Chemical Prevention of Ascocarp Formation in Claviceps purpurea

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This is a report on the current status of research concerning use of chemicals that require registration under the Federal Insecticide, Fungicide, and Rodenticide Act, as amended by the Federal Environmental Pesticide Control Act. Not all of the chemicals mentioned here are presently so registered with the Environmental Protection Agency. No recommendations for use of these chemicals are implied in this report.

ABSTRACT

Prevention of ascocarp formation in *Claviceps purpurea* (ergot) was investigated by application of protectant and systemic fungicides to low-temperature conditioned sclerotia from *Lolium perenne* and *Poa pratensis* at the soil surface. Complete, or a high degree of, control was obtained by a single application of 1-(4-chlorophenoxy)-3,3-dimethyl-1-(1*H*-1,2,4-triazol-1-yl)-2-butanone (BAY MEB 6447) or α-(2-chlorophenyl)-α-cyclohexyl-5-pyrimidine methanol (EL-279) at 1 and 2 mg/92 cm^2 of soil surface, respectively, compared with benomyl at 6 mg, triarimol at 1 mg, and cadmium chloride at 2 mg. Trials of these compounds for ascocarp suppression in field plots appear to be warranted.

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Additional key words: ergot control; fungicides.

Among the several major grass diseases now controlled in Oregon by field burning (1-5), ergot, *Claviceps purpurea* (Fr.) Tul., is the most dangerous and poses a serious threat to grasses grown for seed if burning is discontinued. The need for chemicals as one substitute method for control of ergot became particularly urgent with passage of legislation that outlaws open burning in Oregon after December 31, 1974. Continuation of the excellent ergot control (6) by elimination of ascosporic inoculum through destruction of sclerotia now obtained by fire will depend on the development of a substitute, brief high-temperature treatment that can be applied to the soil surface and/or development of chemical control. Substitute heat treatments may become possible by use of one of several mobile field incinerators now under development. As reported earlier (6), of many chemicals tested, relatively few have suppressed ascocarp formation at reasonable dosages; thus, the choice of chemicals for this purpose is severely restricted. Therefore, the search was continued for additional chemicals that might eliminate ascosporic inoculum by suppressing ascocarp formation.

MATERIALS AND METHODS.—Sclerotia of *C. purpurea* from either *Lolium perenne* L. or *Poa pratensis* L. were placed on the surface of a sandy loam (pH 5.8) soil, 8.5 cm deep in 10-cm square plastic pots (about 200 sclerotia/pot) with four bottom drainage holes. The pots were exposed outdoors over winter or at 5 C in a constant-temperature chamber for 30 to 90 days to condition the moist sclerotia for ascocarp production.

The pots were placed in a greenhouse to force ascocarp formation. Just before chemical treatment, the soil was moistened and pressed firmly to provide a flat surface area of 92 cm^2 and to prevent the chemical solution or suspension from running down the inner walls of the pot. The chemicals were applied once in a 40-ml aqueous suspension or solution to aid distribution of chemical in a uniform layer over the surface after the water was absorbed. Dosages are all expressed as actual chemical.

We kept the soil surface continuously moist by holding the pots in plastic saucers constantly supplied with water. Results from three pots treated with each dosage rate were obtained by counting the perithecial heads and removing these mature ascocarps with attached sclerotia at weekly intervals starting 3 to 4 weeks after chemical application.

RESULTS.—*Protectant fungicides.*—Nearly all protectant-type fungicides tested gave unsatisfactory control, with less than 60% suppression of ascocarp production after a single application of 10 mg/92 cm^2 of soil surface including: *N'*-[(dichlorofluoromethyl)thio]-*N,N*-dimethyl-*N'*-phenylsulfamide (BAY 47531), captan, chloranil, chlorothalonil, sodium *p*-(dimethylamino)benzenediazo sulfonate (Bayer 22555) (Dexon), dichlone, 5,10-dihydro-5,10-dioxonaphtho[2,3b]-*p*-dithiin-2,3-dicarbonitrile (dithianon), captafol, dodine, 2,4-dichloro-6-(*o*-chloroanilino)-*s*-triazine (anilazine) (Dyrene), ferbam, folpet, maneb, thiram, zineb, thiocyanomethylbutyl sulfone (TCMBS), *S*-(2-hydroxypropyl)thiomethanesulfonate (HPMTS), *N*-3,5-di-

chlorophenyl succinimide (Ohric), *N*-(3,5-dichlorophenyl)itaconimide (S-7258), cyclic *S,S*-(6-methyl-2,3-quinozalinediyl) dithiocarbonate (oxythioquinox) (Morestan), *N,N*-diethyl-3-methyl-2-phenylbutyramide (U-35,075), 2,4-dimethyl-5-*o*-phenyl carboxanilido thiazole (UNI-H588), and 2-phenyl thio benzothiophene 1,1-dioxide (UNI-H184).

Protectant fungicides tested at 20 mg that produced less than 60% control included: BAY 47531, captan, chloranil, chlorothalonil, Dexon, dichlone, captafol, dodine, dithianon, zineb, and ziram. Thiram produced 90% control at 20 mg. One chemical, *trans*-1,2-bis(propylsulfonyl)ethylene (CHE-1843), at 4 mg produced 89% control, but at 10 mg, control was increased only to 92%. Incomplete control was obtained at 20 mg with three chemicals: 4,4′-sulfonyldiphenol; 4,4′-thiodiphenol; and 4-mercaptophenol. One chemical, *p*-toluene sulfonamide, gave incomplete control at 10 mg, and no control at 1, 2, and 4 mg.

Seven organic tin compounds that were tested at 2, 4, and 10 mg but failed to suppress ascocarp development, included: bis(tributyltin) oxide (BTBTO), triphenyltin-hydroxide (TPTH), dimethyldiphenyltin (DMDPT), hexaphenylditin (HPDT), allyltributyltin (ATBT), tributyltin dimethyldithiocarbamate (TBTDDC), and triphenyltinacetate (TPTA). In another test at 2, 4, and 6 mg, no control was obtained with BTBTO, TPTH, and TBTDDC.

Systemic fungicides.—Most of the systemic fungicides applied over sclerotia at 2, 4, and 10 mg/92 cm^2 of soil surface either failed to suppress ascocarp formation or provided very poor control, as follows: carboxin, oxycarboxin, chloroneb, 5,6-dihydro-2,2′, 3-trimethyl-1,4-oxathiin-3-carboxanilide (F-827), 5,6-dihydro-2-methyl-*N*-(2-biphenylyl)1,4-oxathiin-3-carboxamide (F-427), 2,4-dimethyl-5-*o*-phenyl carboxanilidothiazole (H-588), 2-(phenylthio)benzothiophene 1,1-dioxide (H-184), 2,5-dimethyl-3-furanilide (BAS-3191F), 2-iodo-benzanilide (BAS-3170F), 2-(*O,O*-diethyl-thionophosphoryl)-5-methyl-6-carbethoxy pyrazolo-(1,5a)pyrimidine (pyrazophos) (HOE-2873), 3-hydroxy-5-methylisoxazole (NIA 24111), 1,2-dibromo-1-cyclohexyl-2-nitroethane (NIA 25050), *O,O*-diethylphthalimidophosphorothioate (EF-106), 4-[(*O*-chlorophenyl)hydrazono]-3-methyl-5-isoxazolinone (PP-395), and *N*-(3-pyridyl) *S*-*n*-butyl-*S*-*tert*-butylbenzyl dithioimidocarbonate (S-1358).

Some systemics at 10 mg provided complete or nearly complete control, but at 4 mg gave inferior or no control, including: *N*-3,5-dichlorophenylcarbamylpyrrolidine (R-24952) and 3-carboxanilido-2,4,5-trimethyl furan (UNI-H719).

Several systemic compounds provided incomplete control at the rates tested, including: 5-methyl-*s*-triazole-(3,4-b)benzothiazole (EL-291) at 4 mg; dichlozoline at 1, 2, and 4 mg; methyl [1-[[(5-cyanopentyl)amino]carbonyl]-1*H*-benzimidazol-2-yl] (BAY DAM 18654) at 2, 3, and 4 mg; and 2-chloro-6-methoxy-4-trichloromethyl-pyridine (DOWCO 269) at 2, 4, 6, and 8 mg. Complete or nearly complete control was obtained at 1 to 2 mg in several tests with α-(2-chlorophenyl)-α-cyclohexyl-5-pyrimidinemethanol (EL-279) and with 1-(4-chlorophenoxy)-3,3-dimethyl-1-(1*H*-1,2,4-triazol-1-yl)-2-butanone - (BAY MEB 6447).

The most active chemicals in the present study, BAY MEB 6447 and EL-279, were compared in a separate test with the most effective chemicals in the previous (6) tests. Complete control was obtained with the following chemicals at the indicated dosages: BAY MEB 6447, 1 mg; EL-279, 1 mg; benomyl, 6 mg; triarimol, 1 mg; and cadmium chloride, 2 mg/92 cm^2 (about 2 lb/acre).

DISCUSSION.—The need for chemical control to substitute for field burning for control of ergot in production of grass seed in Oregon is urgent, because of the projected termination of open burning after the 1974 season. Elimination of primary (ascosporic) inoculum by suppression of ascocarp formation may be a feasible approach to chemical control of ergot, if effective chemicals can be discovered.

Among the better chemicals detected, triarimol (6) and EL-279 exerted strong activity against *C. purpurea* ascocarp formation, but these two pyrimidine derivatives apparently will not be commercially available in the foreseeable future (D. H. Ford, *personal communication,* Eli Lilly & Co.). BAY MEB 6447 demonstrated strong activity that is equal to that of triarimol and EL-279. Several benzimidazoles are effective, but the dosages needed may not be economically feasible. Cadmium chloride might be one of the cheaper chemicals known to be effective, but the compound is not registered for this purpose.

Most of the recognized fungicides currently used in plant disease control and others in advanced stages of development for commercial use have shown little or no promise for suppression of *Claviceps* ascocarps. Except for cadmium chloride, the chemicals with the strong activity have been recent inventions. Although not used to their greater advantage as chemotherapeutants, systemic chemicals generally have shown stronger activity than the protectant-type fungicides.

When effective chemicals become available, obtaining distribution of the chemicals at the soil surface uniformly and in contact with overwintered sclerotia under the leaf canopy will be decisive in suppressing ascocarps. Besides direct disease control, burning grass fields provided a litter-free, clean soil surface that would allow good distribution of fungicides applied for ascocarp suppression. Optimum time and manner of application of fungicides for ascocarp suppression will need to be determined after effective chemicals have been identified.

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