SUMMARY RESEARCH ON BIOLOGY OF HYPOVIRULENT AND VIRULENT *ENDOTHIA PARASITICA* ON BLIGHT-RESISTANT AND BLIGHT-SUSCEPTIBLE CHESTNUT TREES AT VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

F. V. Hebard[1], G. J. Griffin[1], and J. R. Elkins[2]

[1] Department of Plant Pathology and Physiology
Virginia Polytechnic Institute and State University
Blacksburg, VA 24061

[2] Division of Natural Sciences
Concord College
Athens, WV 24712

ABSTRACT.--*Evidence is presented that some degree of blight resistance may be present in a large, surviving American chestnut tree. The evidence includes results of inoculations of stems excised from the tree, of grafted scions of the tree, and of the intact tree, including microscopical observations of such cankers. Additionally, the lengths of naturally caused cankers on the tree were measured over a 3-year period and seedlings of other large, surviving American chestnuts were inoculated. Results also are outlined of histopathological examination of the development of cankers incited by a virulent (V) and a hypovirulent (H) strain of Endothia parasitica on blight-resistant Chinese chestnut and blight-susceptible American chestnut, as are studies of epidemics of V strains of E. parasitica on American chestnut and an experiment on biocontrol by H strains of cankers caused by V strains.*

Introduction

The purpose of the studies detailed in this paper was to determine the cause(s) of survival of a large, American chestnut tree. We measured its blight resistance and the pathogenicity of the strains of *Endothia parasitica* which were attacking it. We hoped to assess the effect of blight resistance on disease control with hypovirulent *E. parasitica;* part of that assessment was a test of the potential of four hypovirulent isolates to biocontrol cankers of four virulent isolates on American chestnut stump sprouts (blight-susceptible). To help our understanding of hypovirulence and blight resistance, we also examined the time-course of canker development of a virulent (V) and hypovirulent (H) isolate of *E. parasitica* on the large, surviving American chestnut, on blight-resistant Chinese chestnut, and on blight-susceptible stump sprouts of American chestnut. Finally, to provide a foundation for understanding spread of hypovirulence, we examined the dynamics of the increase in blight incidence which occurs after clearcutting of

forested areas. Studies of the large, surviving American chestnut tree, the Fldg tree, are reported in detail in this paper and the results of biocontrol, histopathological, and epidemiological studies are summarized.

Methods and Results

Blight resistance in large, surviving American chestnut trees

The Fldg tree measured 39 cm in diameter at breast height (d.b.h.) in 1977, and 85 percent of its crown was alive. The presumed parent of the Fldg tree was located about 10 yards from it; the parent measured over 100 cm d.b.h. without its bark. Patches of bark were clinging to the presumed parent indicating that it had died within the last 10 years (Gravatt and Gill 1930). Another nearby large presumed seedling of the parent tree, the seedling being called the Flds tree, measured 18 cm d.b.h. with 20 percent of its crown alive in 1977. The Flds tree was killed by blight in 1980. Another nearby apparent seedling is approximately 15 cm d.b.h. Three to five other small American chestnut plants also are located near the Fldg tree.

The main bole of the Fldg tree is encased by a blight canker for 2.2 m above ground level. Isolated natural cankers also exist on the Fldg tree. Three natural cankers were treated on June 21, 1978, with the H isolate, EP-66 (Elliston 1978). Inoculations with EP-66 were made every 10 cm around the uninfected periphery of each canker. Three untreated natural cankers served as controls. The treated cankers increased by 8.8 cm in mean length between July 3, 1978, and March 8, 1981, and the untreated cankers increased 13.0 cm in mean length. There was no evidence of callusing on any canker.

In 1980, ten isolates were collected from the main bole of the Fldg tree and two from one of the isolated natural cankers treated with EP-66. The isolates were tested for pathogenicity (Griffin et al. 1978). Two of the isolates from the main bole were hypovirulent, in that the five replicate cankers of each isolate had a mean total length of less than 3.2 cm 4 months after inoculation. The cankers of the H isolates also were superficial in that they did not reach the vascular cambium except around the inoculation point. The cankers of each of the other V isolates had a mean total length exceeding 12 cm 4 months after inoculation. The cankers of the V isolates extended to the vascular cambium over most of their length and produced abundant stromata. Ten or more isolates also were collected from 12 additional large (mean d.b.h. over 50 cm), surviving American chestnut trees in Virginia and West Virginia. From most trees, about 20 percent of the isolates were hypovirulent. However, there were no hypovirulent isolates in 28 isolates from one tree.

Before giving further results of inoculations with the Fldg and Flds trees, it will be helpful to outline the kinetics of canker growth following artificial inoculation. Figure 1 shows the lengths of three individual blight cankers versus days after inoculation. The top two curves in Figure 1 are typical of most of the 24 cankers of this experiment. The bottom curve is the most atypical canker growth curve. In the typical canker growth curve, there is an initial rapid spurt of growth followed by a plateau of no increase in canker length. The plateau started about day 9 in this particular experiment. At day 26, a phase of linear canker expansion began and persisted until the tree was girdled by the canker. At this time, canker elongation often ceased, especially above the canker. Sometimes a short

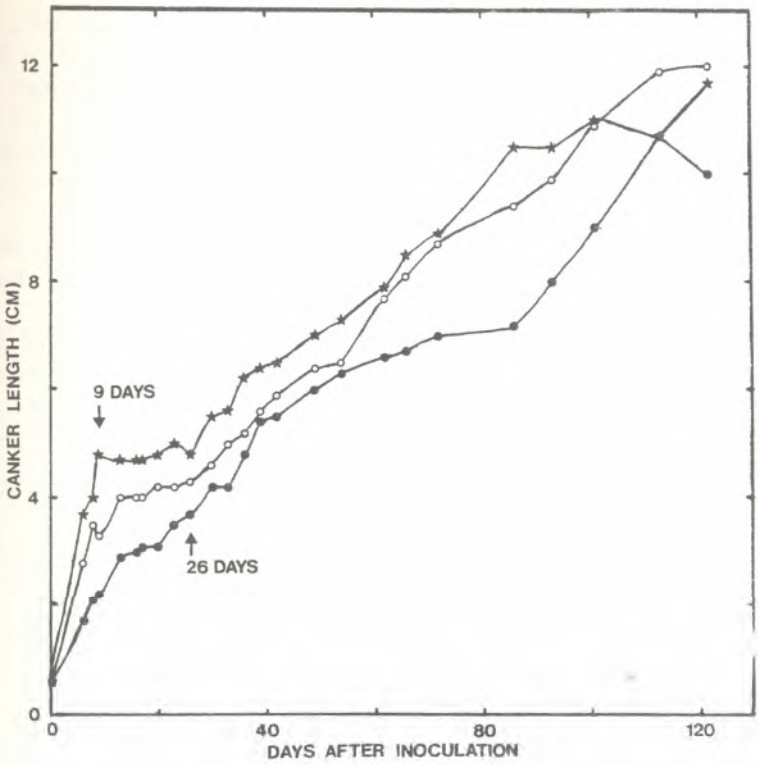

Figure 1. Lengths of three individual cankers on American chestnut seedlings from 6 to 122 days after inoculation with a virulent isolate, CR, of *Endothia parasitica*. Inoculations made June 7, 1979. The bottommost curve was from the most atypical of the 24 cankers. The top two curves are typical.

spurt of extremely rapid growth occurred at the time of canker girdling.

We collected scionwood material from the Fldg and Flds trees. Scions were collected from six other large (mean d.b.h. of 70 cm), surviving American chestnut trees and from two small, blight-susceptible American chestnut seedlings. In addition, seeds were collected from six large (mean d.b.h., 44 cm), surviving trees and three susceptible-type trees. Unfortunately, seeds from the Fld trees were unavailable. The scions were top-worked onto 15- to 30-year-old Chinese chestnut by bark grafting (Elkins et al. 1980). After germination and growth in 13 cm pots in the greenhouse, the seedlings were transplanted to a nursery plot, in rows 90 cm apart with 45 cm between plants in a row. Three years later, in June, 1980, each scion or seedling was inoculated twice to the vascular cambium with agar disks (1.5 mm diameter) of the V isolate of *E. parasitica*, CR. The cankers were measured every 3 to 4 days.

The inoculations of these plants (which included the H isolate, EP-66) were designed to detect blight resistance in them and to assess the effect of blight resistance on biocontrol of the V isolate by the H isolate. Unfortunately, except for two inoculations, the H isolate behaved as a virulent throughout the course of the experiment. Therefore, only the results for the V inoculations on grafted scions are presented here. It is suggested that the use of mycelial ball inoculum (Bazzigher and Schmid 1962) for the

small diameter inoculations would alleviate this complication, since older cultures, possibly with a higher dsRNA titer, could be used. In addition, many of the V inoculations failed on three of the seed sources, probably because the agar plates were allowed to become overheated. It is suggested that more careful attention to the condition of the inoculum might have eliminated the inconsistent results observed on these three seed sources.

Table 1 shows the mean diameter of the grafts at the inoculation point, canker lengths at 10 and 45 days after inoculation and the time of start of linear

Table 1. Canker growth statistics on grafted scions of large, surviving and small American chestnut trees inoculated with the virulent isolate of *Endothia parasitica*, CR

Scion Name [a]	Inoculations	Tree size	Stem diameter inoculation point [b]	Canker length [b] 15 days	Canker length [b] 45 days	Start of linear canker growth [b]
	no.		mm	mm	mm	days
Swent	2	large	4.1b	10.0bc	*	10.0c
GladA	10	small	22.1ab	18.7a	49.5a	12.8c
McDII	8	large	18.1ab	19.8a	49.5a	12.5c
GladB	4	small	10.9b	11.3bc	40.5ab	16.8bc
McDI	8	large	13.7b	18.9a	40.8ab	16.3c
Amhst	10	large	15.3b	12.0b	37.7b	13.2c
Weekly	10	large	15.1b	13.1b	36.9b	16.1c
Fldg	6	large	15.2b	11.0bc	32.0b	23.2bc
Flds	2	large	34.9b	18.0a	31.5b	33.5b
Gaul2	4	large	16.0ab	4.4c	10.3c	78.0a

[a] The start of linear canker growth was determined as the day after inoculation subsequent to which the increase in canker length from the previous observation exceeded 0.2 mm. Observations were 2 to 3 days apart, starting 5 days after inoculation. If one or two increases prior to day 25 were followed by three or more contiguous intervals where growth did not exceed 0.2 mm, the time of start was taken at first increase after that of more than 0.2 mm.

[b] Means within columns followed by the same letter are not significantly ($p < 0.05$) different by Duncan's multiple range test. There were two inoculations per scion.

* This graft was girdled and killed prior to 45 days after inoculation.

canker growth. The mean canker length at 45 days for the Fldg scions was significantly ($p<0.05$) shorter by Dunnett's test than the pooled mean for the two control scions (GladA and GladB) as was the mean time of start of linear canker growth in the Flds scions. However, Duncan's multiple range test revealed no significant ($p<0.05$) differences between the Fld scions and the GladB scions for these statistics. The small sample sizes may have contributed to the lack of sensitivity of the test. This is borne out by the results for the successfully inoculated seedlings (Table 2); there, many of the

Table 2. Canker growth statistics on seedlings of large, surviving blight-susceptible American chestnut trees inoculated with the virulent isolate of *Endothia parasitica*, CR

Seedling[b]	Stem diameter inoculation point[c]	Canker length[b] 15 days	Canker length[b] 45 days	Start of linear canker growth[b]
	mm	mm	mm	days
Wisc (su)	18.0bc	28.6a	64.2a	21.9b
McDI (sr)	25.3a	26.4ab	59.4ab	19.4b
McDII(sr)	17.5bc	21.4b	49.8bc	21.9b
Horn3(sr)	15.9cd	21.8b	48.7bc	25.2b
Gaul2(sr)	21.3ab	22.7b	47.7c	24.6b
Pea3 (sr)	11.6d	11.0c	25.7d	35.3a

[a] The start of linear canker growth was determined as the day after inoculation subsequent to which the increase in canker length from the previous observation exceeded 0.2 mm. Observations were 2 to 3 days apart, starting 15 days after inoculation. If one or two increases prior to day 25 were followed by three or more contiguous intervals where growth did not exceed 0.2 mm, the time of start was taken as the first increase after that of more than 0.2 mm.

[b] Seedling name, surviving (sr) or susceptible (su).

[c] Means within columns followed by the same letter are not significantly ($p < 0.05$) different by Duncan's multiple range test. There were five seedlings of each type with two inoculations per seedling.

seedlings from large, surviving trees gave canker growth statistics significantly ($p<0.05$) different from those for the seedlings of the susceptible-type trees (Wise), even though the magnitude of the differences were not as great as was observed on the grafts.

We have performed additional experiments with the Fldg tree. We excised stems from it and inoculated and incubated them in the laboratory using the method of Elliston (1978). Table 3 shows the results of this experiment. The cankers on the Fldg tree were significantly ($p<0.05$) shorter than the cankers on the control tree and the WA tree, another large, surviving American chestnut.

We also inoculated the Fldg tree *in situ*. Using the EP-66-CR H-V pair, five H+V, V-alone, and H-alone, inoculations were made on 2 cm diameter branches on the Fldg tree, one inoculation per branch. The inoculations were made on June 21, 1978. On November 11, 1978, the V cankers had a mean and standard deviation of 5.1 t 1.95 cm, the H cankers 2.8 t 0.54 cm, and the H+V cankers 6.8 ± 1.41 cm. By October, 1979, two of the branches above the five V cankers were dead and three of the branches above the five H+V cankers were dead. The two lethal V cankers had measured 17 and 22 cm long and the

Table 3. Mean canker lengths on excised stems (branches) of two large, surviving and one small American chestnut tree inoculated with virulent isolates of *Endothia parasitica*, CR and Weekly

Tree[a]	Tree size	Inoculations	Total canker length[c]		Net canker[b]	
			WK	CR	WK	CR
		no.	cm			
ST	Small	6	11.5a	11.8a	9.4a	9.5a
WA	Large surviving	6	8.8b	8.3b	6.6b	6.5b
FG	Large surviving	8	5.8b	6.2b	3.5c	4.2b

a/ Stump sprouts collected from a mature forest area in the Jefferson National Forest, Virginia, on February 21, 1979. Branches from large, surviving trees were collected on March 10, 1979.

b/ Means determined by subtracting initial lesion size, at 16 days, from total lesion size at 37 days at 27 to 28 C.

c/ Means within columns followed by the same letter are not significantly ($p < 0.05$) different by Duncan's multiple range test.

three lethal H+V cankers had measured 27, 30, and 17.5 cm long on June 18, 1979, one year after inoculation. Each of the three non-lethal V cankers were less than 2.5 cm long and the two non-lethal H+V cankers were 8.0 and 2.5 cm long on June 18, 1979. The non-lethal cankers did not grow further through October 31, 1980; most of them had ceased sporulating then, and some were hard to detect. The five cankers incited by the H isolate remained 6 cm long or less, and none of them killed the branches they were on.

Finally, we measured the lengths of cankers which had been made in connection with histopathological studies of canker development on the Fldg tree, on blight susceptible stump sprouts of American chestnut, and on blight-resistant grafted scions of Chinese chestnut, cultivar Nanking. Figure 2 shows the sum of the canker dimensions on the three hosts for the V and H isolates, CR and EP-66, versus days after inoculation in two experiments started July 4, 1978, and June 7, 1979. Each point in Figure 2 is a measurement of a separate canker. It can be seen that the cankers on the Fldg tree (denoted MR in the figure) were approximately the same length as, or shorter than, the cankers on the blight-resistant Chinese chestnut trees (denoted HR in the figure), and that, as expected, the cankers on the Chinese chestnut were shorter than the cankers on the American chestnut stump sprouts.

The canker length data indicate that resistance is playing a role in the survival of the Fldg tree. The findings that cankers on the grafts and seedlings of the G2 tree grew significantly ($p<0.05$) more slowly than cankers on all control trees indicate that there is heritable resistance to blight in

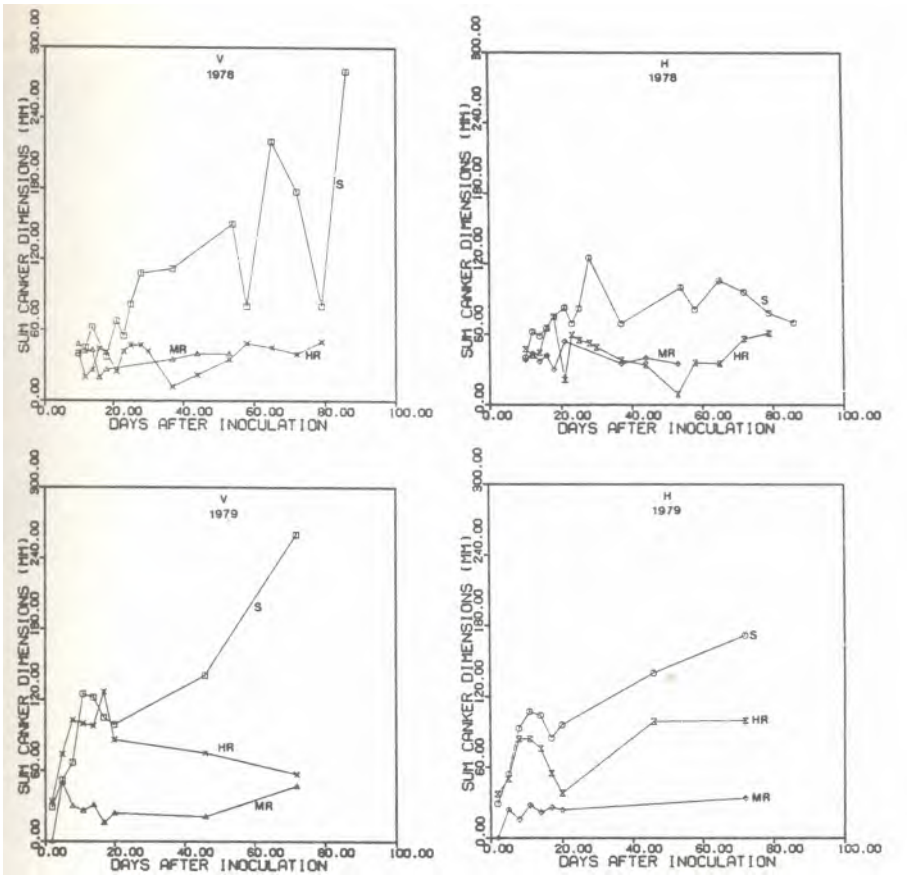

Figure 2. Sum of outer (at the outer periderm) and inner (at the vascular cambium) canker lengths and widths versus days after inoculatiion on July 4, 1978 and June 7, 1979, respectively. Of blight-susceptible (S) American chestnut stump sprouts, a moderately blight-resistant (MR), large, surviving American chestnut (the Fldg tree), and highly blight-resistant (HR) Chinese chestnut, cultivar Nanking, with virulent (V) and hypovirulent (H) strains of *Endothia parasitica*. The scatter in the data occurred because each point represents a separate canker. When there were duplicate cankers on one day for the S tree, the one with the shortest dimension is depicted.

American chestnut. The similar response of the grafts of the Flds and Fldg trees, which apparently are siblings, suggests that these plants also contain heritable blight resistance. Unfortunately, we have not been able to estimate the degree of heritability of blight resistance, because the cankers on the seedlings grew more rapidly than those on the grafts. As stated, about 20 percent of ten or more isolates each of 13 large, surviving American chestnut trees was hypovirulent. This, in conjunction with the above results, suggests that survival may be an interaction of resistance and hypovirulence. We do not know which is more important.

Histopathology

The scatter in the data points for Figure 2 (also for Figures 5 and 6) occurred because each point is a measurement of a separate canker. Each canker was processed for light microscopy to study canker development.

Comparing the curves in Figures 1 and 2, it can be seen that they have the same general outline for the blight-susceptible American chestnut stump sprouts, namely, that there is a rapid early flush of growth followed by a plateau before linear canker expansion begins. The rapid phase of growth was characterized by individual hyphae growing intra- and intercellularly (Figure 3a). The formation of a zone of lignified cells around the point of inoculation (Figure 3b) appeared to halt the expansion of these hyphae. The plateau phase of canker development then began until, in susceptible trees, a mycelial fan formed (Figure 4a) and expanded through and beyond the lignified zone. Linear canker growth was accompanied by the expansion of mycelial fans.

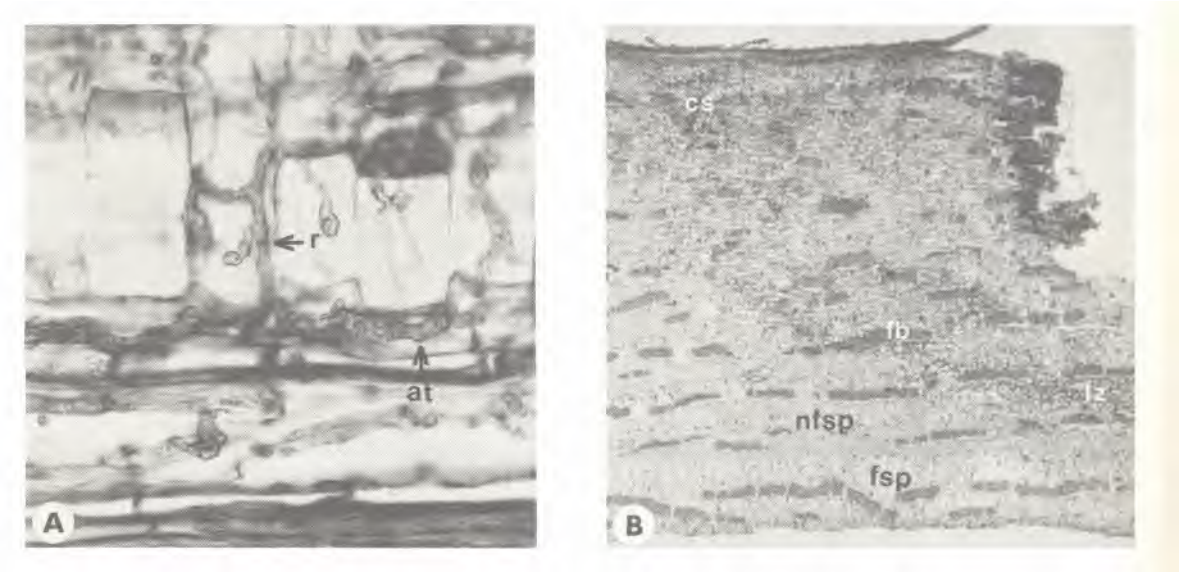

Figure 3. Micrographs of sections of chestnut blight cankers stained with safranin and fast green. Figure 3a. Radial section of an American chestnut stump sprout 12 days after inoculation with a virulent isolate of *Endothia parasitica.* Illustrates general appearance of hyphae in infected tissues. Note hyphae (arrows) in rays (r) and in axial phloem tissues (at) (x550). Figure 3b. Transverse section of a large, surviving American chestnut (the Fldg tree) 12 days after inoculation with a virulent isolate of *E. parasitica.* Inoculation wound is on the right top of section. Functioning (fsp) and nonfunctioning (nfsp) secondary phloem are evident. The infected tissues are surrounded by a dark-staining (safranin) zone of lignified cells (lz), which extends from the point marked by lz, through the noted fiber bundle (fb), and through the cortical sclerids (se) to the outer periderm (x20).

The lignified zone was observed to form at the same time, about 8 to 10 days after inoculation, in all host-treatment combinations. The lignified zone appeared to be a wound periderm induction barrier; wound periderm formed next to it (Figure 4b). In the secondary phloem, wound periderm formation began about 2 to 4 days after lignification when the lignified zone was oriented periclinally. Wound periderm formation began in secondary phloem near the area of deepest canker penetration and progressed outward, taking about 20 days to reach the original periderm in the Fldg tree and the Chinese chestnut trees, and about 30 to 40 days in the American chestnut stump sprouts

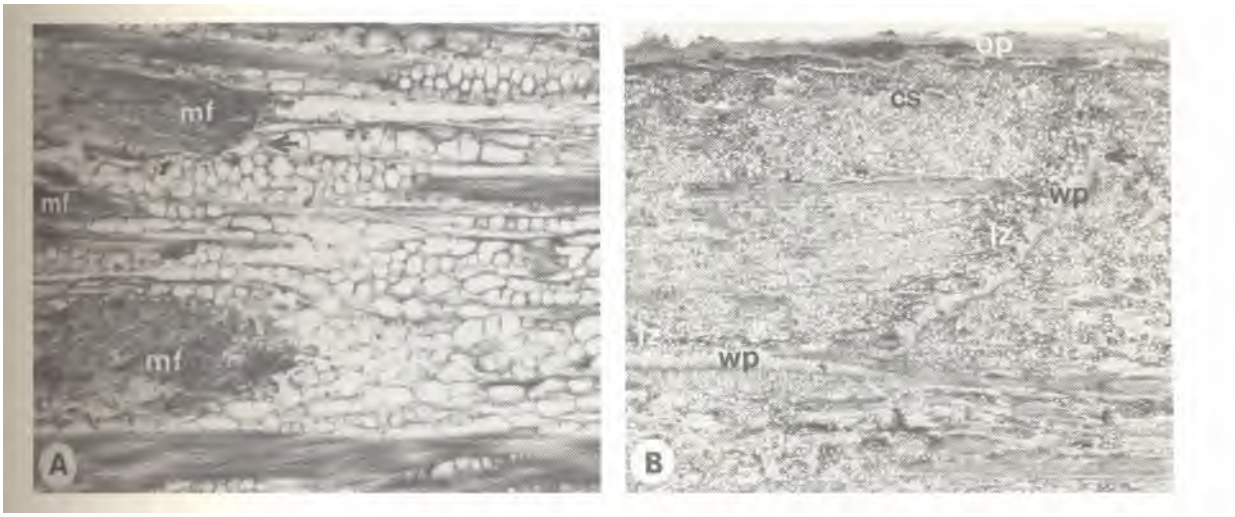

Figure 4. Micrographs of sections of chestnut blight cankers stained with safranin and fast green. Figure 4a. Mycelial fans (mf) of a virulent strain of *Endothia parasitica* in bark of an American chestnut stump sprout 23 days after inoculation. Note discoloration in front of bottommost fan, indicating cell death, and splitting of tissue in front of topmost fan. Arrow there points to a cell being physically crushed by the fan. Distortion of the bark tissues in this region is evident (x60). Figure 4b. Wound periderm (wp) formation next to a lignified zone (lz) in a large, surviving American chestnut (the Fldg tree), 53 days after inoculation with a virulent isolate of *E. parasitica.* Wound periderm does not extend (arrow) beyond cortical sclerids (cs) to the outer periderm (op) (x28).

(Figure 5). At any one place where wound periderm began to form, it took 4 to 8 days before phellem cells began to form and 8 more days before the number of phellem cells reached a maximum. The progress of phellem cell formation next to periclinally oriented lignified zones in secondary phloem is illustrated in Figure 6.

When the lignified zone was oriented anticlinally, the initiation of wound periderm formation was greatly delayed in any host-treatment combination. In secondary phloem, it began approximately 20 days after lignification instead of 4 days. Thus, anticlinally oriented lignified zones resulted in gaps in wound periderm. Anticlinally oriented portions of lignified zones were observed scattered among the samples in all bark tissues in all host-treatment combinations. They were especially prevalent where the lignified zone turned up to connect with the outer periderm (Figure 4b). The propensity of some branches (ca 10 years old) to initiate rhytidome formation appeared to augment the high frequency of gaps in wound periderm observed near the outer perideim.

Canker expansion always was accompanied by mycelial fan formation and expansion. Mycelial fans always penetrated through gaps in the wound periderm as far as could be determined. In the American chestnut stump sprouts, canker expansion beyond the lignified zone was first observed (at day 18 in 1978) with the V isolate and later (at day 28 in 1978) with the H isolate, On the stump sprouts, the cankers incited by the H isolate did not expand

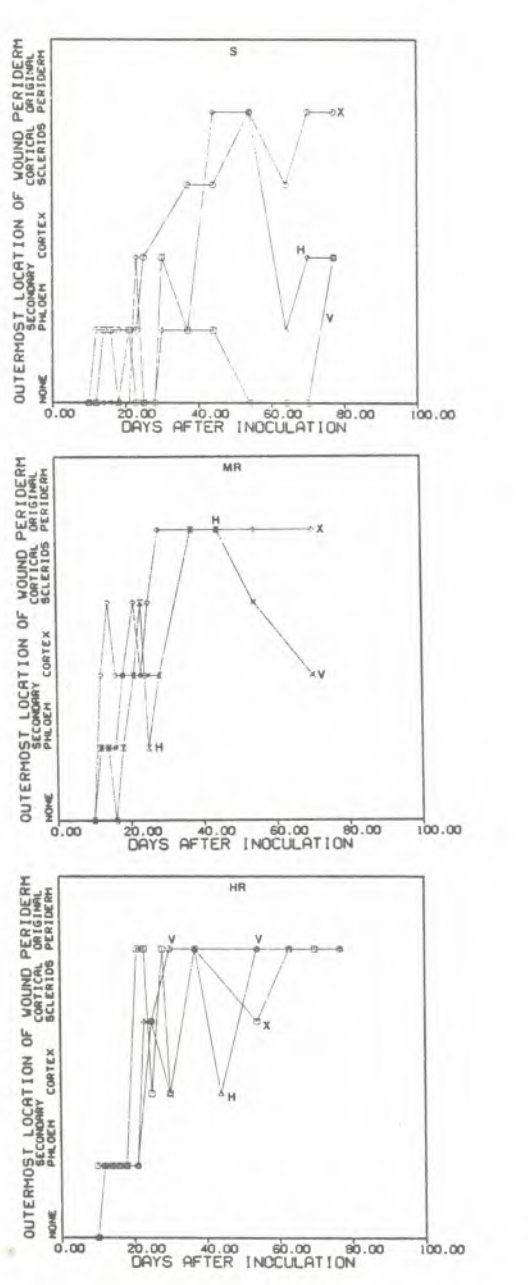

Figure 5. Time-course of region in bark tissue of outermost location of phellem or phelloderm of wound periderm in blight-susceptible (S) American chestnut stump sprouts, a moderately blight-resistant (MR), large, surviving American chestnut (the Fldg tree), and highly blight-resistant (HR) Chinese chestnut, cultivar Nanking, after inoculation on July 3, 1978 with virulent (V) and hypovirulent (H) strains of *Endothia parasitica,* or wounding with no inoculation (X). The scatter in the data occurred because each point represents a separate canker. When there were duplicate cankers on one day for the S trees, the canker with the outermost location is depicted.

as far as the cankers incited by the V isolate (Figure 2), and the H cankers eventually were surrounded by a wound periderm (Figure 5). There was no fan formation or canker expansion with the H isolate on the Fldg and Chinese chestnut trees. There was sporadic occurrence of canker expansion by the V

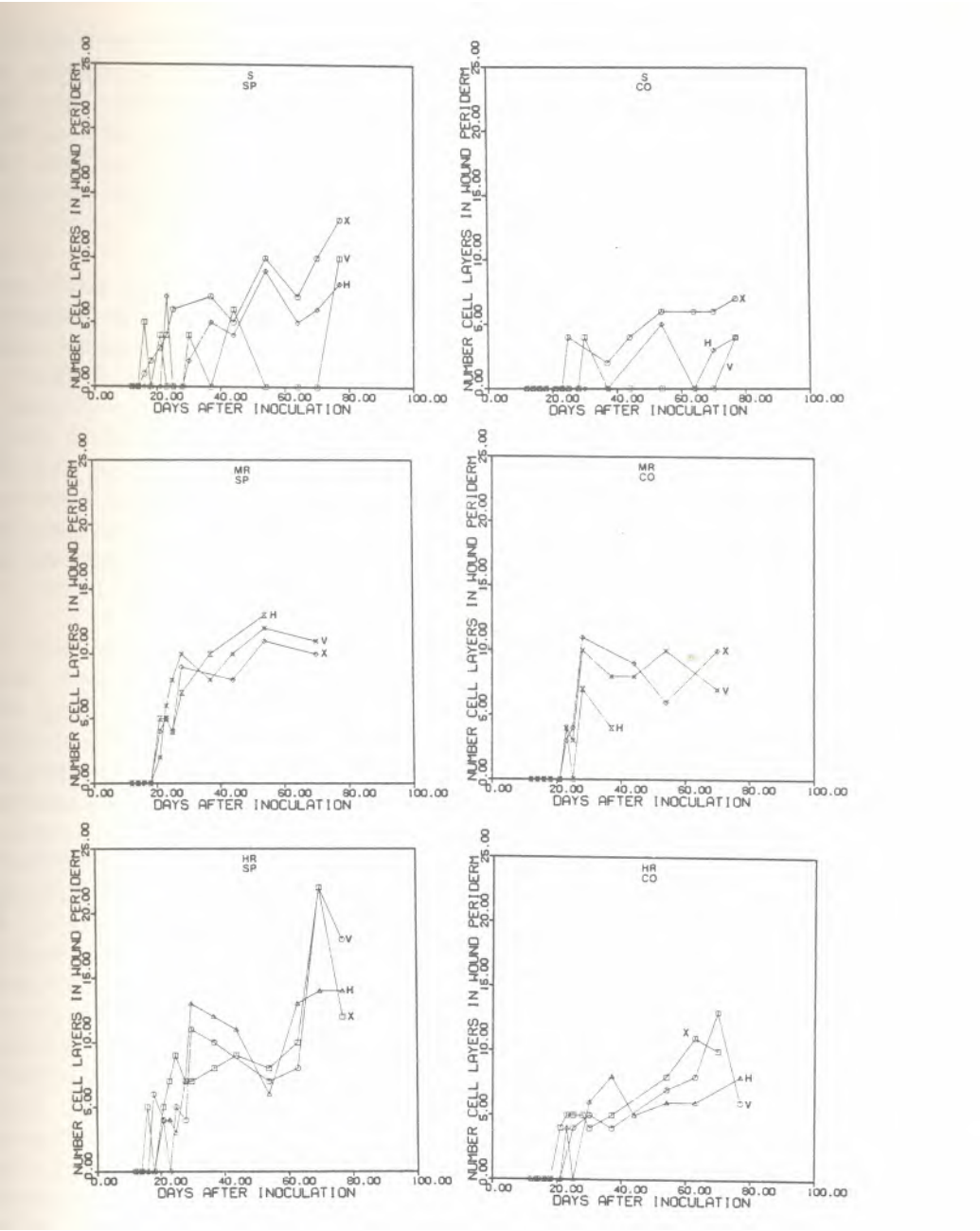

Figure 6. Time-course of change in the number of cell layers in phellem of wound periderm located in the secondary phloem (SP) and cortex (CO) of blight-susceptible (S) American chestnut stump sprouts, a moderately blight-resistant (1a), large, surviving American chestnut (the Fldg tree), and highly blight-resistant (HR) Chinese chestnut, cultivar Nanking, after inoculation on July 3, 1978, with virulent (V) and hypovirulent (H) strains of *Endothia parasitica*, or wounding with no inoculation (X). The scatter in the data occurred because each point represents a separate canker. When there were duplicate cankers on one day for the S trees, the canker with the greatest number of cell layers is depicted.

isolate on the Fldg and Chinese chestnut trees, with more instances of expansion in the Fldg tree. This difference in incidence of occurrence on canker expansion was the only difference we observed between the Fldg tree

and the Chinese chestnut trees. They did not appear to differ with respect to the rate or extent of wound periderm formation when fan expansion did not occur; the inoculated American chestnut stump sprouts, however, did show impairment in wound periderm formation. This is thought to have been due to formation and expansion of mycelial fans in the stump sprouts. The effects of resistance and hypovirulence appeared to be additive as far as rate and frequency of canker expansion were concerned.

The higher frequency of gaps in wound periderm near the outer periderm (Figure 4b), as compared to other bark regions, is part of the mechanism which gives rise to superficial cankers. When we observed canker expansion in the Fldg tree or in the Chinese chestnut trees, the mycelial fans commonly were underlain by a wound periderm. The fans in these two trees were short in comparison to those observed in the stump sprouts (less than 1 cm in length compared to lengths of up to 5 cm). This indicates that the fans expanded more slowly once expansion started, suggesting that the slow rate of fan expansion allowed wound periderm formation to keep pace. Bramble (1936) suggested that fan expansion in blight-susceptible American chestnut proceeds too rapidly for wound periderm formation to keep pace with it.

Biocontrol Tests

In tests using H strains to effect biocontrol of cankers incited by V strains (Hebard et al. 1981a), the rate of deceleration of growth in biocontrolled H+V cankers appeared to be inversely proportional (roughly) to the pathogenicity of the H strain (Hebard et al. 1979). The rate parameters in Hebard et al. (1979) should read cm/day instead of mm/day. Thus, H strains of slight or moderate pathogenicity gave rise to larger biocontrolled cankers than H strains with little pathogenicity. We observed, following dissection, that these larger biocontrolled cankers were superficial to a significant extent. Microscopical examination revealed that the superficial cankers were underlain by a wound periderm. The only instance of biocontrol (confirmed by isolation and pathogenicity tests of the isolates) of a V-alone canker (incited by the CR strain) which we observed occurred on a tree whose H+V canker had been biocontrolled by our most pathogenic H strain (EP-66). We suggest that, in Europe, superficial H cankers arise, in part, during the process of biocontrol of V cankers. We also suggest that the differences at vegetative compatibility loci between V and H strains govern the frequency with which V cankers are biocontrolled but not the rate of deceleration in canker expansion. The results of isolations and pathogenicity tests from biocontrolled cankers indicate that the spread of European hypovirulence factors can be monitored by determining the frequency of white isolates, as has been done in Europe (Grente and Berthelay-Sauret 1978; Mittempergher 1978).

Epidemics in Clearcuts

The main details of the epidemiological studies have been reported previously (Hebard et al. 1981b). Briefly, it takes 9 to 10 years after clearcutting for blight incidence on sprouts over 0.8 cm d.b.h. to increase from 20 percent, the level in forested areas, to 90 to 100 percent. This indicates that caution must be used in relying upon natural infection and mortality as a measure of blight resistance, especially when some blight-resistant trees are present; a blight-free tree at 10 years could well be an escape.

We also found that the increase in tree diameter after clearcutting probably is a major cause of the epidemics in clearcuts. The larger trees appear to allow a larger surface area of *E. parasitica* to develop than occurs in forested areas. This indicates that spread of V strains of *E. parasitica* is relatively inefficient at low population levels. It appears that a large host population surface is necessary for epidemics to occur. Arguing by analogy, we suggest that a large host (V strains of *E. parasitica)* population is necessary to obtain natural spread of hypovirulent factor(s). On the other hand, in a separate study at a 10-year-old clearcut site, chestnut sprouts treated with an H isolate of *E. parasitica* were overwhelmed by additional cankers before disease control could occur. Apparently, the epidemic at that site had progressed too far for biocontrol to be possible on these sprouts. Thus, a moderate population level for virulent *E. parasitica* (less than that at 10-year-old clearcuts but more than at forested areas) appears desirable. It may be possible to manipulate the size of *Endothia* population by regulating the mean diameter of American chestnut sprouts at regeneration sites. This appears to have occurred fortuitously in some European chestnut coppice where hypovirulence-associated blight remission occurred (Grente and Berthelay-Sauret 1978; Mittempergher 1978).

Schuepp (1961) measured canker growth and blight progress in Europe; both were slower than values reported in America (Hunt 1923; Gravatt and Gill 1930). We have developed a mathematical model (Hebard 1981) for assessing the effect of growth of individual cankers on disease progress. Using this model, we have found that the slower canker growth on European chestnut may be a principal cause of the slower rate of blight progress in Europe. It does not appear that hypovirulence was the cause of the slower disease progress and canker growth, since Schuepp's (1961) data were collected during the first 12 years after blight entered an area; Mittempergher (1978) reported that it takes about 15 years after blight enters an area before hypovirulence-associated disease remission begins. We suggest that the slower disease progress in Europe may have been a key factor in remission. Epidemics in clearcuts in the Appalachians probably could be prolonged by removing diseased sprouts and by removing (cutting) some large living trees so as to restrict inoculum production.

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