

Air Pollution Toxicity to Eastern White Pine in Indiana and Wisconsin

ROLAND W. USHER, Biologist, and WAYNE T. WILLIAMS, Plant Pathologist, The Institute of Ecology, Indianapolis, IN 46208

ABSTRACT

Usher, R. W., and Williams, W. T. 1982. Air pollution toxicity to eastern white pine in Indiana and Wisconsin. *Plant Disease* 66:199-204.

Air pollution disease was widespread on eastern white pine (*Pinus strobus*) in Indiana, affecting tolerant as well as sensitive genotypes. Tip necrosis of needles was more prevalent than needle flecking and chlorotic mottling, but both symptom types were considered primary manifestations of air pollution disease. Premature needle abscission was widespread and more common in areas with severe disease. Abscission was not as closely associated with overall disease severity as other symptoms, including chlorotic mottling and flecking, percentage of needles with tip necrosis, and length of necrosis; all of these symptoms correlated with disease severity better than abscission, according to linear regression analyses. A disease index composed of these four components reflected changes in disease severity from 1979 to 1980 and within and among study sites. Significant differences in disease severity between 1979 and 1980 corresponded to significantly different air pollution loads in 1978 and 1979. Changes in frequency distribution of the principal index components are discussed in relation to changes in disease severity along a gradient of differing pollution concentrations and durations. Ozone concentrations and durations of episodes were sufficient to elicit phytotoxicity numerous times each growing season in the region. Sulfur dioxide concentrations considered to be phytotoxic were also common at urban sites and rural areas downwind from coal-fired power plants. Toxicity on trees in south central Wisconsin was compared with that on Indiana trees.

In the middle United States east of the Mississippi River, ozone is generated and moves downwind from numerous urban and industrial sources of nitrogen oxides and hydrocarbons. As weak, high-pressure anticyclones move northeastward from Texas or southeastward from Canada towards the Atlantic coast (5,18,32), polluted air masses with ozone (O_3) concentrations from 0.07 to 0.21 parts per million (ppm) form and pass slowly over the Mississippi and Ohio rivers drainage systems 16 or more times during the growing season (2,9,26,27). These doses are known to be phytotoxic to eastern white pine. Sulfur dioxide (SO_2) emissions, on the other hand, are much more locally distributed, with phytotoxic concentrations of 0.05 ppm and above (23) being limited to areas directly downwind from point sources or

within urban plumes (19,28). The effects of these gaseous pollutants on vegetation on a regional basis would therefore be expected to differ, depending on the location of any given plant community and the accumulated doses during the growing season. Thus, disease symptoms caused by ozone would be expected to be more widespread than those of sulfur dioxide, and multiple effects of pollutants would be expected in urban areas and downwind from point sources.

Numerous studies have demonstrated that many eastern white pine (*Pinus strobus* L.) clones are sensitive to O_3 and SO_2 under present ambient concentrations east of the Mississippi River (3,4,6-8,10,11,13,16). Because of this known sensitivity, formation of visible symptoms of injury when exposed to low doses, comparative abundance in native stands and plantings, and easy access to foliage, this species was selected to serve as a bioindicator of the potential impact of gaseous air pollutants on vegetation in Indiana. Nine stands with suspected differing pollutant exposures were studied in various parts of Indiana in 1979 and 1980. In addition, four stands were surveyed for foliar injury in 1980 in south central Wisconsin where air pollution impacts were suspected to be minor. The Wisconsin sites were four of 15 previously sampled by Karnosky (13) from 1971 to 1977.

Stands were selected to determine whether foliar disease apparently induced

by air pollutants could be associated with rural ozone; point source emissions from neighboring large, coal-fired power plants (in conjunction with rural ozone); and air pollution in urban areas. Our second objective was to determine whether a gradient in disease severity existed from rural to urban localities. The intensity of air pollution injury was studied by comparative analysis of the primary visible symptoms of disease to determine which of those parameters or combinations thereof could be used best in assessing the intensity of air pollution disease on *P. strobus*.

MATERIALS AND METHODS

Between 16 and 50 eastern white pines at each of 13 sites (19) were examined for symptoms of air pollution stress, including chlorotic mottling and flecking, tip necrosis, and premature defoliation (373 trees observed). These symptoms are commonly associated with O_3 , SO_2 , and gaseous fluoride injury. Foliar symptoms of winter injury, deicing salts, hail, drought, scale insects, aphids, sooty molds, foliar rusts, sawflies, casebearers, physical abrasion, herbicides, mites, and spittlebugs were noted when present and were distinguishable from those symptoms attributable to air pollution (12,15). Scale insect and winter injury symptoms are most similar to those produced by experimental and ambient fumigations with O_3 and SO_2 .

Symptoms of all types were assessed on 1-yr-old needles from approximately 10 different branches from all sides of each tree at a height of 1-3 m. Trees in plantations were of similar ages and ranged in diameter of breast height from 3 to 45 cm. Needle retention for 2-yr-old needles was determined to a height of at least 6-8 m and was estimated from branches in the upper and lower crowns. If symptom classification for the majority of needles was uncertain, trees were placed in a less severe category. When doubt existed as to the attribution of symptoms to air pollution, air pollution was not indicated in the assessment.

An index of injury was developed to summarize the severity of pollutant injury to individual trees and stands (Table 1). This index was calculated by assigning numeric values to the sub-categories of the variables of air pollution disease mentioned above. These values

Present address of first author: Eli Lilly & Co., Greenfield Labs, Greenfield, IN 46140. Present address of second author: Black Apple Institute, 920 St. Francis Drive, Petaluma, CA 94952.

Research supported in large part by the U.S. Environmental Protection Agency through Contract A805588 to the University of Illinois for the Ohio River Basin Study.

Accepted for publication 1 May 1981.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

0191-2917/82/03019906/\$03.00/0
©1982 American Phytopathological Society

were then multiplied by the percentage of trees in a stand that belonged to that subcategory. Scores of each subcategory were summed to produce an index of injury for each category, and the category scores were summed to produce a total index of injury for each stand. These indexes for all types of symptoms induced by air pollution were given equal weight, with 80 being the lowest possible index (denoting no visible air pollution disease) and 400 being the highest (denoting severe disease).

A tree with severe air pollution disease would be dwarfed, have shortened needles, retain none of the second-youngest complement of needles, have needles with necrosis more than 5 mm in length on 90–100% of the 1-yr-old foliage, and have severe mottling or chlorosis on portions of the needles that are not necrotic. Trees with index values of 380–400 were considered severely diseased (Table 1).

Pines in the nine stands in Indiana were sampled between 15 May and 5 June 1979 and between 19 and 30 May 1980. The four stands near Portage, WI, were examined on 4 and 5 June 1980. At all sites, candles and new needles were elongating. Sources of O₃ and SO₂ data were the Indiana State Board of Health for five monitoring sites in Indiana and the University of Wisconsin Water Resources Center for two monitoring sites in Wisconsin.

Because of the geographic extent of sampling sites and the known genetic variability of *P. strobus* with regard to air pollution sensitivity (8), disease index variances for the 13 sites in 1980 were tested for homogeneity via Bartlett's

technique (25). The variances were shown to be homogeneous, demonstrating homoscedasticity (χ^2 of 10.45; critical $\chi^2_{0.01(12)} = 26.22$). In an initial analysis of variance, disease indexes for each Indiana site were log-transformed. The resultant F-ratio was only slightly lower than data not transformed, and it was still significantly different. Therefore, the test populations were considered to be normally distributed, and the original index values were analyzed. The Bloomington 1979 data set was excluded from this analysis because of suspected observer error.

Frequency distributions for disease indexes (Fig. 1) and each of the four principal components of the index (percentage of needle retention, percentage of needle necrosis, length of necrosis, and degree of needle chlorosis and flecking) were graphed. The four components were then described for population skewness (coefficients of skewness = g_1), and g_1 statistics were tested for association with the overall index value via linear regression tests (Figs. 2 and 3).

In Figure 2, a positive coefficient of skewness indicates fewer and less severe disease symptoms per sample, and a negative coefficient indicates a more severe disease situation per component measured in the site population; the reverse is true in Figure 3, where positive coefficients indicate more severe disease. A g_1 of zero indicates a normal distribution, which in the case of flecking and chlorosis indicates that most trees were categorized as having slight to moderate mottling and flecking on most needles (Table 1; Fig. 2, Carmel).

Thus, the distribution of injury within each site for each principal component was assessed, and if a gradient of disease severity existed between sites, quantification of the differences was described. This method also permitted assessment of the relative contribution of individual disease symptoms to the overall disease index. General climatic and edaphic parameters were not considered in this paper.

RESULTS

Reactions to air pollution. Symptoms of air pollution disease were present at all locations visited (Tables 2 and 3). At the Indiana sites, 99.6% of the pines showed symptoms of air pollution disease. There

were significant differences in disease intensity among sites [$F = 22.43$; $F_{(12,360)} = 2.74$]. For the Indiana sites, air pollution disease was significantly worse in 1979 than in 1980 [$F = 5.93$; $F_{05(1,16)} = 4.49$; $F_{025(1,16)} = 6.12$] (Fig. 1, Table 2). According to the Student-Newman-Keuls multiple range test, no site couplets were different from year to year; the Duncan's test, however, showed that the Breed 2 and Lizton sites were significantly more diseased in 1979 than 1980. Importantly, the differences between annual disease expression corresponded to significantly different air pollution loads (Table 4) during the growing seasons for the two years.

Trees were significantly healthier in central Wisconsin than Indiana (Fig. 1, Tables 2 and 3) when site indexes were compared with Duncan's and the Student-Newman-Keuls multiple range tests. In Indiana over the 2-yr period, 80.5% of the trees showed tip necrosis on 20% or more of their needles, whereas only 14% of the pines observed had no needle necrosis. Fifty-four percent of the trees had more than 1 mm of tip necrosis on most of their needles, and 81% of the trees had symptoms of chlorotic mottling or flecking on a majority of needles (Table 3). Most trees with needle necrosis had 1–5 mm of dead tissue. In both years, 8% of the trees had needle necrosis greater than 5 mm and were classified as severely diseased. On sensitive trees in Indianapolis, 1-yr-old needles became diseased and abscised as soon as 22 June 1980. Up to that time during the 1980 growing season, the mean maximum hourly O₃ concentrations did not exceed 206 $\mu\text{g}/\text{m}^3$ (0.105 ppm) and the SO₂ concentrations did not exceed 66 $\mu\text{g}/\text{m}^3$ (0.024 ppm) (Indianapolis Air Pollution Board, 1980). Environmental variables that might alter air pollution disease expression were unaccounted for in this study; nevertheless, the disease was present in all stands surveyed.

Frequency distribution analysis. The air pollution disease index allowed us to resolve the different population disease responses of *P. strobus* within and among study areas. Quantification of individual symptoms permitted further analyses of those symptoms that best described overall disease intensity. The following results demonstrate the ways in which the pines differed in response to the ambient

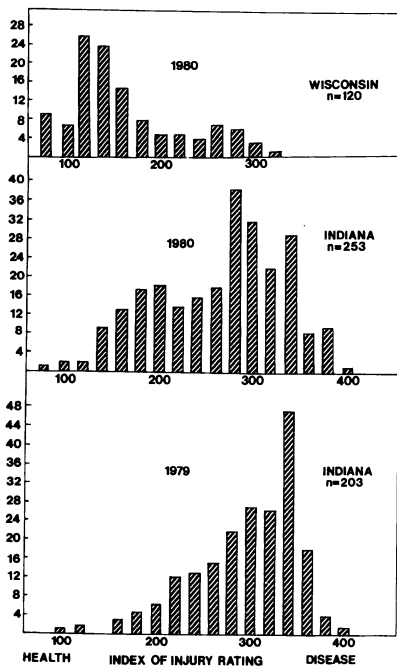


Fig. 1. Frequency histogram of disease index scores for Indiana sites during 1979 and 1980 and Wisconsin sites in 1980.

Table 1. Components of disease indexes for air pollution damage to eastern white pine in Indiana

Component	Numeric value				
	20	40	60	80	100
Mottling/flecking (most needles)	None	Very slight	Slight	Moderate	Severe
Needles necrotic, %	None	1–19	20–49	50–89	90–100
Length of necrosis, mm (most needles)	None	<1	1	1–5	>5
Retention of needles, %	>60	40–59	20–39	1–19	None
Index value	80	160	240	320	400
Disease category	None	Slight		Moderate	Severe

air pollution in terms of fluctuations in response of the overall index and each of the four components.

At the Wisconsin sites, most trees had only slight symptoms of air pollution disease; a subpopulation of 14% of trees was more severely diseased (Fig. 2). These more severely diseased trees, which appeared randomly in the stands, apparently constituted hypersensitive genotypes encountered in other studies (8,13).

In Indiana, the frequency distributions of disease indexes approached a normal distribution in 1980 but were skewed toward more disease in 1979 (Fig. 1). A remnant subpopulation of 8% remained resistant in 1979 but fluctuated in disease expression in 1980. The fluctuation in disease expression would be expected if the pollution load changed or if environmental conditions were more conducive to disease expression in 1979.

Flecking and chlorotic mottling. Flecking and chlorotic mottling on 1-yr-old foliage of *P. strobus* were identical to the classic initial manifestation of disease induced by oxidant air pollution on many kinds of conifers (1,7,22,31). At all sites, 1-yr-old needles were a better indicator of oxidant injury than current year foliage because of a greater exposure time to air pollutants. For example, in Indianapolis on 27 June 1980 an air pollution episode occurred with O_3 at 0.135 ppm and SO_2 at 0.01 ppm. At an additional plantation in Indianapolis, the second complement of foliage (1 yr old) became typically chlorotic mottled and yellowed rapidly in a few days. The current year needles were just completing elongation and had only minor chlorotic mottling, tip chlorosis, and tip burning. The next thunderstorm blew off most of the yellowed needles of the second complement, whereupon the trees appeared to become greener because of the abscission of the more diseased tissue. This is the suggested mode of action for premature leaf fall in *P. strobus* and other conifers (20,30,31) and has been observed with other tree species (21).

The amount of flecking and chlorotic mottling among sites increased with increasing disease severity (Fig. 2), with more trees per site having more severe flecking and chlorotic mottling at those sites with more disease.

Percentage of needles with necrosis. There was an obvious gradient in distribution of percentage of needles with necrosis attributed to air pollution ($r = -0.899$) between the Wisconsin sites (healthy) and the Indiana urban sites (diseased). The two healthiest Wisconsin sites had the least needle necrosis, whereas the urban sites in Indiana, with more complex and concentrated air pollution loads, tended to have more needle necrosis. There was a strong tendency for the frequency of distribution between stands to shift towards more severe disease as the disease index, and

presumably the pollution load, increased.

At the four Wisconsin sites, 67.5% of the second year needles had no needle necrosis; however, on 12.5% of the white pines 90-100% of the needles had necrosis to some degree, and the mean distribution

of necrosis in Wisconsin was strongly skewed ($g_1 = +1.83$).

The Indiana sites had a mean g_1 value of -0.932 , indicating that the majority of the white pines had needle necrosis. At Lizton (1980 $g_1 = -0.923$), 32.5% of the

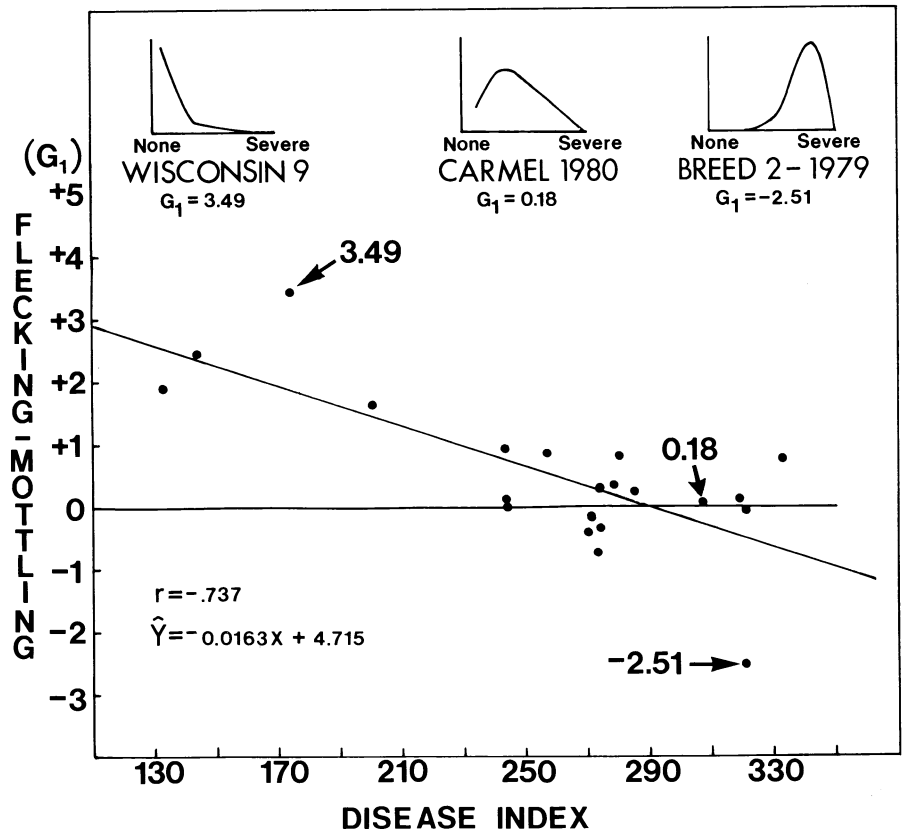


Fig. 2. Linear regression of the coefficients of skewness (g_1) for the symptom of flecking and chlorotic mottling and disease indexes for 13 sites in Indiana (1979 and 1980) and Wisconsin (1980). Inserted graphs display distribution of the symptom per stand in Wisconsin and Carmel and Breed, IN. The g_1 statistics are indicated by arrows.

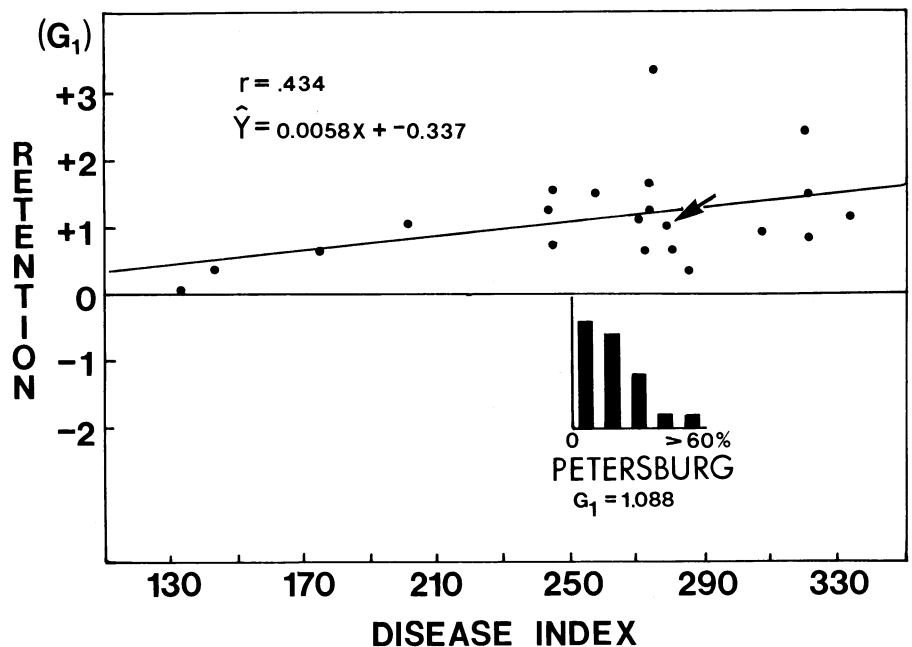


Fig. 3. Linear regression of skewness coefficient (g_1) for needle retention and disease indexes at sites in Wisconsin and Indiana. Inserted graph represents the frequency distribution at the Petersburg, IN, site ($g_1 = 1.088$), which is indicated by the arrow.

trees had necrosis to some degree on 90–100% of their needles, but only 9.3% had no necrosis. Tip necrosis is considered to be an initial symptom of air pollution disease in eastern white pine, and fewer trees had chlorotic mottling and flecking than tip necrosis (Table 3).

Length of necrosis. Length of needle necrosis increased as severity of disease increased, and there were large differences

in the distribution of length of needle necrosis between Wisconsin rural and rural-to-urban sites in Indiana. The linear regression coefficient accounts for 80% of the variation about the line ($r = -0.80$).

The Wisconsin sites had five times as many trees without tip necrosis as the Indiana sites (Table 3) and more than 10 times fewer trees with necrosis over 1 mm in length. On a comparative basis, the

Wisconsin sites would be classified as slightly diseased and thus less prone to long-term decline from additional air pollutants and predisposing factors than more severely injured trees growing under Indiana conditions.

Needle retention. Needle retention had less correlation with overall air pollution disease than the other three variables. At all sites, there was a relatively uniform tendency for premature leaf drop, which would be expected if O₃ were contained in homogeneous air masses over the region during several growing seasons. The correlation coefficient for the linear regression of needle retention × disease index was only 0.435 (Fig. 3). F-tests indicated a significant slope towards less retention in sites with more suspected air pollution (rural-cleaner versus urban-contaminated).

Premature needle abscission from air pollution appeared to be a general phenomenon in the region and was not strongly related to disease severity per se, as demonstrated in Figure 3. The g_1 coefficients for all sites were positive, indicating a general skewness towards less than 20% needle retention of 2-yr-old needles. Because the needles begin to fall early in the growing season as a response to O₃ episodes, the absence or presence of needles would be relatively more independent in assessing disease severity than other variables that are dependent on the presence of needles. These results indicate that needle abscission is not as good a measure of severity of air pollution disease for eastern white pine as flecking and mottling, percentage of needles with necrosis, or length of needle necrosis.

Gerhold (8) reports that normal needle retention is 27 mo for *P. strobus*. At the Indiana sites, 86% of the trees had less than 20% of their 2-yr-old needles, whereas at the Wisconsin sites 49% of the trees had less than 20% retention of this third foliar whorl (Table 3).

Ambient air pollution. Concentrations and durations of O₃ in Indiana and Wisconsin were commonly greater than considered necessary to cause needle chlorosis and necrosis on some eastern white pines (Table 4). Potentially phytotoxic SO₂ concentrations and durations have been measured repeatedly in numerous locations in Indiana (2,9,27). The urban areas of southern Indiana and Indianapolis have levels of SO₂ that regularly exceed 200 µg/m³ during much of the year, and some are greater than 365 µg/m³ (0.074–0.135 ppm) (18,19). O₃ levels were of significantly higher concentrations and durations in 1978 than 1979 [$F_{(1,8)} = 5.357$, $F_{0.05(1,8)} = 5.32$] (Table 4), and the index of injury reflected this air pollution difference (Fig. 1, Table 2).

In Wisconsin, the two ozone monitors (within 5 miles of all study plots and within 2 miles of several plots) varied considerably, with the Messer station

Table 2. Multiple range analysis of site means in 1980 using Duncan's and Student-Newman-Keuls multiple range tests

Geographic aspect	Site	Mean disease index per site		Disease category
		1980 ^a	1979	
Wisconsin				
Rural	Wisconsin 3	132.7		Slight
Rural and power plant	Wisconsin 14	143.7		
Rural and power plant	Wisconsin 9	174.0		
Rural and power plant	Wisconsin 5	200.7		
Indiana				
Rural	Bloomington	243.7	... ^b	Moderate
Rural	Alamo	244.0	256.9	
Rural and power plant	Clifty Creek	244.2	273.0	
Rural and power plant	Breed 2	270.0	321.3 ^c	
Rural and power plant	Breed 1	270.7	274.4	
Rural	Lizton	272.5	321.0 ^c	
Rural and power plant	Petersburg	278.2	280.8	
Urban	Banta	285.7	319.8	
Urban	Carmel	307.2	333.6	

^aBrackets to the left of the column represent maximum insignificant ranges ($P = 0.05$); brackets to the right of the column represent insignificant studentized ranges.

^bNo statistics done on this test site for 1979.

^cSignificant differences between years according to Duncan's multiple range test but not according to the Student-Newman-Keuls test.

Table 3. Percentage of eastern white pine trees at 13 stands in Indiana and Wisconsin with various symptoms attributable to air pollution

Symptom	Trees with symptoms, %	
	Indiana, $n = 457$ (1979–1980)	Wisconsin, $n = 120$ (1980)
Chlorotic mottling/flecking on most needles		
None	18.8	74.2
Very slight	22.1	18.3
Slight	30.0	5.0
Moderate	19.9	2.5
Severe	9.2	0.0
Tip necrosis on		
None of foliage	13.8	67.5
> 20% of foliage	80.5	26.7
Most foliage	70.6	21.0
Length of tip necrosis on most needles		
None	29.4	79.0
1 mm or less	16.7	15.8
1–5 mm	45.8	5.0
> 5 mm	8.1	0.0
Second year needles retained		
None	55.8	18.3
1–19%	30.8	30.8
20–39%	8.7	30.0
40–59%	3.0	11.7
> 60%	1.5	9.1

consistently recording higher O₃ doses than the Genrich Station (Table 4). The Genrich Station O₃ dose for 1979 was similar to Indianapolis in 1979, but the Messer 1979 dose was substantially higher. These two stations were about equidistant from the study plots, but the Messer site was 150 m higher in elevation than the four study sites. According to Karnosky (13), SO₂ concentrations are common near the Wisconsin sites in the range of 0.004 to 0.05 ppm for 1–3 hr episodes during the summer months. The primary difference in air quality between the Indiana and Wisconsin sites would appear to be the amount of SO₂ present.

No monitoring results for gaseous fluorides were available. Twenty-eight samples of *P. strobus* needles collected from Breed 2, Clifty Creek, and Alamo sites in 1979 indicated no foliar fluoride above that considered normal background level (20).

DISCUSSION

Sufficiently high air pollution concentrations existed in rural and urban Indiana to cause extensive foliar injury to eastern white pine. O₃ and SO₂ were considered in this study to be the primary causal agents of the air pollution disease observed. Nitrogen oxides may also be interactive.

Because 99.6% of the trees developed symptoms of disease attributable to air pollution, tolerant as well as sensitive genotypes of white pines were being affected. Chlorosis, necrosis, and premature needle abscission as a result of air pollution stress decrease ornamental and wind shelter values. The intensity of disease observed would be expected to reduce wood and fiber production in forests and plantations (17).

Premature abscission of needles after only 14–17 mo instead of the normal 27 mo would probably reduce wood accretion (14), even without consideration of metabolic stress on the remaining first and second ranks of foliage during pollution episodes. Such an effect is probable because fascicular needles continue to provide substantial amounts of assimilates in their second year and may photosynthesize at up to 65% of their first-year rates into their third year, just before abscission (33). Zelawski (33) stressed that needle retention is very important for productivity. In addition, Wilkinson and Barnes (29) have noted altered carbon dioxide fixation patterns on *P. strobus* with O₃ doses as low as 0.10 ppm for 10 min, and Mann et al (20) have observed photosynthesis diminishment on current and 1-yr-old white pine needles exposed to ambient air pollution. In addition, Mann et al (20) concluded that "differences in growth of symptomatic and non-symptomatic trees appear to be due to reduced foliar biomass resulting from shortened needles and premature needle loss, rather than impaired carbon fixation."

Fluctuations in disease severity attributable to sites were not as pronounced as expected. Stands in rural Wisconsin were not symptom free, and a mosaic of disease existed in Indiana, with the more rural sites distant from known point sources generally being most healthy. Urban plantings in polluted surroundings had the most severe disease condition.

The disease index was sensitive to O₃ fluctuations from year to year. Oxidants were worse in Indiana in 1978 than in 1979, and the air pollution disease on white pine was significantly worse on 1978 than on 1979 needles observed in 1979 and 1980. The combination of high temperatures and stagnant air masses with high concentrations of oxidants and SO₂ appeared to be especially conducive to disease expression. This has been observed elsewhere on pine species exposed to oxidants (4,8,24,30).

With similar amounts of O₃ in the regional polluted air mass, it is surprising that there were such differences in air pollution disease expression between Wisconsin and Indiana. Four factors may be responsible: i) There appeared to be more SO₂ in Indiana than at Wisconsin sites; ii) because Indiana is in the southern part of the range of *P. strobus*, the trees in Indiana may be more sensitive to visible disease expression, especially premature needle abscission; iii) urban areas have more nitrogen oxides, which tend to quench ozone but can react in combination with SO₂ against vegetation (the five ozone monitors in Indiana were all in urban areas, whereas the Wisconsin monitors were in rural settings; reported O₃ values for Indiana may thus be lower than the actual concentrations affecting

the majority of study plots); and iv) greater relative humidity in Indiana than in Wisconsin may predispose trees to air pollution injury.

The widespread O₃ load tends to obscure effects from local sources. Further analysis of the principal components of the index per site may enable us to differentiate site effects at a higher resolution. One additional analysis would be to determine the degree of correlation among the four symptom types as an added measure of confidence in diagnosing air pollution injury.

The broad geographic occurrence of air pollution symptoms on eastern white pine was evidence that O₃ was affecting vegetation on a regional basis. This was supported by observations of visible air pollution symptoms in Indianapolis on red pine (*Pinus resinosa* Ait.), Norway spruce (*Picea abies* (L.) Karst.), Colorado blue spruce (*Picea pungens* Engelm.), sycamore (*Platanus occidentalis* L.), tulip poplar (*Liriodendron tulipifera* L.), sweet corn (*Zea mays* L.), green beans (*Phaseolus vulgaris* L.), milkweed (*Asclepias* sp.), possibly black locust (*Robinia pseudoacacia* L.), sassafras (*Sassafras albidum* (Nutt.) Nees), and black oak (*Quercus velutina* Lam.).

Continued stress from air pollutants on eastern white pine can be expected to cause changes in the ecosystem status of this forest species through depauperization of genotypes, because only 5.7% of the trees can be categorized as being insensitive (0.4% of the trees in Indiana had no visible symptoms). Berry (3) noted that 4.5% of his selected white pine clones were insensitive to air pollution. It is important to consider that tolerant genotypes are not immune and that forest

Table 4. Number of days with episodes^a of ozone ≥ 0.08 ppm and number of hours with ozone ≥ 0.08 or 0.10 at five sites in Indiana and two sites in Wisconsin during May–August in 1978 and 1979

Site	Episodes of ozone ≥ 0.08 ppm (no. of days)	Ozone ≥ 0.08 ppm (hr)	Ozone ≥ 0.10 ppm (hr)	Days with data (%)
Indiana				
Indianapolis Women's Prison				
1978	38	301	63	100
1979	16	117	8	100
New Albany				
1978	19	148	38	72
1979	4	28	1	61
Hammond				
1978	38	259	67	97
1979	28	75	8	87
South Bend				
1978	8	65	9	52
1979	13	116	26	81
Fort Wayne				
1978	27	182	33	48
1979	0	7	5	57
Wisconsin				
Genrich				
1979	16	114	16	100
Messer				
1979	33	318	95	100

^a Episode = three or more continuous hours at or above 0.08 ppm.

tree species reduced in populations or biomass by air pollution are not necessarily replaced by other species of similar physiognomy. Selection against pollution-sensitive tree strains will not necessarily favor those niches being filled by pollution-tolerant tree species. Thus, the native forests and plantings of eastern white pine in polluted airsheds might not provide their full potential benefits to society.

ACKNOWLEDGMENTS

We wish to express thanks to J. P. Bennett and B. G. Woodruff for advice on statistical methods, Melanie Nelson for graphics presentation, and O. L. Loucks for manuscript review.

LITERATURE CITED

1. Anderson, R. L., and Dochinger, L. S. 1978. How to identify white pine susceptible to air pollution. U.S. For. Serv. Northeast. For. Exp. Stn. Delaware, OH.
2. Anonymous. 1969 Air quality criteria for particulate matter. U.S. Dep. Health Educ. Welfare, Public Health Serv. Pub. AP49; National Ambient Air Quality Standards 40 CFR 550.6.
3. Berry, C. R., and Hepting, G. H. 1964. Injury to eastern white pine by unidentified atmospheric constituents. For. Sci. 10(1):213.
4. Blanchard, R. O., Baas, J., and van Cotter, H. 1979. Oxidant damage to eastern white pine in New Hampshire. Plant Dis. Rep. 63:177-182.
5. Bowen, B. B. 1978. A study of the large scale transport of low level ozone across the central and eastern United States. M.S. thesis. University of Wisconsin, Madison.
6. Costonis, A. C. 1970. Acute foliar injury of eastern white pine induced by sulfur dioxide and ozone. Phytopathology 60:994-999.
7. Dochinger, L. S., and Seliskar, C. E. 1970. Air pollution and the chlorotic dwarf disease of eastern white pine. For. Sci. 16:46-55.
8. Gerhold, H. D. 1977. Effect of air pollution on *Pinus strobus* L. and genetic resistance: A literature review. U.S. Environ. Prot. Agency EPA 600/3-77-002.
9. Haering, G. W. 1977. Results of 1975 and 1976

sulfur dioxide and ozone study data analysis. FDL-77-008. Indianapolis Center for Advanced Research, Inc. 88 pp.

10. Hayes, E. M., and Skelly, J. M. 1977. Transport of ozone from the northeastern United States into Virginia and its effects on eastern white pines. Plant Dis. Rep. 61:778-782.
11. Houston, D. B., and Dochinger, L. S. 1977. Effects of ambient air pollution on cone, seed, and pollen characteristics in eastern white and red pines. Environ. Pollut. 12. 5 pp.
12. Johnson, W. T., and Lyon, H. H. 1976. Insects That Feed on Trees and Shrubs: An Illustrated Practical Guide. Comstock Publishing Associates, Ithaca, NY. 464 pp.
13. Karnosky, D. F. 1980. Changes in southern Wisconsin white pine stands related to air pollution sensitivity. Int. Symp. Effects Air Pollut. Mediterr. Temperate For. Ecosystems. June 22-27, University of California at Riverside.
14. Keller, T. 1980. The effect of a continuous spring time fumigation with SO₂ on CO₂ uptake and structure of annual ring of spruce. Can. J. For. Res. 10:16.
15. Lacasse, N. L., and Treshow, M., eds. 1976. Diagnosing vegetation injury caused by air pollution. Developed by Applied Science Associates, Inc., for U.S. Environ. Prot. Agency. 271 pp.
16. Linzon, S. N. 1966. Damage to eastern white pine by sulfur dioxide, semi-mature tissue needle blight, and ozone. J. Air Pollut. Contr. Assoc. 16:140-144.
17. Linzon, S. N. 1966. Economic effects of sulfur dioxide on forest growth. J. Air Pollut. Control Assoc. 21:81-86.
18. Lioy, P. J., and Samson, P. J. 1979. Ozone concentration patterns observed during the 1976-77 long-range transport study. Environ. Int. 2:77-83.
19. Loucks, O. L., Armentano, T. V., Usher, R. W., Williams, W. T., Miller, R. W., and Wong, L. T. K. 1980. Crop and forest losses due to current and projected emissions from coal-fired power plants in the Ohio River Basin. Institute of Ecology, Indianapolis. 266 pp.
20. Mann, L. K., McLaughlin, S. B., and Shriner, D. S. 1980. Seasonal physiological responses of white pine under chronic air pollution stress. Environ. Exp. Bot. 20:99-105.
21. Noble, R. D., and Jensen, K. F. 1980. Effects of

sulfur dioxide and ozone on growth of hybrid poplar leaves. Am. J. Bot. 67:1005-1009.

22. Phillips, S. O., Skelly, J. M., and Burkhart, H. E. 1977. Eastern white pine exhibits growth retardation by fluctuating air pollution levels: Interaction of rainfall, age, and symptom expression. Phytopathology 67:721-725.
23. Roach, M. D. 1980. Montana Ambient Air Quality Standards Study: Final Environmental Impact Statement. 303 pp.
24. Skelly, J. M., Moore, L. D., and Stone, L. L. 1972. Symptom expression of eastern white pine located near a source of oxides of nitrogen and sulfur dioxide. Plant Dis. Rep. 56:3-6.
25. Sokal, R. R., and Rohlf, R. J. 1969. Biometry: The Principles and Practice of Statistics in Biological Research. W. H. Freeman and Co., San Francisco. 776 pp.
26. Teknekron, Inc. 1979. Data base for the evaluation of short-range dispersion models. R-001-EPA-79. Berkeley, CA.
27. U.S. Environmental Protection Agency. National aerometric data bank. Environmental Criteria and Assessment Office. Research Triangle Park, NC.
28. White, W. H., Anderson, J. A., Blumenthal, D. L., Huser, R. B., Gillani, N. V., Huser, J. D., and Wilson W. E., Jr. 1979. Formation and transport of secondary air pollutants: Ozone and aerosols in the St. Louis urban plume. Science 194:187-189.
29. Wilkinson, R. F., and Barnes, R. L. 1973. Effects of ozone on CO₂ fixation patterns in pine. Can. J. Bot. 51:1573-1578.
30. Williams, W. T. 1980. Air pollution disease in the California forests: A base line for smog disease on ponderosa and Jeffrey pines in the Sequoia and Los Padres National Forests, California. Environ. Sci. Tech. 14:179-182.
31. Williams, W. T., Brady, M., and Willison, S. C. 1977. Air pollution damage to the forests of the Sierra Nevada mountains of California. J. Air Pollut. Control Assoc. 27:231-234.
32. Wolff, G. T., and Lioy, P. J. 1980. Development of an ozone river associated with synoptic scale episodes in the eastern United States. Environ. Sci. Tech. 14:1257-1260.
33. Zelawski, W. 1976. Variation in the photosynthetic capacity of *Pinus sylvestris*. Pages 99-109 in: M. G. R. Cannell and R. T. Last, eds. Tree Physiology and Yield Improvement. Academic Press, New York.