Pathogenicity and Relative Virulence of Seven *Phytophthora* spp. on Mahaleb and Mazzard Cherry

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ABSTRACT

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Isolates of *Phytophthora cryptogea*, *P. cambivora*, and *P. megasperma* from cherry, isolates of *P. cinnamomi* and *P. citricola* from walnut, and an isolate of *P. cryptogea* from safflower individually caused 88–100% root rot and 27–100% crown rot on Mahaleb cherry seedlings that were grown for 15 wk in artificially infested UC mix and periodically flooded. In contrast, cherry isolates of *P. drechsleri* and an unidentified *Phytophthora* sp. caused 62 and 41% root rot, respectively, but caused no crown rot under the same conditions. Mazzard cherry seedlings frequently appeared less susceptible than Mahaleb seedlings to root and crown rot, although this varied with the *Phytophthora* species involved. Mazzard appeared to be significantly more resistant than Mahaleb to both root rot and crown rot caused by *P. cambivora*, *P. megasperma*, and the safflower isolate of *P. cryptogea*, and to crown rot caused by *P. cinnamomi* and *P. citricola*. However, Mazzard roots appeared nearly as susceptible as Mahaleb roots to the latter two *Phytophthora* spp., and roots of both cherry species appeared moderately susceptible to *P. drechsleri* and the unidentified *Phytophthora* sp. Roots and crowns of Mazzard also appeared as highly susceptible as Mahaleb to the cherry isolate of *P. cryptogea*. This is believed to be the first report experimentally implicating *P. cryptogea* as a pathogen of a commercial stone fruit tree species in the United States.

Additional key words: *Prunus avium*, *Prunus mahaleb*, soilborne diseases, wet feet.

Phytophthora root and crown rots reached epidemic proportions in California sweet cherry (*Prunus avium* L.) orchards during the early 1970s, and by 1976, *P. megasperma*, *P. cambivora*, and *P. drechsleri* had been identified as causal agents of these diseases (11). Subsequent isolations from numerous sweet cherry trees showing typical root or crown rot symptoms (11) have yielded additional unidentified *Phytophthora* spp. (8,9), although the pathogenicity of these species has not been previously demonstrated. A number of *Phytophthora* spp. have also been isolated from dead and declining peach, apricot, prune, apple, walnut, and almond trees throughout the deciduous fruit and nut tree-growing districts of California (8,9,11,13,14). These isolations have raised concern that such orchards might serve as a source of

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inoculum for cherry root and crown rots. Orchards infested with *P. cinnamomi* and *P. citricola* may warrant special concern, as both fungi are destructive pathogens on several deciduous fruit and nut tree species (8–10,13), although neither has yet been isolated from cherry in the field.

The objectives of this study, therefore, were: to determine which *Phytophthora* spp. other than *P. megasperma*, *P. cambivora*, and *P. drechsleri* are associated with dead and declining cherry trees in California; to determine the pathogenicity and relative virulence on two common cherry rootstocks of the *Phytophthora* spp. most commonly recovered from cherry; and to determine whether *P. cinnamomi* and *P. citricola* are potential pathogens of cherry.

MATERIALS AND METHODS

Recovery and identification of *Phytophthora* spp. *Phytophthora* spp. isolates were recovered from decayed root and crown tissue and from the soil surrounding dead or declining cherry trees using

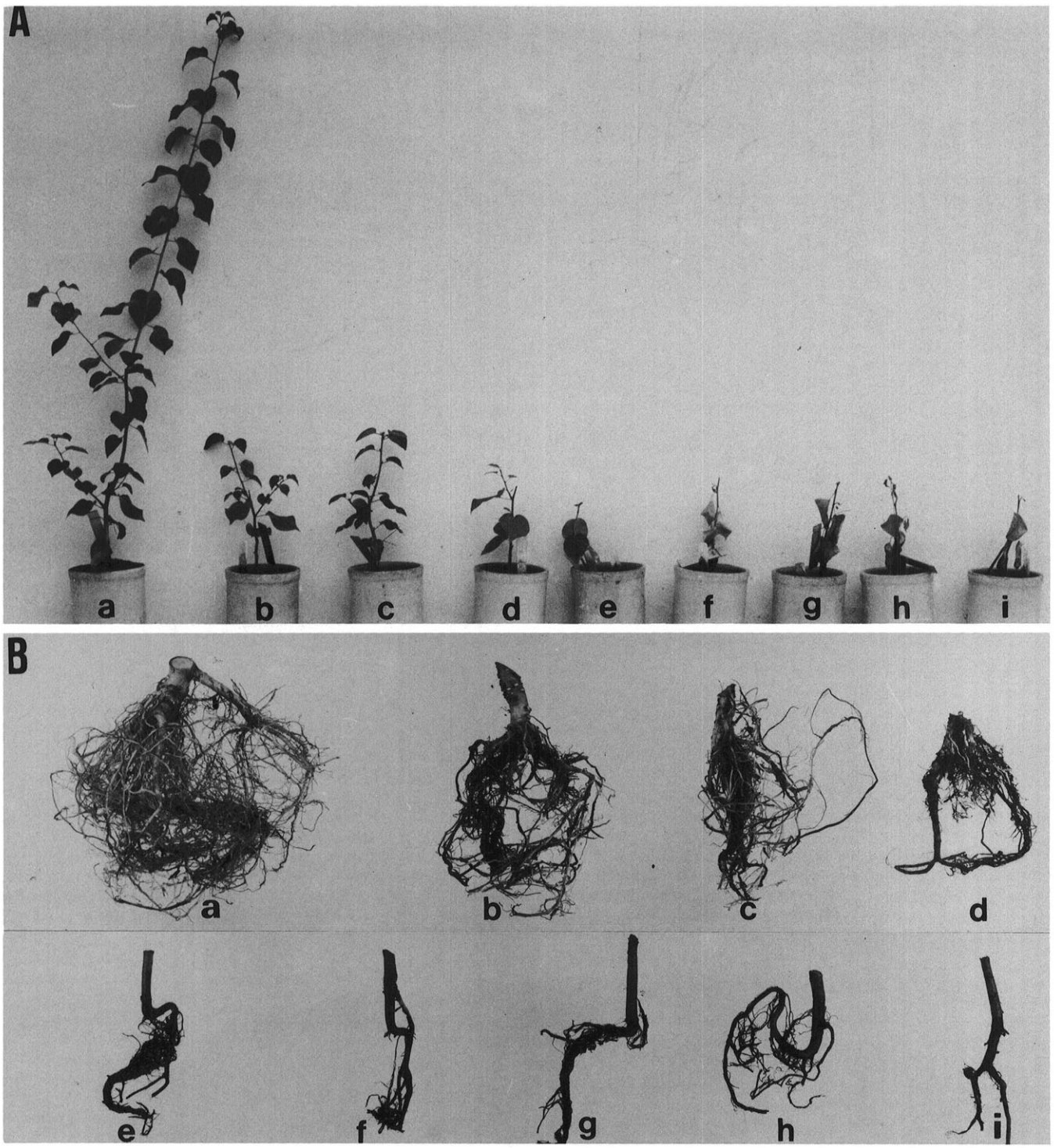

Fig. 1. A, Tops and **B,** roots of representative Mahaleb seedlings grown for 15 wk in UC mix. Soil in the crocks was either (a) uninfested (control), or artificially Infested with (b) a cherry isolate of an unidentified *Phytophthora* sp., (c) a cherry isolate of *P. drechsleri*, (d) a safflower isolate of *P. cryptogea*, (e) a cherry isolate of *P. megasperma*, (f) a walnut isolate of *P. cinnamomi*, (g) a walnut isolate of *P. citricola*, (h) a cherry isolate of *P. cambivora*, or (i) a cherry isolate of *P. cryptogea*.

previously described procedures (11). Isolates were cultured and induced to sporulate by methods employed previously (11), and identified on the basis of Waterhouse's key (20) and original species descriptions (16,19,21).

Pathogenicity and virulence tests. Cherry isolates of *P. megasperma* Drechsler (isolate CH 275C-1), *P. cambivora* (Petri) Buisman (P543), *P. drechsleri* Tucker (P527), *P. cryptogea* Pethyb. and Laff. (P1349), and an unidentified *Phytophthora* sp. (P1347) were used in inoculation tests, as were isolates of *P. cinnamomi* Rands (P1011) and *P. citricola* Sawada (P521) from walnut. A safflower isolate of *P. cryptogea* (P201) obtained from J. M. Duniway (3,4) was also included in the study to compare its pathogenicity on cherry with that of a cherry isolate of the same species.

Inocula were prepared by using methods described previously (11), with the following minor amendments: 250 cc of vermiculite was mixed with 20 cc of whole oat kernels in a 473-ml canning jar, thoroughly moistened with 175 ml of V-8 juice broth, autoclaved twice, and then inoculated with one of the isolates of *Phytophthora* spp. After allowing 4 wk for fungus colonization at 21 C, the substrate was thoroughly rinsed to remove excess nutrients, and mixed with a steam-pasteurized potting medium consisting of UC mix (UCM) (1) and sand (2:1, v/v) at the rate of 20 cc of inoculum per 1,000 cc of potting medium. Controls received an uninoculated vermiculite-oat-broth mixture at the same rate.

Five replicate 8-wk-old Mazzard (*Prunus avium* L. 'Silverbark Mazzard') and Mahaleb (*Prunus mahaleb* L.) cherry seedlings were transplanted individually into 1-L crocks containing infested potting medium, watered immediately, and allowed to drain. Two weeks after transplanting, the potting mix in each crock was flooded by plugging the bottom drainage hole and adding water until approximately 10 mm of water stood on the soil surface. After 48 hr of flooding, all plugs were removed and normal drainage was resumed. This procedure was repeated at 2-wk intervals until the experiment was terminated 15 wk after transplanting. Plants were watered as needed between flooding treatments and fertilized weekly with a modified (no micronutrients except Fe) half-strength Hoagland's solution.

The experiment was repeated three times in a greenhouse, where soil temperatures ranged from 18–22 C in two experiments, and from 18–26 C in the third. Supplementary lighting was used as necessary to provide a 15-hr photoperiod. Pathogenicity and virulence of the different isolates were judged on the basis of final shoot and root fresh weights, a visual estimation of the percent root mass rotted, and the incidences of crown rot and seedling mortality. Root and crown rots were confirmed as resulting from infection by the appropriate *Phytophthora* sp. by reisolating the fungus from decaying tissue on a modified PVP medium (11). The data were combined and analyzed statistically by treating each experiment as a factor in a 3 (experiments) × 9 (isolates) × 5 (replicates) factorial design. There were no significant interactions among any combination of factors.

RESULTS

Identification of *Phytophthora* spp. The four *Phytophthora* spp. most frequently associated with dead or declining cherry trees were identified as *P. megasperma*, *P. cambivora*, and *P. drechsleri* as previously described (11), and as *P. cryptogea* Pethyb. and Laff. An unidentified *Phytophthora* sp. was also associated with diseased trees in several orchards. Additionally, isolates identified as *Phytophthora syringae* (Kleb.) Kleb. and *P. cactorum* (Leb. & Cohn) Schroet. were recovered from diseased trees in single orchards.

Because of reported (2) similarities between isolates of *P. drechsleri* and *P. cryptogea*, the latter are described here in some detail. After 4 days at 21 C, isolates of *P. cryptogea* produced rapidly growing (6–7 mm/day radial increase), radiate colonies with sparse aerial growth on Difco cornmeal agar (CMA), and uniform colonies with moderate-to-abundant aerial growth on V-8

TABLE 1. The relative virulence of seven *Phytophthora* spp. from three hosts on Mahaleb cherry seedlings grown for 15 wk in artificially infested UC mix

Phytophthora sp. and source host[c]	Fresh weight (g)[a]		Root rot[a,d,e] (%)	Number of plants[b]	
	Shoots	Roots		With crown rot[e]	Dead
Control (uninfested)	39.3 A	24.3 A	7 F	0 D	0 D
Phytophthora sp. cherry	13.8 B	9.1 B	41 E	0 D	0 D
P. drechsleri cherry	11.0 B	7.0 B	62 D	0 D	0 D
P. megasperma cherry	3.9 C	2.8 C	98 AB	6 C	7 BC
P. cambivora cherry	2.9 C	2.5 C	99 A	11 B	11 B
P. cryptogea cherry	2.4 C	2.2 C	100 A	15 A	15 A
P. cryptogea safflower	3.1 C	2.6 C	90 BC	4 C	4 C
P. citricola walnut	5.6 C	4.1 C	88 C	11 B	10 B
P. cinnamomi walnut	2.7 C	1.8 C	99 A	12 AB	11 B

[a] Average of 15 observations (five replications per experiment × three experiments) per isolate. Values in each column followed by the same capital letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.
[b] Totals out of 15 plants from three experiments.
[c] Inoculum added at the rate of 20 cc of colonized vermiculite per 1,000 cc of UC mix.
[d] Percent of the root system rotted based on visual estimation.
[e] The *Phytophthora* sp. introduced by inoculation was reisolated from tissue of all symptomatic plants. No *Phytophthora* spp. were recovered from control plants.

TABLE 2. The relative virulence of seven *Phytophthora* spp. from three hosts on Mazzard cherry seedlings grown for 15 wk in artificially infested UC mix

Phytophthora sp. and source host[c]	Fresh weight (g)[a]		Root rot[a,d,e] (%)	Number of plants[b]	
	Shoots	Roots		With crown rot[e]	Dead
Control (uninfested)	33.5 A	35.0 A	6 E	0 D	0 D
Phytophthora sp. cherry	15.5 C	14.2 CD	38 D	0 D	0 D
P. drechsleri cherry	11.4 D	10.4 DE	55 C	0 D	0 D
P. megasperma cherry	11.2 D	8.2 EF	74 B	3 CD	3 CD
P. cambivora cherry	20.6 B	19.4 B	35 D	0 D	0 D
P. cryptogea cherry	3.3 F	3.9 F	99 A	12 A	12 A
P. cryptogea safflower	9.1 DE	8.5 DE	58 C	1 D	1 D
P. citricola walnut	9.3 DE	6.9 EF	79 B	5 BC	5 BC
P. cinnamomi walnut	6.3 EF	4.6 F	96 A	7 B	6 B

[a] Average of 15 observations (five replications per experiment × three experiments) per isolate. Values in each column followed by the same capital letter do not differ significantly ($P = 0.05$) according to Duncan's multiple range test.
[b] Totals out of 15 plants from three experiments.
[c] Inoculum added at the rate of 20 cc of colonized vermiculite per 1,000 cc of UC mix.
[d] Percent of the root system rotted based on visual estimation.
[e] The inoculated *Phytophthora* sp. was reisolated from tissue of all symptomatic plants. No *Phytophthora* spp. were recovered from control plants.

juice agar (V8A). On CMA, the growth rate increased from 9 to 24 C and then decreased with higher temperatures until only a trace of growth occurred at 36 C. No growth occurred after 4 days at 6 or 39 C. Nonpapillate sporangia, borne singly on undifferentiated sporangiophores of uniform width, were typically ovate and well-rounded at the base, and measured 34–60 × 27–39 μm (average 41 × 33 μm) when produced on V8A disks floated in 1.5% nonsterile soil extract at 21 C. Sporangia produced from colonized Mahaleb leaf disks buried in moist UCM were occasionally kidney-shaped as pictured in the report (16) originally describing *P. cryptogea*. No oospores were produced by single cultures grown on clarified V8A amended with β-sitosterol (V8β) (11) after 8 wk in the dark at 20 C, but a few were formed when the same cultures were paired with A^1 mating types of *P. cryptogea*, *P. drechsleri*, or *P. cinnamomi* under the same conditions.

Isolates of the unidentified *Phytophthora* sp. were distinguished by broadly ovate, nonpapillate sporangia that had well-rounded bases and were borne singly on undifferentiated sporangiophores of uniform widths. Sporangia of two typical isolates (P1347 and P1415) were 42–85 μm long × 30–75 μm wide (average 65 × 58 μm) with an average length/width ratio of 1.1 when formed at 21 C. No isolates formed sexual structures in single culture on V8β, but all of those examined did so when paired with known A^2 isolates of *P. cryptogea* and *P. drechsleri*. Several different isolates resembling this *Phytophthora* sp. from cherry have also been recovered from apricot (*Prunus armeniaca* L.) and Duke cherry (*Prunus avium* L. × *P. cerasus* L.) in the eastern United States and from apple (*Malus sylvestris* Mill.) in California (10).

Pathogenicity and relative virulence of *Phytophthora* spp. The isolates of all *Phytophthora* spp. examined were pathogenic on both Mazzard and Mahaleb seedlings. *Phytophthora megasperma*, *P. cambivora*, *P. citricola*, *P. cinnamomi*, and both isolates of *P. cryptogea* were all highly virulent on Mahaleb, causing total or near-total decay of the root system, and often causing crown rot and death of the seedlings as well (Table 1, Fig. 1). The highest incidence of crown rot on Mahaleb was caused by the cherry isolate of *P. cryptogea*, followed by *P. cinnamomi*, *P. cambivora*, and *P. citricola*, whereas *P. megasperma* and the safflower isolate of *P. cryptogea* were less consistent crown invaders (Table 1). The unidentified *Phytophthora* sp. and *P. drechsleri* were less virulent than other species, causing 42 and 61% root rot, respectively, and no crown rot or seedling mortality (Table 1, Fig. 1). Similarly, root and shoot fresh weights were reduced less by the unidentified *Phytophthora* sp. and *P. drechsleri* than by any of the remaining *Phytophthora* spp. (Table 1).

In general, Mazzard seedlings developed less root rot and had

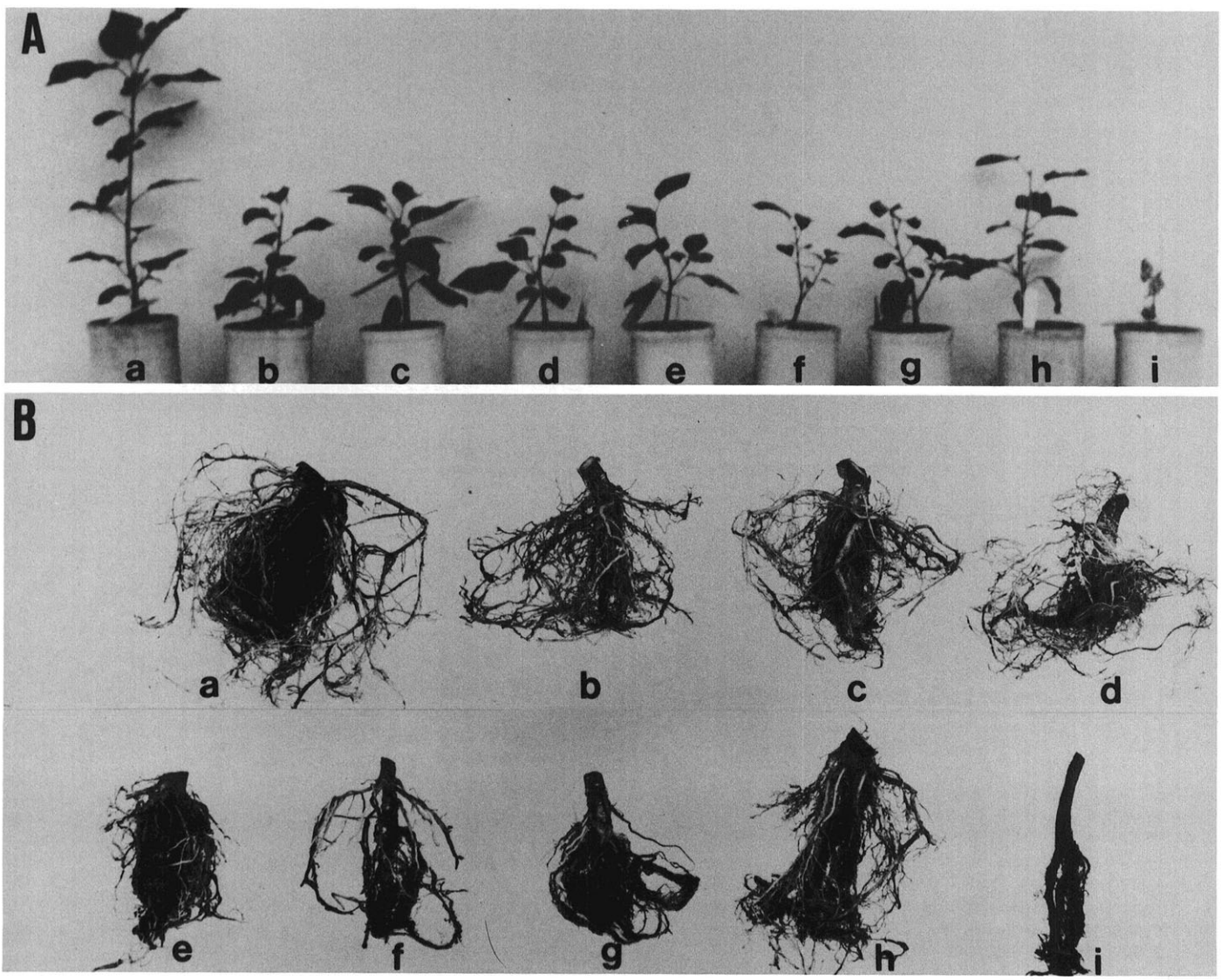

Fig. 2. A, Tops and **B,** roots of representative Mazzard seedlings grown for 15 wk in UC mix. Soil in the crocks was either (a) uninfested (control), or artificially infested with (b) a cherry isolate of an unidentified *Phytophthora* sp., (c) a cherry isolate of *P. drechsleri*, (d) a safflower isolate of *P. cryptogea*, (e) a cherry isolate of *P. megasperma*, (f) a walnut isolate of *P. cinnamomi*, (g) a walnut isolate of *P. citricola*, (h) a cherry isolate of *P. cambivora*, or (i) a cherry isolate of *P. cryptogea*.

lower incidences of crown rot and death than Mahaleb seedlings when both were grown in UCM infested with the same fungus (Tables 1 and 2, Figs. 1 and 2). However, the extent to which Mazzard appeared more disease-resistant varied according to the *Phytophthora* sp. involved. For example, *P. cambivora* caused total root rot and a high incidence of crown rot (11/15 seedlings) on Mahaleb (Table 1), but it caused only moderate root rot and no crown rot on Mazzard (Table 2). Similarly, *P. megasperma* and the safflower isolate of *P. cryptogea* caused substantially less root rot and lower incidences of crown rot on Mazzard (Table 2) than on Mahaleb (Table 1). Mazzard also developed less crown rot caused by *P. citricola* and *P. cinnamomi*, although both hosts appeared to be about equally susceptible to root rot caused by these two species (Tables 1 and 2). In contrast, Mazzard appeared as highly susceptible as Mahaleb to both root and crown rot caused by the cherry isolate of *P. cryptogea* (Tables 1 and 2). Like Mahaleb, Mazzard also appeared only moderately susceptible to root rot caused by *P. drechsleri* and the unidentified *Phytophthora* sp. (Tables 1 and 2).

DISCUSSION

These results confirm an earlier report (11) that *P. megasperma*, *P. cambivora*, and *P. drechsleri* are serious pathogens of cherry in California, and additionally implicate *P. cryptogea* and an unidentified *Phytophthora* sp. as causes of root rot and crown rot in the state's sweet cherry orchards. This appears to be the first report to experimentally implicate *P. cryptogea* as an important pathogen of a commercial fruit tree species other than, possibly, pear (15) in the United States. Although *P. cryptogea* has been shown to cause a serious disease of chestnut in Australia (22) and of Douglas-fir seedlings in the Pacific Northwest (17), it has primarily been reported as a pathogen of herbaceous hosts (18) or as a cause of only minor root rot on woody hosts (5,7). Our research, however, suggests that *P. cryptogea* can be an extremely virulent pathogen on both Mahaleb and Mazzard, which are standard rootstocks for both sweet and sour cherry trees.

Although it has been suggested that *P. cryptogea* and *P. drechsleri* should be considered one species (2), cherry isolates identified as *P. cryptogea* were distinctly different from cherry isolates previously identified as *P. drechsleri* (11). The sporangia of the *P. drechsleri* isolates, which typically had tapered bases and were borne upon sporangiophores that widened at the apices (11), clearly contrasted with the sporangia and sporangiophores of the isolates of *P. cryptogea* described herein. These same differences have been recognized by Klisiewicz (6) and Waterhouse (20), and are illustrated in the reports originally describing the two species (16,19).

The severe root rot and high incidence of crown rot caused by the walnut isolates of *P. cinnamomi* and *P. citricola* suggest that these species also pose a serious potential threat to cherry orchards. Furthermore, the high virulence on cherry of these walnut isolates, of the safflower isolate of *P. cryptogea* (Tables 1 and 2), and of numerous isolates of *P. megasperma* from unrelated hosts (23), collectively indicates that many *Phytophthora* spp. isolates that attack deciduous fruit and nut trees are not host specific. Such nonspecificity should be considered in decisions involving the use of recycled irrigation water, selection and preparation of new orchard planting sites, choice of tree species to be planted, and the movement of equipment between fields and orchards suspected of having Phytophthora disease problems.

The present study further confirms earlier reports that Mazzard is more resistant than Mahaleb to *P. cambivora* and *P. megasperma* (11,12), which commonly cause root and crown rot on cherry in California. Furthermore, this study also indicates that Mazzard is more resistant than Mahaleb to additional *Phytophthora* spp. (Tables 1 and 2) which have the potential to cause severe crown and/or root rot on cherry. It especially appears that Mazzard's higher resistance to crown rot may be an important reason why fewer dead trees are observed in orchards planted on Mazzard than on Mahaleb rootstock (11). A cherry tree can regenerate new roots to replace those lost to infection and thereby recover from the effects of root rot if subsequent infections are minimized. However, a single infection period that results in crown rot can be fatal if it leads to girdling of the tree.

Control of Phytophthora root and crown rots on cherry should include precautions designed to exclude the pathogens from uninfested orchards; appropriate rootstock selection (eg, Mazzard appears preferable to Mahaleb in soil infested with any of a number of different *Phytophthora* spp.); and proper soil water management techniques, particularly those that avoid prolonged periods of soil flooding (12,24). Soil water management appears to control root and crown rots caused by *P. cryptogea*, *P. megasperma*, or *P. drechsleri* more effectively than those caused by *P. cambivora* (24). However, the present study suggests that selecting Mazzard over Mahaleb rootstock may reduce disease severity most effectively when *P. cambivora* is the pathogen involved. These results emphasize the importance of integrating all available control measures in order to minimize losses caused by such potentially destructive pathogens.

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Resistance

Chemical Desiccation of Wheat Plants as a Simulator of Postanthesis Speckled Leaf Blotch Stress

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ABSTRACT

Zilberstein, M., Blum, A., and Eyal, Z. 1985. Chemical desiccation of wheat plants as a simulator of postanthesis speckled leaf blotch stress. Phytopathology 75:226-230.

Field trials were conducted over 2 yr to evaluate whether the postanthesis destruction of the photosynthetic source by chemical desiccation may be used to detect wheat cultivars that sustain kernel growth in the presence of speckled leaf blotch. Spring wheat cultivars of diverse origin were subjected to three treatments: inoculation with virulent isolates of *Septoria tritici* until an epidemic was initiated, postanthesis application of magnesium chlorate (4% a.i.) solution to destroy most of the plant's green tissues, and a control in which a fungicide was applied to protect against loss of green tissue. Grain yield components, kernel growth curves, and harvest index were determined. The progress of the pathogen on the four uppermost leaves was monitored at weekly intervals. For similar Septoria progress values (area under the disease progress curve), various cultivars manifested different rates of loss in kernel weight (as percent of the fungicide-treated control), and harvest index. Cultivars differed in the rate of loss in kernel weight and harvest index when treated with the chemical desiccant. Significant correlations were revealed across the same five cultivars in losses in kernel weight between plants infected by *S. tritici* and those that had been chemically desiccated in 1 yr ($r = 0.925$) but not in the other ($r = 0.804$). Correlation between the two parameters was highly significant ($r = 0.626$) across 16 (out of 18) cultivars that had similar Septoria progress values. The correlations in losses in kernel weight between plants infected by *S. tritici* and those that had been mechanically defoliated (postanthesis) in the five cultivars in 1981-1982 were $r = 0.733$. The potential for utilizing postanthesis chemical desiccation as a measure for revealing wheat cultivars tolerant to speckled leaf blotch in the absence of infection by *S. tritici* is discussed.

Additional key words: tolerance, *Triticum aestivum*, yield components.

Speckled leaf blotch, which is caused in wheat by the fungus *Septoria tritici* Rob. ex Desm. (perfect state: *Mycosphaerella graminicola* (Fuckel) Schroeter), may impose severe limitations on crop yield. In certain environments and years the impact is more pronounced than in others (7).

Under certain environments, early buildup of infection by *S. tritici* on lower plant parts may adversely affect root biomass and plant development (19). Early infection of lower leaves also may reduce yield, especially by altering sink development (the number of tillers per plant, the number of spikes per plant, and the number of grains per spike) and consequently affect assimilate distribution (16,17,19).

Infection of the upper plant parts that are responsible for grain filling is considered to be the most significant factor contributing to losses in yield (18). Infection on upper plant parts usually affects kernel weight and grain number (6,9,22,24). The magnitude of reduction in yield components depends on the pre- and postanthesis level of disease-affected plant tissues, disease progress relative to plant growth stage, and cultivar response to disease stress (1,5,18). Wheat cultivars of similar phenotypic characters may express differential loss in yield under similar apparent disease severity and disease progress (4,22–24). Wheat cultivars may vary in ability to endure (tolerate) severe Septoria epidemics without sustaining significant losses in yield when compared to vulnerable (nontolerant) cultivars (5). This endurance or the tolerance of plants to pathogen-generated stress (10,11,14), though widely mentioned, is poorly understood. One of the major obstacles in evaluating plant responses to disease stresses relates to the inherent difficulties in establishing equivalent disease stresses across cultivars and characterizing the nature and magnitude of the imposed stresses. Accumulating evidence suggests that wheat genotypes vary in capacity to utilize stored assimilates as a source for kernel growth in the absence of postanthesis photosynthesis (2,3,20,21). The differential capacity of wheat cultivars to sustain translocation-based kernel growth in the absence of transient photosynthesis was revealed (3) through postanthesis destruction of the photosynthetic source by chemical desiccation. Since the major effect of postanthesis infection by *S. tritici* is expressed by reduction in the photosynthetic source, it was hypothesized that postanthesis chemical desiccation of the wheat canopy may simulate a uniform disease stress in testing for tolerance. Tolerance is thus estimated as the plant's capacity to sustain appreciable

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