The effect of changes in the environment on the health of the community—an epidemiologist's view

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THE effect of changes in the environment on health is such an immense subject—even from the limited point of view of an epidemiologist—that it cannot be tackled comprehensively in a single paper. The environment in which we live is so complex, is changing so rapidly, and interacts with our genetic constitution in so many ways that I must be selective. Many changes have been listed as likely to affect our health; such as the increase in the lead content of the air from motor exhausts, in the number of particles of asbestos from its thousand uses in factory and home, in pesticides and fertilisers, in sewage from factory farming, in food additives like cyclamates and saccharin, and in noise from aeroplanes and motor traffic; or the reduction in smoke from the combustion of coal in Southern England; or the substitution of machines for manual work.

I suspect, however, this approach may result in missing the most important changes; and I prefer to start from the other end, by considering the distribution of disease and seeing whether it can be accounted for by environmental factors which, if not already changing, can be influenced to do so in the future.

Patterns of mortality

The pattern of mortality can be considered first. Despite the low levels that are now recorded at young ages, mortality remains of primary concern, not only because of its overriding importance to the individual, but also because it continues to be a valuable indicator of morbidity.

It has, of course, the limitation that it fails to reflect morbidity from arthritis and mental disease and is of minor relevance over 65 years of age. I think this is not through lack of concern for old people, but for two reasons. First, because it is more important to deal with the accumulated burden of disability in old age than to prolong the expectation of life by six months, and secondly because the disabilities of old age are to a large extent the result of a life-time's exposure to those factors that have caused mortality at younger ages in the past.

Figure 1 shows the extent to which different diseases contribute to the causes of death at different ages in England and Wales. Only diseases (or groups of diseases) that contribute ten per cent or more of all deaths at one or other age are included.

In infancy nearly half the deaths are attributed to specific conditions associated with pregnancy and the physiological state of the infant, while another fifth are attributed to congenital abnormalities. In the 21 years that followed the introduction of the National Health Service, the infant mortality rate fell by 48 per cent. This is quite impressive until we find that it fell by 75 per cent in Japan, where it is now lower than in England and Wales. In fact, the rate of improvement has been slower in Britain than in several other countries and the British rate deteriorated in position from ninth of the 17 countries whose figures I have examined to twelfth (Doll, 1974).
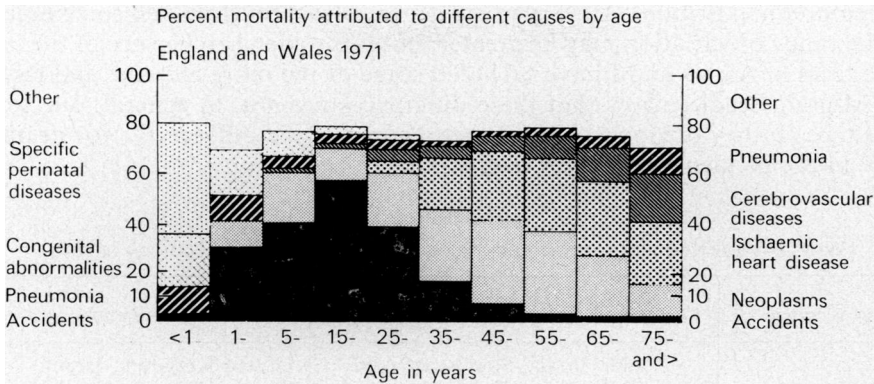
Figure 1
Proportion of deaths due to different causes by age in England and Wales, 1971

There has been, of course, an enormous improvement in neonatal care, and Herculean efforts are now made to save the life of premature infants which would have been written off as stillborn or even, in extreme cases, as abortions, 25 years ago; but it would be unwise to assume that the decline of the British position was due to a disproportionate change in the denominator alone.

In 1970 the Swedish infant mortality and perinatal rates were, respectively, 36 and 31 per cent below those in England and Wales and we need to enquire how far these differences can be attributed to differences in classification, medical care, and the environment—particularly, perhaps, in the socio-economic conditions of the least favoured sections of the community. It is difficult, however, to believe that these were the sole factors determining the differences as there were only two administrative districts in the whole of England and Wales which provided figures as low as those for the whole of Sweden—and these were Ipswich and the rural districts of Cambridgeshire.

Figure 1 shows that primary infections appear as a major cause of death only at the extremes of life and then only under the guise of pneumonia. Neoplasms appear in all age groups after one year of age; ischaemic heart disease appears in all groups after 35 years of age, and accidents and other forms of violence in all groups between one and 44 years of age. It is clear, therefore, that we shall have to make some inroad into these last three disease groups if we are to make any further impression on mortality before old age.

Cancer

Consider first whether it is possible to control cancer. Table 1 lists the ten cancers that

TABLE 1
WORLD-WIDE VARIATION IN INCIDENCE OF CANCERS COMMON IN BRITAIN

Primary cancer	Variation	High incidence	Low incidence
Bronchus	× 40	Scotland	Africa*
Breast	× 6	USA (White)	Africa*
Skin	× 100	Queensland	Indians anywhere
Stomach	× 25	Russia, Japan	Africa*
Colon	× 15	N. America	Africa*
Rectum	× 8	Denmark	Africa*
Prostate	× 10	USA (Black)	India, Japan
Bladder	× 5	USA (White)	India, Japan
Cervix	× 20	Colombia	Israel (Jewish)
Pancreas	× 4	Israel (Jewish)	Israel (non-Jewish)

* Africa South of the Sahara: Uganda, Mozambique, part of South Africa

are most common in Britain. All vary in incidence at least fourfold and some much more. In fact, the range of variation may be greater, but it is difficult to be sure of the incidence of rare cancers in Africa and I have excluded some of the more extreme and less certain figures. Migrant studies show that these differences are not, in general, due to genetic factors, although they obviously contribute to some of the differences; for example, the difference in the incidence of cancer of the skin.

TABLE 2

WORLD-WIDE VARIATION IN INCIDENCE OF SOME CANCERS UNCOMMON IN BRITAIN

Primary cancer	Variation	High incidence	Low incidence
Mouth	× 30	India, South-east Asia	United Kingdom, Israel
Nasopharynx	× 100	South China, Hong Kong	Western Europe, North America
Oesophagus	× 400	Parts of Kazakhstan, Iran, South Africa	Netherlands, Nigeria
Liver	× 1000	Mozambique, Senegal	Western Europe, North America
Kaposi's sarcoma	× 1000	Central Africa	Western Europe
Burkitt's lymphoma	× 200	Parts of East Africa, New Guinea	Europe, North America

Other cancers, at present rare in Britain, vary even more (table 2) and there is, in fact, no cancer that is common anywhere that is not relatively rare somewhere else. It is more difficult to be sure of temporal changes; but it is probably also true that all cancers vary in incidence with time except, perhaps, for a few like acute leukaemia in young adults, that are uncommon everywhere and may be largely due to background factors like cosmic rays and naturally occurring radioactive isotopes.

TABLE 3

TRENDS IN MORTALITY FROM CANCER ENGLAND AND WALES, 1961–1971

Site	Change*	
	Men	Women
Hypopharynx	+	
Pancreas	+	+
Nose and sinuses	+ +	
Bronchus	+	+ +
Melanoma	+	+ +
Testis	+	
Bladder	+	
Kidney	+	
Lympho- and reticulosarcoma	+	
Myelomatosis	+ +	+ +

+ increase in death rate 20–39 per cent
+ + increase in death rate 40 per cent or more
* no cancers showed comparable decreases in death rate

Table 3 lists the cancers whose mortality rates are recorded as having changed materially during the last ten years. Cancers are included only if more than 100 deaths were attributed to them in one or other sex in 1971 and only if the death rate had increased by more than 20 per cent. Mortality rates are, of course, an imperfect measure of incidence and I cite them only to indicate problems that deserve to be investigated.

Melanoma

Increases in the death rate from melanoma, for example, have been reported from

Australia, Canada, and the United States, as well as from Britain. The increase has been most marked in middle age and has occurred despite improvements in treatment. It is confirmed in the incidence data of the Connecticut and New York State Cancer Registries and cannot be accounted for by transfer of deaths from other cancers of the skin. Cohort analysis (Lee and Carter, 1970) suggests that the increase began to be experienced by the generation born in 1906 and continued for the next 20 years. Is it, I wonder, affected in any way by the increased tendency to expose the skin to sunlight?

Cancer of the testis

The increase in mortality attributed to cancer of the testis is particularly surprising in view of the improvements in treatment and the ease with which the disease is diagnosed. An increase in incidence, however, has already been observed in the records of the Danish Cancer Registry. The incidence at ages 25 to 44 years more than doubled in Copenhagen between 1943 and 1962, increased slightly in the provincial towns, and remained constant in the rural areas (Clemmesen, 1968). The English and American mortality figures resemble those in Denmark at the start of the epidemic, which (it now seems) has begun to spread to Britain.

Lung cancer

Detailed examination of the mortality from lung cancer, which now accounts for nine per cent of all deaths in men, shows that the rate continues to increase only above 70 years of age and has been decreasing under 60 years of age for the last ten years (table 4). Indeed it would not be surprising if 1974 was the first year for half a century to show a decrease in the total number of lung cancer deaths. The decrease at young ages confirms the relative unimportance of motor exhausts, and the contrast with the experience in women rules out the possibility that a general factor, like atmospheric

TABLE 4

CHANGE IN DEATH RATE FROM LUNG CANCER ENGLAND AND WALES, 1960–1971 (BY AGE AND SEX)

Age (in years)	Per cent change 1960–1 to 1970–1	
	Men	Women
30–34	−24	−15
35–39	−34	−11
40–44	−12	+11
45–49	−8	+38
50–54	−10	+70
55–59	−8	+66
60–64	+21	+62
65–69	+21	+67
70–74	+53	+62
75–79	+65	+62
80–84	+83	+52

pollution with coal smoke, could have been responsible for the epidemic, except as far as the reduction in smoke may have contributed to the reduction in mortality at young ages in both sexes.

The trends in mortality correspond broadly with the trends in consumption of cigarette tobacco in men and in women, modified by the different habits of different cohorts (Armstrong and Doll, 1974). Only now, for example, are men over 70 years of age coming to be completely composed of cohorts who learnt their smoking habits during

the first world war. The reduction in mortality at young ages is, however, so large that it encourages the hope that the change to filter tipped cigarettes and the reduction in the tar content of the smoke is having an effect, irrespective of a change in size and number of cigarettes consumed.

Such changes should not, of course, affect the incidence of lung cancer alone. Cancers of the mouth, pharynx, larynx, oesophagus, and bladder are also related to smoking, though the position is complicated by differences between the relative importance of cigarettes, cigars, and pipes. It is further complicated by the fact that cancers of the mouth, pharynx, larynx, and oesophagus are also related to the consumption of alcohol, while cancer of the bladder may be related to the consumption of coffee.

Cancer of the oesophagus

No disease presents a more fascinating problem for the epidemiologist than cancer of the oesophagus. The incidence varies 400-fold from parts of Iran, China, and South Africa to Nigeria and the Netherlands. It has appeared as a common disease in South Africa only in the last 40 years, at the same time as it began to disappear from large parts of Europe and North America. More recently, however, the tide has turned again. It is becoming more common among the black population of the United States and has begun to cause an increased number of deaths at young ages in Britain—possibly because of an increased consumption of alcohol.

Cancer of the bowel

A remote but exciting possibility is that we may be able to reduce the incidence of cancer of the large bowel by modifying our diet. Burkitt (1969, 1971) pointed out that cancer was only one of a group of diseases, including polyps, appendicitis, diverticular disease, and ulcerative colitis, which affected the large bowel, were common in developed countries, and were rare in rural Africa; and he suggested that they might all be due, in one way or another, to the fact that the communities in which these diseases were rare consumed a high residue diet, while those in which they were common consumed a low residue diet plus a large amount of refined carbohydrate.

That the difference in diet has an effect on the colon is demonstrated by the difference in stool bulk and transit time, the bulk being greater and the transit time shorter with the high residue diet than with the low.

Another effect has been demonstrated by Williams and his colleagues from examination of the bacterial flora of the bowel (Aries et al., 1969; Hill et al., 1971). Specimens of faeces were collected from healthy adults in the high incidence areas of Connecticut, Scotland, and England, and from the low incidence areas of Uganda, India, and Japan. The same broad groups of bacteria were found in all specimens, save that one type was present only in vegetarians and there were greater numbers of anaerobic bacteroides in the faeces from the high incidence areas. This last group of bacteria have the capacity of degrading steroids by dehydrogenation of the 7α-hydroxyl group, so that the stools in which they were most numerous showed the largest amounts of steroid metabolites, including dihydroxycholnic acids, which may well be precursors of carcinogens, if indeed they are not carcinogenic themselves. Measurement of the amount of these metabolites in the stools showed that pooled values for each group of people correlated closely with the incidence of colon cancer in the community in which they lived. Since this report was published, Professor Williams and his colleagues have obtained specimens from three other populations and the new observations fall readily into place on the graph (Williams, personal communication). We must, therefore, now seek to find out whether the same differences distinguish affected and unaffected individuals within a community and whether the faecal flora can be used to predict the risk of developing

the disease later in life. If these results are equally encouraging, we must then enquire whether the flora, the steroid metabolites, and the risk of cancer can be modified by changing the amount of cereal fibre in the diet.

TABLE 5

FACTORS IN THE AETIOLOGY OF ISCHAEMIC HEART DISEASE

Factor	Genetic component	Environmental component
Hyperlipoproteinaemia	+	Diet: (i) excess calories (ii) excess saturated fat (iii) ? deficient fibre
Hypertension	+	+ ? nature
Diabetes	+	Diet: excess calories
Cigarette smoking		Tobacco smoke ? component
Occupation		Exposure to CS_2
Medical intervention		Drugs: (i) steroid contraceptives (ii) premarin Premenopause oophorectomy
Physical activity		Diminished activity
Mental activity	?	?
Soft water		?

Ischaemic heart disease

I have given a disproportionate space to a consideration of cancer, but even so have hardly scratched the surface of the subject. In the space that is left I can do even less for the other principal components of mortality. Table 5 lists the factors that have so far been shown to affect the incidence of ischaemic heart disease. I have omitted coffee as the evidence is incomplete—although certainly suggestive. Others will doubtless be discovered in the next few years. The outstanding defect of our knowledge is a precise understanding of the way in which saturated fats and total calories interact to produce the differences in risk experienced by the Yemenite and American immigrants to Israel and by the Kalahari bushmen, the Cape Town whites and the Natal Indians. Without this understanding, it is extremely difficult to mount an effective programme of public education.

Smoking is not, I think, a major factor except at young ages. Whether the carbon monoxide in the smoke is responsible, as has been suggested by Wald *et al.* (1973) or whether the amount of carboxyhaemoglobin merely indicates the amount of smoke inhaled better than the number of cigarettes smoked per day, remains to be shown. Wald's observation serves, however, to remind us that cigarette smoke is a complex material and it cannot be assumed that the same component is responsible for cancer of the lung, myocardial infarction, and chronic bronchitis. A reduction in one component of the smoke could conceivably reduce the incidence of one disease while indirectly increasing the incidence of another.

Accidents, poisonings and violence

Figure 1 showed that accidents, poisonings, and violence were the principal causes of death between five and 34 years of age, and indeed accounted for more than half of all deaths during late adolescence and early manhood. At these ages only Malta had a lower death rate from all causes of all the 45 countries whose rates are recorded by the World Health Organisation.

It is, therefore, not surprising to discover that the mortality from accidents, poisoning, and violence is much lower in Britain than in any other developed countries—being, for example, 33 % less than in Sweden, 37 % less than in Australia, 41 % less than in the United States, and 50 % less than in France.
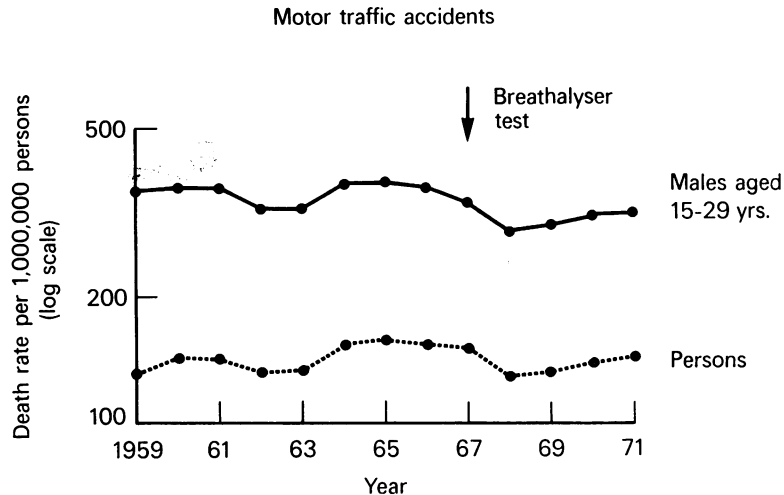
Motor traffic accidents

Figure 2

Death rate from motor traffic accidents in England and Wales, 1959 to 1971; all ages and men aged
15 to 29 years.

Figure 2 shows the mortality from motor traffic accidents during the last 12 years. The relative constancy, despite the great increase in traffic, is a tribute to the efforts that have been made to prevent accidents and to the efficiency of the accident service. A 20 per cent reduction occurred in the year after the introduction of the breathalyser and the setting of an upper limit to the permissible amount of alcohol in a driver's blood; but the rate has been increasing again slowly since. The reduction in 1962 was due almost entirely to a reduction in motorcycle accidents as riders turned for preference to cars.

Figure 3 shows the trend in mortality from suicide. Despite the epidemic of admissions to hospital from self-poisoning among young people, even the mortality at ages 15 to 24 years has remained practically constant, while the rate at other ages has fallen. Examination of the rates attributed to different methods shows that suicide other than by poisoning or domestic gas remained unchanged throughout; suicide by poisoning increased in frequency between 1960 and 1963 and then remained steady; while suicide by inhalation of domestic gas has almost disappeared—due first to the manufacture of gas with less than five per cent carbon monoxide and subsequently to the supply of natural gas which contains no carbon monoxide at all. It is surprising that the use of gas has not been replaced by other methods—or is it that a switch to poisoning has been compensated for by improvements in the treatment of poisoning or of depression or, as Bagley (1968) suggests, by the spread of the Samaritans?

The data in figures 2 and 3 provide some justification for believing that some of the worst aspects of our social environment may be being contained in Britain better than in most developed countries and tempt me to conclude, like Dickens in his *Pictures from Italy* 127 years ago, that " Time is rolling for an end, and that the world is, in all great essentials, better, gentler, more forebearing, and more hopeful, as it rolls."

I wonder, however, if that would be wise. So often before in the last 50 years England has followed in the footsteps of the United States and the position in the United States has been rather different. Unlike Britain, the mortality at ages 15 to 44 years rose between
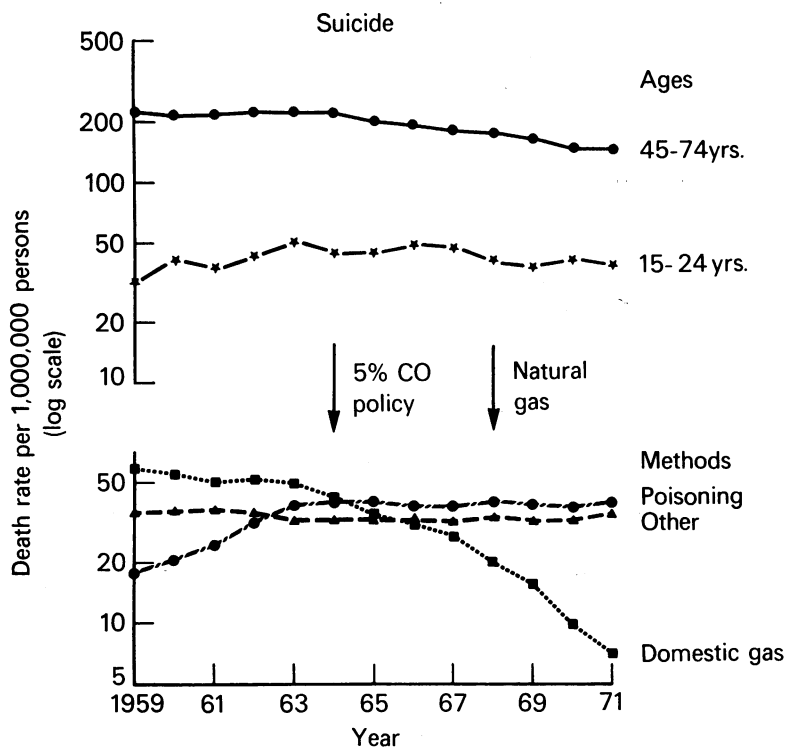
Figure 3

Death rate from suicide in England and Wales 1959 to 1971; all methods, carbon monoxide poisoning, other forms of self poisoning, and all other methods shown separately.

TABLE 6

TRENDS IN MORTALITY IN CHILDREN AND YOUNG ADULTS U.S.A. AND ENGLAND AND WALES 1961–1969

Age (in years)	Death rate in 1969 as per cent of rate in 1961					
	U.S.A.			England and Wales		
	Cirrhosis of liver motor accidents suicide homicide	Other causes	All causes	Cirrhosis of liver motor accidents suicide homicide	Other causes	All causes
5–14	130	88	96	115	82	88
15–24	157	100	127	89	93	92
25–34	138	97	111	90	86	87
35–44	137	100	110	89	98	97

1961 and 1969 (table 6). It is difficult to believe that this could be due to a deterioration in medical services, and it seems more likely to be due to the social conditions which resulted in an increase in deaths from cirrhosis of the liver, motor traffic accidents, suicide, and homicide. At any rate, the mortality from all other causes grouped together remained constant—or fell, as in Britain.

Is there, I wonder, any possibility that the conditions that have given rise to the increase in mortality in the United States will spread to Britain? And if so, how many doctors will be concerned?

We have learnt to control the great majority of infectious diseases and I expect that control will be extended steadily to include influenza, infectious mononucleosis, cytomegalovirus infection, toxocariasis and toxoplasmosis, and such others as remain. Whether we should seek complete control over pneumonia is perhaps something that had better be left to the geriatricians. We have learnt how to avoid the effects of malnutrition and of occupational exposure to industrial hazards and I have little doubt about our ability to prevent the effects of industrial pollution from involving the general population.

We need, I suspect, something equivalent to the Committee on Safety of Medicines to advise on the introduction of new materials into industry, an occupational health service, and a system of record linkage to enable us to detect new hazards quickly; and something equivalent to the American Environmental Pollution Agency to monitor the environment and alert us to change. Given these I see no reason to be anxious about the direct effect on health of the further growth of industrialisation.

The challenge for the future

What we have not learnt to control is the social environment we make for ourselves by living close together in large towns, with a high standard of living, ample leisure, and no need to wrest our daily food from natural resources. Under these conditions we still have to learn how to control our appetite for concentrated food, reduced in bulk by industrial processing, and our need for mental stimulus whether by tobacco, marijuana, alcohol, coffee, or aggressive driving.

Factors such as these are indeed hard to modify—not just because of commercial interests, but also because they require the individual to control himself. In my opinion, however, doctors can no more avoid tackling them now than we could avoid the problem of polluted water in the nineteenth century.

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