

Fungitoxicity of Smoke

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ABSTRACT

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Smoke from burning wheat or barley straw reduced growth rates of all fungi tested, which included representative phycocomycetes, ascomycetes, and basidiomycetes. Gaseous smoke and liquid smoke condensates were equally fungistatic. Low smoke dosages were fungistatic rather than fungicidal, and fungi became tolerant of smoke and were able to grow normally in the presence of smoke condensate deposits. Smoke reduced

Additional key words: fire, *Hordeum vulgare*, *Medicago sativum*.

inoculum of and/or plant infection caused by *Rhizoctonia solani*, *Pythium ultimum*, *Erysiphe graminis* f. sp. *hordei*, and *Colletotrichum trifolii* in greenhouse tests. Smoke was more inhibitory to fungi that cause white-rot than to those that cause brown-rot wood decay. Evidence that phenolic compounds are involved in the fungitoxicity of smoke is discussed.

Fungitoxicity of smoke has long been suspected but has rarely been documented. Romanian peasants let smoke from the hearth fire filter through the roofs of their dwellings, in part to check rot and decay (19). Wolkowskaja and Lapszin (20) reported that liquid condensate of smoke from burning beech wood killed spores of *Mucor* sp., *Aspergillus* sp., and *Penicillium* sp. and that the addition of 0.25% of an unspecified phenolic compound increased the fungicidal properties of the smoke.

Melching et al (12) showed that germination of spores of *Puccinia graminis* Pers. var. *tritici* Eriks. and E. Henn., *P. striiformis* West., *Pyricularia oryzae* Cavara, and an *Alternaria* sp. was inhibited on water agar exposed to 600 cm³ of cigarette smoke per liter of air. Exposure of agar to smoke before seeding with spores, followed by incubation in a smoke-free atmosphere, inhibited germination, depending on smoke concentration and duration of exposure.

Spore germination of *Chromelosporium ollare* (Pers.) Hennebert, *Penicillium expansum* Link, *Fusarium lateritium* Nees, *Fomes annosus* (Fr.) Cke., and a *Trichoderma* sp. and mycelial growth of *F. annosus*, *Verticicladiella wagneri* Kend., *Trichosporium symbioticum* Wright, *Pholiota adiposa* (Fr.) Kummer, and *Rhizoctonia solani* Kuhn were reduced on cellophane previously exposed to smoke from burning pine needles (15,16).

Mihail (13) reported that straw smoke inhibited germination of the spores of *Uromyces appendiculatus* (Pers.) Unger and *Puccinia antirrhini* Diet. and Holw. and that the inhibitory effects of the smoke lasted several days.

Arseculeratne et al (1), working with *Aspergillus flavus* Lk. growing on copra, found that copra smoke slightly inhibited mycelial growth but that aflatoxin production was almost completely inhibited, especially at high substrate moisture contents.

Because reports on the toxicity of smoke to fungi have been mainly qualitative, it was considered useful to demonstrate and

quantify smoke fungitoxicity, thus facilitating evaluation of the potential of smoke to control plant pathogenic fungi.

Studies were designed to determine if smoke was effective in reducing mycelial growth of a broad range of fungi, if the effect of smoke on fungi was primarily fungistatic or fungicidal, and if fungi were able to develop tolerance to smoke.

Uses of smoke in plant disease prevention were investigated in greenhouse tests with a foliar pathogen, a stem canker pathogen, a root pathogen, and a seed pathogen.

In addition, it was observed that the conifer root and butt rot organism, *Fomes annosus*, when grown on agar medium with a liquid smoke condensate added, produced a dark brown stain in the medium very similar to that produced by laccase-producing fungi when grown on gallic acid medium. This suggested that smoke contains phenolic compounds toxic to some fungi and that laccase (polyphenol oxidase) might be involved in the metabolism of phenolic compounds (5,6,8,11,14,17) in smoke. This hypothesis was tested.

MATERIALS AND METHODS

Production of smoke. Smoke was produced as previously described (15). Dried barley or wheat straw was burned in a ventilated incinerator and the resulting smoke was cooled to ambient temperature by passage through stove pipe. The cooled smoke was introduced into a chamber containing test materials. Preliminary studies had shown that burning pine needles, grass clippings, and various straws all produced smoke toxic to fungi. Dried wheat or barley straw provided the most uniform fuel source and was used in all subsequent studies. In some studies, a liquid smoke condensate (LSC), which was collected as it dripped out of the pipe during smoke cooling, was used instead of gaseous smoke.

Survey of smoke fungitoxicity. Petri plates containing potato-dextrose agar (PDA) were placed for varying lengths of time in the smoke chamber and exposed to dense barley straw smoke. A clean glass slide was placed next to the petri plates during smoke exposure and optical density of the slide was measured with a model TBX densitometer (Tobias Associates, Inc. Ivyland, PA). An optical density (OD) of zero represented 100% light transmission; OD = 1 represented 10% transmission, and OD = 2 represented 1% transmission. Other petri plates contained PDA with 0, 0.2, 0.4, 0.6, 0.8, 1.0 or 2.0% (v/v) LSC mixed with the liquid agar before cooling.

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Mycelial plugs (5 mm diameter) from the margins of fungal cultures growing on PDA were placed in the center of petri plates containing PDA and smoke deposits, and the diameters of the growing colonies were measured daily until the petri plate margin was reached or growth ceased. Three replications of each fungus on each smoke concentration were averaged for each day. The fungi tested are presented in Table 1.

Aqueous spore suspensions of *Fusarium solani* f. sp. *phaseoli* were placed on slides previously exposed to barley straw smoke and were incubated in a moist chamber for 2 days. Spores from smoked slides were then washed onto clean slides, onto slides with fresh deposits of smoke, or left on the original smoked slides. All of these slides were put back in the moist chamber for 2 more days and then observed microscopically.

Survey of smoke tolerance. Two isolates of *F. annosus*, one (WF) from white fir (*Abies concolor* (Gord. and Glend.) Lindl.) and one (IC) from incense cedar (*Calocedrus decurrens* Kurz (Torr.)), were grown on PDA or PDA with 1% LSC (v/v). After 8 days of growth, 5-mm-diameter plugs of actively growing fungus were transferred to petri plates containing 0, 0.5, 1.0, or 1.5% LSC in PDA. Two types of liquid smoke were used: LSC as described above and "Wrights Bar-B-Q" smoke (BBQ). BBQ smoke is a commercial food product, obtainable at food markets, with reasonably constant formulation and toxicity. Ingredients are listed as "natural liquid smoke and water." Each treatment was replicated five times. Colony diameter was measured at 15 days.

Effect of smoke on plant disease development. Three-week-old, greenhouse-grown barley plants were exposed to barley straw smoke for 0, 1, 2, 4, or 6 min in the smoke chamber, five replicate

plants per treatment. The plants were then arranged on the greenhouse bench such that each plant was adjacent only to plants of other treatments. A barley plant heavily infected with barley powdery mildew, *Erysiphe graminis* f. sp. *hordei* Em. Marchal, was vigorously shaken over the experimental plants for 1 min to dust them with spores. One week later, mildew infections were counted on each leaf of each plant and the length of each leaf was measured to estimate the number of infections per centimeter of leaf.

Field soil naturally infested with *R. solani* was placed in clay pots 20 cm wide × 5 cm deep. (Isolates of *R. solani* from cotton soils in California usually belong to anastomosis group 4. However, no determination was made in this study.) Cotton seeds and soil-filled pots were then separately exposed to 0, 4, 8, 16, or 32 min of smoke in the smoke chamber. Twenty cotton seeds for each of the five seed treatments were placed in pots, two pots for each soil treatment and 10 seeds per pot, in a factorial array that included all combinations of soil treatment and seed treatment. All pots were placed in the greenhouse and watered with distilled water. Cotton seedlings were counted as they emerged. A week after no further seedlings had emerged, the experiment was terminated. Seedlings were washed, surface sterilized in 10% sodium hypochlorite, and plated on 2% water agar to assay for *R. solani*.

Twelve 1-mo-old Dawson alfalfa plants were sprayed to runoff with an aqueous suspension containing 800 spores of *Colletotrichum trifolii* Bain & Essary per milliliter. Groups of three plants were then immediately sprayed with 20 ml of 100, 10, or 1% LSC. Three control plants were sprayed with 20 ml of sterile, distilled water. Each plant was covered with a plastic bag, left on the greenhouse bench for 2 days, and then examined. Lesions on each plant were counted. Isolations were made from each lesion to confirm infection, and only those from which *C. trifolii* was isolated were included in the analysis. Ten of the isolates were chosen arbitrarily for reinoculation of healthy alfalfa plants, thereby completing Koch's postulates.

In a subsequent study, 5-wk-old Dawson alfalfa plants were exposed to smoke in the smoke chamber for 0, 1, or 2 min before, after, or both before and after being sprayed with an aqueous suspension of spores of *C. trifolii*. Plants were left on the greenhouse bench for a week. Lesions were then counted on each plant and the percentage of necrotic foliage was visually estimated.

Field soil naturally infested with *Pythium ultimum* Trow was placed in wooden flats in the greenhouse. The soil in half of each flat was drenched with 200 ml of BBQ in water dilutions of 10, 1.0, or 0.1%. The other half was drenched with 200 ml of sterile distilled water. Twenty-four hours later, soil from each side of each flat was assayed for viable propagules of *P. ultimum* by using the methods of Stanghellini and Hancock (18). Numbers of propagules per gram of soil were calculated for three replicates of each treatment.

Smoke metabolism by laccase-producing fungi. Each of five wood-decaying fungi that cause brown-rot (*Lentinus lepideus* (Fr.)

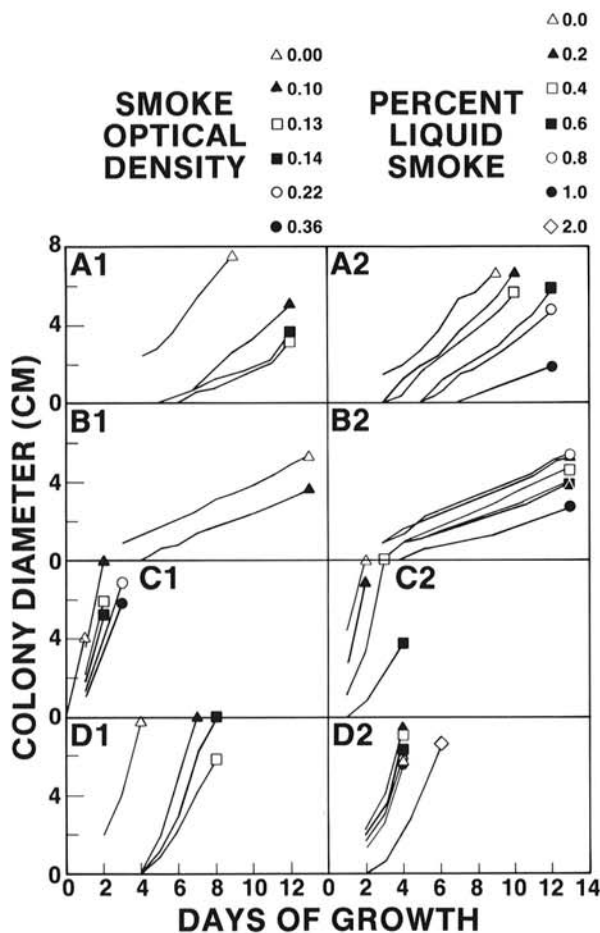


Fig. 1. Colony diameter, in centimeters, of fungi grown on PDA: 1 (left side), previously exposed to wheat straw smoke (measured as optical density of glass slides exposed to smoke adjacent to smoked PDA); or 2 (right side), with liquid smoke condensate mixed with the still-warm agar (measured as percent liquid smoke condensate added). A, *Fomes annosus*, B, *Fusarium solani* f. sp. *phaseoli*, C, *Pythium ultimum*, D, *Sclerotinia sclerotiorum*.

TABLE 1. Smoke tolerance of fungi expressed as slope of the growth curve (centimeters per day)

Fungus	A ^z	B	C
<i>Fomes annosus</i>	0.9	0.4	0.6
<i>Fusarium solani</i>			
f. sp. <i>phaseoli</i>	0.4	0.3	0.3
<i>Ulocladium</i> sp.	0.9	0.8	0.7
<i>Pythium ultimum</i>	4.1	3.5	2.1
<i>Rhizoctonia solani</i>	2.6	1.9	1.7
<i>Sclerotinia sclerotiorum</i>	2.0	1.3	1.4
<i>Fusarium oxysporum</i>			
f. sp. <i>cannibis</i>	1.3	1.2	1.0
<i>Colletotrichum trifolii</i>	0.6		0.5
<i>Verticillium dahliae</i>	0.4		0.3
<i>Trichoderma viride</i>	2.0		1.9

^zTreatments: A, PDA without smoke; B, PDA with smoke deposits resulting in an optical density of 0.10 on smoked glass slides; and C, PDA with 0.4% liquid smoke condensate in the medium. Analysis of variance shows highly significant differences ($P=0.01$) among columns and among rows. Paired *t*-test of treatments shows A is different from B ($P=0.01$), A is different from C ($P=0.03$), but B is not different from C ($P=0.30$).

Fr., *Poria monticola* Murr., *Fomes cajanderi* Karst., *Lenzites trabea* (Pers. ex Fr.) Fr., and *Schizophyllum commune* Fr.) and four wood-decaying fungi that cause white-rot (*Polyporus versicolor* (L.) Fr., the IC isolate of *F. annosus*, the WF isolate of *F. annosus* and *Pholiota adiposa* (Fr.) Kumm.) were grown on PDA containing 0, 1.0, 1.5, or 2.0% (v/v) LSC or on PDA containing 0.5% gallic acid. Each treatment was replicated five times. Fungal colony growth was measured every 2 days or until the colony reached the petri plate margin.

RESULTS

Survey of smoke fungitoxicity. Both liquid smoke condensate and gaseous smoke deposits consistently inhibited the growth of the fungi that were tested (Table 1). For any given fungus, both LSC and smoke chamber deposits resulted in similar growth inhibition. That is, growth curves of a given fungus on each type of smoke are similar in shape and in slope (Fig. 1 and Table 1). Fungi, such as *F. solani* f. sp. *phaseoli* or *F. annosus*, that were strongly inhibited by LSC were also strongly inhibited by smoke chamber deposits. Conversely, fungi such as *P. ultimum* or *S. sclerotiorum*, that seemed relatively tolerant of LSC were also relatively tolerant of smoke deposits. Fungi in the presence of higher dosages of smoke after an initial growth lag often grew nearly as fast as colonies on lower smoke dosages or on no smoke, as indicated by the nearly parallel growth curves for individual fungi, particularly toward the end of the growth period (Fig. 1).

Ungerminated or partially germinated macroconidia of *F. solani* f. sp. *phaseoli* on smoked slides retained viability and resumed growth when removed from smoked slides and placed on unsmoked slides (Fig. 2). Spores placed either on freshly smoked slides or left on the original smoked slides did not grow further. Spores washed from their original smoked slides onto slides

TABLE 2. Colony diameter of isolates of *Fomes annosus* from white fir (WF) and incense cedar (IC)

Liquid smoke in PDA (%)	Colony diameter (mm) on liquid smoke agar at 15 days ^a			
	WF isolate		IC isolate	
	From PDA	From 1% LSC-medium	From PDA	From 1% LSC-medium
BBQ^b				
0.0	60	32	60	60
0.5	0	27	20	60
1.0	0	24	0	35
1.5	0	13	0	0
2.0	0	5	0	0
LSC^b				
0.0	60	30	60	60
0.5	60	45	46	60
1.0	60	60	33	60
1.5	0	10	0	6
2.0	0	0	0	0

^a Each value is the mean of five replicates. First grown on PDA containing 1.0% liquid smoke condensate (LSC), then transferred to PDA containing 0, 0.5, 1.0, 1.5, or 2.0% of either LSC or BBQ type of liquid smoke condensate.

^b BBQ = Wright's Bar-B-Q smoke and LSC = liquid smoke condensate.

TABLE 3. Numbers of powdery mildew lesions on barley plants exposed to different amounts of smoke

Smoke exposure (min)	Lesions (no. per cm)	Leaves showing phytotoxicity (mean %)
0	2.2 a	0
1	0.1 c	3
2	0.2 c	8
4	0.4 bc	9
6	0.6 b	26

^a Values followed by different letters are significantly different ($P=0.05$) by Tukey's HSD test. Each value is the mean for five plants.

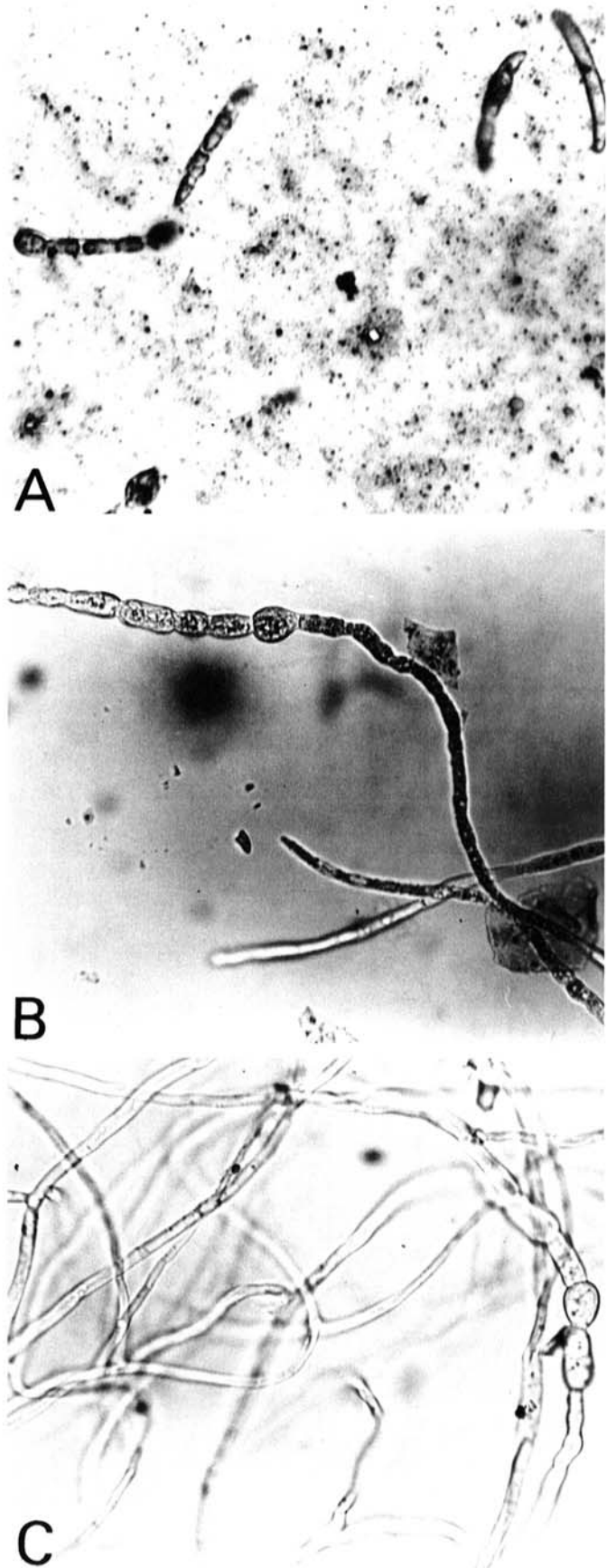


Fig. 2. Spores of *Fusarium solani* f. sp. *phaseoli* after 2 days incubation on glass slides previously exposed to barley smoke. A, Spores on slide exposed to smoke for 6 min and incubated 2 days in a moist chamber. B, Spores as in A, then washed onto slide exposed to smoke for 4 min and incubated 2 days in a moist chamber. C, Spores as in A, then washed onto unsmoked slide and incubated 2 days in a moist chamber. All photos were taken with phase contrast optics at $\times 320$.

smoked 3–4 min showed a small amount of growth, perhaps owing to dilution of smoke compounds in the washing process.

Survey of smoke tolerance. Isolates of *Fomes annosus* previously grown on LSC-medium grew faster than the same isolates previously grown on PDA alone when each was subsequently transferred to LSC- or BBQ-media (Table 2). The WF isolate not previously grown on smoke medium did not grow at all on BBQ medium with 0.5, 1.0, 1.5, or 2.0% liquid smoke. The WF isolate previously grown on 1% LSC medium grew on all concentrations of BBQ medium and on 1.5% LSC medium. The IC isolate, after prior growth on 1% LSC medium, grew on media containing greater concentrations of both LSC and BBQ smokes than did the same fungus previously grown on PDA alone.

Effect of smoke on plant disease development. One minute exposure of barley plants to smoke significantly reduced the number of powdery mildew infections per centimeter of leaf (Table 3). Longer exposures (up to 6 min) did not increase protection. Increasing exposure to smoke beyond 2 min resulted in increased numbers of lesions, apparently because of phytotoxicity.

Exposure of soil infested with *R. solani* to smoke for 16 or 32 min resulted in greater cotton seedling emergence than occurred with low smoke dosages or unsmoked controls ($P = 0.05$) (Table 4). Exposing seeds to smoke did not significantly increase emergence.

Numbers of lesions caused by *C. trifolii* on alfalfa plants sprayed with 0, 1, 10, or 100% LSC averaged 34, 4.3, 0.3, and 0.0, respectively. Differences among the three LSC dilutions were not significant, but all three were significantly different from the 0% control, $P = 0.05$, according to Tukey's HSD test. Plants sprayed with 100% LSC had many dead, scorched-looking leaves and brown pitting of stems, but *C. trifolii* was not isolated from them.

When alfalfa plants were exposed to gaseous smoke in the smoke chamber, longer smoke exposure was associated with fewer lesions (Table 5). Long exposure of alfalfa plants to smoke after inoculation with *C. trifolii* was associated with foliar necrosis, which apparently was due to toxicity of one or more smoke compounds. Least damage occurred when plants were exposed to smoke for 2 min prior to inoculation. However, the results of gaseous smoke treatments were variable and were not statistically significant.

When numbers of propagules of *Pythium* spp. per gram were compared in paired tests of soils treated with water or with 0.1, 1.0, or 10.0% LSC drench, significant differences (t -test, $P = 0.5$) in average count were obtained only for water versus 10.0% LSC treatments (315 versus 94 propagules, respectively). With water versus 1.0% LSC, averages were 344 versus 229, but this difference was not significant ($P = 0.5$).

Smoke metabolism by laccase-producing fungi. Of the five brown-rot fungi tested, only *L. lepideus* failed to grow on 1.5% LSC-medium, and *L. lepideus* and *F. cajanderi* failed to grow on 2.0% LSC-medium. All five grew on 0.5% gallic acid. None of the four white-rot fungi grew on 1.5% or 2.0% LSC-medium or on gallic acid medium.

DISCUSSION

Mycelial growth of a broad range of fungi was consistently retarded, reduced, or prevented on PDA exposed to gaseous smoke

or amended with LSC. Where growth did occur, growth curves showed a lag phase or temporary growth inhibition after which growth proceeded at near normal rates. Thus, at many concentrations, materials in smoke appeared to be fungistatic rather than fungicidal. This hypothesis is further supported by the ability of germ tubes of *Fusarium* spp. to resume growth after removal of inhibitory smoke deposits and by the ability of *F. annosus* to adapt to LSC.

The brown stain that *F. annosus* typically produces in media containing smoke condensates suggests a mechanism of smoke tolerance. This stain resembles that produced in gallic acid medium by laccase-producing fungi. Such a stain is diagnostic for laccase enzymes (the Bavendamm test). Laccase, which is induced in the presence of polyphenols (9), is produced by white-rot wood-decay fungi and is perhaps involved in their ability to metabolize phenolic-based lignin compounds. Wood-decay fungi that cause brown-rot generally lack this ability and generally lack the enzyme. Thus, if laccase is involved in smoke toxicity, white- and brown-rot fungi should show distinctly different tolerances to smoke, and they did. All five brown rot fungi grew on higher smoke dosages than did any of the white rot fungi. Only the brown-rot fungi grew on gallic acid medium.

Laccase enzymes can oxidize phenolic compounds and oxidized phenols may be more fungitoxic than the reduced forms. Phenolic compounds are present in smoke (5,6,8,9,11,14,17) and are thought to be toxic to microorganisms (14). Dion (3) reported that a phenol oxidase enzyme from *Polyporus versicolor* changed the color of ortho- and para-substituted phenols through oxidation to ortho- and para-quinones. Fahraeus (4) had earlier suggested that *Polyporus abietinus* and *P. versicolor* produced dark-colored zones in the presence of phenols through the production of quinonolike substances. Using two mycorrhizal fungi and two litter-decomposing fungi, Lindberg (10) found that those lacking polyphenol oxidase enzymes grew in the presence of gallic acid while those with such enzymes were strongly inhibited. The oxidation product of gallic acid, the ortho-quinone, was more toxic than the gallic acid. These data and observations suggest that, for some fungi, toxicity of smoke is increased by laccase enzymes that convert the phenols to the more toxic quinones.

Exposure of plants to smoke or LSC controlled barley mildew and alfalfa anthracnose, but phytotoxicity at high smoke dosages caused more damage than did infectious disease. It is possible that extended exposures to smoke could predispose the host to infection from pathogens that invade injured tissue. The potential usefulness of smoke or LSC to control diseases of green plants depends in part on the relative sensitivity of pathogens and hosts to smoke toxicity and on the range of safe and effective smoke dosages.

Phytotoxicity is not an important factor in treatment of soil or resistant plant parts. Cotton seedling emergence was increased by exposing soil infested with *R. solani* to smoke. Numbers of propagules of *Pythium* spp. were reduced by drenching soil with LSC. These results suggest that smoke condensate deposits in the upper layers of soil could affect seedling survival after fire by suppressing plant pathogens active near the soil surface.

Similarity in response of individual fungi to deposits from gaseous smoke or to liquid smoke condensates suggests that the

TABLE 4. Emergence^a of cotton seedlings from soil infested with *R. solani* after exposure of seeds and/or the soil to smoke^b

Exposure of seed to smoke (min)	Exposure of soil to smoke (min)					Mean X
	0	4	8	16	32	
0	6	7	11	16	11	10.2
4	9	14	10	14	20	13.4
8	12	11	14	12	12	12.2
16	11	12	11	12	13	11.8
32	8	11	8	16	16	11.8
Mean ^c	9.2 a	11.0 a	10.8 a	14.0 b	14.4 b	

^aEach value is the sum of two replications, twenty seedlings possible.

^bAnalysis of variance F -test gave the following probability levels: soil = 0.028, seed = 0.462, interaction = 0.210.

^cMeans followed by different letters are significantly different ($P = 0.05$) by Tukey's HSD test.

TABLE 5. Number of *Colletotrichum trifolii* lesions on alfalfa plants exposed to smoke

Exposure (min)	Relation to inoculation	Plants (no.)	Lesions/plant ^a	Dead foliage ^b (%)
0		2	30	80
1	before	4	13	52
1	after	3	11	37
2	before	3	6	7
2	after	4	5	39
2	before and after	1	2	30

^aNo significant differences by analysis of variance F -test: $P = 0.146$.

^bEstimated visually.

two forms of smoke are similar in activity and inhibition and that hypotheses based on knowledge of liquid smoke condensates may apply also to surface deposits from gaseous smoke.

Smoke is a common component of some agricultural ecosystems, especially where fire is used regularly (2,7), and it might affect some plant diseases. The magnitude of such an effect and its value in plant disease control is difficult to assess and was not directly addressed in the present studies. However, the temporary nature of smoke-induced fungistasis and the ability of at least some fungi to develop tolerance to smoke suggest that casual or random exposure to smoke is not likely to be an important disease control factor in agricultural systems. Home or farm production and use of smoke condensates for inexpensive control of some plant diseases appears feasible, however, and merits evaluation.

LITERATURE CITED

1. Arseculeratne, S. N., Samarajeewa, U., and Weliana, L. V. 1976. Inhibition of aflatoxin accumulation in smoked substrates. *J. Appl. Bacteriol.* 41:223-233.
2. Darley, E. F., Biswell, H. H., Miller, G., and Goss, J. 1973. Air pollution from forest and agricultural burning. *J. Fire Flammability* 4:74-81.
3. Dion, W. M. 1951. Production and properties of a polyphenol oxidase from the fungus *Polyporus versicolor*. *Can. J. Bot.* 30:9-21.
4. Fahraeus, G. 1962. Aromatic compounds as growth substances for laccase-producing rot fungi. *Physiol. Plant.* 15:572-580.
5. Fiddler, W., Daerr, R. C., Wasserman, A. E., and Salay, J. M. 1966. Composition of hickory sawdust smoke. Furans and phenols. *J. Agric. Food Chem.* 14:659-662.
6. Gilbert, J., and Knowles, M. E. 1975. The chemistry of smoked foods: a review. *J. Food Technol.* 10:245-261.
7. Hardison, J. R. 1976. Fire and flame for plant disease control. *Annu. Rev. Phytopathol.* 14:355-379.
8. Hruza, D. E., Denis, E., van Praag, M., and Heinsohn, H., Jr. 1974. Isolation and identification of the components of the tar of hickory wood smoke. *J. Agric. Food Chem.* 22:123-126.
9. Kaarik, A. 1965. The identification of the mycelia of wood-decay fungi by their oxidation reactions with phenolic compounds. *Stud. For. Suec.* 31. 80 pp.
10. Lindeberg, G. 1949. Influence of enzymatically oxidized gallic acid on the growth of some hymenomycetes. *Svensk Bot. Tidskr.* 43:438-447.
11. Lustre, A. O., and Issenberg, P. 1969. Phenolic components of smoked meat products. *J. Agric. Food Chem.* 18:1056-1060.
12. Melching, J. S., Stanton, J. R., and Koogler, D. L. 1964. Deleterious effects of tobacco smoke on germination and infectivity of spores of *Puccinia graminis tritici*, and on germination of spores of *Puccinia striiformis*, *Pyricularia oryzae*, and an *Alternaria* species. *Phytopathology* 64:1143-1147.
13. Mihail, J. D. 1979. Toxic effects of straw smoke on bean rust, snapdragon rust and on seed germination. Master's thesis, University of California, Berkeley. 52 pp.
14. Olsen, C. Z. 1977. Smoke flavouring and its bacteriological and anti-oxidative effects. *Acta Aliment. Polon.* 3:313-324.
15. Parmeter, J. R., Jr., and Uhrenholdt, B. 1975. Some effects of pine needle and grass smoke on fungi. *Phytopathology* 65:28-31.
16. Parmeter, J. R., Jr., and Uhrenholdt, B. 1975. Effects of smoke on pathogens and other fungi. *Proc. Tall Timbers Fire Ecol. Conf.* 14:299-304.
17. Potthast, K. 1978. Smoking methods and their effect on the content of 3,4-benzopyrene and other constituents in smoked meat products. *Die Fleischwirtschaft* 58:340, 342-348, and 371-375.
18. Stanghellini, M. E., and Hancock, J. G. 1970. A quantitative method for the evaluation of *Pythium ultimum* from soil. *Phytopathology* 60:551-552.
19. Williams, C. 1972. Craftsmen of necessity. *Nat. Hist.* 81:48-59.
20. Wolkowskaja, I. L., and Lapszin, I. I. 1962. Bactericidal and fungicidal properties of smoke solution. *Tehmol. Mesa (special edition):*26-28.