Air Pollution: An Important Issue in Plant Health

And now worse visions of a viler age Loom through the darkness of the future's night

A sickening fog of smoke from British coal Drops in a grimy pall upon the land Befouls the vernal green and chokes to death Each lovely shoot, drifts in low poisoned cloud.

Henrik Ibsen, Brand, Act V

Many people worry about the human health hazards of air pollution. But another aspect of this problem receives less attention: Air pollutants injure many plant species. Air pollution stress can alter plant growth, productivity, and quality. Such effects are often costly.

Generally, air pollution is considered to be a problem in industrialized and urbanized nations. People think of the significant impacts of photochemical smog on the forest ecosystem in the San Bernardino Mountains of southern California or the impacts of industrial emissions in the Ruhr Valley of West Germany. At present, however, air pollution is clearly a global problem. In many developing nations, photochemical smog and sulfur pollutants are of major concern. In Mexico, for example, pine forests in regions downwind from Monterrey and Mexico City show symptoms of ozone-type injury. Similarly, sulfur dioxide appears to be a problem in the vicinity of certain point sources in

Air pollutants occur in several different physical and chemical forms (Table 1). Some air pollutants, such as sulfur dioxide (SO₂) and hydrogen fluoride (HF), are produced as phytotoxic compounds directly from a source (Table 2). Others, such as ozone (O₃) and

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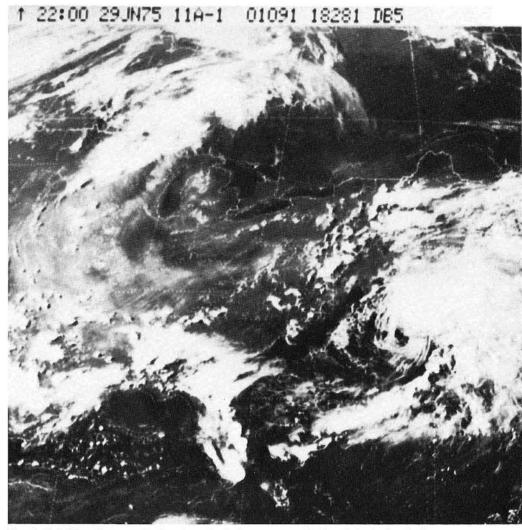


Fig. 1. Satellite photograph showing clockwise movement of pollutant clouds from northeast through central United States into eastern half of Minnesota on 29 June 1975.





Fig. 2. (Left) Contours showing number of miles of visibility (1 mile = 1.61 km) at various locations in eastern and central United States at noon on 29 June 1975. In Minnesota, visibility ranged from 4 to 8 miles (6.4–9.6 km). In the clear area to the northwest, visibility was > 15 miles (> 18 km). Fine particulate sulfates in the air caused the reduction in visibility. (Right) Contours showing maximum hourly ozone concentrations in eastern and central United States on 29 June 1975. In eastern half of Minnesota, maximum hourly ozone concentrations were > 70 ppb (10 ppb = 19.6 $\mu \rm g/m^3$). During this episode, long-range air pollutant transport brought ozone and fine particulate sulfates together into Minnesota; 2 days later, sensitive plant species showed symptoms of ozone injury.

Table 1. Physical forms of air pollutants, with chemical examples

| Physical forms | Chemical examples | | |
|---|--|--|--|
| Gases | Ozone, sulfur dioxide, hydrogen fluoride, oxides of nitrogen, nitric acid, ethylene | | |
| Fine particles ($< 3.0 \mu m \text{ diam}$) | Sulfuric acid, ammonium sulfate, some organics | | |
| Coarse particles (> 3.0 µm diam) | 240 10 00 10 10 10 10 10 | | |
| Dry | Natural and man-made dust, pollen, spores, highway and sea salt, some organics | | |
| Wet | Pollutants in rain and snow | | |

peroxyacetyl nitrate (PAN), are produced secondarily in the atmosphere through chemical reactions involving primary pollutants, such as nitrogen dioxide (NO₂) and hydrocarbons (HC), in the presence of sunlight. Such photochemical air pollutants reach peak concentrations during periods of high solar radiation (1200–1700 hours).

No Boundary Lines

Air pollutants are produced by line sources (highways), single-event point sources (accidental spills and leaks), continuous point sources (smokestacks), area sources (urban centers), and regional sources (eg, Ohio Valley). Air pollutants are also naturally produced by biological processes, volcanic eruptions, etc. Pollutants such as SO₂ produced by a smokestack are rapidly deposited to the ground in the vicinity of the source in elevated concentrations during an atmospheric inversion. More commonly, however, the smoke plume can travel up to 100 km or more downwind. Under these conditions, SO2 is converted to fine sulfate (SO₄) particles that remain in the air for days to weeks and travel hundreds to thousands of kilometers before being deposited to the ground. These fine particles scatter light and reduce visibility (11). From the measurements of visibility and ambient sulfate concentrations

Table 2. Effects of air pollutants on plants

| Air pollutants | Sources | Some sensitive plants | General symptoms of acute injury | |
|--------------------------------|--|--|---|--|
| Ozone | Chemical reactions in atmosphere involving sunlight; storm centers; other natural occurrences in upper atmosphere | Ash, bean, carnation, eastern white pine, lilac, petunia, potato, quaking aspen, radish, tobacco | Pigmented or unpigmented spots or bleaching on upper leaf surface; browning and death of conifer needles starting at tip | |
| Sulfur dioxide | Combustion of fuel; petroleum and natural gas industries; ore smelting and refining processes | Alfalfa, aster, bean, birch, oats, soybean, sunflower, wheat | Death of leaf tissue between veins; death of conifer needles starting at tip | |
| Hydrogen fluoride | Brick plants; refineries; aluminum industries; manufacturers of steel and phosphate fertilizers | Blueberry, corn, gladiolus, Scotch pine, tulip | Death of leaf tissue at tip and margins; browning and death of conifer needles | |
| Peroxyacetyl nitrate | Photochemical reactions in atmosphere | Bean, dahlia, oats, petunia, tomato | Silvering, glazing, or browning of lower leaf surface | |
| Oxides of nitrogen | Exhaust gases of trucks and autos; combustion of natural gas, fuel oil, and coal; refining of petroleum; incineration of organic wastes | To nitrogen dioxide: bean, lettuce, muskmelon, sunflower, tobacco | By nitrogen dioxide: White, tan, or brown dead areas between veins; waxy coating on leaf surface | |
| Particulates* | Cement mills; lime kilns; incinerators; combustion of coal, gasoline, and fuel oil | No specific ones identified | Visible coating; encrustation; marginal burn | |
| Ethylene ^a | Motor vehicles; refuse burning; combustion of coal and oil; leaky natural gas heaters; natural occurrences | Carnation, cream pea, cucumber, Easter lily, orchid, rose, tomato | Yellowing and dropping of leaves; premature leaf drop; failure of flower buds to open; stimulation of lateral growth | |
| Ammonia ^a | Leaks or breakdowns in industrial operations; spillage of anhydrous ammonia | Beet, sunflower, tomato | Cooked green appearance; bleaching and dead spots along margins; yellowing of leaves (injury may be similar to that by sulfur dioxide) | |
| Chlorine and | | | | |
| hydrogen chloride ^a | Refineries; glass industries; scrap burning; accidental spills | Coleus, corn, radish, sugar maple, tomato, tulip, white pine | Dead spots along margins of outer leaves; bleaching of leaves (injury may be similar to that by sulfur dioxide) | |

Minor pollutants.

during satellite and aircraft flights, it is clear that air pollution is a regional and an interregional problem in the United States (Fig. 1) (6).

Generally, O₃ and SO₄ are transported together downwind from the urban centers in the United States (Fig. 2). Meteorological models make possible determinations of the path of movement and the arrival time of a given pollutant-containing air mass in a geographic location of interest (Fig. 3). Such information is vital in identifying the source regions of pollution, climatological conditions, and geographic paths that favor pollutant movement and vegetation impacts at sites far removed from the sources.

How Plants Respond

Since 1872 (9), scientists have examined the effects of air pollutants on plants. Air pollutants affect plants through direct deposition on the foliage and on the soil, with subsequent uptake through the roots. Under proper environmental conditions, exposure of plants to high pollutant concentrations for a few to several hours will result in rapid visible injury. Many gaseous air pollutants tend to produce their own patterns and records of injury on plants (Table 2). The nature and extent of such injury patterns are governed by genetic, physiological, and environmental factors and by the presence of other air pollutants.

Both native and cultivated plant species sensitive to specific air pollutants can be used as biological indicators to monitor or warn against air quality deterioration and its potential impacts on vegetation (5). This approach has been used by scientists in many parts of the world, including the Netherlands, Germany, Canada, the United States, and Mexico. Standardized soil and cultural practices are used in these studies. Injury is estimated visually and appropriate growth parameters are measured in evaluating the pollution impact.

In the United States, O₃ is considered to be the most important phytotoxic air pollutant. Like other gaseous air pollutants, it enters the leaf through stomata. It primarily affects the mesophyll cells in the leaf tissue. Symptoms of O₃ injury on broad-leafed plants include chlorosis, flecking, bleaching, and stippling and, with severe injury, necrosis on both upper and lower leaf surfaces. Symptoms are usually restricted to the upper leaf surface (Fig. 4). Mottling, bands of green and yellow areas, and tip necrosis spreading downward are the symptoms on conifer needles (2).

With coal combustion and metal and oil processing, SO₂ will continue to be an important air pollutant in North America, Europe, and many developing nations. SO₂ injury on broad-leafed

plants consists of interveinal chlorosis or necrosis, whereas on conifer needles, tip necrosis spreads downward (Fig. 5).

As opposed to these short-term effects, exposure of plants to low pollutant concentrations for prolonged periods



Fig. 3. Meteorological model showing movement of pollutant-containing air parcel across central United States into Minnesota on 29 June 1975. Air parcel's path was determined with weather-sounding data collected 600 m above the ground at various locations in the United States. Arrows indicate air parcel location at 12-hour intervals, starting at 0.00 hour Greenwich mean time (GMT).



Fig. 4. Flecks and necrosis on upper leaf surface of soybean denote acute ozone injury.

(growth season) with periodic short-term peaks (eg, 0.5 to 1 or 2 hours) can result in chronic injury. This is generally manifested





Fig. 5. Symptoms of acute sulfur dioxide injury: (A) Interveinal necrosis on raspberry. (B) Necrosis of Scotch pine needles spreading downward from tips. (Courtesy D. B. Drummond)

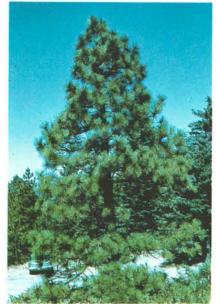




Fig. 6. Ponderosa pine in San Bernardino Mountains of southern California: (Left) in 1961, healthy. (Right) in 1970, after being exposed to photochemical smog for 10 years. (Courtesy P. R. Miller)

by chlorosis, premature senescence, leaf drop, and loss of vigor (growth, productivity, regeneration, etc.). Figure 6 shows a ponderosa pine in the San Bernardino Mountains in 1961 and 1970, respectively. Over the 10-year period the tree was chronically exposed to photochemical smog. Loss in vigor can also occur without these classic symptoms.

Variations in plant response to air pollutants occur within and between

populations. Based on results of laboratory and field studies, several investigators have grouped plants at generic, species, and cultivar levels as relatively sensitive, intermediate, or tolerant to a given air pollutant. For example, soybean cultivars Cursoy and Vickery are relatively more sensitive to O₃ in short-term exposures than are Swift and Evans, whereas Hodgson and Hodgson 78 are intermediate. Such groupings, however, depend on the pollution exposure regimes used and the responses evaluated. It is important in these studies that factors reflecting economic and aesthetic considerations be examined.

Because ambient air contains pollutant mixtures, several investigators have studied the joint effects of two or more pollutants. Plant response to pollutant mixtures can be less than, equal to, or greater than the sum of the response to the individual pollutants in the mixture. These effects, again, depend on the concentration ratio of pollutant combinations used and the criteria examined (8). For example, soybean exposed to 128 μ g/m³ of O₃ for 2 hours daily for five consecutive days showed generalized chlorosis. Plants exposed to $520 \mu g/m^3$ of SO₂ under a similar regime showed no symptoms but a significant accumulation of foliar sulfur. Plants exposed to the pollutant mixture showed a greater than additive visible effect (Fig. 7). These plants, however, did not accumulate foliar sulfur comparable to those exposed to SO₂ alone (7).

Pollutants Plus Parasites

The joint influences of air pollutants and parasites on plant growth and productivity are not well understood. Nevertheless, laboratory experiments and limited field observations show that air pollutants can alter the incidence and severity of parasitic diseases and, reciprocally, parasite infection can modify the plant response to pollutant stress (10). Air pollutants can alter plant-parasite interactions by: 1) influencing the parasite at certain stages of its life

cycle, 2) altering the host susceptibility, and 3) selectively reducing numbers of antagonistic but pollutant-sensitive microflora normally found on the leaf surface. Plant exposure to air pollutants can also alter root microflora.

The type of alterations observed in the plant-parasite interactions depends on the timing of the pollutant stress relative to the stage of pathosystem development. For example, exposure of bean plants to O₃ within 2 days after inoculation with Uromyces phaseoli resulted in a significant reduction in pustule size and number. On the other hand, no effect was observed when exposure to O3 occurred two or more days after the inoculation. Similarly, bean plants inoculated with tobacco mosaic virus and exposed to O₃ immediately or 48 hours after inoculation developed the same number of local lesions as plants grown in charcoalfiltered air. Bean plants exposed 3 or 24 hours after inoculation developed 19 and 16%, respectively, more local lesions than controls did.

Pollutant concentration and exposure duration also influence the pathosystem. Ambient SO₂ concentrations of 100 μg/m³ for 2 days markedly reduced the incidence of Diplocarpon leaf spot on rose, while lower concentrations tended to increase the parasite infection. The effect may vary depending on whether the disease is caused by an obligate or a facultative parasite. Exposure to O₃ decreased the incidence of rust and powdery mildew on wheat but increased the occurrence of Botrytis leaf spot on potato. Air pollution stress to the shoot system can also result in negative effects at the root system. Exposure of legumes to O₃ decreased root nodule size, number, and weight.

Several investigators have shown that parasite infection can modify plant response to air pollutants. Symptoms of O₃ injury generally do not appear in the immediate vicinity of local lesions caused by bacteria or viruses or around localized areas of fungal growth (Fig. 8). This effect can also be produced on soybean with methionine sulfoxamine, a substance that mimics tabtoxin. Thus, localized

Table 3. Ozone-induced loss in alfalfa biomass in selected counties of Minnesota during 1979 and 1980

| Location | County | Loss in 1979 | | Loss in 1980 | |
|---------------|----------|--------------|--------|--------------|---------|
| | | % | Tons | % | Tons |
| Northwest | Marshall | 2.2 | 1,532 | 5.0 | 1,715 |
| North central | Itasca | 0.0 | 0 | 1.5 | 311 |
| Northeast | Lake | 0.0 | 0 | 0.9 | 5 |
| West central | Traverse | 1.7 | 4.3 | 7.7 | 1,601 |
| Central | Wright | 0.0 | 0 | 9.3 | 14,404 |
| East central | Hennepin | 0.0 | 0 | 4.3 | 2,717 |
| Southwest | Nobels | 0.0 | 0 | 8.6 | 4,892 |
| South central | Freeborn | 0.0 | 0 | 7.2 | 2,941 |
| Southeast | Olmsted | 0.0 | 0 | 7.6 | 13,931 |
| State loss | | 0.5 | 35,097 | 7.3 | 415,570 |

suppression of the symptoms of O₃ injury appears to be initiated by nonspecific causal agents. Brennan and Leone (1) reported an interesting case where tobacco inoculated with tobacco mosaic virus and exposed to O₃ 12 days later failed to show symptoms of O₃ injury.

Impact on Crop Yield

Assessment of the amount of crop loss is an extremely important facet of the research on air pollutant impact on vegetation. In analyzing the effects of air pollutants on crop yield, two considerations appear to be critical: 1) Experimental exposures to high pollutant concentrations over the short term, while affecting yield, are less useful than chronic exposures with realistic concentrations lasting over the plant's entire life cycle, and 2) crop loss can occur even in the absence of symptoms of air pollution injury. Therefore, results of experiments where only the intensity of the pollutant stress (concentration/ exposure duration) and visible effects are measured are less useful than results of studies where stress intensity is related to yield reductions. Crop loss due to any stress factor should be viewed as a response surface, where the magnitude of loss to a given intensity of stress varies according to the crop development stage (3). In Minnesota, we are conducting a study to assess the economic impacts of air pollution injury on six crops: alfalfa, corn, oats, potato, soybean, and wheat. This study is unique in using empirically determined stress intensity/yield loss models for loss estimation over an entire region.

In general, the effect of any stress factor at one point in a crop's growth may be expressed in terms of a proportion of response (eg. reduced yield) compared with that of a control. The control may be the crop's response under conditions of zero stress or, in the case of ozone and sulfur dioxide, conditions with background concentrations.

Using this approach, we estimated losses in alfalfa caused by ozone during 1979 and 1980 on a county basis for the state of Minnesota (Table 3). The contribution of daily loss to total loss over the growth season was calculated, and the corrected loss figure was derived by multiplying the daily figure with a correction factor equivalent to the proportion of healthy crop left on a given day. This satisfies a biological requirement that tissue that has been injured does not contribute again to computation of injury at a later date. The contribution of each time interval to overall loss during a growing season varies according to the sensitivity of the crop during that time interval. This approach, where actual monitored pollutant concentrations are used for predicting loss using a model developed from a dose/yield loss experiment, offers a realistic means of

assessing regional crop losses. The model for each crop may also be modified in accordance with any new experimental data, allowing improvement in precision of loss estimation.

Some Critical Issues

The best approach to preventing or reducing air pollutant-induced damage to agriculture and forestry is to significantly control man-made pollutant emissions. But there are two major considerations: 1) With the existing technology, all pollutant emissions cannot be effectively controlled in all cases, and 2) air pollution abatement is expensive and technologically complex. Despite these problems, several nations with advanced industrial and economic status are attempting to reduce air pollutant emissions. Emissions reductions are not internationally uniform, however, and air pollutants are transported across regional and national boundaries. Thus, the effects of air quality on food and fiber production will continue to be important.

At present, there is little or no information on the response of indigenous plant species to air pollution in many developing nations. Additionally, the magnitude and significance of the impact are poorly understood in many of these situations. Plant pathologists and geneticists in many parts of the world routinely screen plants for resistance to biopathogens. Such organized efforts have not been considered with regard to air pollution stress. A basic understanding of plant response to pollutant stress under a wide range of environmental conditions that influence the stress and

the response is necessary in formulating environmental policy.

Numerous studies have been conducted to examine the impacts of emissions from a source (eg, SO₂) on vegetation in the vicinity (4). But few scientists have attempted to partition the specific contribution of a source to the overall multiple stresses to which the vegetation is subjected. Relating high pollutant concentrations to short-term impacts is comparatively easy. With chronic exposures to, for example, SO₂, however, sufficient effort has not been devoted to separating the contributions of atmospheric sulfur from those of soil sulfur in examining the impact. In such studies, chemical tracers



Fig. 7. Joint effects of ozone and sulfur dioxide on soybean: (Left) Unexposed plant. (Center) Plant exposed to ozone at 200 μ g/m³/2 hr/5 days shows generalized chlorosis of leaf. (Right) Plant simultaneously exposed to ozone at 200 μ g/m³/2 hr/5 days and sulfur dioxide at 1,050 μ g/m³/2 hr/5 days shows more than additive symptoms. Symptoms are not typical of those induced by either pollutant alone. Plants exposed to sulfur dioxide alone show no symptoms.

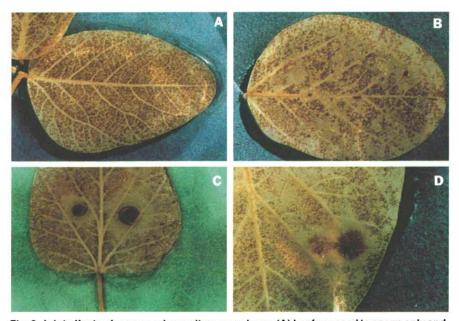


Fig. 8. Joint effects of ozone and parasites on soybean: (A) Leaf exposed to ozone only and with chlorophyll extracted by alcohol shows uniform stippling. (B) Leaf inoculated with *Pseudomonas glycinea*, then exposed to ozone, shows halo areas around bacterial infection sites that are free from ozone stippling. Sites of artificial inoculation with (C) methionine sulfoxamine, which mimics symptoms of wildfire toxin, or (D) *Microsphaera diffusa* (powdery mildew) show halos free from ozone stippling.

that clearly identify the contribution of the source to the stress should be used.

Some progress has been made in field evaluation of air pollutant-induced crop losses. Some critical issues remain: 1) We do not fully understand the long-term joint effects of parasites and air pollutants on host plants; 2) few studies are available where long-term plant responses have been evaluated as a function of pollutant mixtures with dynamic concentration fluxes to mimic ambient temporal and spatial variations; and 3) predictive crop loss assessment models have not been developed that incorporate continuous, measured pollutant concentrations as a function of the plant growth stage, using the total life cycle of the plant.

At present there is a great deal of concern about the increasing global carbon dioxide concentrations and the "greenhouse effect." Similarly, loading of fine particulate matter in our atmosphere is considered to alter temperature, quality, and quantity of light incidence on plants. These phenomena, together with the abundance of phytotoxic air pollutants, should be of significant concern in our approach to plant health.

What are the joint effects of these processes with other disease-causing agents on food production? As we approach the twenty-first century, industrialization will no doubt increase significantly. Thus, plant pathologists should strengthen their efforts in evaluating the role of environmental policies and air quality in plant health.

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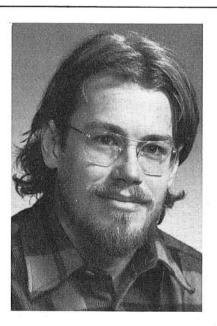
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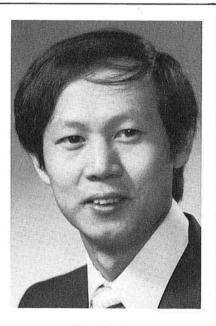
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