THE BIOLOGICAL EFFECTS OF CHEMICAL SUBSTANCES

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*Footnote. The views expressed in this paper are those of the author and not necessarily those of the Department of the Environment.

Pollution may be defined as "the introduction by man into the environment of substances (or energy) liable to cause hazards to human health, harm to living resources or ecological systems, damage to amenity or interference with legitimate uses of the environment". This definition brings out the basic fact that in responding to pollution we are concerned with actual or potential adverse effects - damage to our own physiology, to the living resources on which we depend directly, or to the wider ecological systems of land and sea that play a vital part in the renewal of atmospheric oxygen, the elimination of many of our wastes, and the recycling of vital nutrients. In this paper I am concerned with the effects of chemicals injected into the biosphere by man (whether injected directly or indirectly via the physical environment), and the nature of our response to them.

Even the most apparently stable of living systems exhibits dynamic equilibrium. This is affected by physical and chemical variables at many levels. A pollutant in the environment may affect:

(a) an ecosystem

(b) a population of a single species

(c) a single individual organism

(d) an organ or system within that organism

(e) a biochemical or cellular subsystem

or several of these simultaneously.

MECHANISMS OF POLLUTION

Commonly, we consider pollution in terms of its effect upon whole individual organisms. It is evident that the responses of individuals are in fact the integral of a complex web of subsidiary effects upon biochemical systems, leading to changes in cellular physiology and behaviour which in turn affect the functioning of whole organs. A pollutant may often have a demonstrable effect at biochemical or cellular level which, because of the homeostatic machinery of living creatures, may not be manifest at the level of the whole individual at all. This does not mean that the individual is unaffected by the pollutant: indeed, it means that some of the biological capacity of that individual is being used up in the same way that the discharge of a pollutant into a lake uses some of its dilution capacity even though it does not reach the threshold at which ecological change ensues. Similarly, the responses of whole ecological systems are the integral of many interdependent individual responses. It is quite common for significant effects on individuals not to be manifested at all at the ecosystem level, where so many organisms of so many different species interact to produce a system that is in dynamic equilibrium and which displays considerable inertia.

These levels of effect are arranged hierarchically. The ecosystem level of response is the highest level and a "no response threshold" may yet be maintained while there is a considerable pollution-induced mortality at the individual level, and even at the species level should the species exterminated from the ecosystem not be of major importance in determining the characteristics of the whole. For example, it has been established that exposure of lichen species on the trunks of English oaks to mean annual sulphur dioxide concentra-

tions of 40-180 µg/m³/ can be correlated with their progressive extermination - and, no doubt, that of the small insects that feed on them. This has no effect, so far as we know, on the basic ecological stability of oak woods in the Midlands or South-East England, and, while it clearly leads to a degree of impoverishment compared with the natural situation, does not prevent the establishment of Woodland National Nature Reserves which support an otherwise rich and varied wildlife. Similarly, it would be perfectly possible to contemplate a pollutant effect which exterminated some species from a freshwater or marine environment without affecting either the capacity of those waters to support a balanced and productive ecosystem or creating a system unacceptable to man.

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In this situation pollutants can be considered to impose "stress" on the ecological system just as other forms of human interference do. It seems probable that the very first effect is to increase the species diversity of the system, but thereafter rising levels of pollution are paralleled by reduction in diversity and also by a tendency to change from systems dominated by large, long-lived forms to those dominated by smaller species with shorter life cycles - as when woodland gives way to grasslands. In more technical language, the shift is toward systems in which the annual production is larger in proportion to the standing crop. This is not the place to go into details, but we do now know a good deal about how ecological systems respond to stress and this is valuable in predicting what new levels of pollution may do to patterns of vegetation and fauna.

/ µg/m³ is microgram per cubic metre

At the population level, if one is concerned simply to have "no response" in terms of population size, enhanced mortality is acceptable so long as this is not on so great a scale that it leads to population decline. That is, it is permissible in these terms to substitute pollution for other forms of mortality that would otherwise remove the surplus of offspring produced in most populations of living creatures. If the annual production of young seabirds, for example, is three times that required for recruitment into a stable breeding population and the norm is for the surplus two-thirds to die, in population terms it is immaterial whether this surplus is removed by competition for food, predation or chemical pollution, so long as recruitment is sustained. The size of the population would be equally unaffected if the mortality among breeding adults were increased by pollution, so long as enough young survived to maintain recruitment at the higher rate which would then become necessary.

Indeed it seems very probable that during the early 1960's the heron population in England was in just this position in relation to certain organochlorine pesticides. We know that some adult herons were lethally poisoned and there is considerable evidence to suggest that sub-lethal residues affected egg-shell thickness and chick survival, resulting in a reduced output of young. Neither the adult mortality, nor the decrease in over-all breeding success, however, appear to have reached a level where they affected more than the annual surplus present in the population and so the total heron population of England and Wales of 4,500 pairs remained unchanged.

Again, a demand for "no response" at the individual level permits organ, cellular, or biochemical changes, so long as mortality or a serious retardation of growth or distortion of behaviour does not ensue. The influence of factories emitting fluoride on cattle grazing in their vicinity has been regulated on such a basis: a "no effect" standard at the physiological level has not been demanded, but the exposure has been restricted in such a way that unacceptable effects are avoided. Rather similarly, some pasture grasses and crop plants in industrial areas of Britain may have their growth rates affected by atmospheric pollution, including pollution by SO_2, but this is regarded as acceptable so long as yields are maintained above a certain threshold, determined by economics. Even in man one sees this situation, although the threshold is usually set much more stringently on health grounds: eg, in our acceptance of blood lead levels which affect the activity of the enzyme delta-aminolaevulinic acid dehydratase, so long as these are not permitted to rise above around 36-40 µg/100 ml of blood, which is commonly taken as the point beyond which we become socially concerned. Finally, if we determine that we shall have a "no response" situation at the biochemical level, we are admitting of no detectable effect whatsoever and are faced with the most stringent of all demands for pollution control. Indeed, with an impossible situation, since some substances we release as pollutants are present naturally in our environment at levels which have a biochemical effect.

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In social terms, we tolerate different levels of effect according to the nature of the target and its immediacy to the human situation. For example, it is commonly agreed that we should not accept significant effects at the organ level in man. We are indeed wary about the cumulative effects of lifelong exposure to substances which may only have minor demonstrable effects on human biochemistry, fearing that

they may in some way progressively erode the body's homeostatic machinery and shorten life. We reject significant effects at the individual level - illness or impaired growth - in cattle, sheep, other farm stock and domestic pets. We reject significant ecosystem effects in marine and freshwater ecosystems and in forest and wild vegetation, with their invertebrate and bacterial components. Sometimes, of course, the standard we set at one level affects organisms for which we would otherwise only be seeking lesser degrees of protection: standards set for mercury in fish as human food for example, may lead to curbs on discharges of ~~sheet~~ that metal to the sea which from the point of view of protecting marine ecosystems would not be necessary. It does not follow that our judgement in these matters is always wise (although I believe that it is defensible) but it does follow that we should be aware of the logic of our actions.

EXPOSURE AND EFFECT

The effect on a target depends upon three groups of variables:

(a) the nature of the pollutant,

(b) the biological state of the target with which it is interacting, including the individual and temporal variation which is exhibited by individuals, organs and biochemical systems,

(c) the concentration of the pollutant and the time of exposure (not usually, incidentally, capable of being summarised by a simple arithmetical multiplication. Short term exposures to very high concentrations need not always be the equivalent of exposures to half that concentration for twice the time or one-tenth the concentration for ten times as long. Ozone for example, can affect

sensitive plants in hourly exposures to around 10-20 pphm*: twice the time at half the level may have much less effect). In addition, it is important to know the site of entry of the pollutant (eg skin, lung, gut) and the site of action (nervous system, blood, kidney, liver etc).

[redacted] Such a pathway may be represented as in Figure 1. It is evident that a whole series of physical and chemical properties and interactions influence this movement, and parameters such as diffusion coefficients, absorption properties in and from soil, solubility in water or other liquids, rates of breakdown by ultraviolet radiation and reactivity with other components of air, water or soil can have a major influence. This is well exemplified by the pathway of sulphur dioxide in air (Figure 2) ~~(Holdgate and Reed, 1973)~~. Here transformation to SO_3^{++} or SO_4^{++}, washing out in rain as dilute sulphuric acid and/or combination with amonia to form an ammonium sulphate haze ("Teesside mist") have an important influence on the actual nature of target exposure. Similarly, chlorofluoro hydrocarbon compounds emitted to the environment as aerosol propellants apparently persist for a long time under ground-level conditions but are fairly rapidly degraded by ultraviolet radiation in the upper atmosphere: here physical mixing of layers of air will clearly be an important determinant of persistence. In predicting the scale of a possible pollution problem, therefore, any model we develop must take full account of the pathways, as well as the nature of the actual interaction of the pollutant and the target. It must also cater for individual variations in susceptibility and

*pphm = parts per hundred million

variations with time and circumstance.

Sometimes these variations can be very large. For example, concentrations of lead in air in Britain may reach 20-25 $\mu g/m^3$ in the centre of motorways by day and average 10-15 $\mu g/m^3$ over 24 hours. In ordinary busy streets, and around factories, 24 hour averages are around 1-3 $\mu g/m^3$. In the country levels are much lower - below 0.1 $\mu g/m^3$. Human exposure inevitably varies as people move around. Human intake to the lungs likewise varies according to the volume of air we breathe in the day - and while the average for an adult man is around 15 m^3 this can be doubled by activity or halved by rest. Not all the lead particles passing the nose or mouth reach, or stay in the lungs: various calculations give retentions of 10-60% of the potential intake. Only a proportion of the lead retained is absorbed - maybe 50% on average, but with much individual variation. If you sum the cumulative effect of all these variables, you can readily see how there could be a thousandfold variation in human lead absorption from the air - and the uncertainties in many figures cast doubts on the usefulness of such a broad-brush calculation at all.

Generally speaking, the effect of exposure to a pollutant is related to concentration, although the curve is unlikely to be linear. Moreover, the response to increasing concentration is frequently hierarchical in nature. That is, at the lowest concentrations there is a biochemical effect. With increasing concentration this becomes manifest in whole cell or whole organ changes, then in variations in individual behaviour. At higher concentrations still, individual mortality may ensue while greater exposure leads on to whole population or whole ecosystem changes.

We have partial documentation of many of these curves. Often the information results from laboratory tests, under conditions remote from those in the field, of single pollutants against single target species. This may be reasonable enough for exposures to pollutants with a specific action, like the effects of radiation on genetic systems (Figure 3), but how valid is it as an indicator of whole-population or whole-organism responses to chemical factors? Often the laboratory tests estimate doses that kill 50% of the exposed organisms (LD50) in 24, 48 or 72 hours or over a longer period of days. Often, the dose concentrations used in such tests are relatively high - sometimes this is necessary to demonstrate a response in a convenient time. The test species used are often selected for convenience in the laboratory and basic (and sometimes sweeping) assumptions are made about the similarity of response other organisms may be expected to show.

But what of the situation in nature, when we are dealing with mobile populations, forming part of a much more complex system , and able to move away from areas of high pollution. There can be no confidence in their extrapolation to the real world. Often, too, ecologically significant effects can be sub-lethal and will not show up at all in LD50 tests. For instance, seaweed growth rates can be related to degrees of marine pollution (Figure 4) (Burrows, 49), with consequences for herbivorous marine animals; and fish physiology can be adversely affected by DDT levels below the directly lethal dose, making fish more likely to succumb to cold or to be unsuccessful on migration (Figure 5) (Anderson, 49).

A good example of a complex and imperfect framework of knowledge is provided by sulphur dioxide in air - one of the commonest and most-

studied of atmospheric contaminants. As Figure 6 implies, quite a lot is known about the exposures at which various kinds of plant first show injury and then serious damage. Likewise, the onset of symptoms in the most sensitive people (bronchitics) is predictable. But in both cases allowance must be made for a very wide range of individual variation (in man there is probably a thousandfold range in sensitivity in an average population); furthermore, there is a great deal of variation in response according to the duration of the exposure, and at least in man, the levels of smoke accompanying the SO_2 exposure are critical. Thus, the "threshold" level for SO_2, averaged over 24 hours, is 500 $\mu g/m^3$ when accompanied by 250 $\mu g/m^3$ smoke ~~(Figure 7) (Lawther, ——)~~, whereas laboratory studies have failed to find significant impairment of human respiratory function by SO_2 alone at concentrations below 2500 $\mu g/m^3$ for comparable periods.

In this case we do have enough knowledge of dose/response relationships to conclude that in order to protect vulnerable people we should seek to keep daily average sulphur dioxide levels in air below 500 $\mu g/m^3$ and smoke below 250 $\mu g/m^3$ and these figures have been adopted as short-term objectives by a WHO Working Party. If we consider that the manifest sensitivity of lichens indicates potential biochemical disturbance which, while we cannot prove its parallel existence in man, ought in common prudence to be avoided, we would need to take an annual mean of around 40 $\mu g/m^3$ as our environmental quality goal - and
See that peaks never greatly exceeded this figure. Although we have little information on other species, this would almost certainly also prevent any crop losses and protect most forms of life while it would not eliminate the beneficial action of air-borne SO_2 in correcting sulphur deficiency in some soils. The recently published national

survey of smoke and sulphur dioxide ~~(AMRO 1972)~~ indicates that 500 $\mu g/m^3$ is only exceeded in the south of England, excluding London, for a very few days in the year, whereas in London and in the midland and northern towns it is frequently exceeded. But in many rural areas the lower "target" of an annual mean of 40 $\mu g/m^3$ is exceeded, so that it is not surprising that the British Lichenological Society has recorded considerable impoverishment of the lichen flora in the whole central midland zone of England ~~(Figure 8)~~. The question is whether this is paralleled by any significant agricultural losses (one report placed these at 240 million per annum in England, but with an enormous possible margin of error) and sufficiently serious to justify the major expenditure that would be required to reverse it (especially in the current energy situation).

Only rarely do we have even this degree of detailed information about exposure-effect relationships. More commonly all that we have is a broad, descriptive correlation between pollution levels, generally assessed, and biological response: an example for fresh water, the Trent Biological Index is a well-known ~~is given in Figure 9~~ There is clearly a need for more research to determine rigorous "criteria" on which our social judgement of the need for more (or less) intensive effort to combat pollution must depend. Such work needs to take into account the biochemical means by which the whole-organism responses are mediated. We need a better understanding of individual variations in response, and how variables such as age, nutritional state or reproductive condition of the individual interact with environmental chemical factors in determining them.

THE INTERACTION OF POLLUTANTS AND OTHER FACTORS

This leads to another important generalisation: pollutants rarely operate in isolation. I would like to give a case history that

underlines this rather well and illustrates the true complexity of the kind of situation that confronts us. Between August and November 1969 about 12,000 seabirds died in the Irish Sea. They were almost all of one species (Uria aalge, the Guillemot); they were almost all adults, and nearly all of them came ashore after September gales in an emaciated condition. Investigation ~~(Holgate, 49)~~ showed, however, that the gales were not the primary cause, for while the peak in the number of strandings coincided with the storms, some birds had begun to leave the water in a distressed condition before the gales began and while gales of equal severity had affected the whole western seaboard of Britain, the mortality was concentrated in the Irish Sea north of a line from Holyhead to Dublin and south of a line from Donegal to the Mull of Kintyre ~~(Figure 6)~~.

Histological examination showed that many of the birds had lesions in the kidney and liver and changes in the heart and pericardium which paralleled experimental effects obtained by exposing birds to large concentrations of polychlorinated biphenyls (PCBs). Chemical analysis subsequently showed high levels of PCBs in liver and kidney, and to a lesser extent in brain ~~(Table 4)~~. It would be tempting, therefore, to frame the hypothesis that these birds died of PCB poisoning. This, too, would be an oversimplification, since healthy birds shot off the west coast of Scotland contained whole-body loads of PCBs comparable with those in the victims of the disaster ~~(Table 2)~~. The difference was that in the healthy birds the PCBs were distributed in the body fat and the levels in liver and kidney were low.

The conclusion of the study was that we were dealing with a multivariate situation. The most plausible hypothesis was that something,

possibly climatic change, caused a reduction in the available food supply of these birds in midsummer at a time when their reserves were fairly low after the breeding season and they were about to go into the moult, which imposes a further stress. The oceanographers were unable to detect any change in marine life, but since these birds feed only in the very top layer of the water, a long clear spell with bright sun might have led their food organisms to descend slightly deeper than normal, thereby shutting off the food supply but not creating changes detectable with the marine biologist's tow net. Be that as it may, the initiating cause of the mortality was very possibly an interruption of feeding. This would, of course, have led to the mobilisation of body fat, which would have brought the PCBs stored there into the blood stream and on their way to liver and kidneys. This in turn might well have had minor behavioural effects at the whole-organism level. It might have made the birds a little less efficient at feeding, thus exacerbating the situation, and more prone to exhaustion by storm, especially since their food reserves were already low; any further stress could only lead to the increasing flushing of PCBs into the blood. There would then be the recipe for a localised disaster in the area where the initial imbalance between food intake and food requirement had occurred, and the whole disaster would be attributable not directly to PCB poisoning, but to the impact of this pollutant within the system, tipping the scale between survival and death when the environment imposed external stress. I believe that this is likely to be a model commonly valid in the pollution area and that many pollutants must be looked on as operating in this way, changing the probabilities between health and disease in a situation depending on a great complex of variables.

In predicting what will happen under a variety of circumstances, it is clear that we must take account of the sensitivity of organisms to various levels of exposure to both individual significant pollutants and combinations of pollutants. We must also endeavour to predict the levels of exposure at these target organisms, and these in turn depend on the pathways of the pollutants and the factors determining their rate of input to and removal from the environment.

It is well known that all chemical substances vary in these respects. It is self-evident that the pollutants likely to attain significant concentrations in regions remote from their sources of emission are those released in large quantities, readily soluble in water or easily dispersed in air, having a rapid rate of diffusion and having a low rate of chemical or biological transformation into innocuous products or a low rate of settlement of sedimentation from air or water in which they are suspended and perhaps ultimately exhibiting the phenomenon of concentration via food chains. Those of high persistence, but less ready dispersal, on the other hand, are clearly type-cast to create "hot spots" about points of emission. In both cases, if we know about their inherent toxicity and the way by which that toxicity takes effect, we have the recipe for predicting the likely seriousness of a pollutant substance.

It would be useful were we able to go further than this, and predict from the molecular structure of a substance what its behaviour in the environment was likely to be. Sometimes this can be done to a degree, as with the probability of biodegradation of certain detergents

(Hiatt, 19). But a recent Royal Society discussion exposed the limitations of this approach ( ). Even minor structural changes alter the behaviour of molecules to a surprising degree. For the foreseeable future we can expect to have to rely as the chemical industry now does on laboratory and field testing prior to the introduction of new substances or new formulations. What we can ensure however is that the mass of available information is more effectively retrieved, and this is the aim of the information referral system being developed for the United Nations, with the active participation of the UK Chemical Information Service (Penchey, 197).

The construction of meaningful numerical models of environmental pollution is extremely difficult. Not only is one confronted with the almost endless variation in biological response, but the curves relating emissions to concentrations in the environment at a particular point and time are influenced by a very large number of factors. We have at the present time some relatively crude models for the behaviour of pollutants in water, particularly rivers, which have the merit of being relatively confined and subject to predictable patterns of flow. Such models are being attempted for the sea, but are prone to great difficulty, while for air they have succeeded only at the most superficial level. Clearly, we need many more studies of pathways and effects, and these are the two key areas for research upon which must depend our social response in terms of judging the relative seriousness of a pollution problem and therefore the priority we must give to its control. For we must remind ourselves that we live in a world in which chemical factors are, and always have been, important in ecology and physiology. Many of the pollutants to which we are exposed are natural substances whose concentrations and distributions, rather than

eliminate these substances; indeed many of them are essential at low concentrations to our well-being. We could not expect to take their concentrations back to pre-industrial levels even if we knew what these were. What we can do is seek to predict the points at which their concentrations become socially unacceptable and to work out the most efficient means, in economic and technological terms, of restricting those levels below that threshold. And some pollutants, eg PCBs, are entirely unknown in nature. Here also we must apply the principle of restricting their concentrations to the threshold of social unacceptibility. On the basis of this knowledge, too, we can make a more sensible decision about the parameters we need to monitor in the environment and the frequency of the sampling we require in space and time. Quite evidently, that monitoring must allow us to assess as accurately and precisely as possible the degree of exposure of various targets. It would be logical to see the network of measurements most closely grouped about targets in which we are not prepared to see any significant changes at individual level, while a looser network of monitoring might be acceptable where we are concerned only to prevent changes at the ecosystem level. Moreover, if we are prepared to accept changes at all levels short of the ecosystem level, biological monitoring becomes evidently acceptable. If we monitor the performance of indicator species, we are tacitly accepting that we are prepared to see their numbers or their performance impaired but because the ecosystem level is unlikely to show change until such impairment at individual level has occurred on a substantial scale, the biological monitoring programme yet gives us something of a safety factor and time span in which to effect corrections should that be needed. On the other hand, biological monitoring may be less acceptable where we

are seeking to prevent damage at the individual level unless we can find an organism which is an order of magnitude more susceptible than the targets we are chiefly concerned to protect and displaying a similar sensitivity to the whole interacting matrix of factors. Alternatively, we can monitor at the biochemical level (for example following lead levels in human blood or organochlorine levels in wild life) so as to gain early warning of effects at the organ or individual level. Whatever conclusion we reach, the design of a competent monitoring system must be greatly influenced by our knowledge of the exposure-effect relationship between pollutants and the targets with which we are particularly concerned.

TRENDS IN POLLUTION AND SOCIAL RESPONSE

Pollution has become a subject of increasing national and international concern in the last two decades. But it is fair to point out that the majority of the incidents which have served to focus this concern have been due to localised concentrations of particular pollutants in pollution "hot spots" (eg smoke and sulphur dioxide in the London "smogs" of the 1950s, mercury and cadmium in water receiving effluent from certain Japanese factories, and raised blood lead levels near a number of factories using lead). It is also fair to add that while the number of incidents causing concern has mounted, the actual damage due to pollution has declined: London "pea-soupers" and acute pollution near industries almost certainly harmed more people fifty years ago. [redacted]

There is no evidence of damage to human health or ecological stability on a global scale, due to a globally-distributed pollutant (except perhaps for the statistical prediction of increased radiation damage

atmosphere). The most-quoted examples of global trends in pollution, the gradual increase in the proportion of carbon dioxide in the atmosphere, the trace quantities of DDT and other pesticides to be found in oceans and wildlife throughout the world, and the possible changes in atmospheric turbidity due to the injection of fine dust into the air, are all no more than changes in pollutant level without as yet proven consequences for living "targets".

Nonetheless, it does not follow that increased emissions of pollutants will not have unwelcome effects, both through the creation of more and more unacceptable local "hot spots" and through the elevation of the general level of contamination of ocean and atmosphere to the point at which undesirable climatic or ecological changes ensue. Granted that the latter process, in particular, is likely to build up slowly and require an equally long time and great effort to reverse, it is clearly important to improve our scientific knowledge of pollutants and their effects and our predictive capability, as well as our technical capacity to control pollution. In the past, we have allowed pollution damage to develop, and had to cure it when we no longer found it acceptable; in the future we need to forestall such damage, and this requires better understanding than we now have.

TRENDS IN POLLUTANT EMISSION

It is a historical fact that some pollutants have been emitted in steadily rising quantities, in parallel with mounting human populations and increasing industrialisation. The generation of man's body wastes and food residues, and the body wastes of his livestock, inevitably rise in direct proportion to population. Energy generation through the

burning of fossil fuel releases carbon dioxide, carbon monoxide, oxides of nitrogen and of sulphur, water-vapour, hydrocarbons and a variety of particulates. Total emissions of all these have risen steadily over past decades or even centuries, although man still adds less of many of them to the environment than is contributed by natural processes. There is a similar tendency for the vastly wider range of pollutants generated by industry likewise to rise in direct relation to industrial growth, unless anti-pollution measures are taken, or unless inherently 'cleaner' processes are developed.

[redacted] This is because of recognition of the need for abatement and the development of increasingly efficient pollution control technology. The pollutants still on a rising trend are generally those not known to be harmful at present or projected levels and hence not yet justifying control. Recognition of the need for control, eg on emissions of hydrocarbons, carbon monoxide and lead from cars, lead from industry or sulphur oxides from low level chimneys, has already begun to affect the trends in the products of fossil fuel combustion, until recently looked on as harmless. However, abrupt changes in the cost of various fuels will necessarily have a modifying effect on control policies adopted in future for various forms of energy generation. For we live in a situation where marginal benefits and marginal control costs have to be balanced, and the balance can shift either way.

FACTORS INFLUENCING FUTURE TRENDS

In crude terms the concentration of a pollutant in the environment is the result of emission and removal. In practical terms, the important determining factor is man's deliberate control over emissions. This is in turn governed by the costs and benefits of the activities leading to the generation of the pollution and the actual or predicted cost to the community of the damage it does. Research, leading to a deeper understanding of these matters is likely to be a powerful determinant of the actual future trends in emission, concentration and effects of any substance.

It is impossible to catalogue all major pollutants and predict their future levels. But we can reliably forecast a continuing decline in damaging industrial emissions such as acidic and alkaline vapours, particulates and heavy metals, and in persistent organohalogens such as PCBs and DDTs, for which substitutes either exist or are being sought. We can predict a slowing down in the upward curve of emission of pollutants from motor vehicles since most developed countries are adopting standards that will halt, or even reverse, this trend within their borders. Substitution of nuclear for fossil energy sources will also lead to a net decline in gaseous and particulate emissions, although imposing a demand for continuing stringent controls on radioactive discharges. Recent international agreements seem to lead to a progressive reduction in the dumping or discharge of organohalogens, oils, heavy metals and known carcinogens to the sea and rivers, and the levels of these substances in the ocean and marine life should soon begin to fall.

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Nevertheless, pollutants continue to be an inevitable accompaniment of man's increasingly technological society, and we shall always have wastes to dispose of in the environment. Indeed, it is entirely

legitimate to use the environment for this purpose, so long as we do not over-tax its finite capacity to disperse and degrade wastes. With increasingly stringent demands for the testing of new substances for environmental effects before they are used or discharged, and increasingly precise analytical, toxicological research methods at our disposal, the probability is that pollutant levels will less and less frequently be allowed to mount to the point at which damage begins.

THE TREND IN THE COST OF POLLUTION CONTROL

This essentially optimistic projection is grounded on history, for in Britain and most other developed countries, pollution has become a diminishing problem as our awareness, scientific understanding and technological capabilities have grown. But there is a price to pay for continued optimism. There is a tendency for the energy cost of pollution abatement or prevention to rise disproportionately as populations grow and the standard of living and technological development increase. This is because:

(a) the capacity of the environment to disperse and degrade pollutants is finite, and any increment of pollution over and above that capable of natural degradation demands treatment prior to release,

(b) the larger that increment, the purer the effluent may have to be, volume for volume, if the quality of the environment is to be sustained,

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(c) the costs of removing pollution from an effluent tend to mount exponentially as one moves towards greater purity,

(d) the development of substitute or new products becomes more

and more costly as the community demands increasingly stringent tests of their safety,

(e) the environmental quality which people demand tends also to rise in parallel with the standard of living, and this provides a "positive feed-back" increasing the costs listed above,

(f) as we become more and more dependent on technology to purify emissions and protect the environment, so the consequences of break-down become less acceptable and more thorough "fail-safe" mechanisms are needed, often at substantial cost (c.f. the standards required for nuclear as against convential power stations).

As economic and technological growth proceeds, an increasing proportion of GNP thus tends to be demanded for environmental protection. If this is to be prevented, increasing effort needs to be devoted to the development of better, safer and more efficient technologies, giving higher quality controls at less cost. This is one of the needs of society to which chemistry has shown itself responsive, and must continue to respond.

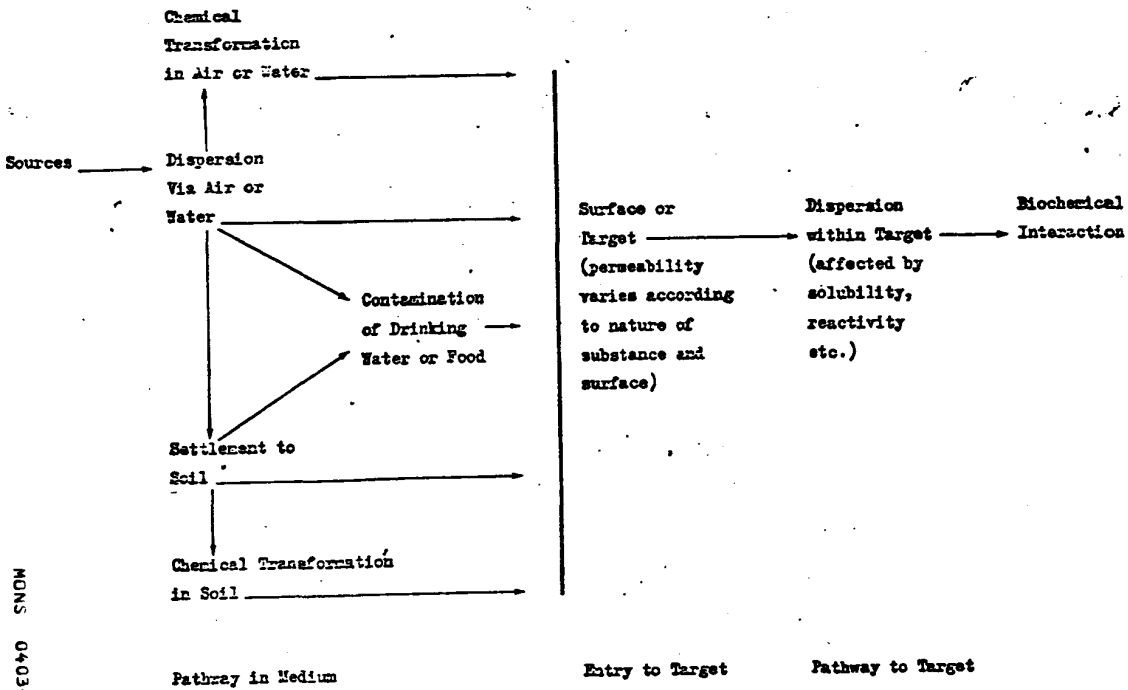
Captions for figures

Figure 1. The pathways of pollutants from source to the point of effect.

Figure 2. The pathway of sulphur dioxide in air (from Kellogg, W W Cudle, R.D., Allen, E R, Lazarus, A L and Martell, E A, Science (February 1972), Vol. 175, No 4022).

Figure 3. Thresholds of sulphur dioxide effect on some plants and people.

FIGURE 1



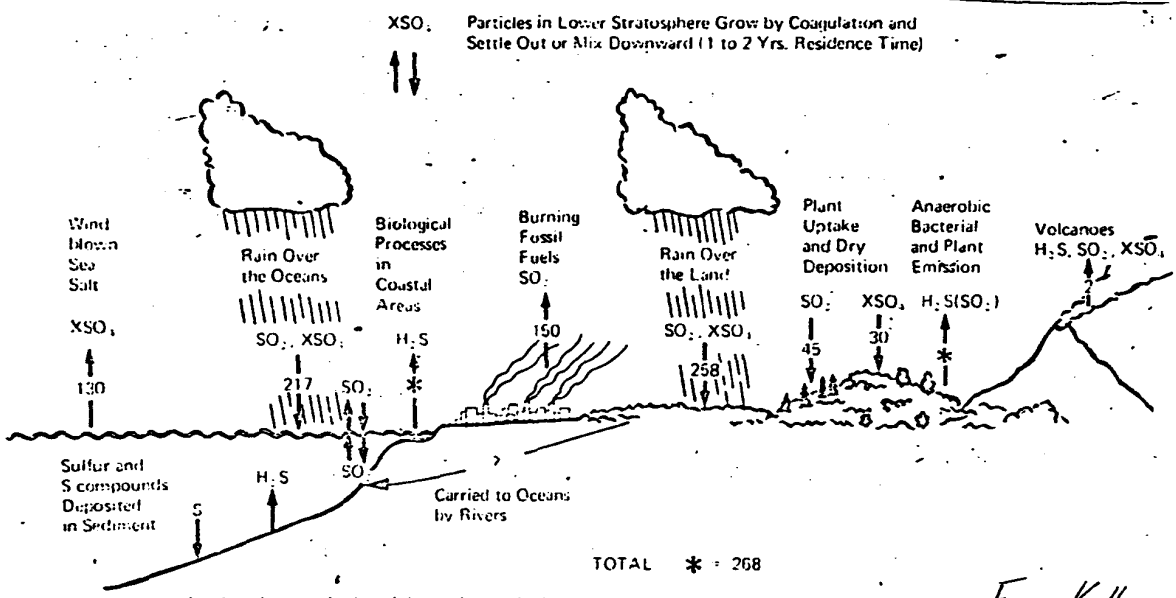
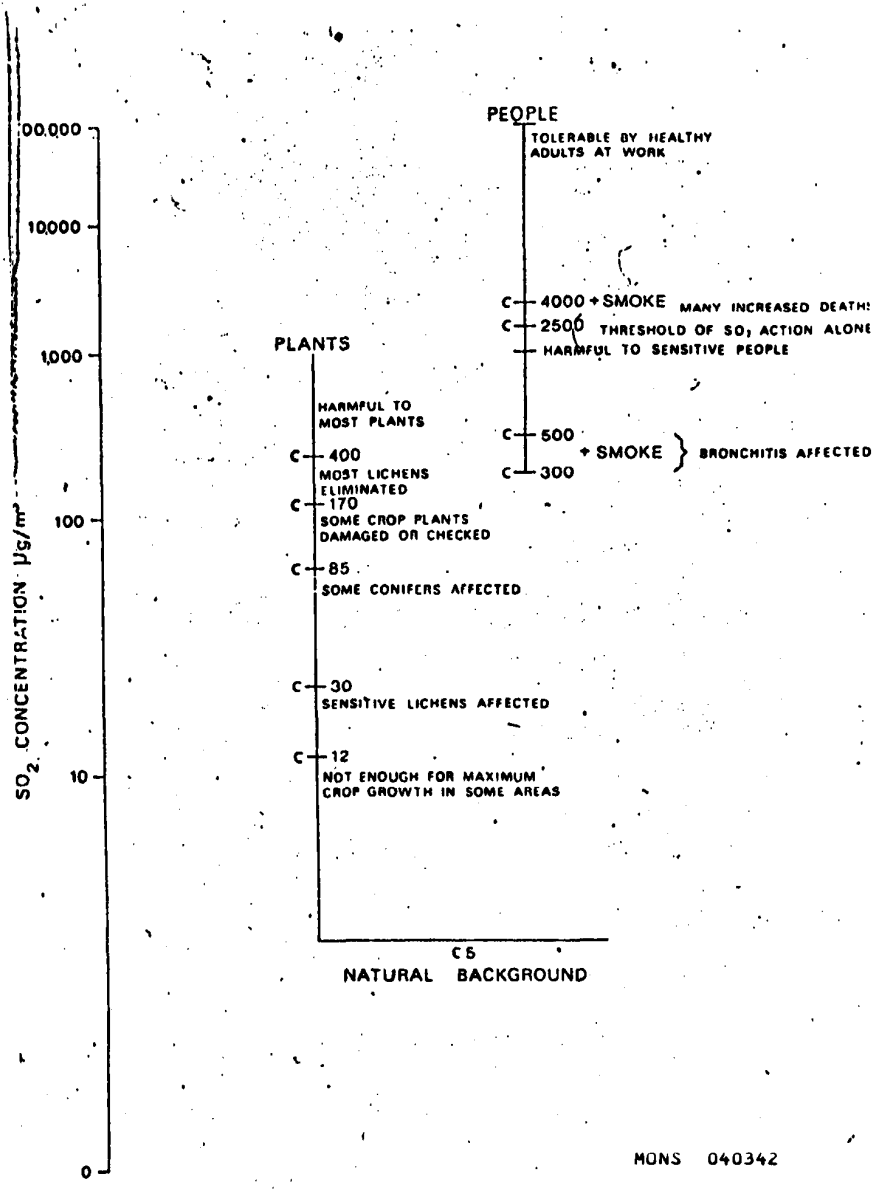


FIG. 2. Sources and sinks of atmospheric sulphur compounds. Units are 10^6 tons calculated as sulphate per year. From Kellogg Critical Science Vol 175, No 4022.

water, vegetation or structures; or they may be transformed in the air into other substances which, in some cases, may be more obnoxious than the original ones.

concern are those emitted in large quantities, especially as a result of fuel combustion. The chief of these are carbon dioxide, carbon monoxide, hydrocarbons, oxide


PEOPLE
TOLERABLE BY HEALTHY ADULTS AT WORK
C 4000 + SMOKE
MANY INCREASED DEATHS
C 2500
THRESHOLD OF SO2 ACTION ALONE
HARMFUL TO SENSITIVE PEOPLE
C 500
+ SMOKE
BRONCHITIS AFFECTED
C 300
PLANTS
HARMFUL TO MOST PLANTS
C 400
MOST LICHENS ELIMINATED
C 170
SOME CROP PLANTS DAMAGED OR CHECKED
C 85
SOME CONIFERS AFFECTED
C 30
SENSITIVE LICHENS AFFECTED
C 12
NOT ENOUGH FOR MAXIMUM CROP GROWTH IN SOME AREAS
C 5
NATURAL BACKGROUND
SO2 CONCENTRATION µg/m³
100,000
10,000
1,000
100
10
0
MONS 040342