SYMPTOMS CAUSED BY PHOTOCHEMICAL AIR POLLUTION INJURIES TO FOREST TREES

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A B S T R A C T

Unexplained needle injuries on eastern white pine and ponderosa pine trees in the forest have been intensively investigated in recent years. Artificial ozone fumigation experiments conducted in the laboratory to duplicate symptoms of injury observed on pine trees were started in Canada in 1959 and in the United States in 1961. Many needle disorders were found to be attributable to atmospheric oxidants of which ozone forms the greatest part. Although symptoms of oxidant injuries on forest trees are specific, confusion may arise in differentiating similar symptoms caused by other agents. For example, semimature-tissue needle blight (SNB) of eastern white pine, a physiogenic disease, produces symptoms closely resembling a portion of those caused by photochemical air pollutants.

This paper describes and compares the symptomatology of ozone injury, chlorotic dwarf, and SNB of eastern white pine, and chlorotic decline of ponderosa pine. The concentrations of atmospheric oxidants and the exposure periods required to cause injury on forest trees are outlined.

The interaction of atmospheric oxidants and sulphur dioxide in causing injury to eastern white pine is also discussed. Additional research is required to explore the action of mixtures of air pollutants in causing injury to forest trees.

I. INTRODUCTION

Injury to vegetation from photochemical air pollution was first observed in the Los Angeles area in 1944 (20). In 1958 (11) atmospheric ozone was found to be the cause of "weather fleck" of tobacco which had caused extensive injury in northeastern U.S.A. and in Ontario, Canada, starting in the early 1950's. Artificial ozone fumigation experiments conducted in the laboratory to duplicate symptoms of injury observed on pine trees in the forest were started in Canada in 1959 (15) and in the United States in 1961 (3).

Ozone is estimated to be responsible for 85 to 90 per cent of the total oxidizing potential of photochemical air pollution (26). Other known phytotoxicants in the smog complex are nitrogen dioxide, peroxyacetyl nitrate (PAN), and sulphur dioxide. Ozone is probably responsible for injuring more plant species than any other phytotoxic air pollutant. Forty plant species have been listed as being relatively sensitive to ozone (14).

Oxidant injury to deciduous hardwood trees is not as well documented as injury found on coniferous trees. Oxidant-type symptoms have been reported to occur on lilac (*Syringa vulgaris*), gingko (*Gingko biloba*) and several species of *Ulmus* in New York City (12). Sugar Maple (*Acer saccharum*) was injured by 20 to 30 pphm ozone for two hours in laboratory experiments, but similar symptoms were not observed on sugar maple trees growing out-of-doors (13). White Ash (*Fraxinus americana*) trees were injured in the laboratory with 10 pphm ozone for 0.5 hours (27).

Needle injuries occurring in white pine (*Pinus strobus*) forests in the east and in ponderosa pine (*Pinus ponderosa*) forests in the west have been the subject of intensive investigations in recent years (3, 5, 8, 17, 23). Many of these disorders have been found to be attributable to atmospheric oxidants. These disorders have been given names and are described in this paper as ozone injury of eastern white pine (5, 17), chlorotic dwarf of eastern white pine (8), and chlorotic decline of ponderosa pine (23). A physiogenic disease affecting eastern white pine is described as semimature-tissue needle blight (SNB) (15), with the injury development resembling a part of the symptom syndrome caused by ozone.

II. AMBIENT AIR OXIDANT CONCENTRATIONS IN THE FOREST REPORTED TO HAVE CAUSED INJURIES ON PINE TREES

Ambient air concentrations of 3 pphm oxidant and over for several hours in eastern United States (5) and in Canada (18) have been found to cause ozone injury symptoms on current-year needles of sensitive eastern white pine trees, and oxidant concentrations of above 15 pphm for approximately 2 hours per day for about 2 months in southern California have been reported to cause chlorotic decline symptoms on one-year-old needles of susceptible ponderosa pine trees(22).

III. OZONE DOSAGES REQUIRED TO CAUSE INJURY ON CONIFEROUS TREES IN ARTIFICIAL FUMIGATION EXPERIMENTS

A number of investigators have attempted to duplicate in the laboratory symptoms of injury observed on pine trees in the forest. Eastern white pine has been fumigated with various dosages of ozone with experiments starting in Canada by Linzon in 1959 (15) and in U.S.A. by Berry and Ripperton in 1961 (3). Injury to eastern white pine was produced by concentrations of ozone as low as 3 pphm for 48 hours (5) in one study, whereas concentrations of 25 pphm for 8 hours (7) and 40 pphm for 2 hours (17) were required in other studies. There are a number of explanations for the widespread differences in ozone dosages required to cause injury in eastern white pine. First there is considerable inherent intraspecific variation among the individual species of eastern white pine. Induced symptoms of ozone injury not only varied from tree-to-tree, but also varied from needle-to-needle, fascicle-to-fascicle, and branch-to-branch on the same tree. Another reason for differences in ozone dosages required to cause injury is apparent from the objectives of the different experiments. Where extremely low concentrations of ozone were responsible for injury the susceptible pine trees were of poor vigor and usually possessed only one year's needles of reduced size on the tree. Higher concentrations of ozone were required to injure white pines of better vigor possessing up to three years of needles of normal size on the trees. These latter trees would be more representative of the forest population. The experiments, however, do show, that strains of eastern white pine exist which are extremely sensitive to air pollution and these are the first to succumb to adverse influences in the forest.

In California, artificial fumigation experiments produced symptoms of injury in ponderosa pine trees similar to those observed in the forest (23). The concentrations of ozone required to cause injury experimentally were higher than the concentrations usually encountered in the forest. However, the period of exposure was less than that required in the forest for susceptible ponderosa pines to develop oxidant injury symptoms.

Artificial ozone fumigation experiments with 18 coniferous species (7) showed that 9 species (spruces, firs, and red pine) were resistant to 25 pphm ozone for 8 hours. The other 9 species exhibited considerable interspecific variation in susceptibility to this dosage of ozone. Virginia pine was most susceptible, followed by jack pine, European larch, Austrian pine, Scotch pine, eastern white pine, eastern hemlock, Japanese larch, and pitch pine in order of decreasing susceptibility.

Table I descibes the ozone dosages required to cause injury on eastern white pine, ponderosa pine, and virginia pine.

IV. GENERAL SYMPTOMATOLOGY OF ABIOTIC INJURIES ENCOUNTERED ON PINE TREES IN THE FOREST

Pine trees in the forest affected by oxidants may display a variety of injury symptoms on their foliage. These symptoms are described as minute silvery flecks, chlorotic flecks, chlorotic mottle, pink spots, pink bands, and tip necrosis (Table 2). In eastern white pine, the overall symptom expression of the disease ozone injury and chlorotic dwarf is different in each case, but some similarities exist. Both injuries affect only the current-year needles, with the older needles senescing early and being cast prematurely. Chlorotic decline of ponderosa pine has symptoms resembling those caused by chlorotic dwarf of eastern white pine, but chlorotic decline affects the older needles first, especially the one-year-old, with the oldest needles being shed prematurely. The physiogenic disease, SNB of eastern white pine affects current-year needles only and the symptoms resemble a portion of those caused by ozone injury. There are, however, some differences between SNB and ozone injury of eastern white pine. The ozone-sensitive trees usually display chlorotic fleck and mottle in addition to distal reddening following exposure to elevated concentrations of ozone. In SNB-susceptible trees the symptoms are typically expressed by a distal reddening only, which can occur during periods of normal levels of ambient concentrations of atmospheric ozone. Histological examination of ozone injury symptoms show mesophyll cell collapse occurring in different locations in the same needle, although it is often more pronounced in semimature needle tissue. In SNB, mesophyll cell collapse initiates only in semimature tissue, which has been defined for the purposes of this disease to be the location in the needle where suberization of the endodermal cells is proceeding.

A more detailed account of the symptom expression and development which would aid in the recognition of these pine injuries is given in Section V.

V. DETAILED DESCRIPTION OF NEEDLE INJURY SYMPTOMS OBSERVED OR INDUCED ON PINE TREES

(1) Ozone Injury of Eastern White Pine

Linzon (17) induced a severe tip necrosis on maturing needles of eastern white pine in laboratory experiments with 40 pphm ozone for 2 hours. In more recent fumigation work (18) Linzon produced chlorotic flecks on new needles of eastern white pine with concentrations of 10 pphm ozone for 6 hrs. In field experiments chlorotic fleck developed on white pine ramets (clonal grafts) exposed to ambient air containing 3 pphm ozone and over, whereas no fleck symptoms developed on ramets which had been covered with polyethylene bags. Berry and Ripperton (3) fumigated newly developing immature needles of eastern white pine with 6.5 pphm ozone for 4 hours and induced a tip necrosis which was called "emergence tipburn". This symptom was described as resembling that caused by the disease needle blight of white

pine (15). Costonis and Sinclair (5) showed that ozone acting on the new needles of certain sensitive strains of eastern white pine could cause a wider range of symptoms, from silvery and chlorotic flecks through chlorotic mottling to tip necrosis. In artifical fumigation experiments this syndrome was induced by controlled doses of ozone as low as 7 pphm for 4 hours or 3 pphm for 48 hours.

The macroscopic symptoms of ozone injury were described (5) as initially occurring as minute silvery flecks radiating from the stomata of current-year needles. These silvery flecks, which are best seen under magnification, develop into larger chlorotic flecks which are visible to the naked eye. The semimature tissue of the needle is the most seriously affected portion of the needle, but the immature and the mature tissues are often simultaneously affected. The chlorotic flecks may develop into pink lesions and bands followed by a distally spreading orange-red necrosis which may take from one to two weeks to reach the needle tips. On less sensitive trees only chlorotic flecks and mottling may occur,whereas severe needle tip necrosis occurs on the most sensitive trees. The microscopic symptoms of ozone injury show that the mesophyll cells adjacent to the stomata are the first to be affected, and the endodermal and stellar cells are the last to be affected.

The new, rapidly growing needles (from about one week after emergence until about six weeks of age) are most sensitive to ozone. Normally, three years of needle ages occur on eastern white pine. However, on ozone-sensitive trees, the older needles become prematurely senescent and discolored, being cast by mid-summer leaving only the current-year needles.

(2) Semimature-Tisssue Needle Blight (SNB) of Eastern White Pine

This disease has been observed affecting inherently susceptible eastern white pine trees since the turn of the century (6). Although numerous causal agents have been postulated as causing the disease, no primary incitant has yet been found. The disease is of physiogenic origin, and the symptoms resemble a portion of those caused by air pollutants (5, 15, 16). Etiological investigations by Linzon (17) demonstrated that SNB-susceptible white pines were tolerant to low levels of ozone.

Chlorotic flecks could be induced on white pine needles with slightly elevated ozone concentrations (10 pphm) but the typical symptoms of SNB did not develop (18). The use of high concentrations of ozone (40 pphm) caused severe needle tip necrosis on both SNB-susceptible and non-susceptible white pines (17).

The macroscopic and microscopic symptoms of SNB were described by Linzon (16). The symptom initiation and development on affected current-year needles are remarkably uniform from fascicle-to-fascicle on the same tree. Although not every needle in a fascicle need be injured, the growth of the entire fascicle is retarded. The initial injury occurs macroscopically on stomata-bearing faces as faint pinkish spots in semimature tissue (about three weeks old). The spots develop

into orange-red bands which spread distally through adjacent, more mature, tissue, reaching the needle tips in a period of a few days. The injury was never observed to spread from semimature tissue to the immature tissue located proximally. Further, the initial injury was never observed in immature or mature needle tissues.

Microscopically, the initiation of SNB occurs in mesophyll cells in semimature needle tissue where suberization of the transverse and radial walls of the endodermal cells is proceeding. Immature needle tissue which occurs below the semimature zone possesses endodermal cells with walls free of suberin. Mesophyll cell collapse spreads from one ventral stomatal face around the adaxial mesophyll tissue to the other ventral stomatal face before involving cells in the non-stomatal dorsal face of the needle. Following the collapse of the mesophyll parenchyma tissue, the endodermal cells and the transfusion cells in the stele are next to become distorted, with the phloem and xylem cells the last to be affected.

Current-year needles possess semimature tissue from about three weeks after emergence until about twelve weeks of age, and are thus susceptible to blighting for a period of about nine weeks. During the period of needle elongation the initial pink spots occur approximately at the same distance from the needle bases, because of the pattern of maturation of tissues laid down by the basal meristems. Blight attacks occur infrequently, and, susceptible trees often escape injury in certain favorable years with needle elongation on these trees not being affected. The oldest needles on affected trees may be shed prematurely, but usually they persist with the necrotic tips often breaking away.

(3) Chlorotic Dwarf of Eastern White Pine

Symptoms typical of the chlorotic dwarf disease have been observed to occur on eastern white pines for over 60 years (25). Etiological studies by Dochinger (8) demonstrated that air pollution, of which ozone forms a part, is the primary causal agent of the disease. Artificial fumigation studies (9) showed that sensitive trees exposed to 10 pphm of ozone for 4-8 hours daily for 2 - 4 weeks could develop chlorotic mottling symptoms similar to those seen in the field. The macroscopic symptoms of the disease have been described (8) and are somewhat different from those described for ozone injury (5, 17) and for SNB (16) of eastern white pine.

On trees genetically susceptible to chlorotic dwarf, the current-year needles emerge normally but not long after they have attained some growth, their natural green color becomes spotted with chlorotic flecks and mottling. The older foliage turns prematurely yellow and are shed before the current needles reach full development. In final stages, particularly following a drought, the affected current-year needles may develop tipburn.

The disease occurs most commonly on young pines in plantations, and severely affected trees usually succumb before they reach 15 years of age. White pines vary considerably in their susceptibility to chlorotic dwarf, with less sensitive trees exhibiting mild mottling symptoms on near-normal length needles, whereas more sensitive trees exhibit severe stunting of all plant parts and yellow mottled, possibly curled, needles of the current-year.

Intergrafting scions affected by SNB and parent stock susceptible to chlorotic dwarf showed that the two disorders are unrelated with each partner of the union continuing to display its own characteristic symptoms (10).

(4) Chlorotic Decline of Ponderosa Pine

The chlorotic decline disease of ponderosa pine trees in the San Bernardino Mountains of southern California was first called "x-disease" (2), then "chlorotic decline"(23), and later "ozone needle mottle" (24). Since the name chlorotic decline was preferred by the plant pathologists most actively involved in determining the etiology of the disease, it is used in this paper to describe the chronic effects of ozone exposure on ponderosa pine trees.

Miller et al (23) have demonstrated that photochemical oxidant smog drifting into the forests from urban areas on the Pacific coast is responsible for the chlorotic decline of ponderosa pine. Extreme variation in susceptibility to chlorotic decline was found in the forests with dead and dying trees growing alongside visibly unaffected trees. In controlled environment exposure experiments, Miller and Parmeter (21) induced chlorotic decline symptoms on sensitive ponderosa pine seedlings utilizing 25 to 35 pphm ozone for nine hours daily for three to four weeks.

Chronic ozone exposure causes the typical chlorotic mottle needle symptoms which develop from the tip to the base on older needles. The chlorotic mottle symptoms are eventually followed by a necrotic tip dieback. The oldest needles become prematurely senescent with the chlorotic areas coalescing and turning to a uniform tan color. Normally, three to five years old needles occur on ponderosa pine. However, on affected trees the oldest needles are abscissed early, leaving the chlorotic mottled one-year-old needles and the visibly unaffected current-year needles on the tree during the summer months.

VI. INTERACTION OF ATMOSPHERIC OXIDANTS AND SULPHUR DIOXIDE IN CAUSING INJURY TO VEGETATION

In 1966, Menser and Heggestad (19) reported injuring susceptible tobacco plants with a mixture of 2.7 pphm ozone and 24.0 pphm sulphur dioxide in which the two gases acted synergistically. The plants were not injured when exposed to either gas alone at the concentrations used in the combined-gas experiment. In 1969, Applegate and Durrant (1) reported the synergistic action of ozone and sulphur dioxide on peanuts. Dochinger et al (9) utilized a mixture of 10 pphm ozone and 10 pphm sulphur dioxide on eastern white pine trees to produce symptoms similar to those caused by the chlorotic dwarf disease. It was suggested that the two pollutants acted synergistically since the degree of injury produced by either of the pollutants alone was much less than that produced by the mixture. Costonis (4) fumigated sulphur dioxide-sensitive strains of eastern white pine with 5 pphm ozone alone, with 5 pphm sulphur dioxide alone, or with a mixture of the two gases. No injury was caused by ozone alone, whereas sulphur dioxide was toxic to the new needles. The severity of injury induced by a mixture of the two gases was less than that produced by sulphur dioxide alone. However, the severity of injury on the trees could be increased by discontinuous fumigations with ozone and sulphur dioxide separately, followed by an ozone-sulphur dioxide mixture.

It is apparent that additional research is required to explore and explain the action of mixtures of air pollutants on forest trees. Ozone is a natural constituent of our air resource. The concentrations fluctuate diurnally in urban, rural, and forested areas, and can persist at high levels over large areas under certain meteorological conditions. More studies are needed to determine the effects on vegetation of mixtures of ozone and other pollutants as sulphur dioxide and fluoride.

TABLE I

OZONE DOSAGES REQUIRED TO CAUSE INJURY ON PINE TREES IN FUMIGATION EXPERIMENTS

Pine Species	Investigators	Ozone Concentration	Exposure Time	Symptoms Produced
Eastern White Pine	Linzon (17)	40 pphm	2 hrs.	tip necrosis
	Davis (7)	25 pphm	8 hrs.	tip chlorosis
	Dochinger et al(9)	10 pphm	4-8 hrs.daily for 2-4 weeks	chlorotic mottle
	Linzon(18)	10 pphm	6 hrs.	chlorotic flecks
	Berry and Ripperton(3)	6.5 pphm	4 hrs.	tip necrosis
	Costonis and Sinclair(5)	7 pphm 3 pphm	4 hrs. 48 hrs.	(chlorotic flecks (to tip necrosis
Ponderosa Pine	Miller et al (23)	50 pphm	9 hrs.daily for 9-18 days	chlorotic mottle
	Miller and Parmeter(21)	25 to 35 pphm	9 hrs.daily for 3-4 weeks	chlorotic mottle
Virginia Pine	Davis (7)	50 pphm 25 pphm 10 pphm	1 hr. 2 hrs. 8 hrs.	(chlorotic mottle (to (tip necrosis

TABLE 2

SYMPTOM EXPRESSION BY PINE TREES IN THE FOREST

Pine Species	Name of Disease	Causal Agent	Type of Injury Exhibited				
			Minute Silvery Flecks	Chlorotic Flecks	Chlorotic Mottle	Pink Spots	Pink Bands	Tip Necrosis
Eastern White Pine	Ozone Injury	Ozone	x	x	x	x	x	x
Eastern White Pine	Semimature-Tissue Needle Blight	Physiogenic (Mimicking symptoms)				x	x	x
Eastern White Pine	Chlorotic Dwarf	Air Pollution (mainly ozone, plus sulphur dioxide)		x	x			x
Ponderosa Pine	Chlorotic Decline	Photochemical Oxidant smog (Mostly ozone)		x	x			x

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