) much of the increase must be due to more accurate methods of diagnosis. In both sexes mortality due to intestinal and gastric cancer has been generally decreasing, particularly in the 1940s and 1950s. Among the genito-urinary sites there has been an increase in mortality due to bladder cancer in men while for women there have been decreases in cancer of the cervix and uterus but a rise in cancer of the ovary. Among women the numerically most important site is the breast which now accounts for 20 per cent of all female cancer mortality.

Mortality from cancer of the stomach, which numerically is the third most important site after the lung and breast, shows a very different picture: death rates are all declining and for both sexes. But this fall is difficult to explain. Five-year survival rates for stomach cancer are well below ten per cent and there is no evidence that they are increasing; thus better treatment is unlikely to account for the decline. With better diagnosis it may be that some of the deaths formerly ascribed to cancer of the stomach are now appearing under such headings as cancer of the pancreas or

Figure 4. Cancer: deaths* by site and sex, 1921-74 (age-standardised death rates)

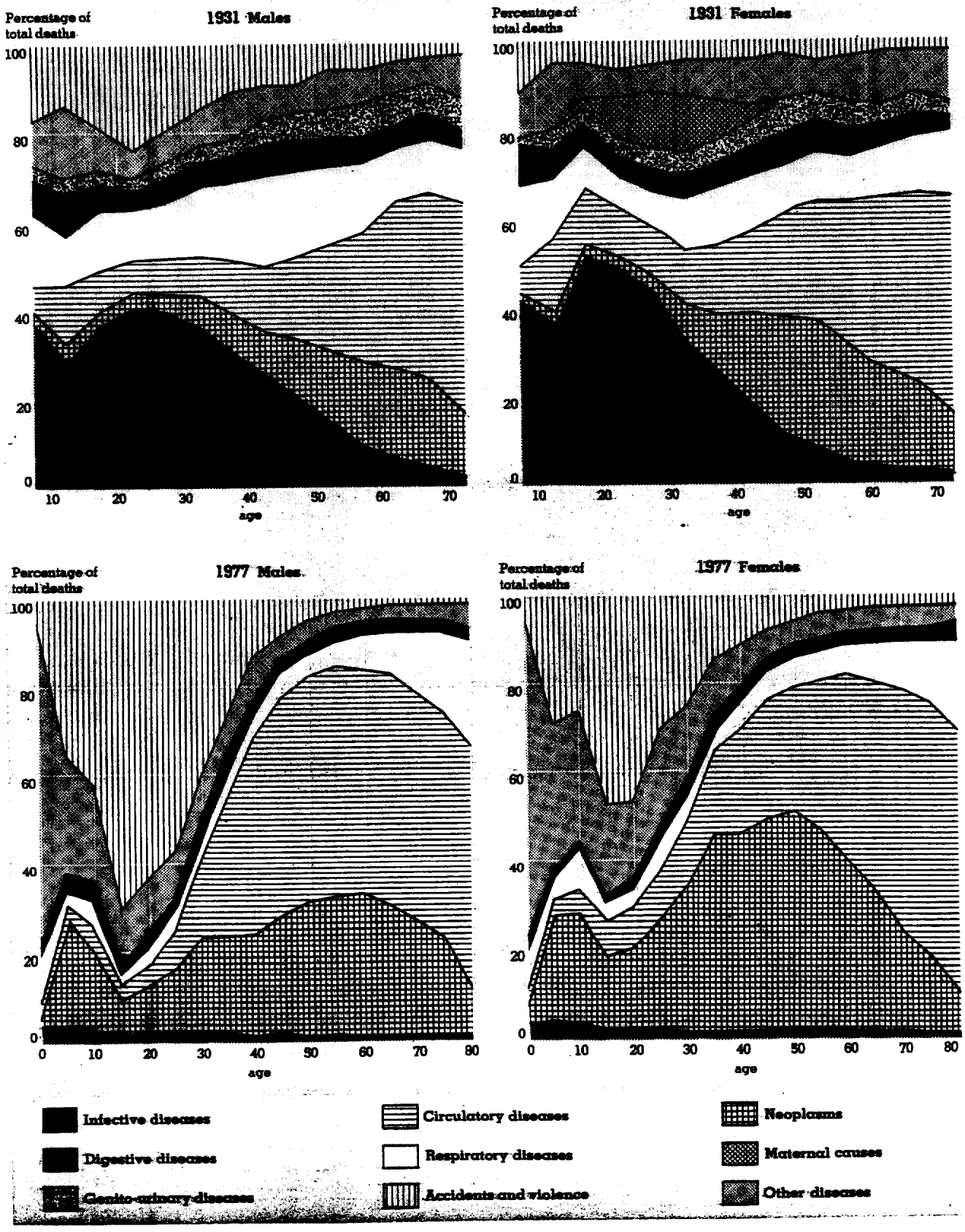


*Age-standardised death rates Source: OPCS

cancer of undetermined site, for both of which the death rates are increasing. But this explanation accounts for only a small part of the decrease. It appears that there has in fact been a true decrease in the incidence of stomach cancer and this is supported by reported registration rates.

Cancer of the large intestine shows a similar picture to that of the stomach. An important difference however is that the decrease in the 1940s and 1950s seems to have been halted; the rates, particularly for men, are now increasing. This form of cancer responds more often to treatment than stomach cancer and it is possible that the falling mortality may have been attributable to radiological and surgical advances. But the subsequent increase can hardly be accounted for by a reversal of the trend. This forces the postulate that any continuing improvement in survival rates is no longer sufficient to counterbalance an underlying tendency for disease incidence to increase – possibly as a consequence of comparatively recent changes in dietetic factors.

Figure 3. Mortality by cause, age and sex, 1931 and 1977



Focussing on breast cancer it is difficult to detect any consistent pattern in a study of the age-specific death rates. At all ages below 70 the general trend has been upwards, a typical increase being 27 per cent in the 50-54 age group between 1911-15 and 1971-74; at the age of 70 and above there has been little overall change during this period. Superimposed on this picture is a cyclical pattern with death rates rising until the 1930s and then falling and now rising again. This is consistent with the hypothesis that cancer of the breast may be related to low fertility. Furthermore it may be worth emphasising that *higher breast cancer mortality rates in the higher social groups* generates strong doubts about suggestions that overall rises in breast cancer mortality may be associated with increasing exposure of women to potential carcinogens in the working environment.

CANCER MORTALITY IN 1977 (ENGLAND AND WALES)

In England and Wales in 1977 neoplasms accounted for 126,448 deaths. This was equivalent to 22 per cent of the 575,928 deaths in that year. Diseases of the circulatory system accounted for 51 per cent and diseases of the respiratory system for 14 per cent. Table 2 indicates that approximately half of all cancer mortality occurs after the age of 70 (which is incidentally slightly more than the average length of the male lifespan). Table 3 shows the main cancer sites – cancer of the bronchus and lung accounts for more than one quarter of these deaths. The 7 sites shown in this table account together for 65 per cent of cancer mortality. (The remaining 35 per cent is spread over more than 90 other specific sites). By contrast, the liver, small intestine, bones, muscles and blood vessels (including all connective and soft tissues) together account for only one per cent or so of all cancers.

Thus the clear fact to have emerged so far is that *there has not been 'a striking increase in overall cancer death rates in this century'*. Furthermore, the relative increase in the proportion of all deaths due to cancer is a reflection of the significantly decreased significance of infectious disease and not of increasing cancer incidence.

A study of mortality patterns for people aged 45-64 years in 1975 by Sir Richard Doll focussed on cancer because it was responsible for 34 per cent of deaths in this age group. He found that for 16 sites the death rate between 1951-55 and 1971-75 decreased progressively by more than 1 per cent per annum (Table 4). For most of these cancers the decrease can be attributed to improved treatment, more precise diagnosis or known changes in personal behaviour. Only the decrease in cancer of the stomach is wholly unexplained.

Table 2. Cancer deaths in 1977, age distribution

Percentage of cancer deaths	
Under 10	0.4
10-19	0.4
20-29	0.6
30-39	1.4
40-49	4.6
50-59	14.5
60-69	29.5
70-79	32.2
80-89	14.5
90 and over	1.7
	100

Table 3. Main cancer sites, percentage of deaths in 1977

ICD		
162.1	Bronchus and lung	26.83
174	Breast	9.34
151	Stomach	8.99
153	Large intestine	8.18
154.1	Rectum	4.38
157	Pancreas	4.50
188	Bladder	3.34
	Others	34.44
		100

For 13 sites the rate has increased progressively by more than 1 per cent per annum (Table 5). The increases in cancer of the pharynx and lung in women and of the oesophagus in both sexes can be attributed to the increased consumption of alcohol and cigarettes. The explanation for the rising mortalities from cancer of the breast and melanoma is uncertain. The former is likely to be due in part to a reduction in fertility, but it may also be due to an increased consumption of meat and fat. The latter has been attributed to more extensive exposure to ultra-violet light. Whether the increases in cancer of the pancreas, non-Hodgkin's lymphoma and myelomatosis are real or spurious is open to question. For myelomatosis, however, the increase has continued for so long and has been so rapid that it would be unwise to ignore the possibility that part of it may be real. Apart from non-Hodgkin's lymphoma, it is the only numerically important type of cancer looking for a new environmental cause.

From this and other evidence Doll concludes that the health of the country has been improving steadily, and that those conditions that have become more common have for the most part done so because of personal and dietary habits that are unrelated to pollution of the environment in the normal sense of the term.

THE ROLE OF INDUSTRIAL CHEMICALS IN CANCER

In the entire history of occupational medicine there can be few industrial hazards that have aroused greater emotion or more muddled thinking than chemically induced cancer. In recent years greater awareness (but not greater understanding) has developed because:

1. Epidemiologists have demonstrated new hazards
2. Publicity arising from litigation
3. Animal experiments have shown that many industrial chemicals are carcinogenic.

However, the frequent assumption that substances which induce tumours when deliberately introduced into animals – often at high dose – will necessarily do so in workmen exposed in industrial conditions is a non sequitur. Thus great caution is needed in extrapolation: for example, carbon tetrachloride is carcinogenic to the mouse, hamster and the rat; chloroform produces inner tumours in the mouse but despite widespread exposure there is no evidence in man. The drug isoniazid induces lung tumours in the mouse.

It should also be borne in mind that:

1. No carcinogen has been described in respect of which it is experimentally impossible to find a dose which will not cause tumours in a finite experimental population. Exposure to sunlight, sex and soot – all carcinogens – is taken for granted without much thought given to the possibility of developing cancer of the skin, cervix or scrotum. (The latter was in fact the first recognised carcinogen – the cause of chimney sweeps' cancer of the scrotum described by Percival Potts in 1775).
2. There is a long and variable latent period from first exposure to diagnosis – in the case of bladder tumours apparently from 2½ years to 30/40 years.
3. Individuals vary in their response to carcinogens.
4. It is impossible to control for confounding variables.

With these considerations in mind it is now possible to look at the evidence for an association between industrial chemicals and cancer. A Royal Society study group (under the chairmanship of the leading British epidemiologist Sir Richard Doll) was set up in July, 1975 to investigate the subject of Long-term Toxic Effects – it presented a final report in July, 1978.

Focussing on carcinogenisis the group noted that a high proportion of all cancers, perhaps 80 per cent, were thought to be environmentally determined in the sense that, given different environments, the incidence of most types of cancer would

Table 4. Trends in cancer mortality at 45-64 years of age, England and Wales, 1931 to 1975: cancers showing a decrease in mortality of more than 1% p.a. since 1951

Type of cancer	Sex	Annual death rate per million†					change (%) 1951-5 to 1971-5‡
		1931-5	1941-5	1951-5	1961-5	1971-5	
Cancer of							
lip	M	17	7.8	3.1	2.0	1.6	−48
lip	F	0.92	0.64	0.23	0.44	0.11	−52
tongue	M	114	45	18	11	8.6	−52
tongue	F	11	9.6	6.5	5.4	5.1	−21
mouth and tonsil	M	63	26	19	13	9.0	−53
mouth and tonsil	F	7.3	5.0	6.9	6.0	4.0	−42
stomach	M	725	685	617	503	376	−49
stomach	F	426	395	272	202	146	−47
rectum	M	298	270	194	153	152	−22
rectum	F	159	156	126	106	101	−20
liver and g.b.	M	139	102	75	61	46	−39
liver and g.b.	F	138	94	63	47	33	−48
larynx	M	108	71	53	42	36	−32
larynx	F	29	27	21	10	9.6	−54
breast	M	6.0	5.4	6.3	5.4	4.1	−35
thyroid	F	17	17	16	13	12	−25

† Standardized for age.
‡ A regular decrease of 1% p.a. produces a decrease of 18% in 20 years.

Table 5. Trends in cancer mortality at 45-64 years of age, England and Wales, 1931 to 1975: cancers showing an increase in mortality of more than 1% p.a. since 1951

Type of cancer	Sex	Annual death rate per million†					Change (%) 1951-5 to 1971-5‡
		1931-5	1941-5	1951-5	1961-5	1971-5	
Cancer of							
pharynx	F	12	10	13	19	17	+ 48
oesophagus	M	194	106	81	83	105	+ 30
oesophagus	F	66	51	42	44	52	+ 24
pancreas	M	102	109	131	147	164	+ 25
pancreas	F	73	69	75	84	93	+ 24
lung	F	69	106	168	257	397	+136
breast	F	709	651	627	687	770	+ 23
melanoma	M	—	—	9.4	13	20	+113
melanoma	F	—	—	11	15	26	+135
non-Hodgkin's lymphoma	M	—	—	45	55	60	+ 33
non-Hodgkin's lymphoma	F	—	—	25	33	41	+ 64
myelomatosis	M	—	—	21	31	39	+ 86
myelomatosis	F	—	—	16	23	26	+ 63

† Standardized for age.
‡ A regular increase of 1% p.a. produces an increase of 22% in 20 years.

vary. Many specific hazards of cancer had been traced to occupational exposure to chemicals in industry, though *such hazards were not likely to account for more than about 1 per cent of all cancers* now occurring in the UK. A few other cancers could be attributed to pollution by industrial products or industrial waste (e.g. respiratory cancer attributable to asbestos dust).

The mass of cancer related to such factors as sunlight, smoking, chewing various types of quid (tobacco or betel), and sexual behaviour would not be affected by new ways of detecting and legislating on carcinogenic chemicals.

All members agreed that it was important to try to detect mutagens and complete carcinogens in the environment, whether natural or man-made; however, there was disagreement about the extent to which such agents were likely to be industrial in origin. There was also universal assent that the social cost of removing many chemicals from industrial use could be far greater than any potential benefit, partly because such social costs are in the end reflected in reduced resources for health and welfare.

A recent study by Fox and Adelstein of the Office of Population Censuses and Surveys considered the extent to which mortality of an occupation group reflects work environment compared to other factors such as 'way of life'. Their results suggested that overall some 18 per cent of variation in mortality between occupation orders was occupationally related. For some causes the proportion associated with work was lower; surprisingly, only 12 per cent of cancer variation appeared to be associated with work. For other causes such as circulatory and respiratory disease the proportion was nearer 30 per cent. For accidents, which incidentally, kill more than 500 people at work each year, the figure was 23 per cent.

CONCLUSIONS

Much concern has stemmed from the use of misleading terminology. Some analyses encourage confusion by failing to explain fully what is meant by such phrases as 'environmental agents are the major cause of cancer'. Many people may interpret such causes as man-made chemicals in a variety of environments, such as the workplace. Other commentators use the term 'environmental' but make clear that it includes such individual habits as smoking and the use of alcohol. Some political commentators seem aware of this distinction and the controversy which it has caused, but slide over it in their use of the word.

So widely misquoted was Professor Higginson (International Agency for Research on Cancer), who coined the phrase 'environmental cancer' that he felt obliged recently to spell out this original meaning: that he meant it to include food, tobacco and life-style and not just occupational environment.

There is no factual evidence of a cancer epidemic. Apparent increases in the overall cancer mortality rate may be attributed to a number of factors, including the disproportionate increase in deaths due to lung cancer attributable to smoking and the ageing of the population due to improvements in life expectancy resulting from the conquest of infectious disease and the interaction of birth and death rates earlier this century.

Focussing on the nature of cancer mortality today a number of observations may be made. While epidemiologists differ somewhat in their estimates of the proportion of cancer deaths attributable to various causes, the majority of analyses suggest that, exclusive of superficial skin cancer, some 30 to 35 per cent of cancer deaths are directly attributable to cigarette smoking (in addition to affecting the lungs, tobacco exerts a carcinogenic effect on other sites, including the bladder); another 30 to 35 per cent of cancer deaths may be related in some yet unexplained ways to factors in total diet. Perhaps, it is now suggested, diets high in fat and total calories may increase the risk of developing malignant tumours of the breast, prostate, colon,

uterus, and possibly other sites. An additional one to two per cent of total cancer mortality has been traced to excessive alcohol consumption (generally in conjunction with tobacco use) and exposure to radiation and cancer-inducing viruses.

The scientific consensus is that exposure to occupational chemicals may be the underlying cause of some one to five per cent of cancer deaths occurring today. However, these deaths are the result of exposure in the workplace 20 or more years ago, when safety measures in chemical plants were not standard procedure as they are today, so that even the one to five per cent figure may overstate the problem existing today. Of all cancer risks we as a society are facing at this time, the occupational risks are likely to be no higher than one per cent of the total, and probably closer to zero.

Pollution and People

Asbestos—can it be used safely?

DAPHNE GLOAG

Asbestos has been mined in small amounts on and off since the Stone Age,[1] having been used for diverse purposes such as making pottery, wicks, and shrouds. Industrially, however, it has been used for only a century. Its production has increased exponentially during this time,[2 3] and it is said to have some 3000 uses.[2] For some purposes, in fact, substitutes are available; and for insulation asbestos has largely been replaced in the last decade. But for many uses there is no equally satisfactory and economic alternative, and for friction materials it is held to be irreplaceable at present. Moreover, millions of tons are already present in our buildings and elsewhere. Thus a ban on all types of asbestos (see box) does not appear to be a present possibility, though some argue that it is.[4] And would such a ban be desirable —are there health hazards at low levels of exposure that outweigh the valuable and sometimes life-saving properties of the material?

As with so many pollutants, several scientific uncertainties remain. There is plenty of evidence that incidental as well as occupational exposure to asbestos may be hazardous; but is there a threshold for cancer, as there appears to be for asbestosis,[3] below which no harm can be expected? Without knowing more about dose-response relationships and the mechanisms of damage it is difficult to be sure how far the risk at low levels can be extrapolated, for the different fibre types, from occupational data. Nevertheless, the persistence of asbestos fibres in the tissues makes it at least theoretically plausible that cancer could on occasion be induced by extremely small quantities.

Though we have considerable data from epidemiological and case studies some provisos have to be made. Firstly, mesotheliomas in particular may have an extremely long latent period —commonly 20-50 years—so that some may be missed in follow-up studies. Secondly, it may be difficult to obtain a full history of exposure to asbestos, at work and elsewhere, covering a person's entire life[5 6]—and no less difficult to exclude such exposure. Some jobs also turn out to have given exposure to asbestos that comes to light only after considerable investigation.[7] Thus associations with the fibre may sometimes be missed, or a tumour may be wrongly attributed to some more casual exposure to asbestos.[1] Thirdly, the control groups used are not always appropriate[8]; and, fourthly, the dust concentrations to which patients have been exposed are often difficult or impossible to estimate. A further problem (see below) is that the types of asbestos associated with particular cases are not always obvious.

Types of cancer associated with asbestos

The main tumour associated with asbestos is lung cancer, though clearly it cannot be attributed to it with confidence in a given case if the patient has smoked and asbestos exposure has been low. The first epidemiological study, of a factory in Rochdale, showing the link beyond doubt was reported in 1955.[9] Mesotheliomas are less common but the relationship is stronger. They were previously thought an extreme rarity (though recognised in the nineteenth century) and were first found in considerable numbers, and associated with crocidolite, in the mining area of Cape Province, South Africa.[10] Apart from lung cancer and pleural and the rather less common peritoneal mesothelioma—found in workers heavily exposed to crocidolite—increases in gastrointestinal cancers and to a lesser extent cancer of the larynx have been reported in some studies.[1-3] Some gastrointestinal cancers, however, may be misdiagnosed peritoneal mesotheliomas. The importance of distinguishing between different types of asbestos, because of their different degrees of hazard, was not realised until the mid-1950s.

People had assumed that the asbestos regulations of 1931, which resulted from the studies of Dr E R A Merewether, had solved the asbestos problem, though cases would continue to occur for a time in workers who had experienced the old conditions. This was optimistic for several reasons. One was the increase in smoking: though lung cancer does occur in some non-smokers exposed to asbestos, smoking may multiply the risk from asbestos exposure many times[11-13] and thus would make the disease much more prominent than before in asbestos

Asbestos and its uses

Asbestos is the name given to several different silicates with a fibrous, crystalline structure,[1 2] widely distributed in the earth's crust and naturally present in dust in small amounts. There are two main types—amphibole and serpentine. The amphiboles used in industry are *crocidolite* ("blue asbestos"), no longer imported into Britain as it is the most dangerous; *amosite* ("brown asbestos"), whose use is now increasing most rapidly; and *anthophyllite* (now hardly used). The serpentine *chrysotile* ("white asbestos") is much the most common type in nature; its fibres have a curly structure making them less apt to penetrate airways and tissues, though they can split into fine fibrils. Because it confers heat resistance (and acid resistance in the case of crocidolite), insulation, and reinforcement and yet is a flexible material, asbestos is extensively used[2]—for example, for fire protection and heat-resistant materials, in cement for building and pipes, for electrical and other insulation, and for friction materials. The risks of asbestosis and cancer come from inhaling free asbestos fibres, and the raw and unbound material (for example, insulation products and sprayed asbestos) is therefore more hazardous than asbestos bound in, say, cement and plastic floor tiles.

British Medical Journal, London WC1H 9JR
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workers. Secondly, less dusty conditions made workers less likely to succumb to asbestosis or tuberculosis and more likely to survive long enough to develop cancers, especially mesotheliomas with their very long latent periods. Moreover, smaller exposures seem to be capable of inducing lung cancer and mesothelioma than asbestosis.[1] Thirdly, many more jobs entailed exposure to asbestos than were covered by the regulations; in some cases indeed workers were being only indirectly exposed, through working near dust-producing operations. Lastly, workers contaminated their relatives; and people living in the neighbourhood of mines and factories could also be at risk. The various sources of evidence have been summarised.[2]

"Environmental" cancers?

The 1960 report from South Africa was the first evidence of the environmental hazard of crocidolite.[10] Some of the victims had merely lived near the mines or a mill, or worked at a clerical job there.[10] But exposure to the dust may have been considerable: children, for instance, played on asbestos dumps. A London study showed that an excessive proportion of patients had lived within half a mile (four-fifths of a kilometre) of a factory processing amphibole asbestos.[5] Some patients had been exposed to the dust through relatives who worked with asbestos and often appeared to have been heavily contaminated—for instance, by washing their work clothes; one said that her husband (a docker) had returned from work "white with asbestos" and that she had brushed him down every evening.[5] Studies including a category of "possible exposure" have included patients who had briefly engaged in, say, "do-it-yourself" jobs handling asbestos.[7] But with these, and with possible "neighbourhood cases," how likely is a causal link?

Though mainly an asbestos cancer, mesothelioma is thought to be "spontaneous" in a proportion of cases.[6] In a particularly thorough study the annual rate in Canada in 1972 was estimated as 2·8 per million in men and 0·7 in women—the latter presumably nearer the "natural" background rate.[14] Interviews to ascertain occupational history and possible exposure to asbestos were conducted "blind," with the interviewers ignorant of which were cases and which controls to avoid bias in the questioning. Occupational exposure to asbestos was found in nearly half the cases in Canada and about two-thirds in the United States; the overall proportion in women, however, was only 5%. This, of course, begs the question of whether any given case with no occupational history might have been caused by some undiscovered "casual" contact with asbestos. A lung fibre study found 87 out of 100 cases of mesothelioma to have high fibre counts.[6] Brief exposure might be harmful, it has been suggested, if sufficiently intense[6]; it is sometimes claimed that intermittent exposure may be more dangerous than continuous, given the same cumulative doses, because it could be the peak dust levels that are responsible.[1 8] There is no information, however, about actual dust concentrations to confirm such a view. In reported cases where mesotheliomas have been associated with only a day working with asbestos (see IARC,[2] table 22) there are other possibilities, notably "spontaneous" tumours and more prolonged occupation that has been missed. On the other hand, the fibre study just mentioned found extremely high fibre counts in the lungs of some who had been exposed in their jobs for only three months.[6] Rather than speculate, however, we may usefully look at information on the relative risks of the different types of asbestos and such dose-response data as may be gleaned from occupational studies.

Fibre types and dose-response relationships

There is a generally accepted gradient of risk, for both lung cancer and mesothelioma, for the three main types of asbestos, from chrysotile through amosite to crocidolite[1 15]; this is reflected in the current industrial limits.[16] But use of a single fibre type is

Sampling asbestos in the atmosphere, Piccadilly, Manchester. Photograph by courtesy of the Asbestos Information Centre.

rare in industry,[17] and different jobs give different risks (insulation work, using dry asbestos, having been particularly hazardous[15]); so the matter is complex.

Most uncertainty has surrounded chrysotile as few factory populations have been exposed to this fibre in isolation. The largest series of workers exposed to chrysotile alone consists of over 11 000 chrysotile miners and millers in Quebec Province; this showed an increase in total mortality and particularly in deaths from lung cancer and asbestosis—but only in those exposed to unacceptable dust concentrations by present standards.[13] Only 11 pleural mesotheliomas occurred in the 50 years. A paper by the same workers[15] reviewing the data concludes that for those engaged in chrysotile production the risk of mesothelioma is perhaps three to six times greater than in the general population, compared with a risk 100-200 times greater in insulation workers, exposed to chrysotile but also crocidolite or amosite.[15] Whether this means that chrysotile has more effect in these conditions or that exposure to the amphiboles was responsible or that these workers were exposed to a cocarcinogen is not clear.[15]

An American investigation was based on one of the few factories that processes almost exclusively chrysotile, which had for long had relatively low dust levels and which possessed good data on the exposure of its textile workers.[18] This showed for lung cancer a standardised mortality ratio of 223 for the lowest cumulative doses. Only one of the 191 deaths was due to mesothelioma. In a cohort of workers using mainly chrysotile there were 10 mesotheliomas, an incidence of 0·05%; but a statistical analysis suggested that for a man employed continuously the life-long risk could have been up to 10%.[19] Some crocidolite is said to have been used in this factory, however, during two appreciable periods. At a factory manufacturing friction materials, where there were also 10 deaths from pleural mesothelioma, all but two of the cases had definitely been exposed to crocidolite even though chrysotile was mainly used, the association with crocidolite having a probability of 0·06.[20] A significant excess of cancer of the lung and pleura occurred in those employed before 1942, when conditions were dustier. The consensus of the various findings is that crocidolite is considerably more carcinogenic than chrysotile. Exposure to amosite alone is rare but the experience of American insulation workers suggests that it is more hazardous than chrysotile[21]; these workers could, however, have been exposed to some crocidolite.

Some reports present enough data to suggest that the risk of cancers is proportional to the accumulated dose of fibre, or at least are not inconsistent with a linear dose-response relationship.[1 3] For lung cancer in chrysotile miners and millers the

Canadian results show a clearly linear relationship[13]; and in the American study risk was also in proportion to exposure.[18] Mesotheliomas have been too few for inferences about dose-response curves.

For crocidolite and for mixtures of fibres the risks of both lung cancer and mesothelioma appear to be roughly proportionate to the likely dose of fibre.[2 22 23] In the amosite workers mentioned above there were increased risks of lung cancer, all cancers, and all asbestos diseases that were related to duration and intensity of exposure, men employed for only a month showing some excess risk.[21]

The EEC report concludes that there is "suggestive evidence" from the epidemiological studies for a threshold limit for asbestos exposure below which excess cancer risk is small or non-existent, but that no adequate data to establish such a limit are available.[1] The report adds that for mesothelioma induction the relevant exposure may be impossible to estimate since the peak dust levels are probably important here. However that may be, for chrysotile at low levels the epidemiological data are reassuring: even if, as with many other carcinogens, there is no threshold the diminishing probability of cancer induction must result in exceedingly small risks. In the next article, after looking at some fibre studies, I will discuss the environmental aspects of exposure to asbestos.

I am grateful for helpful discussion and comments to Dr P C Elmes, Dr J C Wagner, and other members of the MRC Pneumoconiosis Unit, Llandough Hospital, Penarth; Dr J C Gilson; Dr Muriel L Newhouse, TUC Centenary Institute of Occupational Health, London School of Hygiene and Tropical Medicine; Mr Julian Peto, ICRF Cancer Epidemiology and Clinical Trials Unit, University of Oxford; and Mr W Penney, Asbestos Information Centre, London; and to Mrs Nancy Tait, Society for the Prevention of Asbestosis and Industrial Diseases, for helpful discussion and information.

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What is the best form of treatment for a varicose ulcer?

The principle of treating a varicose ulcer is simplicity itself; it is to reverse the high venous pressure in the affected leg. If the patient is put to bed with the leg raised rapid healing will take place, but this is usually not practicable. Ambulant treatment therefore comprises firm continuous elastic compression of the leg, but this can be combined with keeping the leg raised at night in bed or when sitting down. Standing is forbidden, but walking about while under supportive treatment is encouraged, since this favours venous return. The ulcer itself requires no more than gentle cleaning with saline and protection with a simple sterile gauze dressing—no local antibiotics, no steroids, no ointments, no creams, and no lotions. There is no evidence that these help in any way and in too many cases merely produce skin sensitivity. The simplest technique is to use Elastoplast bandaging, but because sensitivity will occur to this in a very high proportion of patients the skin of the leg must be protected from direct contact with the Elastoplast. The ulcer is therefore covered with a simple sterile dressing, the leg then bandaged with viscopaste from the base of the toes to below the knee (zinc paste and coaltar bandage (Coltapaste) may be used when the skin is dry and scaly or zinc paste and ichthamanol bandage (Ichthopaste) when the skin is moist and eczematous). Over this protective layer the Elastoplast bandage is applied from the metatarsophalangeal joints to below the knee, from below upwards, carefully avoiding kinks and constrictions. Normally the bandage may be left in place for two to three weeks, although the maximum is about six weeks. If there is a heavy discharge the bandaging may initially require weekly change. Associated varicose veins may be treated in most cases by injection technique[1] or in some cases, with gross varices extending to the groin, flush ligation of the vein at the saphenofemoral junction at the groin may be necessary. Once the ulcer has healed, it is essential that the patient goes on supporting the leg by a full length elastic stocking worn during the day and is encouraged to continue active exercises and keep the leg raised when resting.

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Are women with Kartagener's syndrome or any of the other immotile cilia syndromes infertile?

There has been at least one pregnancy recorded in a woman with immotile cilia syndrome. In men affected by Kartagener's syndrome and other types of immotile cilia syndrome infertility is the rule, but it appears that women may have children.

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COUNCIL OF EUROPEAN COMMUNITIES:

Proposal of the second directive of the Council concerning protection of workers against risks due to exposure to chemical, physical and biological agents at the working place: ASBESTOS (French)

(Proposition de deuxième directive du Conseil concernant la protection des travailleurs contre les risques dus à l'exposition à des agents chimiques, physiques et biologiques sur le lieu du travail: AMIANTE)

(Presented by the Committee to the Council on 26 September 1980)

(Présentée par la Commission au Conseil le 26 septembre 1980)

Journal officiel des Communautés européennes, 1980 (October 9): No C262/7.

ABSTRACT

"Asbestos" regulated by this proposal is chrysotile, amosite, crocidolite, anthophyllite, tremolite and actinolite and all mixtures containing the above. Asbestos should be replaced by less dangerous substitutes if there are such. Spraying asbestos is prohibited. Crocidolite should be avoided wherever possible. Where not replaceable, the state authority should annually authorise its use. Use of other kinds of asbestos must be notified to authorities. The concentrations in the air not to be exeeded: crocidolite 0,2 fpcc, other kinds of asbestos 1 fpcc as average per 8 hours. Rules for medical surveillance, protective devices for the workers, for keeping files of cases of asbestosis and mesothelioma, etc., follow.

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AA-11