PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY A MATHEMATICAL, PHYSICAL & ENGINEERING SCIENCES

Targets of Pollutants in the Atmosphere [and Discussion]

M. W. Holdgate, R. S. Scorer, C. P. Whittingham and Elizabeth I. Young

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Targets of pollutants in the atmosphere

By M. W. Holdgate†
Departments of the Environment and of Transport,
2 Marsham Street, London SW1P 3EB, U.K.

People, animals, plants, ecological systems and human artefacts are all potential targets (or receptors) for pollutants. The amount of damage caused can generally be related to the dose received (defined as the product of concentration and time of exposure), and the properties of the receptor that govern uptake of the pollutant and susceptibility to it.

Plants and animals vary widely in their sensitivity to gaseous pollutants and to particulates, aerosols, and substances deposited in rain. Atmospheric pollutants may affect targets directly, or indirectly through changes in climate and, after deposition, via aquatic systems and soils. In predicting potential damage to targets each substance and type of receptor needs separate evaluation, and what usually matters is the total dose of a substance received, over what period, and in the presence of what other factors, rather than the particular proportion transmitted via the atmosphere.

Introduction

Pollutants provoke concern and social action because of the damage they are considered likely to cause. 'Damage' includes all adverse effects on man, his crops and livestock, his artefacts and his environment (Saunders 1976). It may be *acute*, resulting from short exposures to high concentrations of pollutants, or *chronic*, caused by exposures to lower concentrations over longer periods. Acute damage usually results from the near-instantaneous and irreversible response of the target or receptor, and is clear-cut: the causative agent is not too difficult to determine and controls can also be fairly direct. Chronic damage is much less easy to assess: it may involve the combination of many factors and result from sources of pollution that are distant in space and time: it may also be temporary or reversible to various degrees.

Atmospheric pollutants may affect targets or receptors (the terms are used interchangeably in this paper):

(*a*) *directly*, by (i) chemical effects on organisms; (ii) chemical effects on artefacts (corrosion); (iii) physical effects on organisms and artefacts (e.g. abrasion of surfaces, obstruction of stomata, and soiling of buildings by particles);

(*b*) *indirectly*, by (i) chemical effects on habitats (e.g. changes in acidity of soil or water); (ii) physical effects on habitats (e.g. temperature changes, changes in climate).

It is not unusual for a substance to have both direct and indirect effects. Sulphur dioxide, for example, is directly toxic to plant tissues, but may also exert an indirect effect when deposited as sulphate by wet deposition, dissolved in rain, on to soil or fresh waters. In considering the likely effect of atmospheric pollutants on targets it is therefore necessary to consider the whole series of pathways that they may follow.

† The views expressed in this paper are those of the author and not necessarily those of the Departments of Environment and Transport.

Many substances that we regard as atmospheric pollutants because their concentrations have been elevated by man are also natural components of the air. The proportion of natural and man-made sources in any particular area clearly needs to be considered in any analysis. So must the interactions between atmospheric pollutants, and the changes in ecological balance that result from their uneven impact on living targets.

In this paper no attempt is made to catalogue all the possible direct and indirect effects of atmospheric pollutants acting singly or in combination against the millions of different potential targets in the living world. Illustrations are given of some of the more important types of effects, drawing heavily on papers presented to the Study Group on Pollution in the Atmosphere over the past two years. Effects on plants receive more emphasis than those on animals because more information is available about them. Effects on the atmosphere itself are excluded because they are reviewed in other papers: in a sense, ozone in the stratosphere can be regarded as a 'target' for chlorofluorocarbons, climate and polar ice-caps as 'targets' affected by carbon dioxide, and biogeochemical cycles as targets of almost anything, but it would broaden (and weaken) the discussion too much to pursue such ramifications here.

Exposure, dose, uptake and effect

The *exposure* of a target to a pollutant is defined in terms of the concentration in the medium (or media) in contact with it at a particular time. *Dose* is defined as the total amount of the pollutant received by the target over a period. Exposures commonly fluctuate with time, many pollutants reaching short-lived peaks determined by climatic factors that affect dispersion (e.g. temperature inversions), or by sunlight, affecting photochemical reactions (figure 1). Damage commonly increases markedly with exposure concentrations, even when exposure times are short. Ozone at a concentration of 10 parts/10^8 can cause acute injury to leaves of potato (*Solanum tuberosum*) after 4 h exposure (Brennan, Leone & Daines 1964), while white pine (*Pinus strobus*) needles are damaged after the same period of exposure to only 6.5 parts/10^8 (Berry & Ripperton 1963). In contrast, exposures of pinto bean (*Phaseolus vulgaris*), radish (*Raphanus sativus*) and the pines *Pinus elliotti* and *P. taeda* to ozone at 5 parts/10^8 over 3 days, 5 weeks and 18 weeks respectively caused no visible leaf injury but resulted in chronic effects such as reduction in leaf growth, fresh mass and photosynthesis (data from Engle & Gabelman 1967; Tingey, Heck & Reinert 1971; Barnes 1972; summarized by Bell 1974). Ashenden & Mansfield (1977) review evidence that SO_2 generally does not cause visible injury at concentrations below 25 parts/10^8, but may depress growth in sensitive plants like S 23 ryegrass exposed to 15 parts/10^8 for 77 days. The time over which the dose is delivered is obviously critical, and a particular effect may *not* be produced by half the exposure over twice the time, even though the cumulative doses are identical.

The concepts of *dose commitment* and *harm commitment* (see, for example, Machta 1976) developed originally for radiation protection purposes, may be applied to other pollution problems (M.A.R.C. 1977). They imply a further step. Dose commitment is the predicted dose that a target population or populations will receive assuming a specified emission of pollutant over a period and allowing for the various factors that govern dispersion and transformation along pathways (table 1). Harm commitment is a statement of the damage that is predicted to result from that dose. As Machta (1976) emphasises, there are obvious dangers in predictions of this kind, involving extrapolation from data of limited reliability, and this is also well illustrated

by table 1 which includes doubtful assumptions about the impacts of aircraft, fertilizers and chlorofluorocarbons. Even when dose commitment can be defined, it cannot be translated into harm commitment without knowledge of the interactions that occur at and within the the receptor. These interactions begin at the surface of the receptor and depend critically on its composition and on the nature of the pollutant and whether it is gaseous, particulate, a liquid droplet, or a solution in such a droplet.

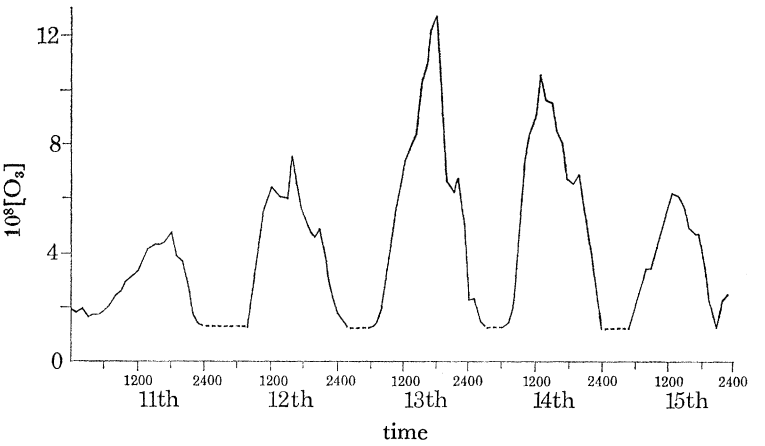

FIGURE 1. Hourly mean ozone concentrations at Silwood Park, 11–15 July 1972. Note that 1.3 parts/10^8 is the lowest value recorded because the zero of the meter was offset during calibration. From Bell & Cox (1975), with permission.

The chemistry of corrosion of many artefacts by atmospheric pollutants is well established: for example, the tarnishing of silver by hydrogen sulphide, corrosion of limestone by sulphur dioxide and acid aerosols, damage to nylon fabrics by acid aerosols, or the cracking of rubber and fading of dyes due to the ozone in photochemical smog (Jaffe 1967). Means of protection of exposed wood, metal and stone from atmospheric pollution and fungal attack have received major investment. Substitution has solved some problems: in the early 1960s, for example, stress cracking of nickel–brass alloy relays in Los Angeles telephone offices due to high concentrations of nitrates in urban dusts was solved by replacement with cupro-nickel and better humidity and ventilation controls (American Chemical Society 1969). Despite relatively good scientific understanding of the interactions, however, the costs of air pollution damage to materials was estimated at $\$4.8 \times 10^9$ in the United States in 1968 (Council on Environmental Quality 1973), while in Britain the corrosion and protection of metals and accelerated degradation of textiles, paper, leather, etc., were together estimated to cost $£75 \times 10^6$ p.a. in 1970 (Programmes Analysis Unit 1972).

With living organisms both the interactions and the consequent damage are much less understood. Many terrestrial plants and insects are waterproofed by surface wax layers, variously protected by layers of lipoprotein and other substances. When clean, such surfaces are not readily wetted by water and gas exchange through them is slow: in contrast, they are readily wetted by oily droplets, may be rendered permeable by abrasion by some mineral particles and adsorption by others (including carbon dusts), and may trap particles readily because they are rough. As leaves age, they often support an increasing growth of algae, fungi, yeasts and bacteria and the effects of this 'phyllosphere' on surface permeability are uncertain.

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TABLE 1. ESTIMATED ENVIRONMENTAL EFFECTS (DOSE COMMITMENT) AND INCIDENCE OF AND MORTALITY FROM SKIN CANCERS (HARM COMMITMENT) DUE TO STEADY STATE RELEASE OF SELECTED CHEMICALS (FROM MACHTA 1976).

				dose commitment		harm commitment, additional cases per year†		
						incidence‡		
chemical	input 10^{11} g/a	altitude of input km	source	% ozone depletion	% increase u.v.-B	non-melanoma skin cancers	melanoma skin cancers	mortality melanoma skin cancers‡
NO_x	156	10.5	400 wide-bodied sub-sonic aircraft	0.082	0.12	2000	40	10
NO_x	1	16–18	100 Concorde, TU-144 aircraft	0.11–0.57	0.17–0.86	2500–15000	50–250	10–50
NO_x	164	19.5	100 large supersonic aircraft	3.27	4.91	75000	1500	300
N_2O	2000(N_2)	0	fertilizers in 2000 A.D. and constant there-after	< 1.8–23.0	< 2.7–35.0	< 40000–500000	< 900–10000	< 150–2000
F-11§	2	0	aerosols, re-frigerants, etc., at 1973 rate	6.5–18.0	9.8–27.0	150000	30000	600–1500
F-12§	3					400000		

† Estimates assume a constant world light-skinned population of 10^9 persons.
‡ Based on current non-melanoma skin cancer incidence of 150/100000 light-skinned persons per year; melanoma incidence of 4/100000; melanoma mortality of 1.5/100000.
§ Chlorofluorocarbons ('freons').

In contrast to these waterproof surfaces, the stomata of green plants, the spiracles and trachea of insects, and the lungs of vertebrates are adapted for rapid gas exchange, culminating in the solution of the gas in shielded, moist surfaces.

It follows that although many pollutants, including gaseous sulphur dioxide, fluorine, nitrogen oxides and photochemical oxidants, acid aerosols, and particulate carbon, lead or fluoride may be present in polluted air, the points of contact between target organisms and

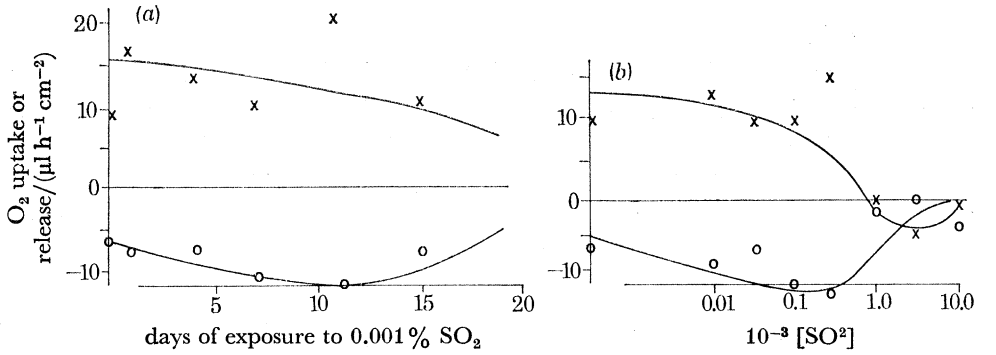

FIGURE 2. Photosynthetic activity (×) and respiratory activity (○) in disks of lichen thallus. (*a*) The effects of different duration of exposure to constant SO_2 concentration; (*b*) the response to increasing SO_2. From Ramade (1977).

pollutants and the rates of pollutant uptake must vary widely. Gaseous pollutants reach plant and animal surfaces at a rate dependent on the resistance to diffusion within the boundary layer, which in turn depends on the velocity and turbulence of the airflow over the surface (Ashenden & Mansfield 1977). Penetration within plant and animal tissues is generally fastest at sites of respiratory and photosynthetic exchange, and is greatly affected by the rates of lung and insect tracheal ventilation and on whether stomata or spiracles are open: these factors may themselves be influenced by exposure concentrations. Pollutants deposited on surfaces by wet deposition may well penetrate more slowly (pesticides and perhaps fluorine are exceptions) and may largely affect surface layers and superficial organisms like those of the phyllosphere. Particles may physically impede stomata and reduce the efficiency of animal respiratory exchange, but except where they have damaging mechanical effects, may be of more significance to animal herbivores than to plants on which they are deposited: this is certainly true of fluoride and lead particles. There are, however, some indications of interactions between dusts deposited on tree leaves and sulphur dioxide. Particles of soluble materials (like lead sulphate) naturally form an intermediate category, moving under physical diffusion in an airflow (for example within a lung) and then through solution at the tissue surface. More research is needed to document more precisely the sites and rates of entry of atmospheric pollutants into organisms.

Within target organisms, pollutants follow pathways (usually in circulating or diffusing fluids) to reach the biochemical systems on which they exert their primary effects. Oxides of nitrogen, SO_2, peroxyacetyl nitrates (PAN) and ozone all damage the mesophyll tissues of plant leaves, and SO_2 damage is manifest in the disruption of chloroplasts and depression of photosynthesis (Ramade 1977; figure 2). Ozone causes the palisade cells of the leaf to collapse through some unknown mechanism: there is some evidence that it affects the permeability of cell membranes (Bell 1976). In all these instances the pathways of the pollutant within the

target are short, involving gaseous diffusion through the airspaces of the leaf and movement in solution in the cytoplasm.

In animals, too, sulphur oxides, ozone and PAN appear to act fairly directly on the cells of the mucous membranes in the respiratory tract and other sensitive tissues (like the conjunctiva). They are irritants, and provoke mucus secretion and can consequently aggravate respiratory disorders like bronchitis, emphysema and asthma. Smoke particles also aggravate such conditions, perhaps as much through the actual physical clogging of lung surfaces as through irritation, although the capacity of some particulates (like silica dust or asbestos) to cause specific disorders is well known. In contrast, some other atmospheric pollutants follow a somewhat longer pathway within animal targets in solution in the blood (whose oxygen carrying capacity carbon monoxide directly impairs) to reach the biochemical systems upon which they operate. Organochlorine compounds and metals like lead appear to have primary lesions on a range of enzyme systems, the latter affecting those involved in blood synthesis and many other functions and the former interfering with nerve impulse transmission in insects (Moriarty 1975; Clayton 1975). These effects were reviewed in detail in another Discussion Meeting convened by Sir Richard Doll,† and will not be analysed further here. The important point for us to recognize is the need to define the sites and rates of entry of airborne pollutants into targets, and their pathways and impacts there, before harm commitment can be calculated.

Variation in target sensitivity

It is a matter of common observation that plants and animals differ in their sensitivity to atmospheric pollutants: the widespread growth of the London plane (*Platanus* × *hispanica*) and the corresponding rarity of Scots pine and other conifers in the centre of the city is a direct expression of such variation. The differences in sensitivity between particular targets may reflect intrinsic genetic factors, or age and physiological condition (the latter depending on many other factors, such as nutritional state, reproductive condition, disease, parasite loading, etc.).

A number of authors have examined the range of variation between plant genotypes in their susceptibility to air pollution. Figure 3, based on work by Bell (1976) demonstrates that the tobacco strain Bel W-3 is much more sensitive to ozone than the strains Bel-C and Bel-B. Table 2 repeats well known data on the relative susceptibility of lichen species to SO_2 (Hawkesworth & Rose 1976; Ferry, Baddeley & Hawkesworth 1973). Within *Homo sapiens* the average annual incidence rate of malignant melanoma (which can be induced by u.v.-B radiation, and is hence one of the causes of concern over possible depletion of stratospheric ozone) varies in the United States from 4.6 and 4.4 per 10^5 (age-adjusted data) in white males and females respectively to only 0.9 and 0.7 per 10^5 in black males and females. (C.I.S.C. 1976).

These genetic differences are not necessarily static. Bell & Mudd (1976) demonstrated that in an area of Lancashire with a long history of coal-smoke pollution, ryegrass from a less-polluted coastal locality is adversely affected by prevailing pollution conditions to which indigenous ryegrass is immune (table 3). This effect probably depends on differential response to SO_2 of the processes of chlorophyll synthesis or degradation. A similar rapid selection of tolerant plant

† 'Long-term hazards to man from man-made chemicals in the environment', 13 and 14 December 1977. To be published in 1979 in *Proceedings*, series B.

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strains is well known in areas highly polluted by metals (Roberts 1976; Wainwright & Woolhouse 1975).

There are many examples of variations in target susceptibility with age and physiological condition. Cooke (1970) has shown that frog tadpoles dosed with DDT in early life show a marked initial mortality, but then no further deaths until the time of metamorphosis when mobilization of body reserves brings the pesticide into circulation and promotes a second peak of mortality. On a larger scale, about 12000 guillemots (*Uria aalge*) died in the Irish Sea in

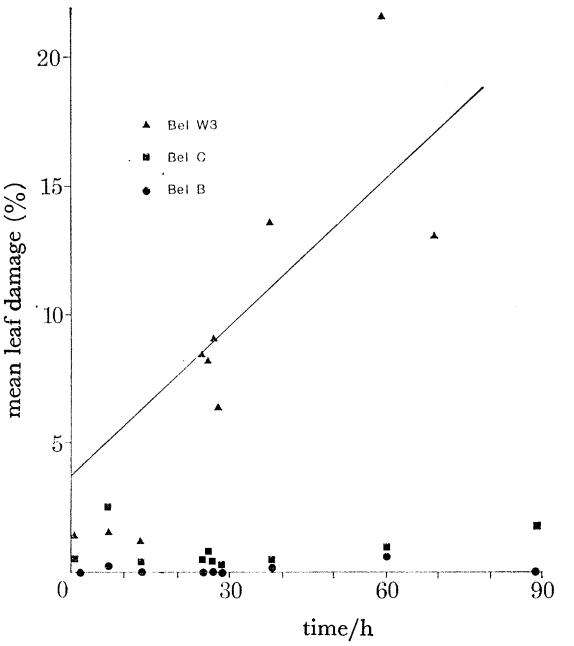

FIGURE 3. Mean leaf damage in three tobacco varieties in relation to the time that the atmospheric ozone concentration exceeded 4 parts/10^8. From Bell (1976), with permission.

1969: almost all of the dead birds were adults and the mortality probably resulted from a combination of high body burdens of polychlorinated biphenyls (p.c.bs) and strain imposed by storms at a time when the birds had already been stressed by breeding and by the moult which follows breeding (Holdgate 1971; Moriarty 1975). In man, it is well known that susceptibility to urban smogs depends very much on physiological condition: of the 3500–4000 excess deaths in London in 1952/3, most were among the elderly with histories of bronchitis, emphysema or cardiac weakness (Royal College of Physicians 1970), but the death rate of children in the first year of life also rose abruptly. Lead is another pollutant whose uptake and effect in man vary with age. Lead compounds are abortifacients and women working with lead have a high miscarriage rate, perhaps because lead passes the placenta and accumulates in the foetus throughout pregnancy (Clayton 1975). Children appear to take up more lead from the gut (perhaps because of dietary differences) and may be more susceptible to its neurological effects, although there are major uncertainties here.

A rigorous basis for comparing the susceptibility of targets to pollutant damage is often lacking. This arises because of the capacity of organisms for physiological homeostasis and reproductive and ecological adaptation, and because effects are manifest at many 'levels'. The primary lesion of any pollutant on a target organism is commonly biochemical: through

the perturbation of enzyme systems and consequent effects on nerve impulse transmission, membrane permeabilities, or the rates of synthesis or breakdown of materials. We lack detailed knowledge of many of these effects, and they are hard to quantify and use as a basis for interspecific or interorganism comparison. Consequently many of the stated 'measured effects' of pollutants on organisms are at a more general level, involving measurements of gross physio-

TABLE 2. RELATION BETWEEN WINTER SULPHUR DIOXIDE CONCENTRATIONS AND THE CORTICOLOUS LICHEN FLORA OF TREE TRUNKS WITH MODERATELY ACID BARK (FROM HAWKESWORTH & ROSE 1976)

zone	epiphyte flora	mean winter SO_2 $\mu g\ m^{-3}$
0	none	?
1	*Pleurococcus viridis* s.l. confined to the base	> 170
2	*Pleurococcus viridis* s.l. extends up the trunk: *Lecanora conizaeoides* confined to the bases	about 150
3	*Lecanora conizaeoides* extends up the trunk; *Lepraria incana* becomes frequent on the bases	about 125
4	*Hypogymnia physodes* and/or *Parmelia saxatilis* or *P. sulcata* appear on bases but do not extend up trunk. *Lecidea scalaris*, *Lecanora expallens* and *Chaenotheca ferruginea* often present	about 70
5	*Hypogymnia physodes* or *P. saxatilis* extends up the trunk to 2.5 m or more. *P. glabratula*, *P. subrudecta*, *Parmeliopsis ambigua* and *Lecanora chlarotera* appear. *Calicium viride*, *Lepraria candelaris* and *Pertusaria amara* may occur. *Ramalina farinacea* and *Evernia prunastri*, if present, confined to the bases: *Plantismatia glauca* may be present on horizontal branches	about 60
6	*P. caperata* present at least on base: rich in *Pertusaria* species (e.g. *P. albescens*, *P. hymenea*) and *Parmelia* (e.g. *P. revoluta*, except in NE, *P. tiliacea*, *P. exasperatula* (in N)). *Graphis elegans* appearing; *Pseudevernia furfuracea* and *Alectoria fuscescens* present in upland areas	about 50
7	*Parmelia caperata*, *P. revoluta* (except in NE), *P. tiliacea*, *P. exasperatula* (in N) extend up trunk: *Usnea subfloridana*, *Pertusaria hemisphaerica*, *Rinodina roboris* (in S) and *Arthonia impolita* (in E) appear	about 40
8	*Usnea ceratina*, *Parmelia perlata*, or *P. reticulata* (S and W) appear: *Rinodina roboris* extends up trunk (in S); *Normandina pulchella* and *U. rubiginea* (in S) usually present	about 35
9	*Lobaria pulmonaria*, *L. amplissima*, *Pachyphiale cornae*, *Dimerella lutea* or *Usnea florida* present: if these absent, crustose flora well developed, often with more than 25 species on larger, well lit trees	under 30
10	*L. amplissima*, *L. scrobiculata*, *Sticta limbata*, *Pannaria* spp., *Usnea articulata*, *U. filipendula*, or *Teloschistes flavicans* present to locally abundant.	'pure'

A different series of indicators has been defined for trees with basic or nutrient-enriched bark (see Hawkesworth & Rose 1976).

TABLE 3. DRY MASS (grams) PER POT OF RYE GRASS (*LOLIUM PERENNE*) FROM AN AREA WITH A LONG HISTORY OF AIR POLLUTION (HELMSHORE) AND A COASTAL AREA OF LOW POLLUTION (FYLDE), AT FIRST CUT FOLLOWING THE YEAR OF ESTABLISHMENT (FROM BELL & MUDD 1976)

	locality of experiment	
locality of origin	Helmshore	Fylde
Helmshore	83.9	55.1
Fylde	68.3	105.9

logical malfunction (like diminished growth rates in plants or reduced reproductive success in birds). But these physiological effects are the resultant of many biochemical interactions, and because organisms possess a capacity to break down, eliminate or compensate for the effects of many toxic materials, there may be no manifest change at the whole organism level despite appreciable biochemical impact. For example, it is well known that the process of haem synthesis in mammals is interfered with by lead, which blocks the enzyme delta-aminolaevulinic acid dehydratase, and that this effect is measurable in man at concentrations of lead in blood above about 20 μg/100 ml, but although such concentrations are not infrequently attained within what are considered normal limits, there is no evidence of grosser impairment at the whole organism level. Similarly, even when growth rates, reproductive rates or longevity are diminished, there may be no effect on the overall population size in a target species because species possess an excess reproductive capacity. The British heron (*Ardea cinerea*) population demonstrated this in the early 1960s, when high organochlorine pesticide residue levels were associated with reduced eggshell thicknesses, causing higher breakage rates in the nest, yet although there was reduced chick survival and at the same time enhanced adult mortality, enough young were still reared to keep total numbers stable. Finally, ecosystems as a whole have a certain resilience and even when pollutant stresses reduce the abundance of some component species there may be little effect on overall appearance. In areas with mean winter sulphur dioxide levels above 170 μg m^{-3}, woodlands are likely to lack sensitive conifers and epiphytic lichens, and have only the alga *Pleurococcus viridis* on the tree trunks (Hawkesworth & Rose 1976), yet despite such impoverishment the most obvious features of the forest ecosystem would remain. Critical measurement of the nature and scale of each effect at each level in the system is thus needed if comparisons of pollutant damage are to be made.

Multiple and single impacts on targets

The classical experimental approach has been employed by many authors to investigate dose–effect relations for particular targets and air pollutants. Early work was reviewed by Webster (1967), and more recent studies on sulphur oxides by a D.O.E./N.E.R.C. Study Group (D.O.E. 1976).

This work has indicated, for example, that the ryegrass S 23, chosen as a sensitive target, can display marked yield reductions when fumigated with air containing over 50, and especially over 100, μg m^{-3}. The effect appears to be more marked in winter than in summer (Bell & Mudd 1976). When soil is deficient in sulphur, however, prolonged exposure to air containing about 50 μg SO_2 m^{-3} may favour growth, and there is increasing evidence that crops in some intensively managed areas of the English Midlands and Scottish borders would suffer sulphur deficiency in the absence of pollutant sulphur deposited from the air. There are numerous apparent inconsistencies between different experimental results, perhaps reflecting differences in technique and environmental conditions, and even the effects of other pollutants that were not strictly controlled (D.O.E. 1976). Experiments may have been conducted with closed rather than open stomata and with different rates of air movement, thus greatly modifying leaf boundary resistance and the dose received by the tissues (Mansfield 1976; Ashenden & Mansfield 1977). Extrapolation from these laboratory studies to the field, moreover, is often hampered by the use of unrealistically constant gas concentrations instead of attempting to reproduce the changes that occur over seasonal and diurnal cycles and under different weather conditions.

One way round this problem may be the use of pairs of 'open top' containers placed in the field and supplying ambient and filtered air respectively to matched crop samples: preliminary experiments have shown considerable differences in yield (Whittingham 1977).

Sulphur dioxide is rarely likely to be the sole phytotoxic pollutant present in the air. In the United States it and ozone often occur together at concentrations known to cause damage when studied separately in the laboratory. Additive, synergistic or neutralizing interactions

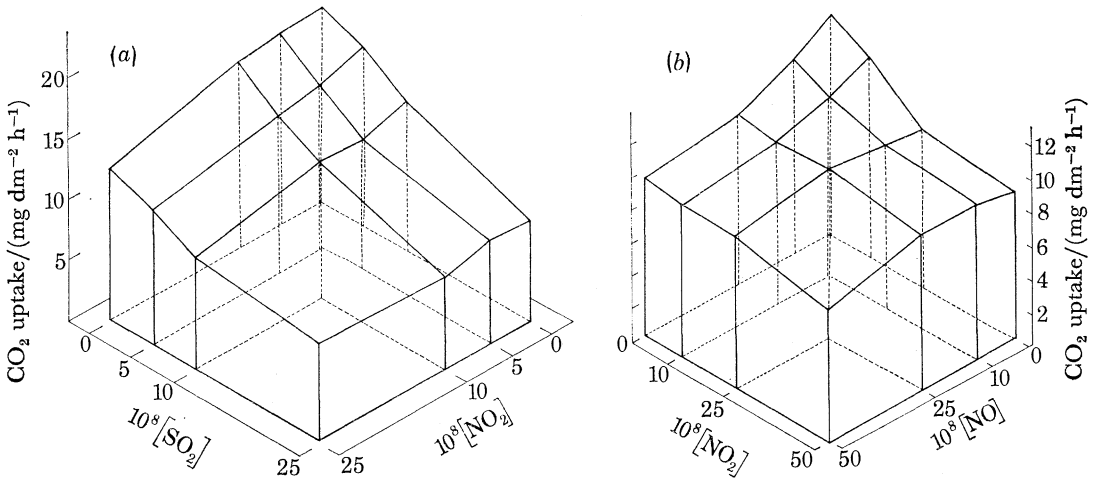

FIGURE 4. (*a*) Effects of SO_2 and NO_2 pollution on the rate of net photosynthesis in pea (*Pisum sativum*). Fumigation conditions: 1.20 air changes/min; temperature, 21 °C; light intensity, 73 J m^{-2} s^{-1}; water vapour pressure deficit 995 Pa. From Bull & Mansfield (1974). (*b*) Effects of NO and NO_2 pollution on the rate of net photosynthesis in tomato. From Capron & Mansfield (1977).

between these pollutants and others, including PAN and nitrogen oxides, consequently need to be considered. Mansfield (1974, 1977) showed that the effects of NO_2 can be additive to, or synergistic with, those of SO_2 and vice versa (figure 4*a*), and Capron & Mansfield (1976) have demonstrated a nearly additive response to NO and NO_2 (figure 4*b*). Bell (1974; also figure 3) pointed out that ozone has been shown to damage the most sensitive plants at concentrations well within the range known to occur in Britain in summer, and Menser & Heggestad (1966) demonstrated a synergistic interaction between ozone and SO_2, the two together damaging 25 and 38 % respectively of the leaves of two tobacco varieties at concentrations that were ineffective when the gases occurred singly. More recent work (J. N. B. Bell, personal communication) has indicated that depending on the target species, environmental conditions and the concentrations of SO_2 and O_3 it is possible to get a synergistic, additive or antagonistic action! Sulphur dioxide and hydrogen fluoride at high concentrations are reported as interacting additively by Saunders (1976), while ammonia and sulphur dioxide can react to neutralize one another, although the resulting ammonium sulphate haze can in special circumstances become a notorious local disamenity (e.g. as 'Teesside mist'). In case these examples appear unduly botanical, it may be worth recalling one suggestion of synergistic interaction of SO_2 and O_3 in man. Huzucha & Bates (1975) stated that healthy subjects exposed to 0.37 parts/10^6 of ozone for 2 h showed a 10 % reduction in mid-expiratory flow rate: 0.37 parts/10^6 of SO_2 had no effect over a similar period but a mixture of the two gases at the same concentration produced a more than 30 % reduction in flow rate. This effect needs further examination.

Another kind of multiple effect arises because a pollutant may affect both a target and one of its diseases or predators. Some fungi are among the plants particularly sensitive to sulphur dioxide, and rose mildew, rose black spot and maple leaf tar spot are well known to be reduced in urban areas. Oat and wheat rusts and barley powdery mildew are also sensitive to low pollutant concentrations (Bell 1976). There are also reports of pest infestations of forests after air pollution damage to foliage. Differential effects of pesticides on insect herbivores and the predators that feed on them, sometimes leading to increases in pest populations after spraying, are well known (see, for example, Moriarty 1976).

Targets are affected by many environmental variables which can alter their susceptibility to pollutants. The extent to which plants are damaged by phytotoxic gases naturally depends on exposure, which is affected by wind speed and by whether stomata are open or shut. In many plants, stomata open in the presence of SO_2, but behaviour is influenced by the water relations of the plant and there appears to be a critical relative humidity below which closure occurs. In the field bean *Phaseolus vulgaris*, radish and maize this is around 40 % while in tobacco and sunflower the critical value is nearer 50 %. Pollutants can also affect the tolerance by organisms of other variables: for example the capacity of trout to withstand cold autumn temperatures in New Brunswick was shown by Anderson (1971) to be reduced by low concentrations of DDT derived from airborne spraying of forests. Considering the complexity of the possible interactions and the inconsistency of the results obtained so far, the main conclusion to be drawn is that our understanding of pollutant interactions and the factors predisposing to sensitivity or resistance is far from adequate.

These criticisms apply especially to knowledge of the indirect affects of atmospheric pollutants. The most widely argued of these is the possibility that sulphur dioxide, oxidized and deposited as dilute sulphuric acid in rain, may be responsible for observed increases in the acidity of soils and freshwaters in some areas, with consequential changes in forest productivity and aquatic ecosystems (e.g. Sweden 1972). Research has established that long range transport of SO_2 can occur (O.E.C.D. 1977; D.O.E. 1976) and lead to wet and dry deposition at sites remote from sources, but the environmental effects of such deposition are very uncertain and there is no confirmation of any change in forest growth. Obviously, the relative contributions of local and distant sources of sulphate and hydrogen ions need to be analysed, as must the contributions to the acidification of fresh water from inorganic acids in rain, sulphate in snow melt, and humic and fulvic acids from soils. The effects of acid precipitation on soils likewise need to be compared with those of humic and fulvic acids derived from litter decomposition. Land use changes in catchment areas, climatic changes, and natural ecological trends and cycles all need to be evaluated before such indirect effects are likely to be seen in proportion.

Conclusion: the need for an integrated approach

Living targets integrate the influence upon them of many variables, and the effects of pollutants are modified by genetic, physiological and environmental factors. Three kinds of research are needed in order to unravel this complex of interactions: epidemiological studies documenting variations in target performance (and hence damage), controlled experiments to explore particular effects and mechanisms of interaction (and hence relate dose to effect) and modelling, to provide a framework for interpreting the system as a whole and guiding selection of the interactions that most require detailed study.

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Some epidemiological studies have been undertaken, the most complete being that by Hawkesworth & Rose (1976) (see also Ferry *et al.* (1973) on corticolous lichens). The zones defined in table 2 can be used to predict air pollution (figure 5) and the resulting pattern correlates fairly closely with sulphur dioxide concentrations (Warren Spring 1972) but it is above all a map of industrialization. Isercutant & Margot (quoted by Ramade 1977) proposed an *Index de pureté atmospherique* (I.P.A.) on the formula:

$$\text{I.P.A.} = \tfrac{1}{100} n \left(\sum_{1}^{n} Qf \right),$$

where n is the number of lichen species present at a locality, f the frequency of each species, and Q the index of pollution tolerance for each species. A map produced by using this index is also shown in figure 5.

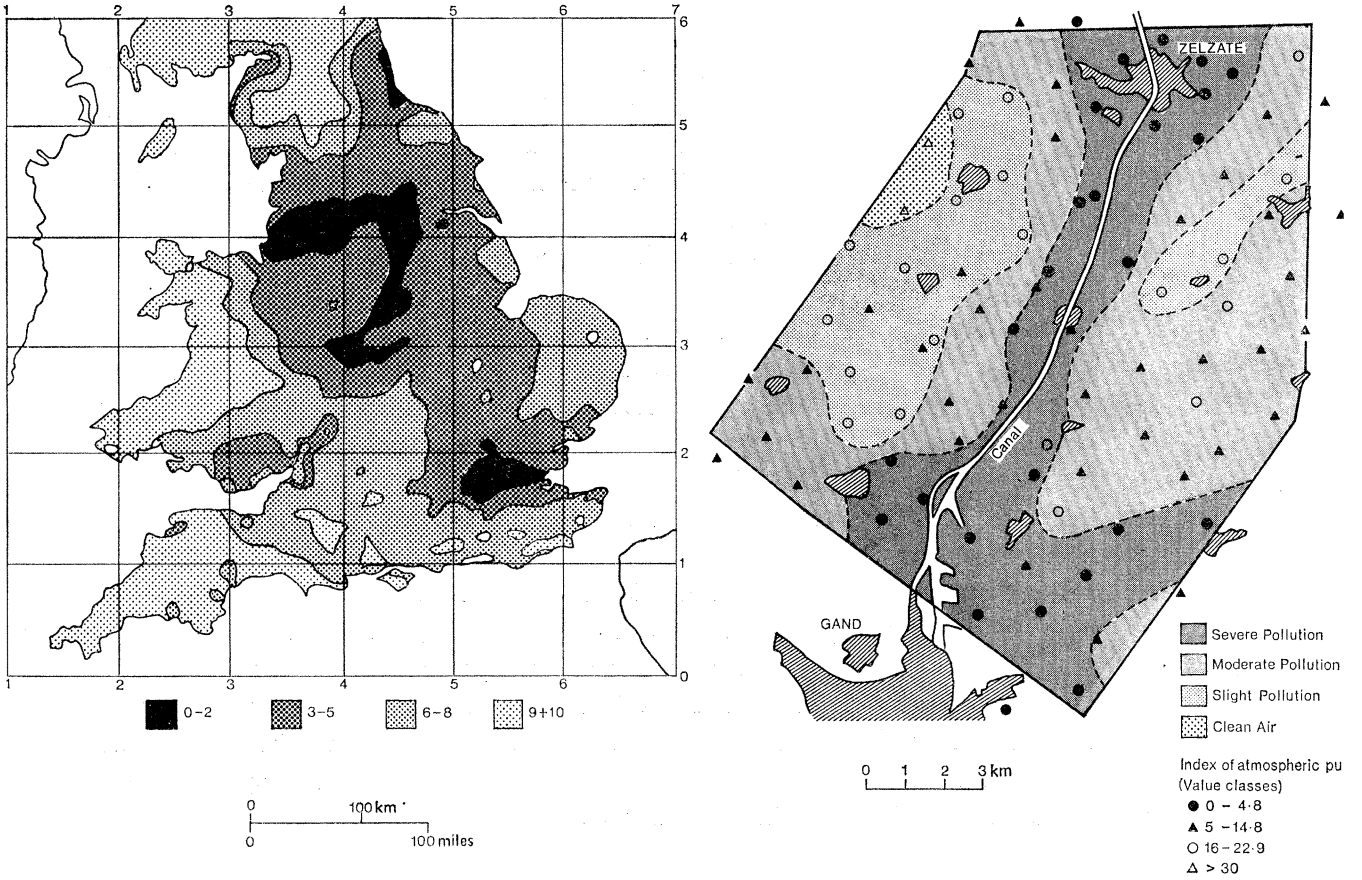

FIGURE 5. Mapping of zones of intensity of air pollution with the use of lichens as indicators. The pattern over England and Wales (left) was derived by using the criteria set out in table 2, and a comparable series for basic or nutrient-enriched bark (Hawkesworth & Rose 1976). The pattern in the map on the right is for the area north of Gand in Belgium, by using the I.P.A. index of Isercutant & Margot (cited by Ramade 1977).

There are fewer comparative records of the performance of sensitive tobacco strains or S 23 ryegrass or other crop plants over a wide area. High damage functions have been revealed in some areas, as in South East Lancashire where Bleasdale (1959) estimated that in the 1950s air pollution could be blamed for lost argicultural production worth £2.6×10^6 p.a. The lack of

such epidemiological information about crop damage over the whole country may be surprising when it is recalled that the Programmes Analysis Unit (1972) provisionally estimated possible total damage costs in the region of $£40 \times 10^6$ p.a., but may be explained in part by the practical response of farmers in not growing the most sensitive varieties in polluted areas, and by the natural development there of tolerance in pasture species. The most helpful approach may lie in the use of 'open top' containers (Whittingham 1977) to compare growth of normal crops on soil prepared by using normal husbandry methods, in filtered and ambient air. In contrast, epidemiological research on man, leading to correlations like that between bronchitic illness and air pollution (figure 6) is well established and worth extending to other relations between pollution and mortality/morbidity records. We have far less information of this kind about other animal targets.

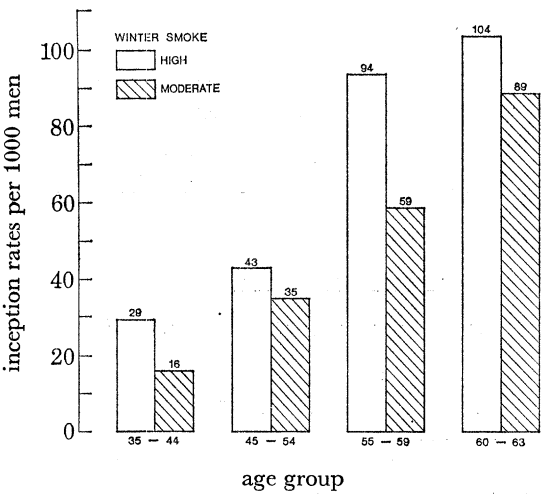

FIGURE 6. Sickness rates from bronchitis in areas in London with different pollutant levels. From Royal College of Physicians (1970), based on data from Ministry of Pensions and National Insurance Morbidity Survey.

Epidemiological studies demand establishment of pollutant concentrations and target performance with the use of sampling that permits rigorous correlation: they therefore make demands on a monitoring programme. Very often the programme proves inadequate. For example, the pattern of impaired lichen performance in figure 5 conforms with that of smoke and SO_2 pollution – but these are the only phytotoxic air pollutants extensively monitored in Britain today. It is now evident that PAN, NO_x and ozone also occur in British air, in concentrations capable of causing damage to sensitive crop plants and trees, acting in combination as well as singly, and they might also contribute to the pattern of damage, but without monitoring such relations can only be a matter of speculation.

Epidemiological surveys can only suggest relations: to define causative mechanisms experiments are essential. Classical experiments, examining dose–effect relations for one pollutant and target at a time, are necessary but are not easy to extrapolate to the field, where so many variables interact. This paper has demonstrated that laboratory research is picking its way slowly through these complex interactions, but if target performance surveys, pollutant monitoring, and experimental research are to be brought together in a synthesis, models will be required. So far, modelling has been developed for the dispersion and transformation of atmospheric pollutants, but not their impact on targets. Input–output models can be extended

to the point where they define dose commitment, and it is important that this be done for all the pathways by which a pollutant reaches a target since some (lead is a good example) follow several routes and it is the total dose from all sources that matters. But models describing harm commitment need to relate pollutant dose to other environmental factors modifying target response, and allow examinination of their relative significance. It may well prove, if this is done, that air pollutants are much less significant than climate, soil, or husbandry techniques in affecting the performance of most plant targets, and that outside the most polluted urban zones they are of little concern for human health. But these hypotheses may equally be wrong, and until models allowing the exploration of the properties of pollutant–environment–target systems are built, and laboratory experiments structured to define the key component mechanisms, this field is likely to remain speculative, and one in which laboratory research is hard to relate to the real world.

Much of the information in this paper comes from contributions to the Study Group made in advance of publication. I am grateful especially to Dr T. A. Mansfield and Dr J. N. B. Bell for permission to refer to work that is still incomplete. I am also indebted to Professor F. T. Last, Dr P. J. W. Saunders and Dr W. S. Clough for their kindness in commenting on the text during preparation.

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Discussion

R. S. Scorer (*Department of Mathematics, Imperial College of Science and Technology, London SW7 2BZ, U.K.*). It is one thing, in the early stages of an industrial society, to wait for pollution to make its harmful effects apparent before restricting an industrial activity, but clearly we cannot continue that way with industry operating on the present scale. But it is quite another thing to require any theoretical hazard, however far-fetched, to be taken into account just because it has not been proved to be of no consequence. Are we not seriously in danger of this latter course? People are easily scared and seem to equate a danger which is imagined and described as a 'potential threat' as real. How do we decide whether the cry of a group that a threat is real must be taken seriously? Surely not on the basis that we must have a proof that it is not real that will convince everyone?

M. W. Holdgate. I agree that there are two philosophical starting points. One often adopted by governments, industries and others with a large stake in things as they are, assumes that the present is satisfactory until proved otherwise: changes in pollution control should depend on the demonstration of unacceptable damage or risk under present conditions. The other argues that in our present state of ignorance of environmental systems we should be cautious, never allowing air or water quality to deteriorate, prohibiting release of new substances until they have been tested exhaustively, and taking all possible opportunities to reduce pollution. In practice most Governments and agencies pursue a middle road. New chemicals are being subjected to increasingly stringent scrutiny in Europe and North America. In Britain, H.M. Alkali and Clean Air Inspectorate revise 'best practicable means' when new technology allows more effective pollution control at acceptable cost and under acceptable operating conditions. Water Authorities pursue a general policy of not allowing water quality to decline,

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and impose more stringent conditions on discharges when environmental needs require this or better technology permits. Pollution is, let us recognize, generally defined in damage terms. The U.N. and International Conventions state that it is 'the release by man to the environment of matter or energy liable to cause hazard to human health, damage to living resources or ecological systems, harm to amenity, or interference with legitimate uses of the environment'. There will, of course, be value judgements about the acceptability of such hazard or damage, but the fact remains that pollution is defined, and reacted to, in terms of its effects.

R. S. Scorer. In thinking about damage to the environment, which we take for granted (e.g. the air we breathe, but there are many other examples), the magnitude of the part of the environment in question is of basic importance. We can afford to put small areas of land out of action for ever, but there must be 'a big wide world out there' somewhere if the regeneration processes are to be relied upon. Consequently it is the magnitude of the human population concentrated in an area that is crucial. A city can be as large as you like if it is serviced by an appropriate area that is not built up, and this applies to each of food, mineral resources, waste disposal, etc. Do we not therefore have to develop a greater consciousness that the sheer magnitude of our population and the tonnage of its turnover have become the major cause of concern already in some areas and will soon do so in many others?

M. W. Holdgate. I do not agree with Professor Scorer that there is a direct, inevitable, relation between population and pollution. Most developed countries have experienced population growth over the past century, yet their urban air and rivers are cleaner now than they were a few decades ago. It is not the number of people that matters but the nature of their activities and the amount of money and effort they decide – and can afford – to devote to their environment. It is true that as population densities and industrialization increase we can less and less count on the dilution capacity of the environment as a 'free good' and may have to purify emissions more and more to sustain the same environmental quality. Despite some worries about relying on them too uncritically, technological solutions have so far allowed such improvement at acceptable cost. (For a fuller discussion see M. W. Holdgate (1973) in *Population and the quality of life in Britain*, London: Royal Society of Arts.)

C. P. Whittingham (*Rothamsted Experimental Station, Harpenden, Herts., U.K.*). The words 'synergistic' and 'additive' are meaningful in relation to rate measurements concerning single reaction steps. I would suggest, however, that when the parameter measured, e.g. growth, is the result of the interaction of a series of processes, each of which are inhibited by a specific pollutant to a different extent, then the use of these general terms could be misleading.

M. W. Holdgate. This is an important point. But if we are to use these terms in the strict sense Dr Whittingham suggests we may need to find new terms to apply to the interactive effects of pollutants on multi-step processes. The literature certainly mixes these two things up. In the same way, the capacity of pollutants to interact with one another and with other variables and to affect different steps in a complex process makes the concepts of dose commitment and harm commitment hard to apply unambiguously.

Elizabeth I. Young (*Fair Acres, Tydcombe Road, Warlingham, Surrey, U.K.*). In May 1976 I gave Dr Holdgate a paper reporting my research into the apparent effects of television transmission and reception during daylight on world climate. I now have a good deal of further evidence,

although it is difficult to put into strict statistical form. Has Dr Holdgate been able to take any action on this matter?

M. W. Holdgate. I appreciate Mrs Young's concern, but know of no atmospheric or climatic changes that can be related to the modern increase in the use of radio waves for telecommunication: nor am I aware of any mechanism by which such an effect could come about. Indeed, as far as I know it is the properties of the ionosphere that control the telecommunication, not the other way about. Others here are, however, more qualified than I to reply.

Concluding remarks

M. W. Holdgate. The basic problem addressed in my paper is how to measure the effect of pollution quantitatively. There are so many potential targets and interactions that we cannot possibly measure them all: nor should we. Although in theory everything may be connected to everything else, it does not follow that it is good science to put equal emphasis on all these interconnections. Selection is vital, and I think there are some rational starting points. I suggest that we concentrate, at least initially, on:

(*a*) manifest effects on targets we know to be of significance (people, livestock, crops and components we have reason to believe important in environmental systems), seeking to define the scale of the effect spatially and temporally and then search for cause;

(*b*) mechanisms which we have observational or theoretical grounds for believing important, again defining the scale on which they operate, the controlling factors, and the targets involved.

Models will be needed in both fields. These need not be massive ecosystem models, but descriptions in strict mathematical language of the interactions we are concerned with, as a test of understanding, a guide to gaps in knowledge, and a means of probing relative significances of components.

Our aim, in the end, is risk assessment. We shall never eliminate risk, but we should try to define more precisely the risks we are running, or asking others to run. We can decide, in the end, to accept the risks, but we ought to know what we are doing.

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