Chemically Induced Root Injury Correlated With a Reduction of Fusarium Wilt of Tomato

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ABSTRACT

Tomato plants showed less Fusarium wilt when compounds that injure roots were applied before root inoculation with *Fusarium oxysporum* f. sp. *lycopersici*. Tests of 272 compounds showed that the severity of Fusarium wilt and root injury were negatively correlated to a significant degree. Results from studies in which a split-root technique was used suggested that, with the exception of plant growth regulators, compounds which caused root injury or an inhibition of root elongation generally had a localized effect. Phytopathology 63:655-656

Additional key words: disease resistance, chemotherapy.

Keyworth & Dimond (9) observed that some chemical or physical treatments that severely injured roots also greatly reduced the subsequent severity of Fusarium wilt of tomato. In this note, we report on the correlation between the degree of chemically induced root injury and the severity of Fusarium wilt of tomato for 272 compounds. Some of the major groups of compounds tested were phenols, benzoic acids and esters, aliphatic compounds including acids, miscellaneous benzene ring compounds, compounds with two or more carbon-containing rings in the molecule, heterocyclic compounds containing mostly two or three rings, and compounds with growth-modifying effects.

Tomato plants, cultivar 'Bonny Best', were grown in sand in 16-oz styrofoam cups, treated with compounds, and the roots inoculated and later assessed for Fusarium wilt by methods previously described (7).

The plants were rated for the severity of root injury prior to root inoculation with *F. oxysporum* f. sp. *lycopersici*. Ratings of the severity of root injury were as follows: 1 = 0%, 2 = 1-33%, 3 = 33-66% and 4 = 66-100%.

Fusarium wilt decreased with an increase in the amount of root injury (Fig. 1). Severity of Fusarium wilt versus root injury was negatively correlated ($r = -0.61$) to a highly significant degree (1% level). The root injury symptoms caused by the compounds were usually a reduction in the amount of roots, sometimes with browning, or browning alone in a few instances.

Some compounds greatly reduced Fusarium wilt in this assay and resulted in zero or only slight injury to roots, or reduced Fusarium wilt more than would be expected from the degree of root injury observed. Examples of such compounds, with Fusarium wilt ratings which fell more than one standard deviation below the regression line, are as follows: 2,4,5-trichlorophenoxyacetic acid, α-naphthalene acetamide; *N*-dimethylaminosuccinic acid; tetrachlorophenylacetic acid; parahydroxybenzoic acid; *n*-butyl parahydroxybenzoate; *N*-(2-cyanoethyl)-DL-methionine; triphenyltin acetate; 9-chloro-9-phenyl fluorene; 3,4-dichlorobenzoic acid hydrazide; D-galactose; D-mannose; DL-norleucine; nitrobenzene; ethepon [2-(chloroethyl)phosphonic acid] and 2,4-DEP [a mixture of bis- and tris-(2,4-dichlorophenoxyethyl) phosphite].

Examples of compounds that caused the severity of Fusarium wilt to rise more than one standard deviation above the regression line are as follows: picloram (4-amino-3,5,6-trichloropicolinic acid); hippuronitrile; *N*-chlorosuccinimide; 2,2-dimethyl-1,2-cyclobutane-diacetic acid; 2-chloro-4,6-di-tert-amylphenol; propyl gallate, 2-butoxy ethanol; 4-tert-butyl-2-chloro-6-nitrophenol, and salicylanilide.

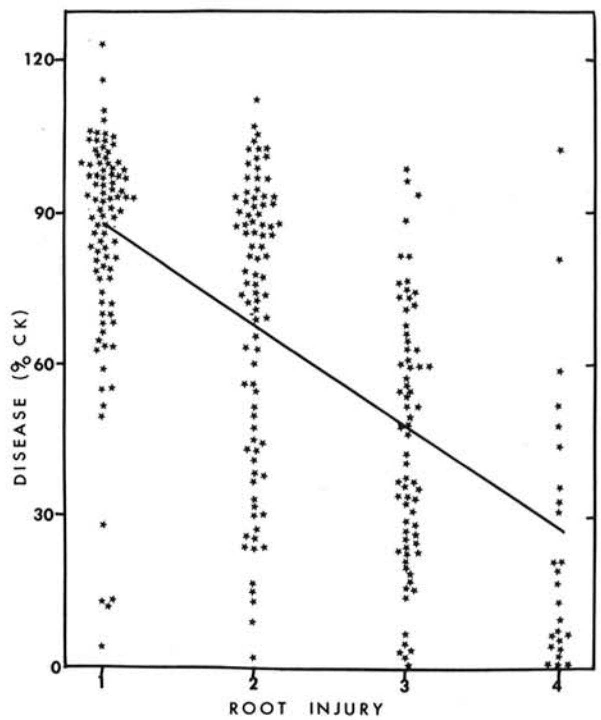

Fig. 1. Scatter diagram of the root injury to tomatoes caused by 272 chemical compounds and the subsequent severity of Fusarium wilt developing on these plants. The plants were root-inoculated after the period of treatment with the compounds. Severity of root injury was rated as follows: 1 = 0%, 2 = 1-33%, 3 = 33-66%, 4 = 66-100%. The severity of wilt was estimated by the method of Dimond et al., and expressed here as a percent of the severity of wilt that developed in comparable, inoculated plants not previously treated with chemical compounds. Correlation coefficient (r) is 0.61, significant beyond the 1% level.

A split-root technique (4) was used to determine whether host resistance due to chemically induced root injury was localized or systemic. Some of the plants were inoculated through the chemically treated half of the root system and some were inoculated through the untreated half of the root system. For most compounds tested in this way, the severity of Fusarium wilt was reduced only when both the chemical treatment and inoculation were applied to the same portion of the root system. This suggests that the compounds which injured or inhibited the roots generally had a localized phytotoxic effect. However, some of the growth regulators, like methyl dichlorobenzoic acid, reduced Fusarium wilt symptoms when applied to the noninoculated root system in the split-root experiment. This compound was not fungitoxic at the concentration used and inhibited root elongation in the opposite, untreated root system.

The split-root technique may be a method for establishing whether the activity of a compound against Fusarium wilt of tomato is systemic. For example, benomyl reduced Fusarium wilt symptoms 74% when applications were initiated through the noninoculated root system the same day the adjacent root system was inoculated (W. L. Biehn, *unpublished data*).

In addition to other growth regulators previously reported to reduce the severity of Fusarium wilt (3, 5), we found that the following growth regulators also reduce Fusarium wilt symptoms: methyl chlorophenoxyacetic acid; *N*-dimethylaminosuccinic acid; ethepon; 2,4-DEP; and 2,4-(dichlorophenoxy) acetamide. They all inhibited root elongation or reduced the amount of roots. They also caused both a localized and general swelling of the basal portion of the stem, probably due in part to cell proliferation (6).

Growth regulators may induce resistance by altering the structure of the xylem vessels, by increasing tylose formation, or both. Such changes might physically impede the systemic movement of wilt fungi in plants (1) and have been associated with the protective action of growth regulators against Dutch elm disease (2, 8, 10).

LITERATURE CITED

1. BECKMAN, C. H. 1966. Cell irritability and localization of vascular infections in plants. Phytopathology 56:821-824.
2. BRENER, W. D., & C. H. BECKMAN. 1968. A mechanism of enhanced resistance to Ceratocystis ulmi in American elms treated with sodium trichlorophenylacetate. Phytopathology 58:555-561.
3. CORDEN, M. E., & A. E. DIMOND. 1959. The effect of growth regulating substances on disease resistance and plant growth. Phytopathology 49:68-72.
4. COUCH, H. B., & J. R. BLOOM. 1960. Influence of soil moisture stresses on the development of the root-knot nematode. Phytopathology 50:319-321.
5. DAVIS, D., & A. E. DIMOND. 1953. Inducing disease resistance with plant-growth regulators. Phytopathology 43:137-140.
6. DAVIS, D., & A. E. DIMOND. 1956. Site of disease resistance induced by plant-growth regulators in tomato. Phytopathology 46:551-552.
7. DIMOND, A. E., D. DAVIS, R. A. CHAPMAN, & E. M. STODDARD. 1952. Plant chemotherapy as evaluated by the Fusarium wilt assay on tomatoes. Conn. Agr. Exp. Sta. Bull. 557. 82 p.
8. EDGINGTON, L. V. 1963. A chemical that retards development of Dutch elm disease. Phytopathology 53:349 (Abstr.).
9. KEYWORTH, W. G., & A. E. DIMOND. 1952. Root injury as a factor in the assessment of chemotherapeutants. Phytopathology 42:311-315.
10. SMALLEY, E. B. 1962. Prevention of Dutch elm disease by treatments with 2,3,6-trichlorophenylacetic acid. Phytopathology 52:1090-1091.