Effects of Ambient Sulfur Dioxide and Ozone on Eastern White Pine in a Rural Environment

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ABSTRACT

Symptoms observed on current and old foliage of eastern white pine (Pinus strobus) were found in field and laboratory studies to be the syndrome of acute and chronic injury from sulfur dioxide. Occurrences of acute symptoms on susceptible trees in a plantation and on those in adjacent pots were related to increases in the concentration of ambient sulfur dioxide. Sulfur dioxide at a concentration of 6 parts per hundred million of air by volume (pphm) for 4 hr induced acute injury to new needles of field trees and adjacent potted indicator P. strobus plants. Ozone concentrations monitored with sulfur dioxide did not exceed 4 pphm. No injury was correlated with fluctuations in ozone concentrations.

Additional key words: air pollutants, ramets, gas analyzers, lesion severity ratings.

Recently it was demonstrated that atmospheric pollutants can cause injury to conifers (1, 2, 3, 4, 5, 6). Costonis (3) was unable to distinguish acute foliar injury independently induced by sulfur dioxide and ozone on new needles of eastern white pine (Pinus strobus L.) once the affected tissue became necrotic. He demonstrated in controlled laboratory experiments that concentrations of sulfur dioxide as low as 5.0 pphm for 1 hr were sufficient to cause acute foliar injury to sensitive white pines, and he also pointed out that foresters must recognize the potential of small local sources of air pollutants, such as manufacturing industries and coal-burning power plants. Stack emissions from such industries can radically change the makeup of an atmosphere previously free of air pollutants. The opportunity to investigate this problem presented itself in a plantation of eastern white pine in the Pisgah National Forest in North Carolina. The plantation is located about 1.5 miles from such an industrialized area. Several of the trees in the plantation were exhibiting typical symptoms of injury from air pollution. This study was designed to determine whether the injury to eastern white pines growing in the plantation was caused by air pollutants and, if so, to determine the relative importance of sulfur dioxide and ozone.

MATERIALS AND METHODS.—Observations were made on each of 40 pollution-resistant, 40 intermediate sensitive, and 39 highly sensitive trees in a 10-year-old plantation of eastern white pine in the Pisgah National Forest, Pisgah Forest, N.C., in the summer of 1969. These trees had previously been rated for sensitivity to pollutant injury (4). In addition, 10 potted ramets of eastern white pine (scions grafted on 2-year-old nursery-run rootstock) of known susceptibility to sulfur dioxide and ozone and 5 pollutant-resistant ramets were maintained in a plastic greenhouse under positive pressure with the air filtered for the removal of sulfur dioxide and oxidants. The use of ramets provided genetic homogeneity throughout the study. The ramets, placed in 1-gal metal pots containing forest topsoil, were watered to field capacity each day. Temperature in the greenhouse varied from a min of −2 °C at night to a max of 38 °C during early afternoon hr.

Preliminary laboratory tests had previously shown that sensitive ramets developed pink necrotic lesions on the new needles after exposure to sulfur dioxide at concentrations as low as three parts/hundred million of air by volume (pphm) for 1 hr. At the same concentration for a 3-hr exposure, the current needles developed severe necrosis. Under similar environmental conditions, at least 15 pphm of ozone for 4 hr was required to induce equivalent injury on these same ramets. Resistant ramets were uninjured by any treatments.

When the current year’s foliage (1969) was approximately 3 weeks old, the ramets were removed from the greenhouse and sunk to the pot rims in the field plot. Two branchlets/ramet were tagged and observed for injury during the remainder of the growing season. A completely randomized block design was employed throughout the study. The field tree (or ramet) was the block, the branchlet the test unit, and the fascicle the sampling unit. For data collection, three branch-
lets/tree were randomly selected and tagged. The basal scales of 10 current season's fascicles were randomly selected and marked on each branchlet. Data were recorded on the plantation trees and potted ramets at approximately monthly intervals from the time of needle emergence (20 May) until cessation of needle elongation (30 August). Data enumerated were as follows: exposed length of the new fascicle; color according to the Munsell scale (8); occurrence of new symptoms; severity of new and older lesions; and percentage of 1-year-old needles retained. To determine needle retention, 10 randomly selected 1-year-old fascicles on each branchlet were marked as previously described and the number remaining was recorded at each observation period. Trees were under observation almost daily. Severity of foliar symptoms was quantified by the use of numerical scores, as follows: 1 = no visible symptoms; 2 = minute silvery flecks; 3 = yellow lesions up to 3 mm long (accompanied by resin secretion and a collapse of the stomatal face); 4 = pink lesions 3-5 mm long; 5 = brown lesions 3-5 mm long; 6 = tip necrosis of up to 1 cm of the tip; 7 = tip necrosis involving 1-2 cm of the needle tip; and 8 = tip necrosis involving 2-3 cm of the needle tip. A fascicle was considered symptomatic if any of its needles had lesions, and the most severe lesion was the one rated. Fascicles were examined in the field with a ×10 hand lens or in the laboratory with a stereo-microroscope at magnifications up to ×200.

Just prior to elongation of new needles, three branchlets on each of five resistant, five intermediately sensitive, and five sensitive trees were tagged and marked as previously described. Two branchlets/tree were enclosed in polyethylene bags which had been shown by preliminary testing to be impermeable to sulfur dioxide and ozone. The one remaining branchlet was left exposed to ambient air. The bags were fastened in an upright position with a piece of twine to ensure that the foliage did not contact the sides of the bags (foliage is severely burned if it contacts the bags). The bags were opened only when necessary to drain condensate. All branchlets were tagged on 22 May 1969, and the bags were removed on 30 August 1969.

Ambient sulfur dioxide and oxidant in the study area were monitored continuously from 22 May to 31 August 1969, with Atlas iodometric sulfur dioxide and ozone analyzers (Atlas Electric Devices Company, Chicago, Ill. 60613). These analyzers are equipped with preferential filters which remove ozone in the sulfur dioxide sequence and sulfur dioxide in the ozone sequence.

Results.—Symptoms.—Field studies and laboratory work strongly indicated that the injury observed on eastern white pine in Pisgah Forest, N. C., was caused mainly by sulfur dioxide. Symptoms artificially induced on new needles of sensitive trees with sulfur dioxide are similar to those resulting from injury in ambient air containing phytotoxic concentrations of sulfur dioxide. Lesions initially develop as slightly collapsed areas on the stomatal faces of the needle, and are accompanied by an internal resin secretion. Lesions may be produced as soon as the current needles emerge from the bud scales. Lesions may be seen as early as 8-24 hr after a fumigation, and are best seen against a black background with a stereoscopic microscope at ×60-120. Within 24-48 hr after fumigations, lesions become readily apparent to the naked eye as pink spots on the stomatal faces of the needles. Collapsed resin-soaked areas are then apparent on the nonstomatal faces of the needles. Necrosis of the needle tissue progresses distally in the needle. Injury is most severe, and usually initiated in the semimature tissue (7). Injured tissue changes in color from an olivaceous green (5 Y 5/4 on the Munsell scale) to light brown (2.5 Y 7/6), and finally to bright orange-brown (2.5 Y 4/6) about 2 weeks after fumigation. It is common to find all stages of lesion development on needles within a single fascicle.

Eastern white pines vary greatly in sensitivity to injury from sulfur dioxide. On the extremely sensitive trees at Pisgah Forest, the current year's needles were often chlorotic, with mean lengths as short as 46 mm at maturity. Often only the current needles were present on these trees after July (Fig. 1). By early July, one or more needles in most new fascicles on these trees was necrotic along the distal fourth to half, and most other needles had several yellow-to-brown lesions. Severely injured new needles were often cast singly or in fascicles as early as the latter part of August.

On the trees immediately sensitive to sulfur dioxide, new needles often attained a mean length of 58 mm at maturity. Close examination of the stomatal faces of needles from these trees revealed abundant yellow-to-brown lesions. When sulfur dioxide concentrations rose high enough during the period of rapid needle elongation, some necrosis of the needles developed. Generally, more 1-year-old needles were retained by trees of intermediate sensitivity than by highly sensitive trees (Fig. 1).

Resistant trees were easily recognized because of their generally high degree of vigor. The current year's needles attained a mean length of 72 mm at maturity and were dark green. Often, 2 years' needles were retained. The 1- and 2-year-old needles did not start casting profusely until mid-September, although some casting of the 1-year-old needles did occur during the growing season (Fig. 1). Symptoms ranging from inconspicuous flecks to yellow spots were seen on needles of all age classes.

Relationship of symptoms to fluctuations in ambient sulfur dioxide and ozone.—During the growing season, discrete new lesions developed 7 times subsequent to recorded increases in the concentration of ambient sulfur dioxide (Fig. 2). There were several other occurrences of phytotoxic concentrations of sulfur dioxide while new needle tissue was sensitive, but fumigations occurred so close together in time that no new semimature tissue was available to be injured. After the severe fumigations of 6-10 June, necrosis of the distal portions of the current year's needles on highly sensitive trees was conspicuous, while needle tip necrosis developed on some trees of intermediate sensitivity. Some resistant trees were also affected, but lesions on these trees did not progress beyond flecks to yellow
spots. During the growing season, trees of each susceptibility class were injured simultaneously when phytotoxic concentrations of ambient sulfur dioxide occurred (Fig. 2, 3). Repeated fumigations caused a coalescence and intensification of existing lesions.

Average daily concentrations of sulfur dioxide as low as 6-10 ppm for a 4-hr period during the period of prime needle sensitivity, which is a 6- to 8-week period beginning about 1 week after needle emergence, were toxic to new needles of eastern white pine (Fig. 2). Concentrations of sulfur dioxide of 6-10 ppm occurred on 26% of the days during this period of high sensitivity. No relationship was detected between increases in ambient ozone and injury to needles on the trees under study. Ozone concentrations were generally low during the period of prime needle sensitivity. The highest 4-hr average, which occurred only twice during this time period, was only 4.0 ppm. On 33 of the days of record, ozone concentrations averaged between 1-1.9 ppm.

From their introduction on 4 June until the end of the growing season, all the sensitive ramets developed symptoms typical of acute sulfur dioxide injury simultaneously with sensitive field trees (Fig. 3). All the resistant ramets remained free of injury.

Isolation of foliage within polyethylene bags.—No lesions characteristic of sulfur dioxide injury developed on isolated foliage, whereas, on the same trees, foliage exposed to ambient air developed symptoms typical of such injury. Current season's needles on isolated branchlets were generally darker green than exposed needles, and grew at comparable rates until about the 2nd week in August, when most new needles within the bags became somewhat lighter green as they matured.

Discussion.—The symptoms observed on injured trees were apparently induced by brief exposures to concentrations of sulfur dioxide in the range of 6 to 10 ppm for a 4-hr daylight period. Symptoms observed appeared to be identical with those induced on eastern white pine in laboratory tests; in those tests, clones sensitive to sulfur dioxide were subjected to controlled fumigations employing time-concentration combinations of sulfur dioxide of similar magnitude to those monitored in the ambient atmosphere. Concentrations of sulfur dioxide which averaged 6 ppm or

Fig. 1-3. 1) Retention of 1-year-old needles on eastern white pines resistant, intermediate sensitive, and highly sensitive to sulfur dioxide. Data are from 1,250 fascicles on 40 resistant trees, 1,250 fascicles on 40 inter-
higher for a 4-hr daylight period occurred on 26% of the days of prime sensitivity of new needles, a period of approx 8 weeks beginning about 1 week after needle emergence. These field data corroborate recent laboratory results (3) which demonstrated that sulfur dioxide at concentrations as low as 5 ppm for 1 hr was toxic to new needles of strains of eastern white pine sensitive to sulfur dioxide.

The syndrome induced by low concentrations of ambient sulfur dioxide is remarkably similar to the syndrome recently described for ozone (4). With continuous records of ambient gases and daily field observations, however, it should be possible to determine the gas causing the most injury in a particular situation.

This study has further demonstrated that in a typical population of planting stock of eastern white pine, many of the trees can be expected to be sensitive in varying degrees to injury by low concentrations of sulfur dioxide. Concentrations of 6-10 ppm or higher for 2-4 hr can be encountered near certain industries, and, if air drainage is poor, sulfur dioxide can accumulate to toxic levels before dissipation.

LITERATURE CITED