# Relationship of Number of Cytospora Infections on Prunus domestica to Rate of Expansion of Individual Cankers

A. W. Helton and J. W. Braun

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

ABSTRACT

Two-year-old Italian prune trees were infected simultaneously with *Cytospora cincta* at 1, 2, 3, 4, 5, 6, or 7 locations per tree. Subsequent development of cankers suggested that the rate of canker expansion varies inversely with number of infections initiated in a single tree. Phytopathology 60:1700-1701.

*Additional key words:* Induced resistance, resistance, mass action.

Little has been reported on the effect of lesion numbers on development of individual infections in plants. In cases where the host possesses preformed resistance, however, increasing numbers of lesions may result in proportionate weakening of the host with a corresponding decline in resistance (7, p. 111) so that more rapid expansion of individual infections can occur. The converse would become likely in cases where the resistance is preformed but develops upon invasion by the pathogen, as reported for *Cytospora cincta* Fr. in Italian prune (*Prunus domestica* L.) by Helton & Hubert (4, 5). Discovery of this induced resistance phenomenon in Italian prune trees raised the question as to whether increasing numbers of infections would result in increased resistance. Such a quantitative increase in resistance incitation should lead to reduced expansion rates of individual infections. Support for this idea was found in the report of Cruickshank & Perrin (2) in which they described a direct relationship between concn of spores in an inoculum preparation and the concn of pisatin (2), a phytoalexin produced by pea pods in response to the inoculum.

The present study, therefore, was undertaken to determine the effect of multiple simultaneous *Cytospora cincta* infections on development of individual cankers in Italian prune trees.

On 18 June, 2-year-old Italian prune trees growing on seedling rootstocks of Lovell peach (*P. persica* [L.] Batsch) were selected and indexed on Shirofugen flowering cherry (*P. serrulata* Lindl.) for the presence of *Prunus* ringspot virus (PRSV). Exclusion of PRSV-infected trees was necessary because PRSV influences development of *Cytospora* cankers (4) by induction of a similar if not identical resistance reaction. The indexed trees were divided into seven groups of six trees each for the experiment. Treatments consisted of 1, 2, 3, 4, 5, 6, or 7 *Cytospora* infections/tree, one canker/scaffold branch. Inoculations were made with *Cytospora cincta* Fr. (isolate Cy-59) using an impact-wound (ca. 1 $cm^2$) and rubber tape wrapping technique previously described (3).

Expansion of individual cankers was followed by measuring the length and width to the nearest 0.1 cm and using the product of these dimensions, minus the impact-wound area (also length $\times$ width), as a standardized expression of approx canker-necrosis area. Measurements were made 2 weeks after inoculation and at 2-week intervals thereafter for the remainder of the study.

Data are presented (Fig. 1) as mean canker size (MCS) per treatment (i.e., for 1, 2, 3, 4, 5, 6, or 7 cankers/tree).

Decrease in MCS after the 12 August observation date was the result of marginal healing (formation of callus tissue by the host at the canker margins, resulting in a progressive decrease in canker dimensions). When marginal healing became evident, the necrotic tissue was removed to expose the constricting margins and allow more accurate measurement of the shrinking canker dimensions.

Duncan's multiple range test (5% level) for unequal subclass numbers was applied, using the MCS per tree, for each observation date. The use of unequal subclass numbers became necessary when subsequent detection of PRSV infection necessitated discarding of 4 of the 42 trees used in the experiment. Data from the four virus-infected trees were discarded.

MCS measurements showed that the more cankers initiated simultaneously per tree the smaller the individual cankers tended to be after a given period of time (Fig. 1). Results of Duncan's multiple range test generally verified this trend. Significant difference (5% level) was found between the MCS for 1 or 3 cankers/tree and the MCS for 5, 6, or 7 cankers/tree until the marginal healing reaction halted increases in canker size. The 2- and 4-cankers/tree treatments were not significantly different from each other and generally not different from the 1 or 3 canker group or the 5, 6, or 7 canker group.

In 1942, Allen (1) reported similar results when he demonstrated that lesions of powdery mildew (*Erysiphe graminis*) on heavily infected leaves of wheat never became as large as those on lightly infected leaves; however, because localized areas (individual leaves) were involved, factors such as competition for nutrients and moisture and production of self-inhibiting toxins by the pathogen probably were largely responsible for differences in lesion size (6).

It is doubtful that these factors were responsible for the differences in canker size reported in the present study, where whole trees and relatively small amounts of infection were involved. Rather, it is probable that these differences are an expression of the same induced resistance reported earlier (4, 5), and that the induced resistance, or inhibitory effect, reacts on initiating infections as well as on subsequent infections. The present study suggests that the induced resistance increases with the number of cankers initiated in a given tree at the same time. The magnitude of the resistance

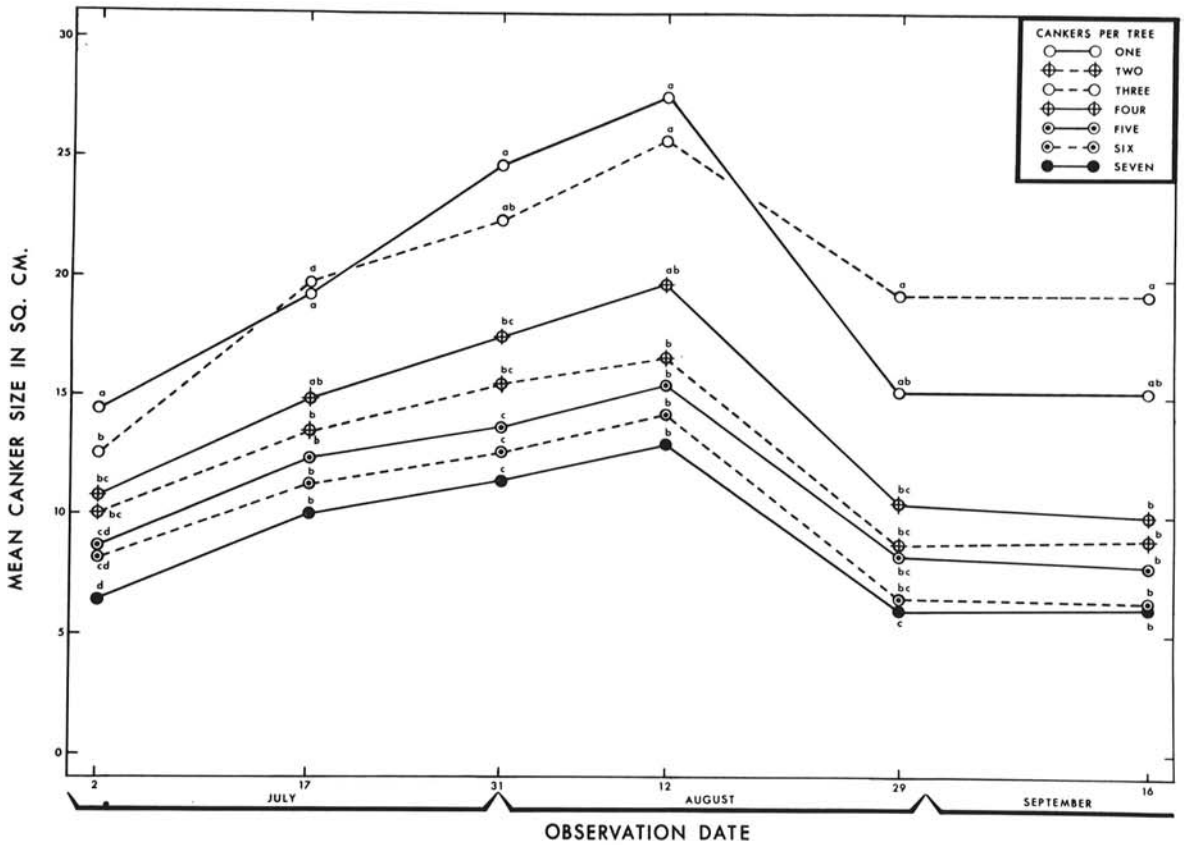


**Fig. 1.** Effect of various numbers of *Cytospora* cankers per tree on size of individual cankers when 1, 2, 3, 4, 5, 6, or 7 cankers (1/branch) were initiated on 2-year-old Italian prune trees on 18 June; each value represents mean canker size (MCS) per treatment; lower case letters a, b, c, and d indicate significant differences (5% level) between MCS values for the seven treatments at any one observation date (same letter indicates no significant difference).

effect in an infected tree therefore seems to be a function of the number of cankers on the tree as well as a function of the time elapsed after initial invasion as demonstrated previously (2).

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