

Induced Resistance to *Cytospora* in *Prunus persica*

J. W. Braun and A. W. Helton

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

Present address of senior author: Department of Pomology, Cornell University, Ithaca, New York 14850.

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ABSTRACT

Three-year-old J. H. Hale peach (*Prunus persica*) trees were artificially infected with *Cytospora cincta* early in the growing season, and groups of these trees (three in a group) were reinfected once at intervals throughout the remainder of the growing season.

An induced resistance response occurs at a distance of at least 18 cm from the site of primary *Cytospora* invasion; the maximum canker size achieved prior to the onset of marginal healing varied directly with the initial vigor of the *Cytospora* invasion. Phytopathology 61:685-687.

Induction of disease resistance in plants by pathogenic organisms has been considered by several reviewers (3, 6, 8, 11). Resistance has been induced by one pathogen and measured by its effects on disease production by the same pathogen or by another of the same type (e.g., if both initial and challenge pathogens are fungi) or by another pathogen of a different type (e.g., if the initial pathogen is a fungus and the challenge pathogen is a virus or bacterium).

Most reports demonstrate a localized response in herbaceous plants. In 1967, however, Hubert & Helton (4) reported that initial infection of Italian prune trees by *Cytospora cincta* induced resistance to subsequent *Cytospora* infections, and that this resistance occurred at least 18 cm from the initial infection. Since this was the first report that demonstrated systemic resistance to fungus infection in woody plants induced by prior infection by the same fungus, we wished to discover whether the same degree of systemic response could be initiated in another species.

MATERIALS AND METHODS.—Three-year-old J. H. Hale peach trees growing on seedling rootstocks of Lovell peach (*Prunus persica* [L.] Batsch) were inoculated with an isolate of *Cytospora cincta* Fr. as described previously (4). On 13 June, twenty-four 3-year-old trees were inoculated on three branches at one site/branch (hereafter referred to as "primary cankers"). The inoculated trees were arranged in eight groups of three trees each, with one group being re-inoculated at intervals of 0, 1, 2, 3, 5, 7, 9, or 11 weeks after primary inoculation. Reinoculation consisted of placing another inoculation 18 cm directly below the primary canker on each infected branch (the challenge inoculations). Each time one group of trees received challenge inoculations, another group of three trees bearing no primary cankers was inoculated at three comparable sites/tree (control inoculations).

First measurements of canker expansion were taken at 1 week after inoculation, then at intervals during the remainder of the study. Canker measurement involved removal of the electrician's tape and measuring the length and width of each canker to the nearest 0.1 cm. A standardized expression of canker expansion was obtained by multiplying the length by the width and

subtracting the impact-wound area (length \times width) made by the tack hammer at the time of inoculation (4).

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. Decrease in TCN, after a short period of no change in canker dimensions in midseason, was the result of formation of callus tissue by the host at the canker margins (marginal healing). When marginal healing became evident, the necrotic bark was removed to expose the margins and permit more accurate measurement of the shrinking canker dimensions.

One-tailed t-tests at the 10% level of significance were performed for each observation date (Fig. 1).

RESULTS.—*Cytospora* cankers generally developed most rapidly during the first 2 weeks after inoculation, often achieving approx half their max size within 1 week, and max size within 2 to 8 weeks (Fig. 1). Maximum TCN was not dependent upon duration of the canker expansion period but varied during the growing season, apparently influenced most by the rate of canker expansion during the first week after inoculation (Fig. 2). Generally, the greater the expansion rate during this period the greater the max size achieved. After attaining max size, cankers decreased in dimensions during the remainder of the growing season as a result of marginal healing.

Where significant differences were found in TCN per tree (as determined by one-tailed t-tests at the 10% level), the TCN values for control cankers were larger than those for corresponding challenge cankers with the exception of cankers initiated 29 August (Fig. 1).

Significant differences between control and corresponding challenge canker TCN maxima were found for inoculations made at intervals of 0, 1, 2, 3, 5, or 7 weeks after initiation of primary cankers. No difference existed between cankers initiated at the 9-week interval. Cankers initiated at the 11 week interval did not reach a max TCN development peak until the following summer. Significant differences occurred throughout the season except for inoculations made at the 9-week interval (Fig. 1).

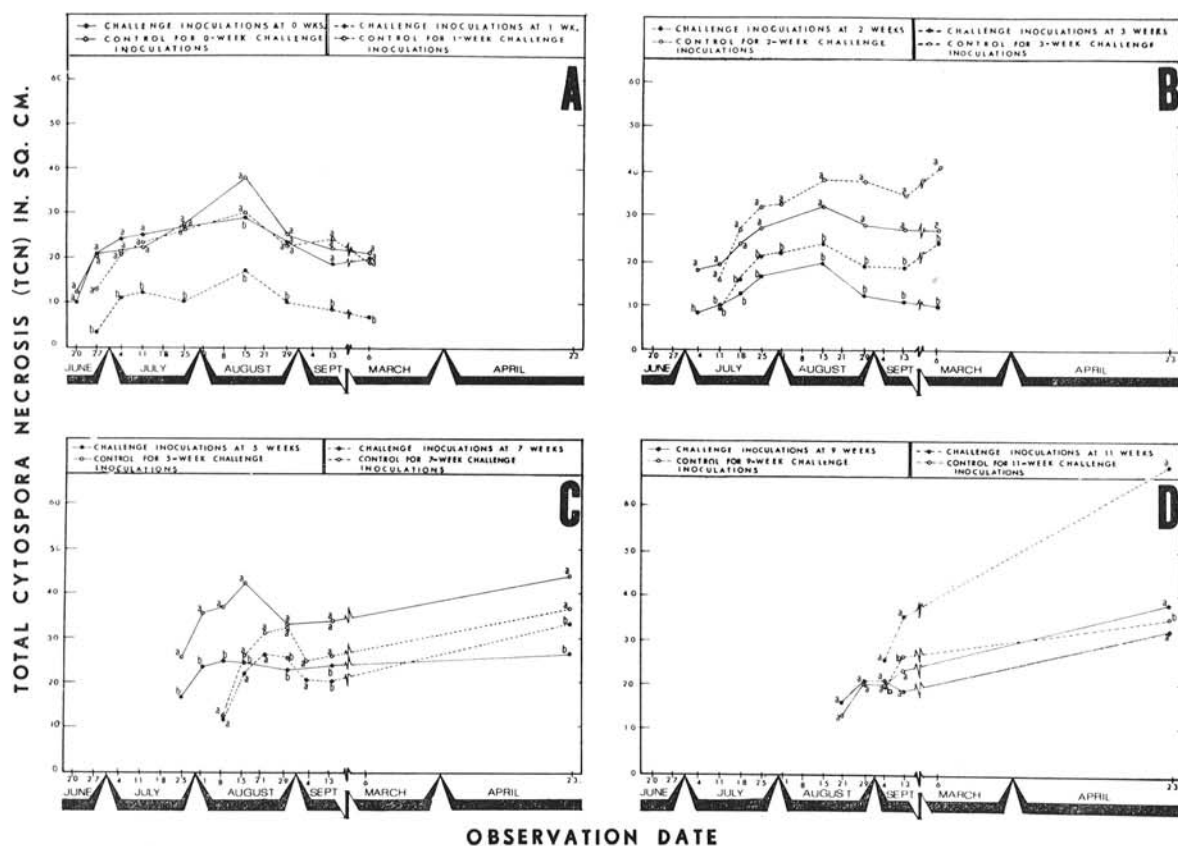


Fig. 1. Effect of *Cytospora* infection on total *Cytospora* necrosis (TCN) induced by reinoculations in 3-year-old J. H. Hale peach trees. One primary infection was initiated on three branches of each tree, and a challenge infection was initiated 18 cm below each primary infection at various intervals thereafter. Primary infections were initiated on 13 June, and challenge (solid dots) and control (open dots) infections were initiated simultaneously at intervals of **A**) 0 and 1 weeks; **B**) 2 and 3 weeks; **C**) 5 and 7 weeks; and **D**) 9 and 11 weeks later. Each TCN value represents the average TCN per tree for three trees; (a) and (b) indicate significant difference between TCN values for challenge and control infections.

DISCUSSION.—Our results (Fig. 1) indicate that primary invasion of J. H. Hale peach trees by *Cytospora* results in an inhibitory effect on the development of subsequent *Cytospora* invasions. This inhibitory response is evident for 7 weeks after primary infection, which suggests that peach trees support the induced-resistance reaction more effectively than Italian prune trees (*P. domestica*) (4).

A number of mechanisms have been reported or suggested to be responsible for induced inhibitory responses to fungal infection. Muller (5), Weber & Stahmann (9), and Cruickshank (2) attributed such a response to formation of phytoalexins. Other reports suggest that accumulation of phenols in adjacent tissues, production of toxins in host tissues, or alteration of host enzyme systems are responsible (3, 6). The *Cytospora* induced-resistance mechanism probably is not associated with the wound-response (marginal healing phenomenon), a process that seems to be governed by the presence of certain growth substances (1, p. 161-164). While it is possible that the primary *Cytospora* invasion might alter the growth-substance

content of the tissues and thereby induce the host to heal more readily with respect to future invasions, Wensley's results (10) showed that cultivars of peach more resistant to *Cytospora* invasion already possess a greater ability to heal uninfected wounds. His results suggest that healing is due to a passive wound-response mechanism present before *Cytospora* invasion and stimulated to action by invasive disruption of the cells (1, 2). Whether *Cytospora* toxin(s) (7) can similarly disrupt host cells and thereby result in a similar wound-response is not known.

Additional evidence that the *Cytospora*-induced resistance mechanism is not directly associated with the marginal healing response in prune trees is the fact that marginal healing was observed to occur at approx the same time and rate for both control and challenge cankers (Fig. 1). If the resistance and healing phenomena were directly associated, marginal healing probably would have occurred sooner where challenge cankers were initiated, as infections already had been established in those trees. If the primary cankers had

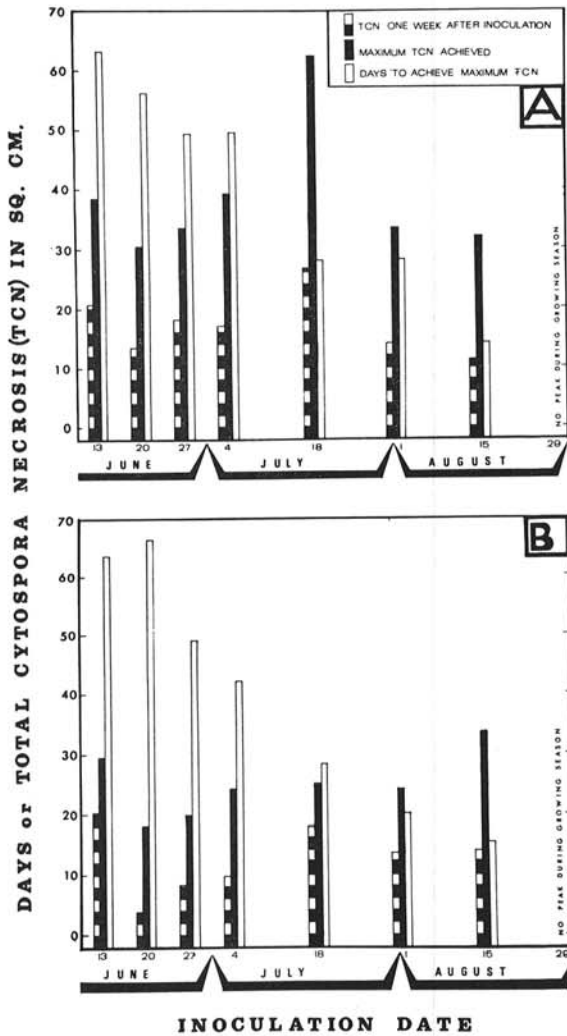


Fig. 2. Relationship of initial vigor of infection (as represented by average total *Cytospora* necrosis [TCN] after 1 week) to maximum TCN achieved by inoculations made on A) uninfected or B) infected 3-year-old J. H. Hale peach trees at intervals after initial inoculation of the infected trees.

induced significant increase in growth substances, this would already have been accomplished.

We conclude that (i) the induced-resistance phenomenon reported by Hubert & Helton (4) for Italian prune trees as a result of *Cytospora* invasion also occurs in J. H. Hale peach trees; (ii) the induced resistance response is more prominent in J. H. Hale peach trees at a distance of at least 18 cm from the site of primary invasion than it is in Italian prune trees (9); (iii) the max canker size achieved prior to the onset of marginal healing varies directly with the initial vigor of *Cytospora* invasion.

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