rich organ. It is the outstanding characteristic of the human animal, yet the lipid nutrients involved in its construction have been overlooked in contemporary food policies.

If my suggestions are correct, then there is a fundamental error in our food structure which sooner or later must produce some long-term consequences for the population. The specialisation of our structural fats distinguished the human from other animals. The continued deterioration in the materials used is likely to lead to degeneration. The most marvellous genetic plan is like an architect's blueprint and it will be worthless without the correct building materials.

Acknowledgement

This lecture was based on the book *What we eat today* by M. A. Crawford and S. M. Crawford, with a foreword by Sir Frank Fraser-Darling published by Neville Spearman, London.

DEATH IN THE AIR WE BREATHE

Dr L. M. Swinburne
Consultant pathologist, St. James Hospital, Leeds

It is a terrible indictment of human attitudes that when air pollution was first noted in the Los Angeles area it was not because of its effects on human life and health but because of its effect on crops. Only when the lettuces became spotty, the oats were wilting, and there was a nasty bronze shine on the sugar-beet leaves did people stop and think. In other words, it was only when Popeye's spinach began to curl at the edges that people began to wonder what was happening to Popeye himself and also to take note of the smarting eyes, running noses and the shortage of breath that ordinary people were experiencing. Other people had also commented on the effects of atmospheric pollution on rubber, no doubt worrying about their precious motor-car tyres, but quite heedless of the fact that what rots rubber must certainly rot lungs.

Nor can we flatter ourselves that things have been any different in Britain. The gardens and parks in our industrial towns are largely furnished with privet, spotty laurels, rhododendrons and the so-called London Pride. These plants have very thick wax-coated leaves which can stand up to soot and regular rinsing in sulphuric acid, which are the chief pollutants in our atmosphere. Similarly the rose is particularly suitable as a national emblem and also as the emblem of our two industrialised northern counties, Lancashire and Yorkshire. Roses bloom very happily in these areas because they are preserved from fungal disease and blackspot by the high sulphur content of the atmosphere.

It is also significant that the cauliflower and cabbage, almost our national vegetables, are highly resistant to some forms of pollution. Long ago we should have considered whether we were soot-tolerant like the rhododendron or more like the cupressus species which thrive in clean country air. I am not being facetious in quoting these parallels because we need to emphasise that there are two main types of growth-air pollution which differ markedly in effects on plants, animals and man.

Major pollutants

The extreme forms of atmospheric conditions combined with wind, temperature, combustion, decay of organic materials and industrial processes can produce what is

politely known as a major pollution disaster, our old enemy, smog. In Los Angeles the atmosphere contains excessive oxidant materials whilst the London type is equally irritant but has reducing properties rather than oxidant properties.

These episodes are rare, but the atmosphere contains the same pollutants in varying proportions but far below the level of the acute episodes. The primary pollutants of Los Angeles are the direct products of the combustion of coal, oil, petrol and other materials which are retained in the atmosphere as a fine suspension; under the influence of sunlight a photochemical oxidation takes place to yield secondary pollutants, in the form of gases, such as ozone, peroxy-acyl nitrite and other materials.

Contrary to popular belief, ozone is not beneficial as it can cause conjunctivitis, tightness of breath, an irritative cough and low-grade headache. The small amount in sea air is harmless, but in a high concentration, both in man and experimental animals, it is highly toxic and unpleasant. The acute pollution episode or smog has a temporary effect in reducing lung function by lowering the diffusion capacity of the lungs as a result of congestion and oedema. These episodes do not produce structural changes in man, which is why they have been so difficult to detect. There have been varying effects in experimental animals; in the rat there is little effect and no structural change, but in certain strains of mice acute exposure causes premature ageing, pulmonary oedema, pneumonitis, and septal fibrosis. In the long term the mice also develop squamous metaplasia, hyperplasia of the bronchial epithelium and pulmonary adenomata.

We must consider the effects of these pollutants on the human and whether they are chronic or acute. An interesting study in the United States of America compared the athletic performance of boys from two schools in different areas. It was found that the boys' performance in a given race correlated best with the ozone content of the atmosphere in the hour preceding the race; whether this was due to some toxic effect or a decrease in the maximal effort has yet to be decided.

The ozone type of pollution requires sunlight for its production, whereas in Britain the pollution is mainly due to reducing substances. Our problem is the accumulation of irritant gases such as sulphur dioxide, aerosol suspensions of tars, resins, and particulate matter like carbon and asbestos fibres. Sources of industrial waste produce hydrogen sulphide and nitrites which can lead to the formation of formaldehyde, ammonia and other irritant gases. The acute episodes of the London smog type cause eye and bronchial irritation, bronchospasm, shortness of breath and an increased susceptibility to infections which often lead to the death of children or elderly people.

The salient point that emerges from the various reports of these episodes is that people who already had some degree of respiratory damage or difficulty were more likely to be affected or to die during these episodes. Major episodes, with a high morbidity rate, are rare but we are still being subjected to a steady but less intense pollution of the atmosphere by the same materials. There is good reason to believe that, combined with other factors, it adds to the toll in the form of chronic bronchitis, emphysema and the inevitable cardiac sequelae. A certain professor of philology has attributed the Birmingham accent to the effects of air pollution, because of the tendency of the local population to talk down permanently closed nostrils.

The sources of these pollutants are complex as instanced by one episode in France where at least 30 different items were enumerated as being responsible for pollution. In general, 95 per cent of pollutants are due to the products of combustion of coal and oil. One analysis showed that domestic heating produced 21 per cent of the pollution but this could increase in cold weather. Power plants supplying indirect heating and industrial power were responsible for 39 per cent and direct industrial usage led to a further 22 per cent.

The motor car as a source of pollution

That survey did not include the automobile as a source of combustion. In Los Angeles and Tokyo the automobile is blamed for some of the worst features of atmospheric pollution by producing an aerosol of partially burnt gases, an excess of carbon dioxide, tetraethyl lead residues, and a fine suspension of rubber and rubber degenerative products due to the friction of the tyres. It has also been suggested that the rubbing of millions of brake linings causes asbestos fibre pollution of the atmosphere, but this has never been substantiated. Traffic policemen in Tokyo have to use an oxygen mask for five minutes in every hour to offset the effects of the inhalation of excess carbon monoxide.

In addition to car pollutants there is temporary local pollution by noxious substances which can be caused either deliberately or accidentally. In one town a whole hedge suddenly turned black and died, while a row of poplar trees nearby withered and shrivelled up. This was due to the dumping of chemical waste from a local factory in what was supposed to be a safe open space, but the wind wafted the waste back into the local gardens and hedgerows. It is easy to see that the causes and effects, of pollution are slow and difficult to detect.

Besides the general atmosphere we must also consider the local atmosphere surrounding each individual and the contribution of domestic and occupational factors to this local environment. The effect of the irritant gases producing exudation in the lung with bronchial irritation is well known, but in the more confined personal environment particulate matter, both organic and inorganic, begins to play an important part. These materials are released into the general atmosphere but their effect is lessened by dilution in most instances. Under factory, workshop, or workbench conditions these particulate substances can produce a far-reaching effect on the individual worker.

While the effects of the inhalation of gases are often ill-defined, subjective or functional, the reactions to particulate matter are long-lasting and structural, since a proportion of the solid matter is usually retained in the lungs. The response mechanism to the inhalation of organic and inorganic dusts depends on particle size, the chemical structure, and physical form. There is a different pulmonary response between the species so that the guinea pig retains dust in the bronchial tree and alveoli whilst the rat, under similar conditions, shows a far greater ability to wash out and clear the dust from its lungs.

The immediate human response to particle inhalation is bronchial irritation and coughing. If the amount is below the irritant level a certain amount is returned to the trachea by ciliary action, but when dust is inhaled more deeply it is then phagocytosed by the macrophages in the periphery of the lung. My own work leads me to believe that the fate of this ingested material is very closely linked to its effect on the ingesting cell.

Grades of particle toxicity

Comparing the behaviour of particulate matter of different types in order of cell toxicity, we find that carbon in the form of soot appears to be almost inert. The ingesting phagocytes are unaffected by the carbon, they migrate normally to the local lymph follicles and lymph nodes, then they travel to the lymphoid tissue at the lung surface. It is common to see deposits of carbon in portal tracts, the liver, the splenic pulp and the peritoneal lymph nodes, which is evidence that carbon in these circumstances is quite harmless. There is no evidence that carbon by itself produces fibrosis of the lung, although bronchitis is a common finding.

In a town atmosphere we do not inhale pure carbon and it is possible that the particles act as a vehicle for the absorption of other pollutants such as benzpyrine which

could explain the higher incidence of carcinoma of the bronchus in town dwellers and soft-coal workers.

Glass fibre is also inert but it can irritate the skin, as anyone who has tried to insulate a roof will know. The particles are readily phagocytosed by the macrophages and carried to the regional lymph nodes. They can also be coated with protein by the macrophages to form bodies resembling asbestos bodies, but there is no tendency to fibrosis of the lung and no evidence of a carcinogenic effect. Chemically, glass fibre is more inert than carbon.

Asbestos

Asbestos, in its various forms, is again phagocytosed after inhalation and a proportion of the largest fibres are coated with protein and haemosiderin to produce the well-known asbestos body. The fibres and the asbestos bodies persist in the macrophages for many years after the original period of exposure to the dusty atmosphere, and since it is unlikely that the macrophage can exist for 20 to 30 years, the bodies must be re-ingested by fresh cells or by a process of mitosis passed on to a daughter cell.

A certain amount of fibre is deposited in intestinal tissues of the lung after the death of the macrophages, and the cells which carry the fibre seem to undergo a transformation into fibroblasts. Fibrosis of the lung is a conspicuous feature of the inhalation of asbestos fibre in quantity. The ingesting macrophages retain their mobility for a time while they travel to the surface of the lung and the local lymph nodes, but there is little evidence that the cells travel any further. This probably means that the asbestos has some toxic effect on the cells and where it is deposited fibrosis can occur. This happens particularly at the pleura, the surface of the lung, and the diaphragm.

The long-term effect is to produce a high incidence of carcinoma of the lung and, in many cases, the special tumour, mesothelioma of the pleura or peritoneum. We have evidence of very long-term cell damage caused by asbestos but we do not know what the actual process is.

Silica

Silica is an entirely different problem because of its toxicity. When phagocytosis takes place, silica leads to rapid death of the cell, giving rise to an interstitial deposition of the material at the site of cell death. There is less transfer of silica to the surface of the lung or to the lymph tissue than of the previous three pollutants, so that fibrosis occurs in a diffuse or focal manner that can affect the upper lobe or any part of the lung. There is no long-term carcinogenic effect, although the fibrosis can be a serious and devastating problem.

The four substances tend to be deposited permanently in the lung so that the only defence is to stop their inhalation. Every effort is being made, at least in industrial conditions, to cut down the sources of these dusts, because reaction to these inorganic materials is usually permanent and progressive.

Organic dusts

Organic dusts are a much more difficult problem because the effects are often subjective, temporary, and non-lethal. The materials inhaled can include virtually everything that can occur in a fine particulate form or as an aerosol. The largest particles are phagocytosed while the smaller particles are carried by the air stream to the periphery of the lung and give rise to very unpleasant symptoms due to the development of local immunological reactions.

Examples of such particles are pollens, fungal spores, metallic dust such as beryllium, bacterial products such as *Bacillus subtilis* which is found in the so-called biologically-active washing powders, actinomycetes, malt dust, cork dust, wood dust of various

kinds, and many other particulate materials. Some of these materials are not in themselves allergens but provide a medium for the growth of the fungi which cause the symptoms. Byssinosis is due to the inhalation of damp cotton fibre and the fungi on it.

Farmer's lung is a further example, in which mouldy hay is the vehicle for several fungi and their by-products. My favourite example of this type of disease is ' cheese-washer's lung ' which occurs in Swiss cheese factories; I can imagine little men with tiny hoses and brushes hosing down the holes in the gruyères and the emmentaler. With these conditions the symptoms occur five to six hours after exposure and they consist of tightness of the chest, a cough and malaise which can persist for several hours. Repeated exposure leads to structural changes in the periphery of the lung due to an excess of antibody-producing cells.

The diagnosis of these conditions is best undertaken by provocative testing—by the inhalation of the suspected material. The inhalation of budgerigar droppings or pigeon feathers will provoke the diagnostic symptoms in cases of ' bird-fancier's lung ', while the use of different types of wood dust will help to differentiate such diverse conditions as ' maple bark stripper's lung ' from the more common wood allergies. Some patients develop an immediate asthmatic response in addition to delayed peripheral response, and all these patients develop serologically-detectable antibodies which can be used for diagnostic purposes. It is fortunate that the removal of the offending material, or taking the worker away from the polluted atmosphere, will usually resolve the symptoms.

So far I have mentioned a number of separate items which affect individuals but many symptoms are due to build-up of a little pinch of asbestos, a whiff of insecticide, a puff of sulphur dioxide, and a mountain of soot.

Personal environments

People who smoke cigarettes were worse off in the major pollution episodes than the non-smokers. Smokers were also worse off when they had a dusty occupation or when exposed to one of the organic dusts. The moral is—do not poison your cilia, they are doing their best. One of the effects of cigarette smoke is to cause temporary paralysis of the cilia, thus diminishing the defence against these other pollutants. Many factors are beyond our control, but we should encourage our patients to control their own personal environment as far as possible.

The slow recognition of the harmful effects of smoking is due to the fact that the human animal is a comparatively poor experimental animal. It is too long-lived and irregular in its habits, as was found in a comparative study of bronchitis in a rural and a town area in Britain and the United States of America. This study was almost ruined because the Americans moved away from the towns when they developed bronchitis and thus spoilt the statistics, whilst the British sufferers obligingly stayed in the places where they were born and bred.

Resolving these environmental problems on an individual basis is not easy. A recent case concerned a colleague who complained of regular Sunday-morning headaches. This was not a well-timed allergy to car shampoo or lawn-mower sweepings, but was due to another Sunday chore, lighting the drawing-room fire and clearing out the kitchen boiler. This condition was due to a sudden rise in the level of carbon monoxide and not a personal silicosis. The down-draft from an old-fashioned flue allowed carbon monoxide saturation of his blood to build up to a dangerous level of about 11 per cent. I do not know whether he repaired the chimney or made his wife do it! The main point is that we have a special responsibility to listen to what our patients say about their symptoms and the circumstances in which they occur, while keeping an open mind on our interpretation. In this way we shall be able to spot diseases such as ' cheesewasher's lung ' or the

pollutant effects of substituting oil for coal under the ' Clean ' Air Act at a much earlier stage than if we have to wait for the agriculturalists to report a new rot in aspidistras, brussel sprouts, or rhubarb.

Dr G. I. Watson—Chairman

Dr Pinsent needs no introduction but his subject may seem unusual unless you know of his particular interest in the soil and water of the South-west peninsula. He has also established a transatlantic link with a colleague in Vancouver where similar investigations are being conducted. Dr Pinsent is going to tell us about the hazards of soil and water.

HAZARDS OF SOIL AND WATER

Dr R. J. F. H. Pinsent
General practitioner, Birmingham

It is impossible to consider all the hazardous interactions which can occur between man and his surroundings. I have completely omitted much that we know of flood, tempest, volcano and earthquake, so I shall deal with two variants of the three common biological ecological cycles. This is the old story of natural forces such as rain dissolving naturally occurring organic and inorganic substances which are carried by water to the varied plant and food chains, until they eventually reach man. When man appeared he began to make hostile surroundings by digging holes to bring combustible minerals to the surface. Dr Swinburne has told us that " what goes up is breathed and what is not breathed comes down ", and so we get surface contamination. We sink oil tankers and we dump tin cans and these are hazards with which you are all quite familiar.

There is a range of 41 major and minor trace substances of which 18 are supposed to have biological functions which are essential to life. The remaining 23 are in the comfortable position of being ' not proven ' because we cannot say that a substance is absolutely inert and free from hazard. The community is exposed to hazards to which it has to adapt and this multiple series of adjustments is the cumulative process which we regard as adaptation. Adjustment to a change in environment can also be a problem. It is well known that when cattle are moved into a neighbouring pasture they may very well suffer brief gastrointestinal upsets. Of the two processes of adjustment and adaptation we can say that people adjust while populations adapt.

How do people adjust and adapt and what mechanisms are involved? Many of the biological processes are conditioned by enzyme sequences, and the trace substances form a normal part of many of the large enzyme molecules. It is known that substitution of one molecule, at a given valency, for another may occur and that this may also alter the pattern of the action of that enzyme. The lipid metabolism which led to the incorrect differential distribution of fats in muscle may be due to an altered enzyme sequence as a result of a molecular replacement. Definite relationships have been established between the intake of chromium, glucose tolerance, and diabetes. Cadmium has been shown to play a part in hypertension, and the relationship between hypertension and zinc is also being investigated.

Wilson's disease

This allows us to speculate on some of the many illnesses that may be unrecognised evidence of such changes. Wilson's disease is a disease due to an autosomal recessive trait in which copper is deposited. This affects both monoamine oxidase and ceruloplasmin metabolism, but the interesting point is that very few people suffer from Wilson's