Induced Resistance to Cytospora in Bearing Trees of Prunus domestica

A. W. Helton and J. W. Braun

Plant Pathologist and former Graduate Fellow, respectively, Department of Plant Sciences, University of Idaho, Moscow 83843.

Portion of an M.S. thesis, University of Idaho, Moscow; submitted with the approval of the Director of the Idaho Agricultural Experiment Station as Research Paper No. 840.

Accepted for publication 26 January 1971.

ABSTRACT

Eight-year-old Italian prune (*Prunus domestica*) trees were inoculated with *Cytospora cincta* early in the growing season, and groups of these trees (three in a group) were reinoculated once at intervals throughout the remainder of the growing season. The results showed that (i) the maximum canker size achieved prior to the onset of marginal healing

varies directly with the initial vigor of the individual *Cytospora* invasions; and (ii) invasion of bearing Italian prune trees by *Cytospora* fungi induces a resistance reaction in vivo which is detectable as far as 120 cm away from the initiating infection, and appears to be truly systemic in nature. Phytopathology 61:721-723.

Additional key words: Cytospora canker, systemic resistance.

Induction of disease resistance in plants by pathogenic organisms has been studied in several test systems (5, 10, 11, 13), but most reports have been concerned with a localized response in herbaceous plants. Two exceptions were studies of induced resistance in Italian prune (Prunus domestica L.) trees (6) and J. H. Hale peach trees (P. persica) (2) in which initial infection of young trees by Cytospora cincta induced resistance to subsequent Cytospora infections at a distance of 18 cm from the initial infection. Further investigation was desirable to determine whether this resistance response could be measured at greater distances from the site of primary invasion, i.e., whether the response is truly systemic.

MATERIALS AND METHODS.—The 8-year-old Italian prune trees selected for this investigation were growing on seedling rootstocks of Lovell peach (P. persica [L.] Batsch). The trees were inoculated with an isolate of Cytospora cincta Fr. as previously described (6). On 9 June, 18 8-year-old trees received one primary inoculation on one scaffold branch at a site 60 cm above the main crotch (hereafter referred to as the primary canker). The inoculated trees were arranged in six groups of three trees each, with one group receiving challenge inoculations at intervals of 1, 2, 3, 5, 7, or 9 weeks after 9 June. One challenge inoculation was made/branch on each of three other scaffold branches/ tree so that the total distance between the primary canker and each of the three challenge cankers was 120 cm. This scheme was intended to reduce the induced-resistance effect (2, 6), as measured by the challenge cankers, in two ways. First, the "dilution factor" would be increased, both by the distance separating primary and challenge cankers and by the greater stem volume [i.e., the cross-sectional area of the larger stems of these older trees (6)]. Second, the "induction potential" would be reduced in magnitude by use of only one primary canker/tree rather than three as in the case for the 18-cm studies (2, 6). Each time challenge inoculations were made, a group of three trees containing no primary cankers received control inoculations at locations within the tree comparable to those of the challenge inoculations.

In addition, a group of 24 3-year-old Italian trees was inoculated so that each tree bore three primary cankers, one on each of three scaffold branches, and challenge inoculations were initiated 18 cm below the primary infections at intervals of 0, 1, 2, 3, 5, 7, 9, or 11 weeks after the primary inoculations. This portion of the study was executed for comparative and verificational purposes only, and the results are recorded elsewhere (1).

Canker expansion was measured first at 1 week after inoculation, then at intervals during the remainder of the study. This was done by measuring the length and width of each canker to the nearest 0.1 cm, then computing a standardized expression of canker expansion by multiplying the length by the width and subtracting the impact-wound area (length x width) made by the tack hammer at the time of inoculation (9). Data are presented (Fig. 1, 2) as total Cytospora necrosis (TCN) per tree resulting from the three cankers established in each tree at any one time. When lateseasonal marginal healing became evident, all necrotic tissues (bark) were removed to expose the margins and allow more accurate measurements of the shrinking canker dimensions. One-tailed t-tests (10% level) were performed for each observation date (Fig. 1) for the control and challenge cankers initiated simultaneously at the various reinoculation intervals.

RESULTS.—Cankers developed most rapidly during the first 2 weeks after inoculation, often achieving approx half their max size within 1 week and their max size 2 to 8 weeks later (Fig. 1). Maximum canker size (max TCN) achieved was independent of the length of the canker expansion period, but varied during the growing season, apparently influenced primarily by the canker expansion rate during the 1st week after inoculation (Fig. 2). Cankers exhibiting greater expansion rates during this period generally achieved greater size. After attaining max size, cankers decreased in dimensions during the rest of the growing season as a result of marginal healing by the host tissue.

TCN values for control cankers generally were larger than those for challenge cankers initiated simulta-

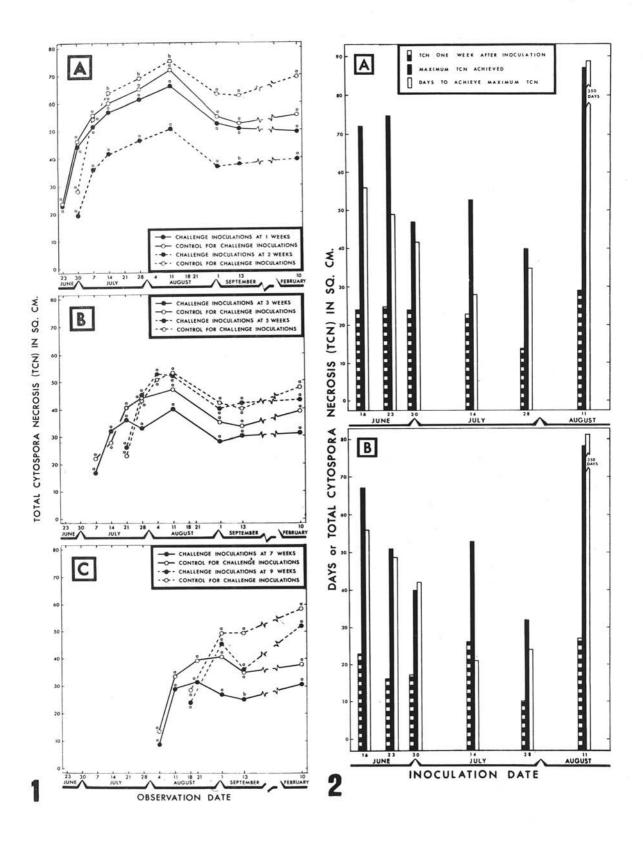


Fig. 1-2. 1) Effect of Cytospora infection on subsequent Cytospora infections in 8-year-old Italian prune trees when a primary infection was initiated 60 cm above the base of a single branch and challenge infections were initiated at a distance of 60 cm from the base of three adjacent branches. Primary infections were initiated on 9 June, and challenge (solid dots) and control (open dots) infections were initiated simultaneously at intervals of 1, 2, 3, 5, 7, or 9 weeks later; control trees were not previously infected. Each value represents the average total Cytospora necrosis (TCN) per tree for three trees. The lower case letters a and b indicate significant difference (as determined by one-tailed t-tests at the 10% level) between TCN values for simultaneously initiated challenge and control infections at any one observation date. 2) Relationship of initial vigor of infection (as represented by TCN after 1 week) to max TCN achieved by inoculations made on uninfected (A) or infected (B) 8-year-old Italian prune trees at intervals after initial inoculation of the infected trees.

neously, whatever the interval separating primary and challenge inoculations (Fig. 1), which is important only in its appearance of consistency. When one-tailed t-tests were computed, these differences were found to have no statistical significance (10% level) except for cankers initiated 2 weeks after the primary inoculations (Fig. 1).

In the 18-cm study, significant differences between control-canker TCN max and corresponding challengecanker TCN max were found for inoculations made in prune trees at intervals of 0, 1, 2, or 3 weeks after primary inoculations were initiated (1).

Discussion.—This study suggests (Fig. 1) that primary invasion of bearing Italian prune trees by Cytospora results in an inhibitory effect on the development of subsequent Cytospora invasions even when the distance separating primary and challenge cankers is 120 cm, the tissue volume separating these infection points is enormously greater than previously reported (6), and one inciting infection is used instead of three. The effect was stronger and more consistent in the 3year-old trees 18 cm from the primary canker sites; but the consistency of the greater size of the primary infections in the bearing trees throughout the season (Fig. 1), and the significance of this greater size after 2 weeks, indicate that the induced resistance effect is measurable 120 cm from the primary infections. The responsible factor(s) therefore must be translocated over considerable distances, at least in trace quantities, and should be regarded as truly systemic in nature. Such a distribution pattern in vivo is greatly different from the localized inhibitory effects reported for various other fungus invasions of plant tissues (3, 4, 7, 8, 9, 12, 14).

LITERATURE CITED

1. Braun, J. W. 1969. Characteristics of the Cytosporainduced, disease-resistance phenomenon in Prunus domestica L. and the sugar-polyol content of the bark of affected trees. M.S. Thesis, Univ. Idaho,

Moscow. 59 p.
2. Braun, J. W., & A. W. Helton. 1971. Induced resistance to Cytospora in Prunus persica. Phytopathology 61:685-687.

3. CRUICKSHANK, I. A. M., & M. MANDRYK. 1960. The effect of stem infection of tobacco with Peronospora tabacina Adam on foliage reaction to blue mold. J. Australian Inst. Agr. Sci. 26:369-372.

4. Davis, D. 1964. Cross-protection of Fusarium wilt suscepts with eight formae specialis of Fusarium oxysporum. Phytopathology 54:891 (Abstr.).

HARE, R. C. 1966. Physiology of resistance to fungal diseases in plants. Bot. Rev. 32:95-137.

 Hubert, J. J., & A. W. Helton. 1967. A translocated resistance phenomenon in Prunus domestica induced by initial infection with Cytospora cincta. Phytopathology 57:1094-1098.

JOHNSTON, C. O., & M. D. HUFFMAN. 1958. Evidence of local antagonism between two cereal rust fungi.

Phytopathology 48:69-70.

- 8. Muller, K. O. 1958. Studies on phytoalexins. I. The formation and the immunological significance of phytoalexin produced by Phaseolus vulgaris in response to infections with Sclerotinia fructicola and Phytophthora infestans. Australian J. Biol. Sci. 11:
- 9. Schnathorst, W. C., & D. E. Mathre. 1966. Crossprotection in cotton with strains of Verticillium albo-atrum. Phytopathology 56:1204-1209.

TOMIYAMA, K. 1963. Physiology and biochemistry of disease resistance of plants. Annu. Rev. Phytopathol. 1:295-324.

11. Walker, J. C. 1963. The physiology of disease resistance. p. 1-25. The physiology of fungi and fungus diseases. West Virginia Univ. Agr. Exp. Sta. Bull. 488T. 106 p.

Weber, D. J., & M. A. Stahmann. 1966. Induced immunity to Ceratocystis infection in sweet potato

root tissue. Phytopathology 56:1066-1070.

 Wood, R. K. S. 1967. Physiological plant pathology, p. 464-509. In W. O. James and J. H. Burnett [ed.] Botanical Monographs, Vol. VI. Blackwell Sci. Publ. Oxford, Edinburgh, Great Britain. 570 p.

14. YARWOOD, C. E. 1965. Translocated effect of two fungus infections of bean. Phytopathology 55:330-332.