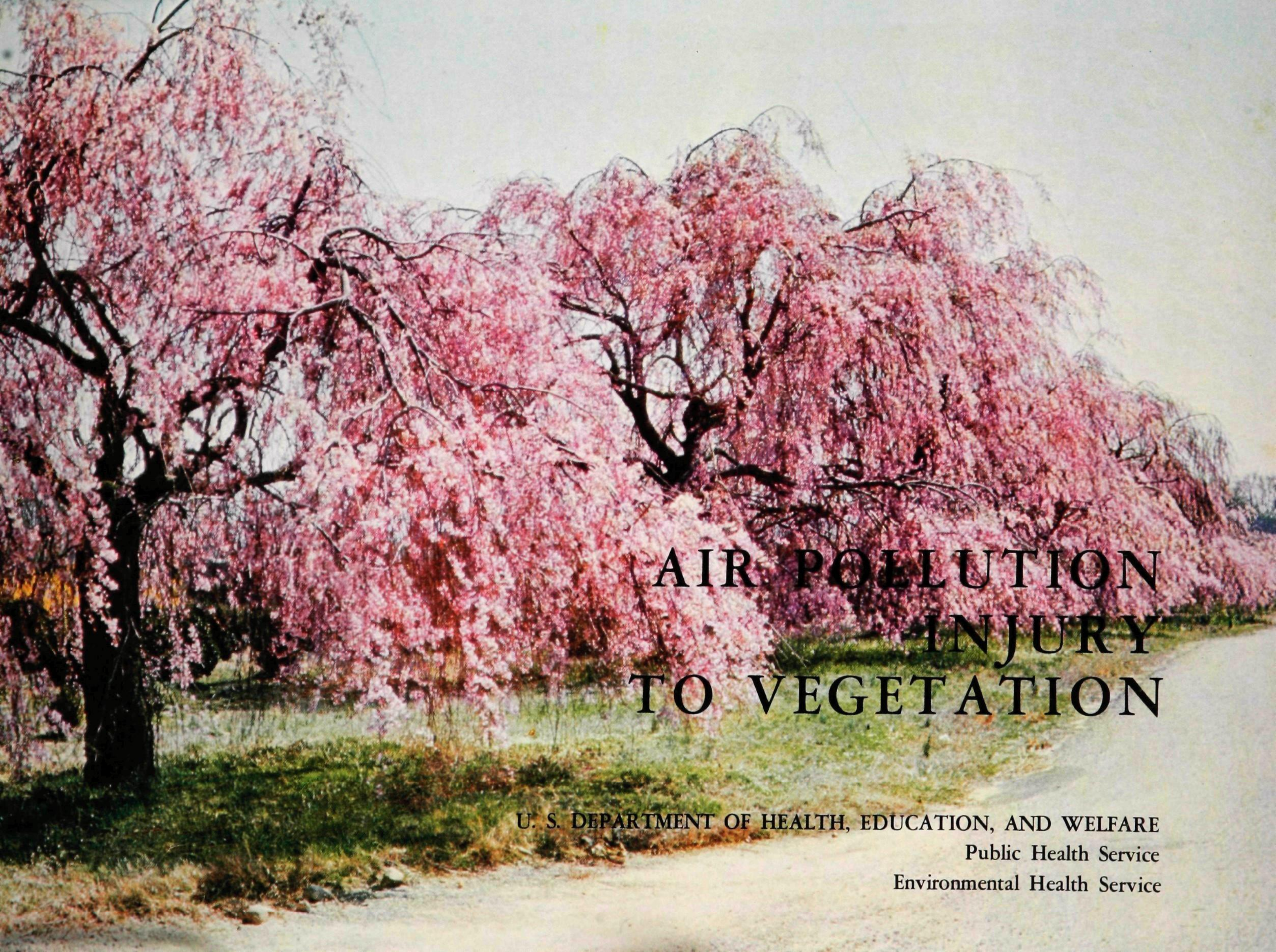

AIR POLLUTION INJURY TO VEGETATION

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Environmental Health Service

AIR POLLUTION INJURY TO VEGETATION

Ibrahim Joseph Hindawi

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
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National Air Pollution Control Administration Publication No. AP-71

TABLE OF CONTENTS

INTRODUCTION

Air pollution fouls the air in every community of more than 50,000 population in the United States as well as in a major proportion of smaller towns.[1] A number of natural variables play an important role in the formation, duration, and location of air pollution. Because of variation in wind direction and speed, air pollution respects no arbitrary boundaries. Pollutants may travel freely from their sources, cross city and state lines, and contaminate other communities many miles away. Meteorological and topographical conditions that cause stagnant air masses may cause a buildup of pollution at certain times and places.

In Los Angeles, pollution from automobile exhaust is the major problem. In London, it is the smoke and gases from high-sulfur-content coal. In New York and New Jersey, air pollution has been traced to petroleum refineries, automobile exhaust, power-generating facilities, waste disposal systems, and many other sources. In some cases the problem arises from a series of chemical reactions in the atmosphere involving by-products of combustion and other processes. A chemical reaction takes place, for instance, between hydrocarbons and nitrogen oxides, with sunlight acting as an energy source, to produce photochemical smog. Pollutants from these various sources injure crops, trees, and property and reduce the amount of sunlight reaching the earth's surface.

Since a threat to human health is implicit in air pollution, and since agricultural and other economic losses can be attributed to pollution, means must be found to detect and control atmospheric pollutants. The study of the effects of pollutants on vegetation is important not only because of the obvious relationship of such effects to agricultural production, but also because many plants can serve as indicators of the presence of pollutants. The usefulness of plants as indicators is based primarily on the sensitivity to pollutants of selected species or varieties.

For the past 20 years, scientists have been growing plants under controlled conditions and exposing them to various concentrations and combinations of certain chemicals. In this way they have been able to determine the relative effects of these chemicals on plant life. By now, characteristic injuries to certain plants can be interpreted not only to determine the presence but even the relative concentrations of aerial pollutants that cause injury. In some instances when analytical techniques and measuring instruments are inadequate, plant injury alone may be used to determine the presence of pollutants.

One variety of tobacco is used by many investigators as an effective ozone indicator. Pinto bean plants are used to detect peroxyacetyl nitrate (PAN); petunias, to investigate total oxidants; and gladioli, to determine fluoride accumulations. Dahlias, petunias, alfalfa, and cotton are excellent detectors of sulfur dioxide injury.

Actual leaf injury and its relationship to crop losses have been measured quantitatively. In fact, alfalfa and grain crop losses have been shown to be directly proportional to visible damage caused by sulfur dioxide.[2] Field vegetation surveys and plant damage reports have been used to estimate the presence, distribution, and general levels of pollution.[3]

STRUCTURE AND ACTIVITIES OF PLANTS

Air pollutants are divided into two major types, gases and particulates. Gaseous pollutants account for the most widespread injury to plant life. Gases known to damage vegetation include: ozone, PAN, nitrogen dioxide, sulfur dioxide, hydrogen fluoride, ethylene, and chlorine. These pollutants destroy plant chlorophyll, disrupt the photosynthesis process, and consequently reduce food production. In severely polluted areas, adjacent to certain industrial operations, plant life has been almost entirely exterminated.

An *interaction of even low concentrations of these gases can cause* injury that is not readily apparent. This injury may show up as growth suppression, dwarfing, or early maturation or as symptoms similar to those of nitrogen deficiency or virus infection.

The leaf is the primary indicator of the injurious effects of air pollution. Its structure plays an important role in building carbohydrates and other vital plant products and foods. Some of the basic anatomy, morphology, and physiology of the leaf are discussed on the following pages to facilitate understanding of leaf activity in relation to air pollution symptoms and injuries.

PLANT CELLS

Plants and animals alike are composed of tiny cells, which may be compared to the bricks or stones that make up the walls of a house. Cell sizes range from 0.01 to 0.1 millimeter in diameter. A typical plant cell (Figure 1) has three main components: the cell wall; the protoplast, which is the protoplasm of one cell; and the inclusions, which are composed of nonliving structures.

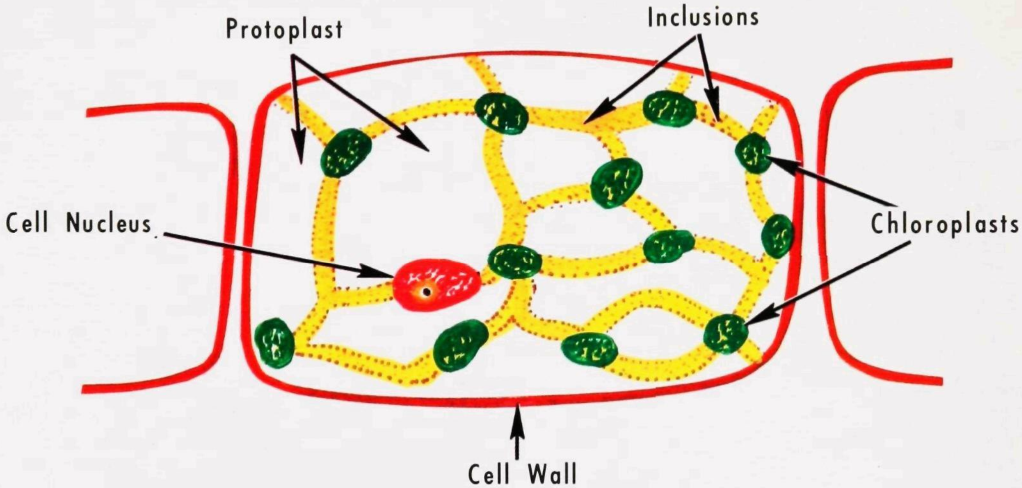


Figure 1. All or part of a cell may be injured by air pollutants.

Cell Walls

Cell walls are extremely thin when they are first formed, but they become relatively thick with age. The cell wall, produced by the protoplast that it encloses, is made up almost entirely of cellulose.

Protoplasm

Protoplasm, usually colorless, is a jelly-like substance that is found in all living plant and animal matter. It is considered living matter in its simplest form.

Protoplasm is composed of several chemical compounds. The most important is protein; others are fat and carbohydrate. The water content of the protoplasm ranges from 60 to 90 percent. The dense bit of protoplasm found in the center of the cell is the nucleus, and the protoplasm located outside the nucleus is the cytoplasm. Within the cytoplasm are three types of tiny bodies, or plastids, that perform vital functions in the life of a plant: chloroplasts, leucoplasts, and chromoplasts.

All green plant cells contain chloroplasts, oval microscopic bodies that contain the green pigment chlorophyll plus some yellow pigment. Chloroplasts are of enormous biological importance because they are key structures in the plant's food manufacturing process, photosynthesis.

Leucoplasts are colorless plastids that convert starch into starch grains. As these grains increase in size and number, the leucoplasts stretch to many times their normal size.

Chromoplasts are responsible for the red, yellow, or orange colors appearing in many flowers and fruits.

Inclusions

Inclusions are the nonliving structures of cells. They are seldom found in very young cells, but appear in increasing numbers as cells mature. They are particularly evident in old cells with large food-storage capacity. Inclusions may be crystals, noncrystals, or sacs containing liquid mixtures. These sacs, called vacuoles, contain mixtures of water and substances such as nitrogen oxides, carbon dioxide, inorganic salts, soluble protein, and simple nitrogen compounds.

ANATOMY OF THE LEAF

Plant structure is formed by the leaf, stem, and root. The leaf (Figure 2) is the principal organ involved in photosynthesis. Microscopic examination of a typical mature leaf reveals three primary tissue systems: the outer epidermis, the mesophyll, and the vascular bundle (veins).

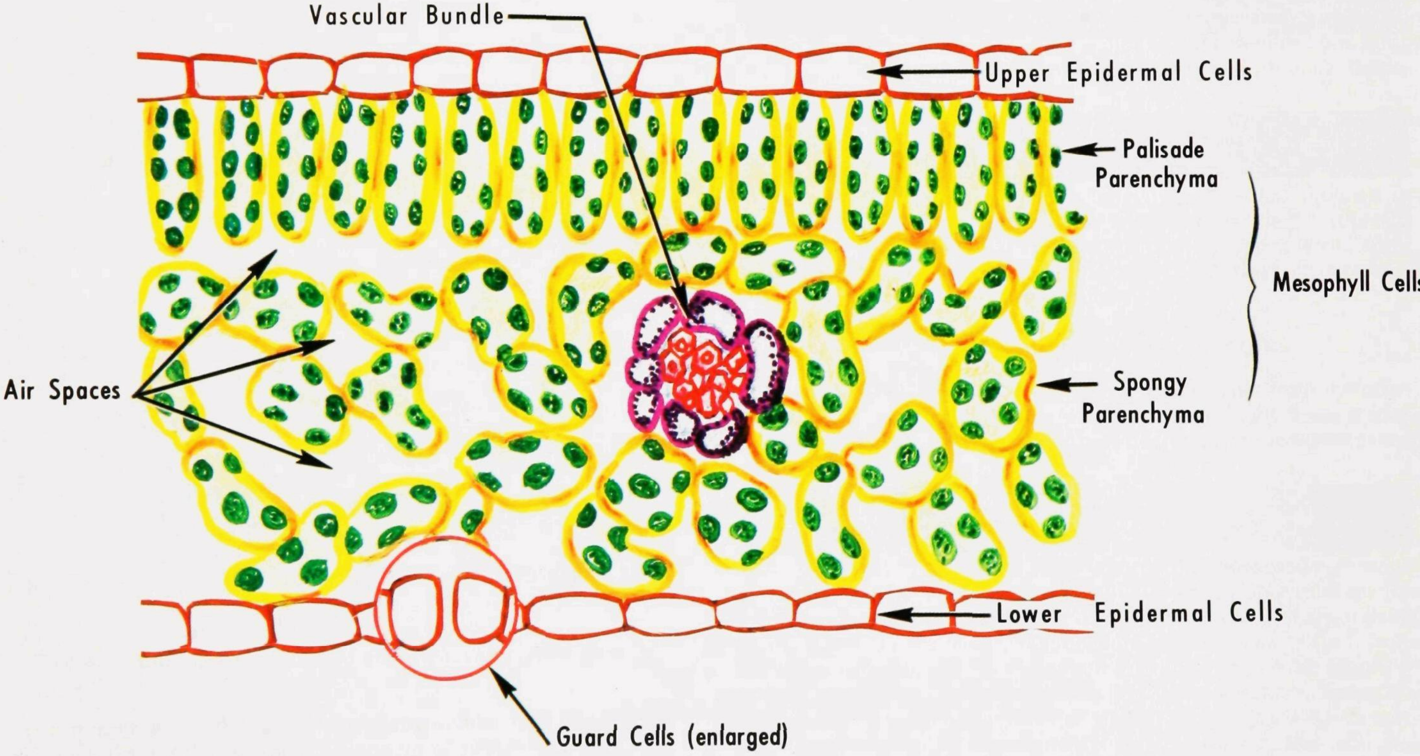


Figure 2. Cross section of intact leaf shows air spaces within leaf that serve as passages for pollutants that may subsequently injure leaf.

The epidermis covers the entire surface and protects the tissue within from injury caused by such things as excessive water loss and wind and rain. Epidermal cells are relatively long-lived, dying just before the leaf falls. Chloroplasts are usually absent from epidermal cells. Oval openings in the leaf epidermis are called stomata. Bounded by two special cells called guard cells (Figures 3 and 4), stomata are found among ordinary epidermal cells on the lower side of a leaf and occasionally on the upper epidermis. These openings regulate the entrance and exit of gases. The function of the guard cells in opening and closing stomata depends upon change in the turgor (normal distention and resiliency) of the cell.

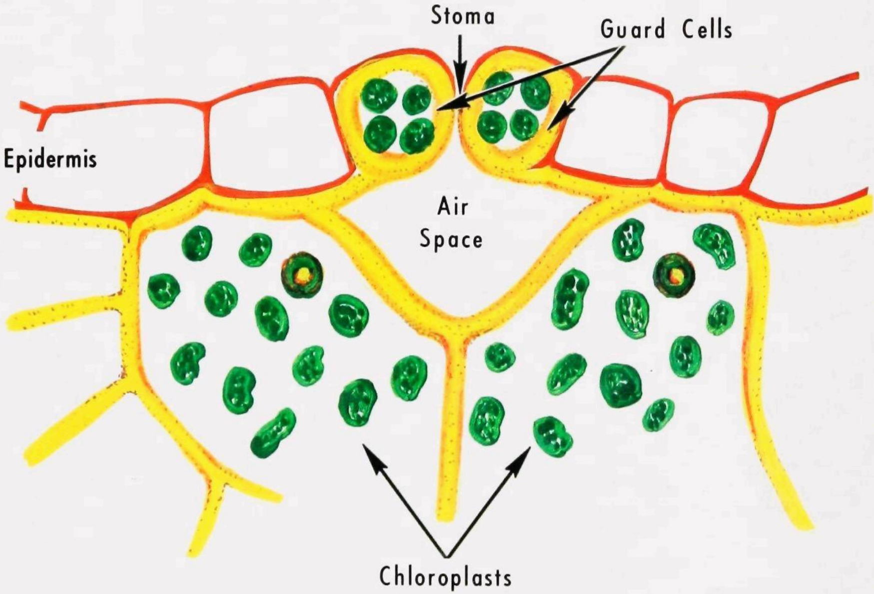


Figure 3. Cross section of leaf inward from epidermis illustrates stoma through which pollutants enter.

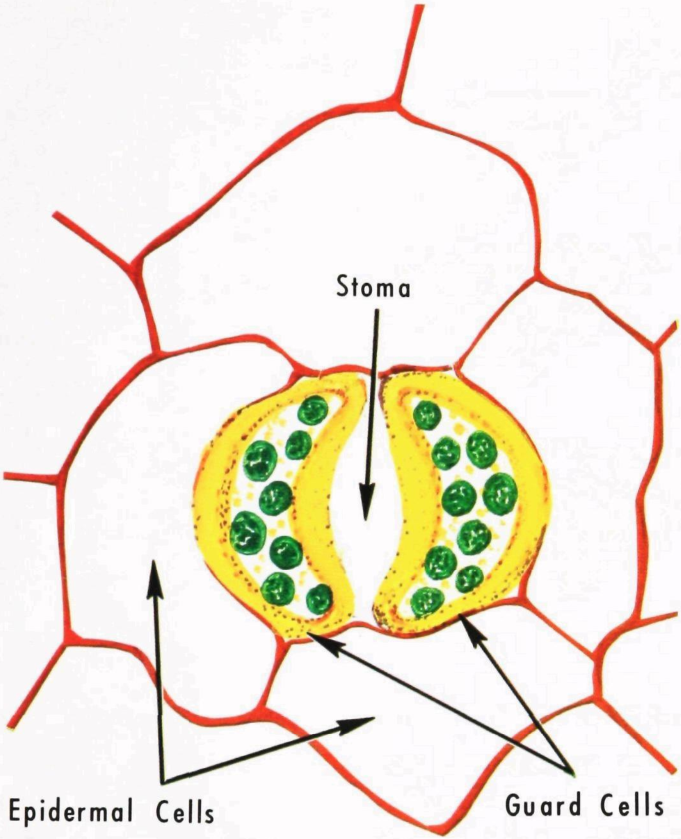


Figure 4. Enlarged view of leaf surface reveals open stoma.

The mesophyll is the soft tissue between the lower and upper epidermis of typical leaves and is made up of two principal types of tissue, the palisade parenchyma and the spongy parenchyma. Palisade parenchyma consists of one or several layers of elongated cells located between the upper epidermis and the spongy cells. Spongy parenchyma cells are not elongated. They are formed and arranged so that many large spaces exist between them. The cells of both palisade and spongy parenchyma contain chloroplasts.

Vascular bundles carry water, minerals, and carbohydrates throughout the leaf area. The mid-rib is the largest vein.

ACTIVITIES OF THE LEAF

The primary functions within a typical leaf are photosynthesis, transpiration, and respiration.

Photosynthesis: Basic Food Manufacturing Process

The mesophyll tissue contains a large number of chloroplasts. In the presence of light, photosynthesis takes place in the chloroplasts. This involves the union of water and carbon dioxide to form sugar (glucose) and then starch. Photosynthesis is described by the following equation:

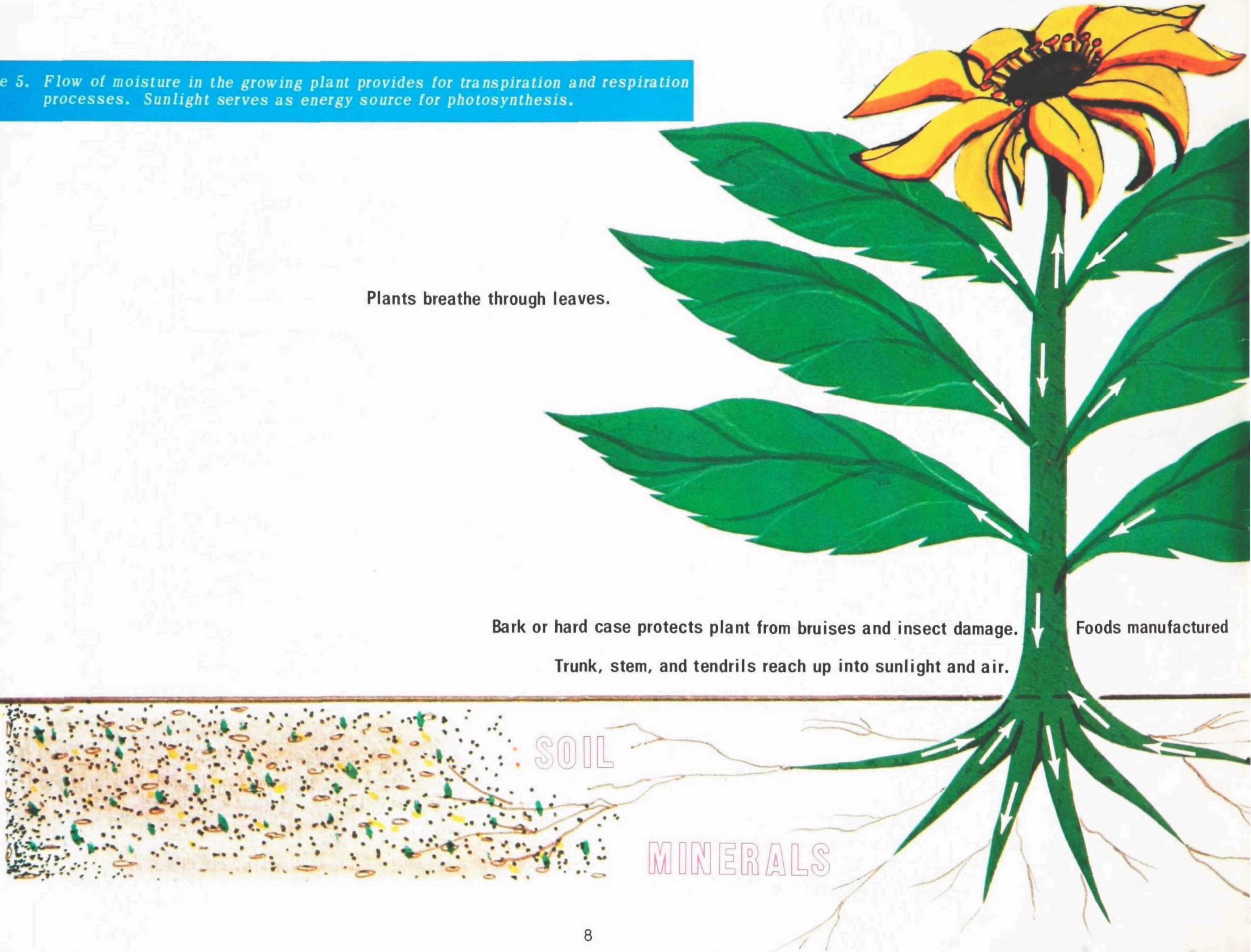
$$6CO_2 + 6H_2O \longrightarrow C_6H_{12}O_6 + 6O_2$$

The excess oxygen generated in the process escapes from the plant and into the atmosphere during photosynthesis. Light is the source of energy for photosynthesis, and part of this energy is stored in the molecules of glucose.

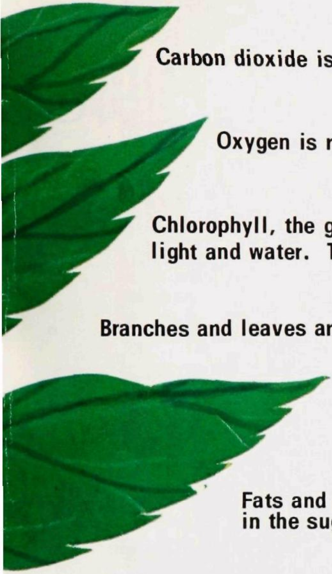
Transpiration: Cooling and Nutritional Processes

A plant suffers constant water loss to the atmosphere through evaporation. As shown in Figure 5, the water is siphoned from the soil through the roots and moves through conducting tissue or veins to the leaves. In the leaves, the moisture escapes in vapor form from the moist cell walls of the mesophyll into the intercellular spaces. It is then released through the stomata into the atmosphere. Transpiration causes a continuous stream of water to move from the roots to the top of the plant. It transports soil minerals through the plant and also cools the plant. These processes increase during hot sunny weather and greater-than-normal air movement.

Figure 5. Flow of moisture in the growing plant provides for transpiration and respiration processes. Sunlight serves as energy source for photosynthesis.



Blossom uses food in production of seeds.



Carbon dioxide is absorbed.

Oxygen is released.

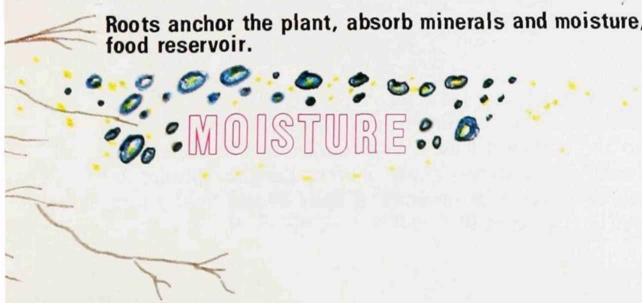
Chlorophyll, the green substance in the plant, absorbs energy from light and water. This energy is used to make starch and sugar.

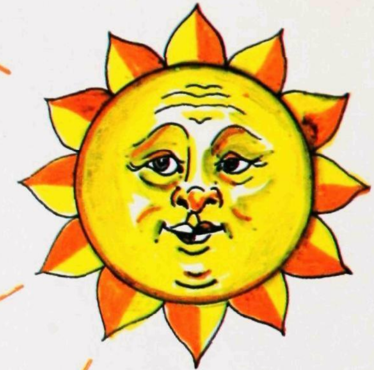
Branches and leaves are arranged to expose greatest possible area to light.

Fats and proteins are made by chemical changes in the sugar and starches in various parts of the plant.

in leaves and branches are carried down stem.

Roots anchor the plant, absorb minerals and moisture, and serve as food reservoir.





Respiration: Energy-Producing Mechanism

Respiration, a continuous process in the protoplasts of all living cells, involves the reaction of oxygen with carbohydrates to release energy. In effect, oxygen acts as a fuel to burn carbohydrates and produce energy. Thus, photosynthesis (see Figure 5) is an energy-absorbing or storing process and respiration is an energy-releasing process.

Because they obtain all the energy necessary for food production and storage directly from the sun, plants form the beginning of the food chain that sustains all animal life, for animals derive all their food, and thus all their energy, directly or indirectly from plants. The immediate end product of photosynthesis in plants is glucose, which, conversely, is the substrate oxidized in respiration. The equation for respiration, then, is the reverse of that for photosynthesis:

$$C_6H_{12}O_6 + 6O_2 \longrightarrow 6H_2O + 6CO_2$$

While the process of photosynthesis is taking place, plants release oxygen into the air and help maintain the correct atmospheric balance. At night, or at other times of photosynthesis inactivity, plant cells take in oxygen and release carbon dioxide to the atmosphere.

PHOTOCHEMICAL REACTION OF HYDROC
AND NITROGEN OXIDES
PRODUCES OZONE AND PAN

The photochemical reaction of hydrocarbons and nitrogen oxides produces ozone and PAN. When exposed to sunlight, nitrogen dioxide absorbs ultraviolet light, which furnishes the energy to break the bond between oxygen and nitrogen in the gas molecule. The result is the formation of nitric oxide and atomic oxygen. This reaction causes a number of oxidations, with ozone the principal oxidant produced.

Major sources of hydrocarbons and nitrogen oxides are automobile engines and industrial plants.

OZONE INJURES PLANT CELLS AND ALTERS COLOR

Ozone severely injures many forms of plant life (Figures 6-15). "Weather flecks" on tobacco leaves have been attributed to ozone.[4] These marks appear first on mature leaves of Tobacco Bel W3 and then progress to the youngest leaves. The first lesions to appear on tobacco leaves are dark (Figure 7), and remain dark for about 24 hours. Then they become lighter in color (Figure 8) and finally turn white several days later (Figure 9).

ARBONS

Figure 6. *Tobacco plant displayed oxidant (ozone) injury at Staten Island, New York, in 1966. Injury progressed from oldest to youngest leaves.*

Figure 7. *Tobacco leaves exposed to ambient air in New York City developed dark lesions attributed to ozone.*

Figure 8. *Ozone-injured tobacco leaves develop light-colored lesions approximately 3 or 4 days after exposure.*

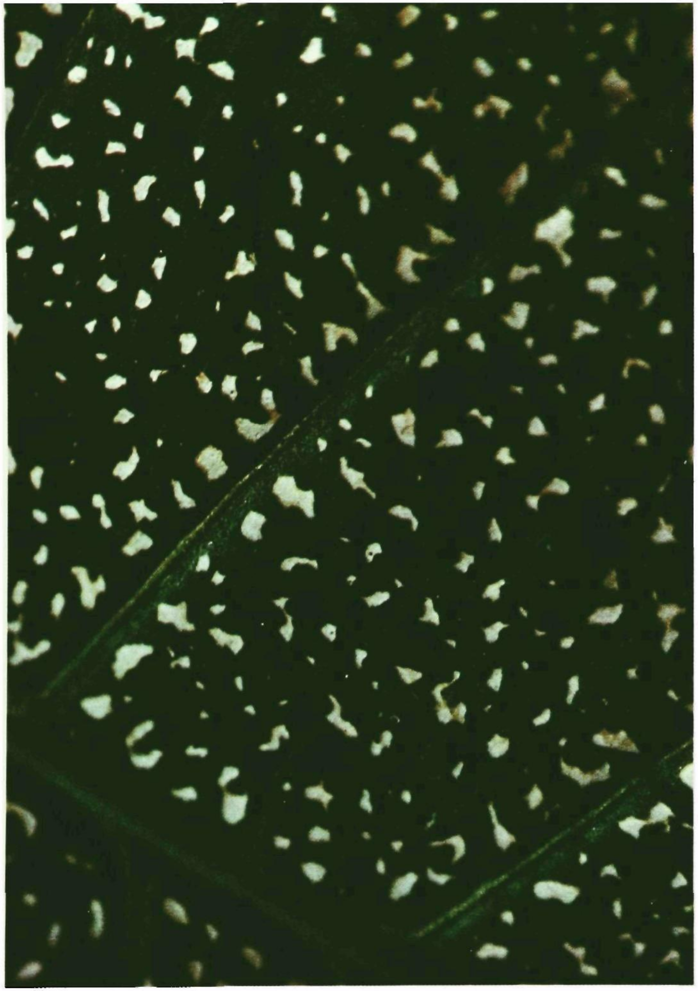


Figure 9. Lesions on tobacco leaves appear white several days after exposure to ozone.

Figure 10. Enlarged surface of pinto bean leaf shows stipple (pigmentation) along veins of plant grown outside at Staten Island, New York.

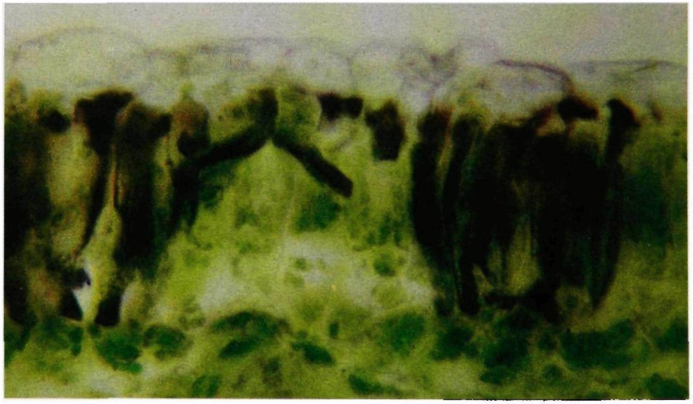


Figure 11. Microscopic cross section of pinto bean leaf shows severe ozone injury. Palisade cells plasmolyzed and lost their integrity, but spongy and epidermal cells remained intact.

Figure 12. Fleck and symptoms of dehydration appeared on old primary leaf of pinto bean plant exposed to natural pollutants.

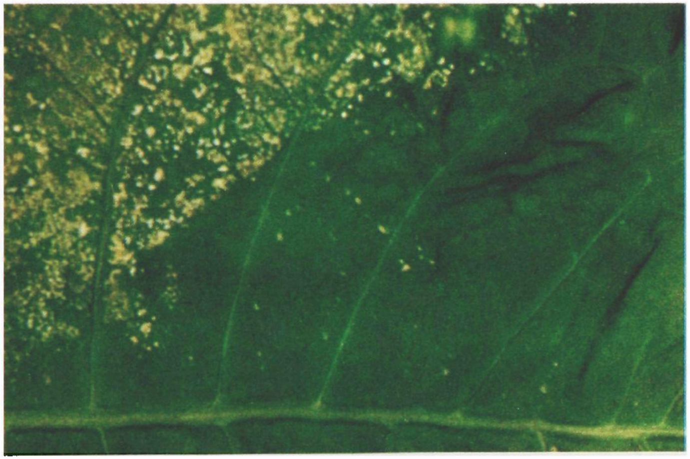


Figure 13. Injury on tobacco leaf exposed to ozone in laboratory did not develop on upper part of leaf because it was shaded during ozone exposure. Severe ozone injury appeared on unshaded part of leaf. Light caused the stomata to open, thus a greater amount of ozone entered and injured this portion.

Ozone causes stipple and bleaching on the upper surface of leaves of the pinto bean plant (Figure 10). Fleck and stipple caused by ozone also appear on the upper surface of grapevines. These symptoms are similar to those of ozone injury in other species of plants and were first recognized in 1958.[5]

Ozone injures the palisade cells of plants (Figure 11). Initially, the chloroplasts condense and accumulate at the center or at both ends of the cell. This is followed by collapse of the cell wall. Occasionally, the chloroplasts granulate and form a jelly-like mass. As the result of pigment breakdown in the damaged cells, a reddish-brown color is produced. When exposed to ozone, leaves of woody plants develop red-brown spots from the formation of new pigment.[6] When small islands of palisade tissues are injured, discrete punctate spots appear.

Studies made at the greenhouse facilities of the National Air Pollution Control Administration in Cincinnati revealed that sensitivity of Tobacco Bel W3 and pinto bean plants to ozone exposure is influenced by environmental conditions and the time of day of exposure.[7] Plants were significantly more sensitive during the mid-portion of the day. The importance of the time factor is recorded in Tables 1 and 2. Table 3 indicates the effect of light intensity during ozone exposure tests on pinto bean plants.

Published information concerning tobacco varieties indicates that test plants grown in California and Utah are not as sensitive to ozone as are the same plant varieties when grown in the middle and eastern regions of the United States.[4] Studies in California showed that 25 parts per hundred million (pphm) of ozone for 6 hours causes injury to plants.[4] In Utah in 1961, it was found that an average of 24 pphm of ozone for 2 hours injured tobacco plants.[8] In 1959 in New York City, however, tobacco injury was reported at 10 pphm for 8 hours,[9] and in 1962 in Beltsville, Maryland, studies showed that Tobacco Bel W3 was injured after 4 hours fumigation at an ozone concentration of only 5 to 7 pphm.[10] In 1965 it was found that greenhouse-grown Tobacco Bel W3 plants were injured after 4 hours' fumigation at 3.5 pphm of ozone,[7] a concentration about half of that used at Beltsville.

This variation suggests that the plant injury threshold of ozone in one location may vary from day to day and that a concentration of the gas that would not cause injury in a certain location may be injurious to comparable plants in other locations. Conditions that may cause the injury threshold to vary include light intensity, temperature, humidity, available nutrition, and season. Examples of ozone injury to agricultural specimens are shown in Figures 14 and 15.

Figure 14. Symptoms on spinach leaves indicate injury from oxidants, mainly ozone.

Figure 15. Injured leaf shows reddish-brown bleach attributed to atmospheric oxidants.

Table 1. EFFECT OF TIME OF DAY OF EXPOSURE ON SENSITIVITY OF PINTO BEAN TO OZONE [7]

Time of day	Portion of leaf damaged, %
9 a.m.	39
11 a.m.	63
1 p.m.	60
3 p.m.	47

Table 2. SENSITIVITY OF TOBACCO BEL W3 TO LOW LEVELS OF OZONE [7]

Concentration, pphm	Time, hours	Portion of leaf damaged, %
3.5	4	12
3.5	8	56
5.0	2	1
5.0	4	14
9.0	2	11
9.0	4	45

Table 3. EFFECT OF LIGHT INTENSITY ON SENSITIVITY OF PINTO BEAN TO OZONE EXPOSED AT CONSTANT TEMPERATURE [7]

Light intensity, ft-c	Portion of leaf damaged, %
2,600	89
1,800	73
100	10
Dark	2

PAN IS HIGHLY TOXIC TO MANY PLANT SPECIES

Peroxyacetyl nitrate, commonly called PAN, is extremely toxic to many plant species, especially small plants and young leaves. PAN is unstable, particularly at temperatures above 90° F. Visible symptoms of PAN injury include bronzing, silvering, and glazing on lower leaf surfaces (Figures 16-19).

In this kind of damage, some of the spongy or the lower epidermis cells or both become plasmolyzed, and the chloroplasts tend to lose their integrity. After severe damage, additional spongy cells become plasmolyzed and the cytoplasm changes color to light or dark brown. The microscopic pattern of injury associated with PAN is shown in Figure 19.

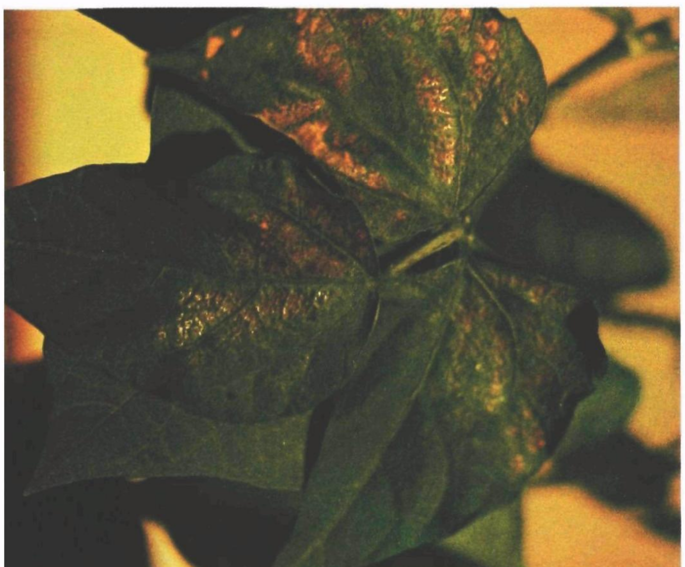


Figure 16. Trifoliate leaf of pinto bean plant exposed to ambient air shows bronzing symptoms caused by severe PAN injury.

Studies have shown that injury varies directly with concentration and length of exposure to PAN. Exposure to high concentrations of PAN (0.8 ppm for 30 minutes or more) can cause severe injury on young pinto bean plants. A low concentration of PAN, 0.5 ppm for 1 hour or 0.1 ppm for 5 hours, caused severe injury to pinto bean, petunia, and tomato plants.[11] Glazing or bronzing on the lower leaf surface with little or no symptom visible on the upper surface developed when bean leaves were exposed to 0.8 ppm for 15 minutes.[12] PAN concentrations as low as 0.01 ppm for 6 hours can cause plant injury.[13]

Physical changes have been found in the chloroplast structure of pinto bean leaves exposed to 1 ppm for 30 minutes although injury was not evident to the unaided eye.[14]

The light intensity under which pinto bean plants are grown and exposed to PAN has a very pronounced effect on development of visible symptoms. For the visible symptom to develop light is required by the plant for at least 3 hours preceding exposure, during exposure, and for at least 2 to 3 hours following exposure.[15]

Figure 17. Lower surface of primary leaf of pinto bean plant shows silvering.

Figure 18. Severe oxidant injury developed on lettuce plants grown in Los Angeles, California, area.

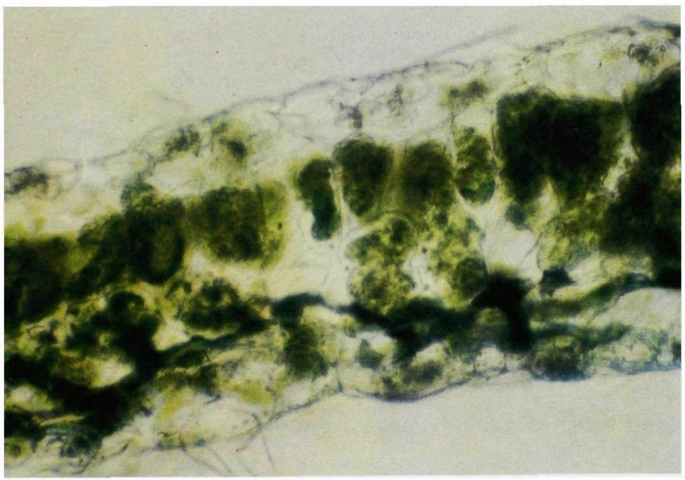


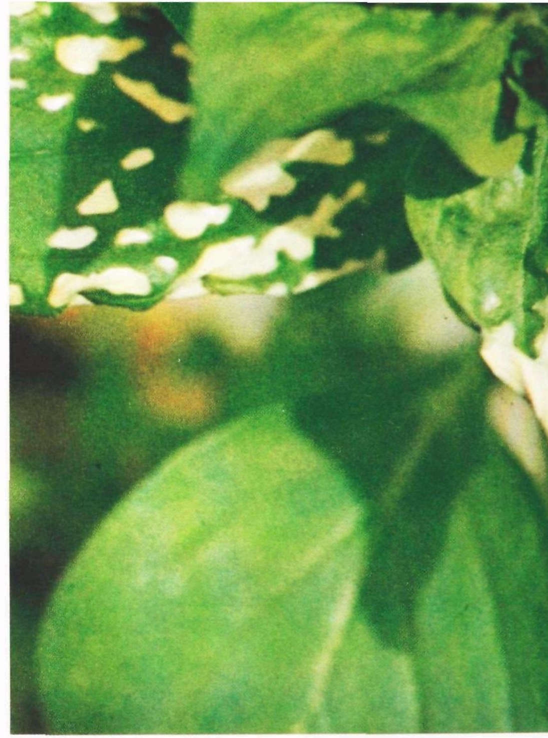
Figure 19. Microscopic cross section of petunia leaf exposed in laboratory shows PAN injury. Spongy cells adjacent to lower epidermis plasmolyzed, and chloroplasts lost their integrity. Upper and lower epidermis and palasade cells remained intact.

Overt symptoms of injury by PAN may be indistinguishable from those produced by other pollutants. For example, PAN causes glazing and silvering on the lower surface of young trifoliate and primary leaves of the pinto bean plant, but identical symptoms develop on comparable plants exposed to irradiated automobile exhaust fumes and irradiated pure components such as propylene and nitrogen oxide, when tested under controlled conditions.[16,17]

Other studies demonstrated that plant injury similar to that found on the lower surfaces of old leaves of pinto beans grown in Los Angeles could be reproduced by exposing similar plants to a mixture of gasoline vapor and ozone.[18] Comparable results were obtained with mixtures of ozone and certain olefins.[16,17,19,20] Mixtures of nitrogen dioxide and gasoline vapor also produced the same type of injury.

Reddening and bronzing developed on the lower surfaces of old primary and trifoliate leaves of pinto beans grown and exposed to ambient air at Staten Island, New York, and Perth Amboy, New Jersey.[21] These symptoms also resemble those produced by ozone-olefin products. It has been reported that a non-irradiated mixture of ozone and 1-pentane, 1-hexane, and 3-heptane caused typical PAN-type injuries to 14-day-old primary leaves of pinto beans, but did not injure 8-day-old bean leaves or petunia leaves.[22] During the course of the studies at Staten Island and Perth Amboy[21] in 1965 and 1966, cloud cover and heavy concentrations of particulate matter in the atmosphere were encountered. Because light intensity was reduced, the ozone-olefin mixture was not fully irradiated.

NITROGEN DIOXIDE CAUSES DIRECT VEGETATION DAMAGE



Combustion of coal, fuel oil, natural gas, and gasolines used in power-generating operations is the source of nitrogen dioxide. In addition to its role in producing ozone and PAN in the presence of light and hydrocarbons, nitrogen dioxide alone injures vegetation. Symptoms of nitrogen dioxide injury appear as irregular white or brown collapsed lesions on tissue between the veins and near the leaf margin (Figure 20).

Figure 20. Petunia plant exposed to 13.5 ppm nitrogen dioxide for 1 hour shows injury that resembles sulfur dioxide damage.

Studies indicate that visible leaf damage to tomato seedlings is caused by a concentration of 2.5 ppm nitrogen dioxide after 4 hours exposure.[15] Pinto bean plants are reportedly injured by a 3-ppm concentration after 4 to 8 hours exposure.[3] Nitrogen dioxide injury to a tomato plant exposed to ambient air is shown in Figure 21.

Acute symptoms found on bean, tomato, and tobacco seedlings, induced by an exposure to 2.5 ppm of nitrogen dioxide, also closely resemble necrotic lesions (surface spotting) caused by sulfur dioxide or injuries caused by excessive concentrations of ozone.[11] A nitrogen dioxide concentration of 25 ppm injures most indigenous vegetation.[23]

Plant growth may be inhibited by continuous exposure to 0.5 ppm nitrogen dioxide or perhaps less.[15] When applied continuously, nitrogen dioxide in concentrations below 0.5 ppm (and equivalent to the levels recorded in the Los Angeles atmosphere) cause marked chlorosis of basal leaves of tobacco.[12]

Figure 21. Tomato plant exposed to ambient air in Chattanooga, Tennessee, was injured by nitrogen dioxide.

SULFUR DIOXIDE CAUSES ACUTE AND CHRONIC INJURY

Major sources of sulfur dioxide are residential and commercial buildings heated by coal or fuel oil of high-sulfur content, industrial facilities such as petroleum refineries and some chemical plants, and power-generating plants that burn sulfur-bearing fuel.

Sulfur dioxide causes both acute and chronic plant injury (Figures 22-29). Acute injury is characterized by clearly marked dead tissue between the veins or on the margins of leaves. Chronic injury is marked by brownish-red, turgid, or bleached white areas on the blade of the leaf. The plant injury threshold for sulfur dioxide is 0.3 ppm for 8 hours.

Plant injury is thought to be caused by sulfur dioxide, sulfuric acid that forms as a by-product of sulfur dioxide in the atmosphere, or a combination of both. It has been reported that for each 25 to 30 parts of sulfur dioxide formed during combustion of fossil fuel, one part of sulfur trioxide is formed. It continues to form as the combustion products mix with the atmosphere. Sulfur trioxide, in turn, mav rapidly combine with atmospheric moisture to form sulfuric acid.[24] This acid may be suspended as small droplets, which cause distinct punctate spots to appear on leaves. Most often, acid aerosol damage occurs during foggy weather and the injury develops on non-waxy leaves (Figure 25). This type of spotted injury has been reported in the Los Angeles area during a period of heavy air pollution accompanied by fog or light rain.[14] Similar injury may also occur without fog, and its attendant moisture, in an area where there are combustion by-products containing sulfur oxides and where the effluent dew-point permits acid droplets to form. Usually the upper leaf surfaces display the initial necrosis. Cellular collapse and punctate spots develop progressively through the upper epidermis, mesophyll layers, and the lower epidermis of the leaf. No glazing or bleaching accompanies this injury.

Figure 22. Rose leaves in Independence, Missouri, show marginal and interveinal necrotic injury from sulfur dioxide.

Figure 23. Laboratory-exposed potato leaves reveal typical sulfur dioxide injury.

Figure 24. Salt bush plant was exposed to sulfur dioxide in laboratory; this plant is excellent indicator of excess SO_2.

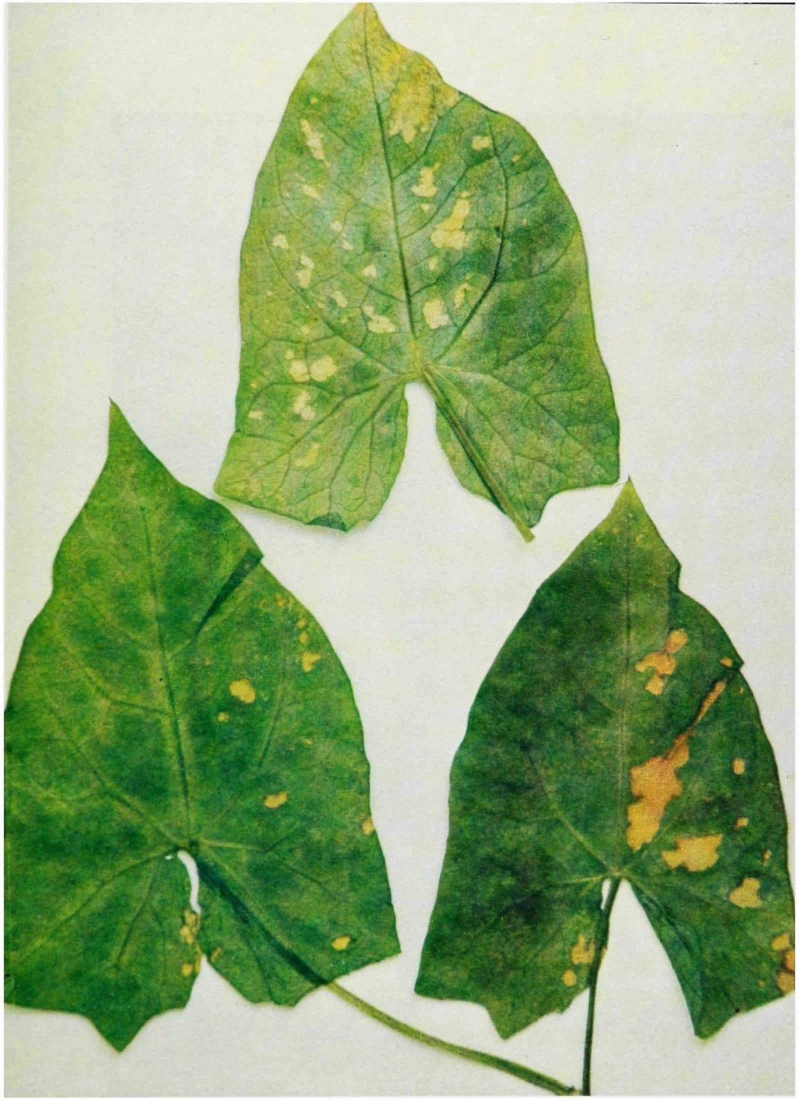


Figure 25. Bindweed injury in Staten Island, New York, was caused by sulfuric acid droplets.

Figure 26. Pinto bean plant was sprayed with water before sulfur dioxide exposure in laboratory.

Figure 27. Apple tree leaves near power plant in Pennsylvania shows typical sulfur dioxide injury.

Figure 28. Castor bean leaf injury resulted from laboratory exposure.

Figure 29. Forsythia in upper Ohio River Valley near Steubenville, Ohio, was injured by sulfur dioxide.

Figure 30. Necrotic injury on hydrangea leaf was caused by fly ash from New Jersey power plant.

Plants are particularly sensitive to sulfur dioxide during periods of intense light, high relative humidity, adequate plant moisture, and moderate temperature. Plants are, therefore, especially sensitive to sulfur dioxide during the growing season in late spring and early summer.

Time and levels of exposure affect plant sensitivity to sulfur dioxide. Plants exposed to high sulfur dioxide concentrations during the early or late daylight hours are less affected by the gas than plants exposed from 10:00 a.m. to 2:00 p.m. At night, when the stomata of most plants are closed, the plants are much less susceptible to sulfur dioxide injury.

The degree of turgidity of test plants is extremely important in sulfur dioxide sensitivity. Soil dry enough to cause a slight wilting increases plant resistance to sulfur dioxide injury. Turgid tomato leaves are severely injured by sulfur dioxide, but slightly wilted leaves are uninjured by the same concentration of the toxicant. Young plants are more resistant than old plants, and middle-aged leaves are most susceptible to injury. These differences are probably caused by variations in the number, size, and activity of the stomata and the quality of the cytoplasmic contents of the cells.

Microscopic examination of leaf tissue injured by sulfur dioxide reveals that the mesophyll cells are affected and the chloroplasts become plasmolyzed or bleached out. The spongy cells are often more readily affected than the palisades. Under severe conditions the epidermis cells are also plasmolyzed. The mid-rib and large veins remain intact and green, even though most of the leaf has collapsed.

Injury to agricultural crops exposed to a given concentration of sulfur dioxide is greater than the injury to laboratory-exposed plants subjected to the same concentration because of possible additional toxicants present in the uncontrolled ambient atmosphere. For this reason, injury to agricultural crops is usually greater than can be projected from laboratory experiments involving only one toxicant.

Low concentrations of sulfur dioxide can interfere with the growth and functioning of a plant without leaving visible injury. This injury may interfere with or reduce photosynthesis. Grain crops may suffer a reduction in yield, especially if crops are damaged by sulfur dioxide at the blossom stage.[2]

SYNERGISTIC EFFECT OF OZONE
MORE SEVERE

AND SULFUR DIOXIDE CAN CAUSE INJURY THAN EITHER POLLUTANT ALONE

Ozone and sulfur dioxide together produce a synergistic action that reduces the injury threshold of leaf tissue. It has been reported that an ordinarily harmless concentration of ozone, when combined with sulfur dioxide, produced ozone-type injury to Tobacco Bel W3 plants.[25] No injury developed from exposure of similar plants to identical concentrations of the individual gases. See Table 4.

Table 4. SYNERGISTIC EFFECT OF OZONE AND SULFUR DIOXIDE ON TOBACCO BEL W3 PLANTS[25]

Duration, hr	Toxicants, ppm O_3		Toxicants, ppm SO_2	Leaf damage, %
2	0.03			0
2			0.24	0
2	0.027	+	0.24	38
4	0.031			0
4			0.26	0
4	0.28	+	0.28	75

Laboratory work has indicated that a single 4-hour exposure to nitrogen dioxide below 2 ppm and to sulfur dioxide below 0.7 ppm will not injure tobacco. Exposure for 4 hours to a mixture of 0.1 ppm of nitrogen dioxide and 0.1 ppm of sulfur dioxide produces moderate injury to the older leaves of Tobacco Bel W3. Preliminary experiments with ozone, nitrogen dioxide, and sulfur dioxide suggest that a mixture containing 0.05 ppm of each of these toxicants injures tobacco.[26]

In 1966 it was revealed that tobacco exposed to ambient air in the Brooklyn Botanical Garden; Staten Island, New York; and Bayonne and Roselle, New Jersey, showed ozone injury on plant leaves although the ozone concentration was far below the injury threshold.[27, 28] Sulfur dioxide was shown to be present, however. Figure 31 shows a tobacco leaf exposed to ambient air and another to a controlled, uncontaminated atmosphere during the New York-New Jersey study.

Figure 31. Tobacco Bel W3 plants exposed to clean atmosphere (left) and to ambient air (right) dramatize the possibility of ozone - sulfur dioxide synergistic action.

AEROSOLS—SULFUR DIOXIDE

The presence of an aerosol may substantially increase the toxic effects of sulfur dioxide. A study has indicated that in the presence of dew, mist, or very light rain, a lower level of sulfur dioxide concentration than that required during a dry period could cause plant injury.[29] Heavy rains, however, might wash away the gas rather than concentrate it on leaf surfaces.

PLANT DAMAGE APPEARS FROM LOW CONCENTRATIONS OF FLUORIDES

Fluorides in concentrations as low as 0.1 part per billion are toxic to some plants.[2] Fluorides in either gaseous or particulate form can accumulate outside or inside plant leaves and cause leaf injury (Figures 32 through 39). Fluorides do not translocate from the leaf to other parts of the plant. Even when high fluoride concentrations exist in the leaf tissue, the root systems, flower seeds, and all other plant parts remain very low in fluoride content.

Gaseous fluoride compounds are responsible for most fluoride injuries suffered by plants. Hydrogen fluoride and other gaseous compounds are readily absorbed by the leaves. An accumulation of a solid fluoride on its surface will not injure the leaf, however, unless the fluoride on the leaf is dissolved by moisture. Dew or light rain can provide the necessary moisture. The soluble fluoride then can be absorbed readily by the tissues.

Principal sources of fluorides emitted to the atmosphere are fertilizer manufacturing, aluminum reduction, ore smelting, and ceramic manufacturing.

Figure 32. *Gladiolus plant shows beginning of fluoride injury on tips of leaves.*

Figure 33. *In advanced stage, injury progresses from tip to base of gladiolus leaves.*

Figure 34. *Fluoride-injured pear leaves were collected in Staten Island, New York.*

Figure 35. Prune leaves turn brown at edges when exposed to fluoride.

Figure 37. Maple leaves from West Virginia were injured by fluoride from nearby industry.

Figure 36. Italian prune leaves were injured during laboratory exposure to fluoride.

Figure 38. Pine needles show sporadic injury.

Figure 39. Young needles of ponderosa pine exposed to fluoride (right) were injured; control on left is healthy plant.

Vegetation closest to and downwind from a fluoride source suffers the most severe injury.[30] Measurements taken over the years from such locations have disclosed alarmingly high fluoride concentrations. Concentrations as high as 300 ppm of fluorine were found in vegetation near aluminum and fertilizer plants in Tennessee.[31] In another case the fluoride level was 462 ppm. Nine miles from this source the concentration on grape leaves was still 114 ppm. In still another investigation, concentrations of more that 10,000 ppm fluoride were found on pine trees in Montana.

Fluoride contamination of plants is manifested in several ways. In gladioli, for instance, necrotic injury of the tissue starts at the tip of the leaf and advances downward. On broad-leaf plants, the injury is along the margin, and there is often a sharp demarcation line between injured and intact tissue. Injury to some plants appears as spots of injured tissue surrounded by healthy tissue.

A study of citrus trees exposed to 2 to 3 ppm fluoride for several months revealed leaf chlorosis. Compared with comparable trees growing in fluoride-free air, the trees studied produced smaller leaves and lower fruit yields, and grew less vigorously. In other plants studied, fluoride accumulations also caused dwarfing, leaf abscission, dropping of fruit, and lower yields.[32]

In 1965, Snow Princess gladioli plants were used to locate sources of fluoride pollution at Staten Island, New York.[21] Gladioli bulbs were planted at five sites on June 27, 1965. Tip and margin burn developed by July 20, and injury on the leaf blades gradually extended downward and maintained a fairly uniform front. The gladioli plants were harvested September 20, 1965, and leaf and petal samples from each of the five test stations were analyzed chemically. The results are shown in Table 5. The amount of fluoride found and the predominance of fluoride injury that appeared on the tips of the gladioli leaves indicated that gaseous fluorides were present in the atmosphere.

Table 5. RESULTS OF FLUORINE ANALYSIS OF GLADIOLUS LEAF SAMPLES[a]

Location	Location of sample analyzed, inches from blade tip	Fluoride concentration,[b,c] ppm
Port Richmond	0 - 2	58.9
	2 - 4	23.8
	4 - 8	3.3
Willowbrook	0 - 2	82.9
	2 - 4	38.2
	4 - 8	9.3
Willowbrook (Control)	0 - 2	37.9
	2 - 4	6.7
	4 - 8	10.7
Princess Bay	0 - 2	31.3
	2 - 4	11.4
	4 - 8	3.4
Greenhouse at U. S. Public Health Hospital	0 - 2	123.4
	2 - 4	30.8
	4 - 8	10.4

[a]Gladiolus petals collected from all five locations and analyzed for fluorine were found to contain 3.9 ppm, calculated on a dry weight basis.

[b]Calculated on a dry weight basis.

[c]Values above 10 ppm are considered accurate to about ± 5 ppm; values under 10 ppm are accurate to about ± 3 ppm.

CHLORINE DAMAGE OCCURS CLOSE TO SOURCE

Chlorine is found in the atmosphere primarily in those areas where it is being used as a disinfectant or where it is involved in a chemical process. For example, chlorine-injured plants are often observed near swimming pools and sewage disposal systems.

Chlorine pollution causes marginal and tip necrosis, which may be similar to the plant injury caused by sulfur dioxide. Middle-aged leaves are most susceptible to chlorine injury, followed by the oldest, then the youngest leaves. Photographs of chlorine injury are shown in Figures 40-43.

The chlorine concentration required to injure plants is greater than that reported for hydrogen fluoride, but less than the level reported for sulfur dioxide.[33]

In a study at Rutgers University, tomato plants were exposed for 2 and 3 hours at three different chlorine levels.[34] Half of the plants were periodically sprayed with water. Response of plants, wet or dry, was similar. A chlorine concentration of 0.31 ppm caused no injury, 0.61 ppm caused slight injury, and 1.38 ppm caused severe injury. Stanford Research Institute reported injury to alfalfa and radishes that were exposed for 2 hours to 0.10 ppm chlorine.[33]

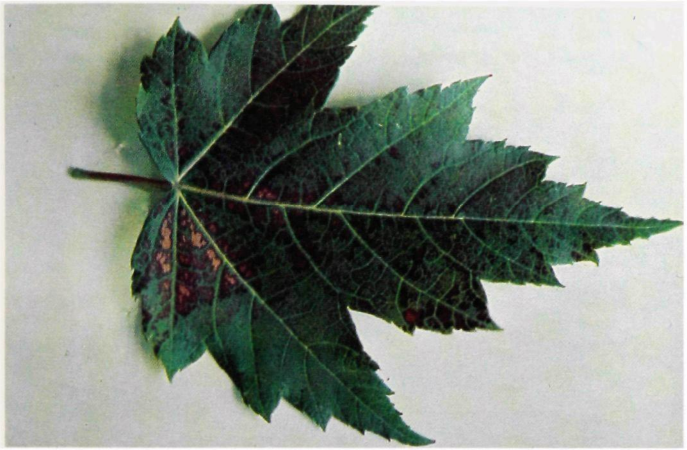


Figure 40. Chlorine injury to silver maple leaves is similar to sulfur dioxide injury.

Figure 41. Cucumber leaf exposed to 0.75 ppm Cl_2 for 4 hours shows injury similar to sulfur dioxide injury.

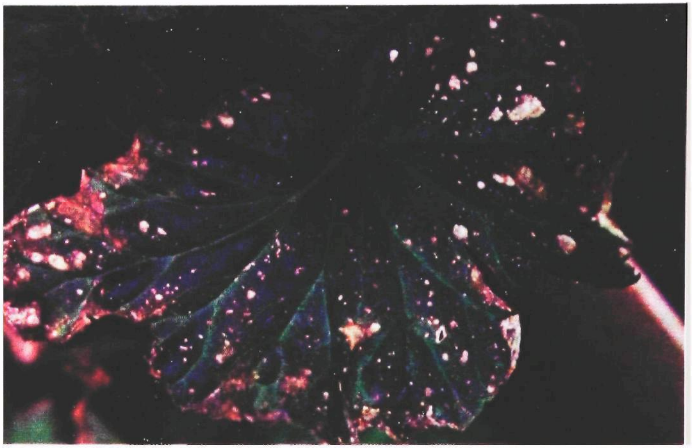


Figure 42. Begonia was injured by chlorine and by hydrochloric acid mist in Pennsylvania.

Figure 43. Redbud leaves from same area as begonia in Figure 42 were also injured.

ETHYLENE INJURES VEGETATION IN URBAN AREAS

Ethylene is one of the few hydrocarbons possessing the ability to injure plants without undergoing photochemical reaction with nitrogen oxides.[35] Unlike PAN and ozone, ethylene is difficult to extract by carbon-filtering processes.

Ethylene is found principally in the atmospheres of larger cities and urban areas because of the high concentrations of automobile and truck exhaust, natural-gas and fuel-oil heating systems, and coal-burning industrial processes.

Ethylene injury to orchids and azaleas is shown in Figures 44 and 45. In the Los Angeles area and Hawaii, ethylene has been reported to cause the sepals of orchid flowers to become brown or withered and dry. In an experimental study, the sepals of orchid flowers became withered and dry after a 24-hour exposure to 5 ppm of ethylene.[36] Exposure to 0.3 ppm ethylene for 1 hour or 0.05 ppm for 6 hours causes abnormalities in the sepals of cattleya orchids.

Ethylene interferes with the activities of plant hormones and thereby causes growth retardation.[6] In addition, carnation flowers, for example, failed to open properly after a 6-hour exposure to 0.1 ppm.[37] A 0.05-ppm ethylene exposure impaired normal development of marigold leaves.[38] Abnormalities of tomato leaves and loss of flower buds were observed after exposure of tomato and pepper plants to 0.01 ppm ethylene for several hours.

Extensive injury to a cotton field adjacent to a polyethylene manufacturing area has been reported.[39] The pollutant traveled 1 mile downwind from the source, and the damaged field suffered almost complete loss of yield.

Figure 44. Orchid at top was exposed to ethylene in laboratory; intact orchid of same variety is shown at bottom.

Figure 45. Control azalea on left has rigorous blooms, but plant on right dropped blooms when exposed to ethylene.

AIR POLLUTION RETARDS GROWTH OF VEGETATION

In addition to the visible injuries caused by toxicants in the atmosphere, atmospheric pollutants or combinations of pollutants may cause considerable injuries that disturb plant function and that alter or suppress growth. An example of growth suppression is shown in Figure 46.

Figure 46. Petunia on left was grown out of doors. Larger plant on right was grown in controlled environment.

In one experiment, the action of ozone and 1-hexane in combination reduced tomato plant growth, but no visible symptoms of leaf injury were observed. Tomato plants exposed to naturally occurring oxidants in contaminated air had abnormally small leaves. Blossom initiation was completely suppressed. Comparable plants grown in carbon-filtered air developed normal blossoms and set fruit.

In 1966, it was found that growth was suppressed in Tobacco Bel W3, pinto beans, petunias, and geraniums when grown for 8 weeks in ambient air at Steubenville, Weirton, and Wheeling and for 10 weeks in New York and New Jersey. These plants were compared to similar plants grown in ambient air that was passed through an activated-carbon filter. Leaves on plants grown in carbon-filtered air were longer, more vigorous, and darker green. Root systems of plants grown in carbon-filtered air also were larger and more vigorous than those grown in unfiltered air[26, 27] as shown in Figure 47.

In a later study, chlorosis, dwarfing, and growth suppression of petunia, columbine, geranium, and pinto bean plants developed. These plants were exposed to ambient air, and the injuries probably were caused by the interaction of a combination of toxicants.

It also has been shown that tree growth may be reduced by exposure to ozone-olefin reaction products and to naturally occurring oxidants.[40] Exposure to ozone and hexane[41] for 280 hours over a period of 8 weeks caused the following growth reductions in avocado seedlings: stem growth, 56 percent; leaf width, 35 percent; and stem diameter, 21 percent. The fresh weight of seedlings, including the root system, was reduced 52 percent. The dry weight of seedlings, including the root system, was reduced 58 percent. All comparisons were made with comparable lots of avocados grown in carbon-filtered air.

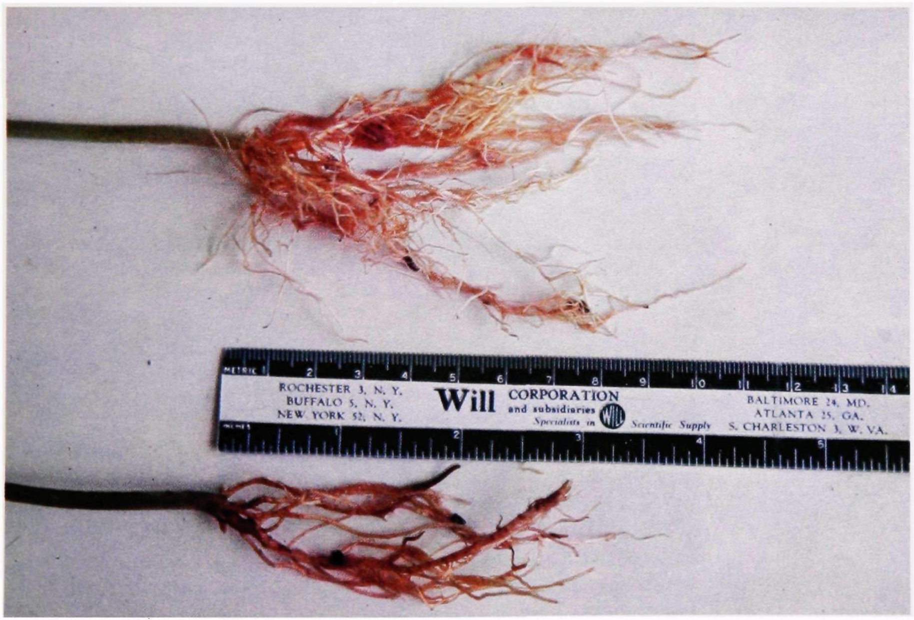


Figure 47. Root systems of pinto bean plants grown with (above) and without (below) activated-charcoal-filtered air.

VEGETATION INJURY FROM OTHER AGENTS CAN BE CONFUSED WITH AIR POLLUTION INJURY

Some striking similarities exist between visible injury from air pollution and injury from other agents. Natural factors that may be injurious are drought, frost, and mineral deficiencies. Other agents of plant injury and disease include insects, nematodes, and viruses. Not only can natural factors mimic pollution-like damage, but pollutants themselves can sometimes mimic each other.

In 1965, injury by natural agents was thoroughly explored.[42] It was pointed out, for example, that terminal bleach disease in cereal plants was caused by excessive water loss associated with hot winds. Symptoms similar to sulfur dioxide injury can be induced by frost (Figure 48) or even by mineral deficiency (Figure 49). Symptoms of virus or nematode attack (Figure 50) also have to be taken into consideration in diagnosis. Virus-like symptoms (Figure 51) are sometimes confused with ozone injury.

In addition to the major air pollutants, other agents may cause extensive damage to vegetation. One such agent is the herbicide 2, 4-D. Very low concentrations of 2, 4-D cause defoliation, dwarfing, curling, stiffening, and twisting (Figure 52). Cotton, grape, and tomato plants are especially sensitive to 2, 4-D. Some insecticides are also harmful to vegetation (Figure 53).

Because of the many possible mechanisms and sources of injury to vegetation, investigators determining damage attributable to air pollution should have extensive experience in the field.

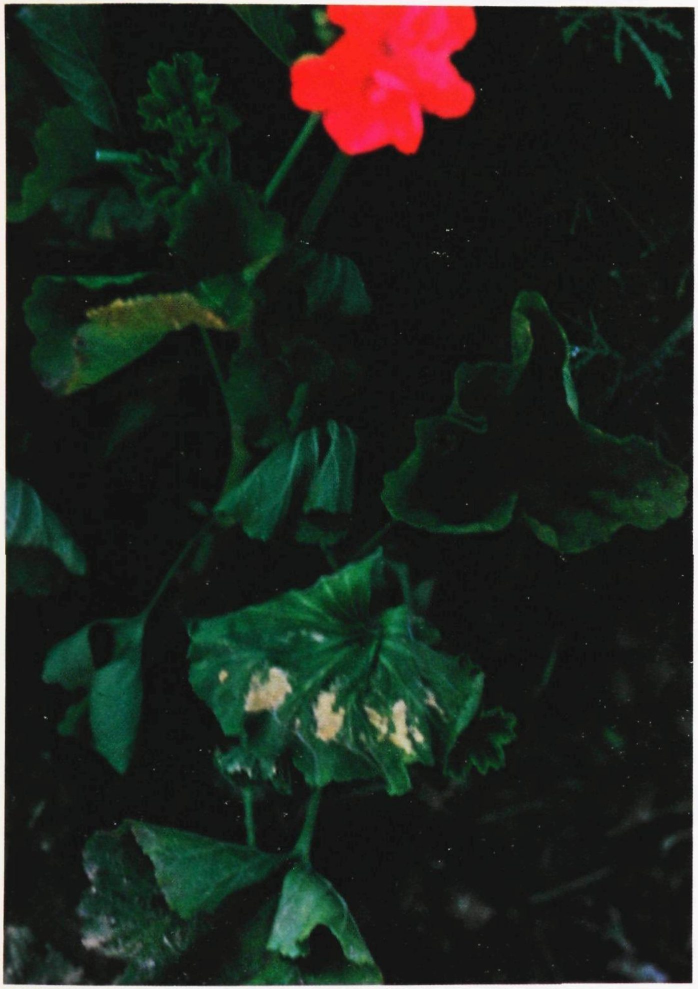


Figure 48. Frost injury to geranium plants is similar to sulfur dioxide injury.

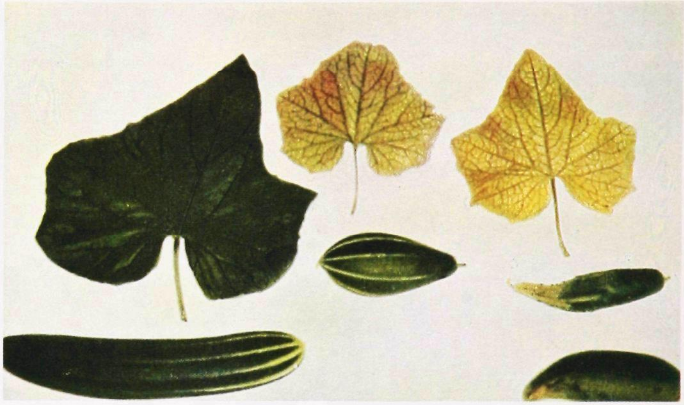


Figure 49. Cucumbers and leaves from plants with potash (center) and nitrogen (right) deficiencies are shown with healthy leaf and cucumber (left).

Figure 50. Suspected sulfur dioxide injury to Texas cotton was rightfully identified as nematode attack.

Figure 51. Strikingly similar tobacco injury was from ozone (top) and mosaic virus (right). Ozone attacks old leaves first, then young leaves. Mosaic virus attacks young leaves first and progresses to old leaves.

Figure 52. Cotton leaves exposed to 2, 4-D are subject to "crow-footing," shown at right of photograph; healthy leaves are shown on the left.

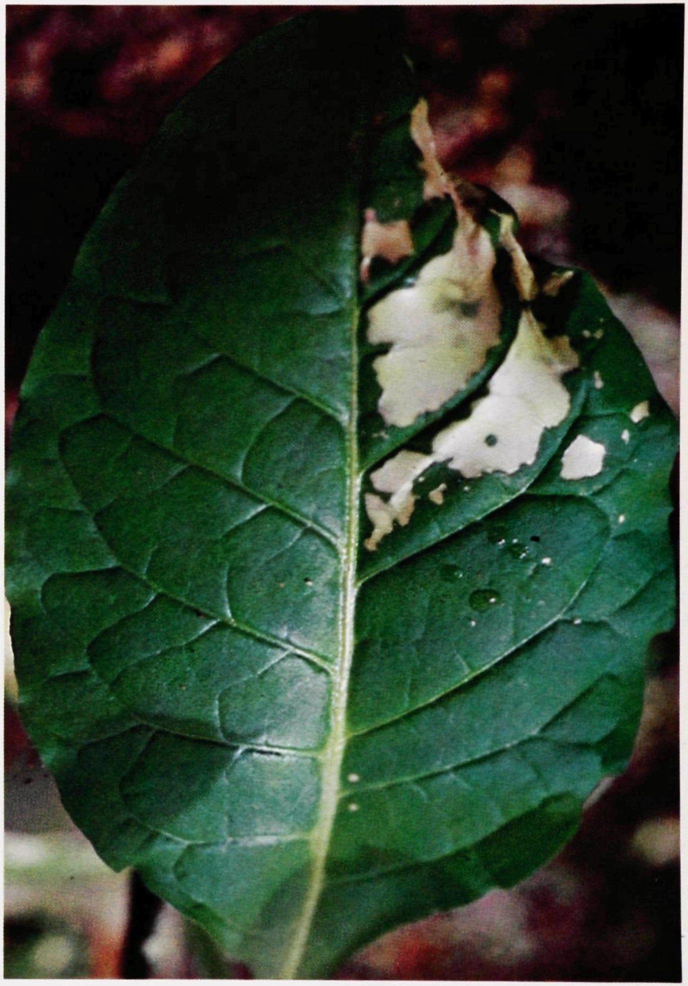


Figure 53. Tobacco was exposed to methyl parathion.

AIR POLLUTION INJURY TO VEGETATION INDICATES DEGRADATION OF ENVIRONMENT

Air pollution injury to vegetation is not only important for the economic losses it causes agriculture, but because vegetation injury is a sign or forewarning of air pollution problems that can affect man and his well being. Plants are considered to be a sensitive tool by which the presence of several airborne toxicants in low concentrations can be detected and evaluated.

The photographs in this document illustrate the damage that air pollution can cause to vegetation. A number of the most serious pollution offenders have been identified, and their effects have been determined. Synergistic effects of multiple pollutants have also been revealed. In most cases injury observed in indigenous vegetation has been reproduced in laboratory experiments. Table 6 is a summary of the well-known airborne pollutants that cause vegetation injury.

The investigator will need to use caution in diagnosing pollution injury symptoms; and where there are uncertainties, he should obtain help from scientific experts. Special care must be taken to differentiate between pollutant symptoms and problems associated with mineral deficiency, plant disease, and other agents.

Toxicants stemming from photochemical reactions and the injurious effects of these toxicants have been found in at least 27 states and the District of Columbia. Canada, Mexico, and Europe also are known to suffer from similar problems.

The cost of agricultural losses in the United States was recently estimated to be in the neighborhood of $500 million annually. Losses to agronomic species in California alone are estimated to amount to $132 million, with a 50 percent loss in citrus fruit.[43] No estimates have been made of the real economic loss caused by suppression of growth, delayed maturity, reduction in yield, and the attendant increase in the cost of crop production. Since air pollution is growing in intensity in many areas of the country, the losses from vegetation damage also are undoubtedly increasing.

Table 6. SUMMARY OF POLLUTANTS, SOURCES, SYMPTOMS, VEGETATION AFFECTED, INJURY THRESHOLDS, AND CHEMICAL ANALYSES

Pollutants	Source	Symptom	Type of leaf affected	Part of leaf affected	Injury threshold[a]				Chemical analysis for pollutants in plants
					ppm	$\mu g/m^3$	Sustained exposure	Reference	
Ozone (O_3)	Photochemical reaction of hydrocarbon and nitrogen oxides from fuel combustion, refuse burning, and evaporation from petroleum products and organic solvents.	Fleck, stipple, bleaching bleached spotting, pigmentation, growth suppression, and early abscission. Tips of conifer needles become brown and necrotic.	Old, progressing to young	Palisade	0.03	70	4 hours	7	None
Peroxyacetyl nitrate (PAN)	Same sources as ozone	Glazing, silvering, or bronzing on lower surface leaves.	Young	Spongy cells	0.01	250	6 hours	13	None
Nitrogen dioxide (NO_2)	High-temperature combustion of coal, oil, gas, and gasoline in power plants and internal combustion engines.	Irregular, white or brown collapsed lesion on intercostal tissue and near leaf margin.	Middle-aged	Mesophyll cells	2.5	4700	4 hours	15	None
Sulfur dioxide (SO_2)	Coal, fuel oil, and petroleum.	Bleached spots, bleached areas between veins, bleached margin, chlorosis, growth suppression, early abscission, and reduction in yield.	Middle-aged	Mesophyll cells	0.3	800	8 hours	24	b
Hydrogen fluoride (HF)	Phosphate rock processing, aluminum industry, iron smelting, brick and ceramic works, and fiber-glass manufacturing.	Tip and margin burn, chlorosis, dwarfing, leaf abscission, and lower yield.	Mature	Epidermis and mesophyll	0.1 (ppb)	0.2	5 weeks	2	Distillation and titration
Chlorine (Cl_2)	Leaks in chlorine storage tanks; hydrochloric acid mist.	Bleaching between veins, tip and margin burn, and leaf abscission.	Mature	Epidermis and mesophyll	0.10	300	2 hours	34	b
Ethylene (CH_2)	Incomplete combustion of coal, gas, and oil for heating, and automobile and truck exhaust.	Sepal withering, leaf abnormalities; flower dropping, and failure of flower to open properly.	(Flower)	All	0.05	60	6 hours	35	None

[a]Metric equivalent based on 25°C and 760 mm mercury.

[b]Chemical analysis often is not reliable for diagnosing chloride or sulfate accumulation in leaf tissue because undamaged plants often contain higher concentrations of these pollutants than are found in damaged plants.

REFERENCES

1. Tetzlaff, F. S., M. Rogers, and S. Edelman, "Guiding Principles—State Air Pollution Legislation," presented at 87th meeting of American Public Health Association, Atlantic City, N.J., October 23, 1959.

2. Thomas, M. D., "Effects of Air Pollution on Plants," World Health Organization, Monograph Series, No. 46, Columbia University Press, New York, 1961.

3. Middleton, J. T., J. B. Kendrick, Jr., and E. F. Darley, "Airborne Oxidants as Plant-Damaging Agent," National Air Pollution Symposium, Stanford Research Institute, 1955.

4. Heggestad, H. E., R. R. Burleson, J. T. Middleton, and E. F. Darley, "Leaf Injury on Tobacco Varieties Resulting from Ozone, Ozonated Hexene-1, and Ambient Air of Metropolitan Areas," Int. J. Air and Water Pollution, 8:1-10, January 1964.

5. Richards, B. L., J. T. Middleton, and W. B. Hewitt, "Air Pollution with Relation to Agronomic Crops: V. Oxidant Stipple of Grape," Agron. J., 50:559-561, 1958.

6. Darley, E. F., C. W. Nicholas, and J. T. Middleton, "Identification of Air Pollution Damage to Agricultural Crops," Bulletin, Dept. of Agriculture, State of California, Volume 55, 1966.

7. Heck, W. W., J. A. Dunning, and I. J. Hindawi, "Interaction of Environmental Factors on the Sensitivity of Plants to Air Pollution," JAPCA, 15:511-515, November 1965.

8. Hill, A. C., M. R. Pack, M. Threshow, R. J. Downs, and L. G. Transtrum, "Plant Injury Induced by Ozone," Phytopathology, 51:356-363, June 1961.

9. Ledbetter, M. C., P. W. Zimmerman, and A. E. Hitchcock, "The Histopathological Effects of Ozone on Plant Foliage," Contributions of Boyce-Thompson Institute 20(4):225-282, Oct.-Dec. 1959.

10. Heggestad, H. E., and H. A. Menser, "Leaf Spots-Sensitive Tobacco Strain Bel W-3, A Biological Indicator of the Air Pollutant Ozone," Phytopathology, 52:735, August 1962.

11. Taylor, O. C., and F. M. Eaton, "Suppression of Plant Growth by Nitrogen Dioxide," Plant Physiol., 41:132-135, January 1966.

12. Bush, A. F., R. A. Glater, J. Dyer, and G. Richards, "The Effects of Engine Exhaust on the Atmosphere When Automobiles are Equipped with Afterburners," Dept. Eng. Report No. 62-63, Univ. of Calif., L. A., pp. 1-35.

13. Darley, E. F., W. M. Dugger, J. B. Mudd, L. Orchi, O. C. Taylor, and E. R. Stephens, "Plant Damage by Pollution Derived from Automobiles," A.M.A. Arch. Environmental Health, 6:761-770, June, 1963.

14. Middleton, J. T., E. F. Darley, and R. F. Brewer, "Damage to Vegetation from Polluted Atmospheres," JAPCA, 8:9-15, May 1958.

15. Taylor, O. C., "Oxidant Air Pollutants as Phytotoxicants," Presented at 57th Annual Meeting of Air Pollution Control Association, Houston, Texas, June 1967.

16. Hindawi, I. J., J. A. Dunning, and C. S. Brandt, "Morphological and Microscopical Changes in Tobacco, Bean, and Petunia Leaves Exposed to Irradiated Automobile Exhaust," Phytopathology, 55:27-30, January 1965.

17. Altshuller, A. P., S. L. Kopczynski, W. Lonneman, T. L. Becker, T. A. Bellar, and I. J. Hindawi, "Air Pollution Aspects of the Propylene-Nitrogen Oxides Photochemical Reaction," presented at 149th National ACS Meeting, Detroit, Michigan, May 1965.

18. Haagen-Smit, A. J., and M. M. Fox, "Ozone Formation in Photochemical Oxidation of Organic Substances," Ind. Eng. Chem., 48:1484-7, September 1956.

19. Haagen-Smit, A. J., E. F. Darley, M. Zaitlin, H. Hull, and W. H. Noble, "Investigation on Injury to Plants for Air Pollution in the Los Angeles Area," Plant Physiol. 27:18-34, January 1952.

20. Darley, E. F., E. R. Stephens, J. T. Middleton, and P. L. Hanst, "Oxidant Plant Damage from Ozone-Olefin Reactions," Int. J. Air Pollution, 1:155-162, January 1958.

21. Hindawi, I. J., "Natural Pollutants in Staten Island, New York, and Perth Amboy, New Jersey, as Reflected on Vegetation," in: report submitted by Abatement Branch, Division of Air Pollution, Public Health Service, to Commissioner of the City of New York, 1965.

22. Stephens, E. R., E. F. Darley, O. C. Taylor, and W. E. Scott, "Photochemical Reaction Product in Air Pollution," Int. J. Air and Water Pollution, 4:79-100, June 1961.

23. Air Quality Criteria for Sulfur Oxides, U.S. DHEW, NAPCA, Publication No. AP-50, Washington, D.C., March 1967.

24. Thomas, M. D., and R. H. Hendricks, "Effect of Air Pollutants on Plants," in: *Air Pollution Handbook*, ed. P. L. Magill, F. R. Holden, and C. Ackley, pp. 9.1-9.44, New York, 1956.

25. Menser, H. A., and H. E. Heggestad, "Ozone and Sulfur Dioxide Synergism: Injury to Tobacco Plants," Science, 153(3734):424-425, July 1966.

26. Heck, W. W., "Discussion of O. C. Taylor's Paper, "Effects of Oxidant Air Pollutants," Occupational Med., 10:496-499, May 1968.

27. Hindawi, I. J., "Injury by Sulfur Dioxide, Hydrogen Fluoride, and Chlorine as Observed and Reflected on Vegetation in the Field," JAPCA, 18:307-312, May 1968.

28. Hindawi, I. J. "The Product of Photochemical Reaction as Reflected on Vegetation Grown in Brooklyn Botanical Gardens, Willowbrook, New York, Roselle and Bayonne, New Jersey," presented at Cooper Union Colloquium, New York, April 17, 1967.

29. Brewer, R. F., R. C. McColooch, and P. H. Sutherland, Proc. Am. Soc. Hort. Science, 70:259, 1957.

30. Leone, I. A., E. G. Brennan, R. H. Daines, and W. R. Robbins, "Some Effects of Fluorine on Peach, Tomato, and Buckwheat When Absorbed Through the Roots," Soil Science, 66:259-266, December 1948.

31. McIntire, W. H., et al., "Effects of Fluorine in Tennessee Soil and Crops," Ind. Eng. Chem. 41:2466-75, November 1949.

32. Brewer, R. F., et al., "Some Effects of Hydrogen Fluoride Gas on Bearing Navel Orange Trees," Proc. Am. Soc. Hort. Sci., 76:208-214, 1960.

33. Zimmerman, P. W., Proceedings of the First National Air Pollution Symposium, Los Angeles, Stanford Research Institute, p. 135, 1949.

34. Brennan, E., I. A. Leone, and R. H. Daines, "Chlorine as a

Phytotoxic Air Pollutant," Int. J. Air and Water Pollution, 9:791-797, December 1965.

35. Brennan, E. G., I. A. Leone, and R. H. Daines, "Fluorine Toxicity in Tomato as Modified by Alternations with Nitrogen, Calcium, and Phosphorus Nutrition of the Plant," Plant Physiol. 25:736-47, October 1950.

36. Davidson, O. W., "Effects of Ethylene on Orchid Flowers," Proc. Am. Soc. Hort. Sci., 53:440-446, 1949.

37. Middleton, J. T., L. O. Emik, and O. C. Taylor, "Air Quality Criteria and Standards for Agriculture," JAPCA, 15:476-80, October 1965.

38. Croker, W., *Growth of Plants*, Reingold, New York, 1948.

39. Heck, W. W., E. G. Pires, and W. C. Hall, "The Effects of a Low Ethylene Concentration on the Growth of Cotton," presented at 54th Annual Meeting of the Air Pollution Control Assoc., New York, June 1961.

40. Todd, G. W., J. T. Middleton, and R. F. Brewer, California Agr. 10:7-8, 14, 1956.

41. Taylor, O. C., E. A. Cardiff, J. D. Mersereau, and J. T. Middleton, Proc. Am. Soc. Hort. Sci., 71:320-25, 1958.

42. Threshow, M., "Evaluation of Vegetation Injury as an Air Pollution Criterion," JAPCA, 15:266-269, June 1965.

43. Stern, A. C., *Air Pollution and Its Effects*, Academic Press, New York and London, 1968.

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