Indicator Hosts for Pear Decline: Symptomatology, Histopathology, and Distribution of Mycoplasmalike Organisms in Leaf Veins

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ABSTRACT

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The *Pyrus* cultivars Variolosa, Magness, and Precocious all were susceptible to pear decline, and each exhibited a distinctive type of browning of leaf veins. In Variolosa, the abaxial ribs of major lateral veins became brown; in Magness, the minor veinlets discolored; and in Precocious, a new and unusual indicator host, the mesophyll bordering adaxial ribs of framework veins became yellow and later brown. Pathological features common to the three hosts were: (i) a tendency for mycoplasmalike organisms (MLO) to be abundant in finer minor veinlets and sparse in sieve tubes of the secondary phloem of framework veins; (ii) the occurrence of lesions in vein encasing tissues; and (iii) necrosis of sieve tubes of the secondary phloem. In Variolosa and Precocious, sieve-tube necrosis occurred early and abundantly in the secondary phloem of framework veins and was accompanied by excessive phloem formation. The occurrence of sieve-tube necrosis in Magness was erratic, and no replacement phloem formed. There appears to be an anomaly among the pathological features. Pathosis was absent or rare in fine minor veins where MLO occurred most abundantly; conversely, pathosis was severe in tissues that encased veins where MLO did not occur and in secondary sieve tubes where MLO were rare or absent. One possible explanation is that MLO do not cause pear decline. A more plausible hypothesis is that toxins are produced by MLO in the specialized phloem of fine minor veins or that host tissues are stimulated to produce them. The toxins then are translocated to other sites where they cause pathosis.

In 1959 and following years, pear decline was recognized as a bud-union disorder affecting French pears (*Pyrus communis* L.) grafted on certain rootstocks including *P. serotina* Rehd., *P. ussuriensis* Maxim, and *Cydonia oblonga* Mill. (1, 3, 8, 16, 17). Later, other trees without bud-union pathosis were observed to exhibit leaf symptoms different from trees with the bud-union disorder. Curling and sometimes purpling of leaves were observed in French pears such as *P. communis* 'Comice' (14, 15). The cultivar Magness drops its leaves prematurely (13). In the greenhouse, several patterns of vein browning were observed (17, 21). The pear decline agent is transmitted by grafting and by *Psylla pyricola* Foerster, and mycoplasmalike organisms (MLO) are associated with both infected trees and vectors (2, 9, 10, 12, 19).

Although MLO were first found associated with diseased plants in 1967, anatomical and histochemical aberrations (pathoses) have yet to be related to MLO (4, 6, 18). In this paper, three indicator hosts for pear decline were studied to determine the manner in which brown veins developed symptomatically, how pathoses developed, and where MLO could be found in the veinal network; and through these to seek an hypothesis for a causal relationship between the presence of MLO and the occurrence of pathoses.

MATERIALS AND METHODS

Three pear cultivars with different symptoms were studied. They were: Variolosa, browning of the abaxial ribs of major lateral veins; Magness, early browning of the abaxial bundle-sheath extensions of minor veinlets; and Precocious, adaxial yellowing along the ribs of the midvein and major lateral veins culminating in conspicuous browning. Variolosa, a pear of unknown parentage (20), has been under study for 10 yr; Magness, an hybrid of P. I. 49490 (Seckel × O. P.) × Comice (Howard J. Brooks, *personal communication*) for 9 years; and Precocious, a seedling line from Winter Nelis × O. P., for 3 years. Some other pears exhibited symptoms similar to the types expressed by Variolosa and Magness.

Systemically infected trees that had been graft inoculated were used for the studies. They were grown in containers in the greenhouse and were from earlier experiments. Each year when the foliage aged and older leaves began to abscise, trees were chilled at 4 C for 60 days or longer in a cold room to complete their defoliation and to condition their buds to break dormancy. After the chilling they were pruned; and, if necessary, transplanted to larger containers and brought to a greenhouse.

Tissues for electron microscopy were fixed in glutaraldehyde, postfixed in osmic acid, and embedded in Spurr's low viscosity resin (10). For examination by light microscopy, monitor sections 0.5 μm thick were

stained with methylene blue, Azure II, and basic fuchsin (11). For electron microscopy, ultrathin sections were cut alternately with the monitor sections and stained with uranyl acetate and lead citrate. Occurrence and density of MLO in veins were determined by studies of the ultrathin sections with the electron microscope. Polyphenols (tannins) were fixed and stained by the above procedure. For a further study of polyphenols, freehand sections were treated with the following mixture for 2 to 12 hr: water – 89 ml; glacial acetic acid – 0.25 ml; 37% formaldehyde – 10 ml; $FeSO_4$ – 2.0 g. Sections then were cleared in and mounted in glycerine.

RESULTS

Venation and anatomy of pear leaves.—The pear leaf is simple, ovate, and its venation is pinnately reticulate (Fig. 1-C; 2-A, B; 3-A; 4-A and D). Veins and veinlets were classified arbitrarily as follows: The *midvein* extended from the petiole to the tip of the leaf and its branches were the major lateral veins (*major veins*). The midvein and major veins are sometimes referred to as *framework veins*. Networks of *minor veinlets* occurred between major veins. *First-order minor veinlets* were the larger, uniformly-sized minor veinlets that branched off major veins. These veinlets anastomosed with each other and thus formed a network (Fig. 1-C and 3-A, B). Sometimes three first-order veins anastomosed at a point. *Second-order minor veinlets* were veinlets that branched from first-order veinlets; those selected were of uniform size and slightly smaller than those classified first order. Veinlets finer than second order (*finer veinlets*) were not selected, but leaf samples for first- and second-order minor veinlets included several finer veinlets. These veinlets often appeared in cross or longitudinal section because they tended to lie approximately in squares (Fig. 3-B and 5-A). Except for veinlet endings, each subbranch anastomosed with another veinlet of similar size to form a cross strand or other configuration in the network. The finer veinlets are inconspicuous from outside the leaf because they are embedded in the mesophyll and lack ribs or sheath extensions (e. g., Fig. 5-A, vein at left).

In mature leaves, veins of different sizes differed in degree of development and in structure. The midvein and major veins contained secondary xylem and phloem and they were supported by ribs containing collenchyma (Fig. 1-A and 2-C). Fibers occurred between the adaxial ribs and the xylem and between the abaxial ribs and the phloem. Sieve tubes of secondary phloem and of metaphloem were wide (approximately 4 μm diameter) and had nacreous walls; in the secondary phloem, sieve tubes were associated with companion cells (20). First order veinlets sometimes contained secondary phloem, but some metaphloem usually remained functional; instead of ribs, these veinlets had parenchymatous bundle-sheath extensions (Fig. 3-E). As veinlets branched and rebranched, they decreased in size and their bundle-sheath extensions disappeared, first the upper one (Fig. 3-F), then also the lower (Fig. 5-A, left). Some of the finest veinlets contained a few tracheary elements and phloem with narrow sieve tubes (approximately 2 μm in diameter) with slightly and evenly thickened walls. Intimately associated with the sieve tubes of fine veinlets were large parenchyma cells (intermediary cells); their cytoplasm was often thick and densely packed with ribosomes and other organelles (Fig. 5-B and E). Looking through the epidermis at a vein, one sees either rib tissues or bundle-sheath extension tissue. These tissues are referred to sometimes as veins; e.g., brown veins, and sometimes either as ribs or as sheath extensions.

Symptomatology and anatomical aberrations in three cultivars representing three types of host reactions.—In the greenhouse, three patterns of brown veins were recognized in leaves of hosts affected by pear decline. Two of the patterns were associated with two groups of hosts. A third pattern occurred in only Precocious.

Hosts with browning of abaxial ribs of major veins as with Variolosa.—Tan to brown discoloration appeared on abaxial ribs of the major veins at 3-4 mo after dormant Variolosa trees were brought from the cold room to the greenhouse (compare Fig. 1-C and 2-B) (17, 21). In some cases, browning extended over the length of the veins; in others, it was confined to shorter segments. The discoloration eventually intensified to dark brown, and the veinal surfaces became russeted due to splitting of the epidermis. Pathosis occurred in immature collenchyma cells of the abaxial ribs but not after they matured. These cells underwent hypertrophy, hyperplasia, or necrosis. Polyphenols were abundant (compare Fig. 1-A and 1-B) and in some cells they oxidized to a brown color. Walls of pathological cells at breaks in the epidermis were suberized. Similar symptoms and pathoses sometimes occurred on the adaxial ribs of major veins.

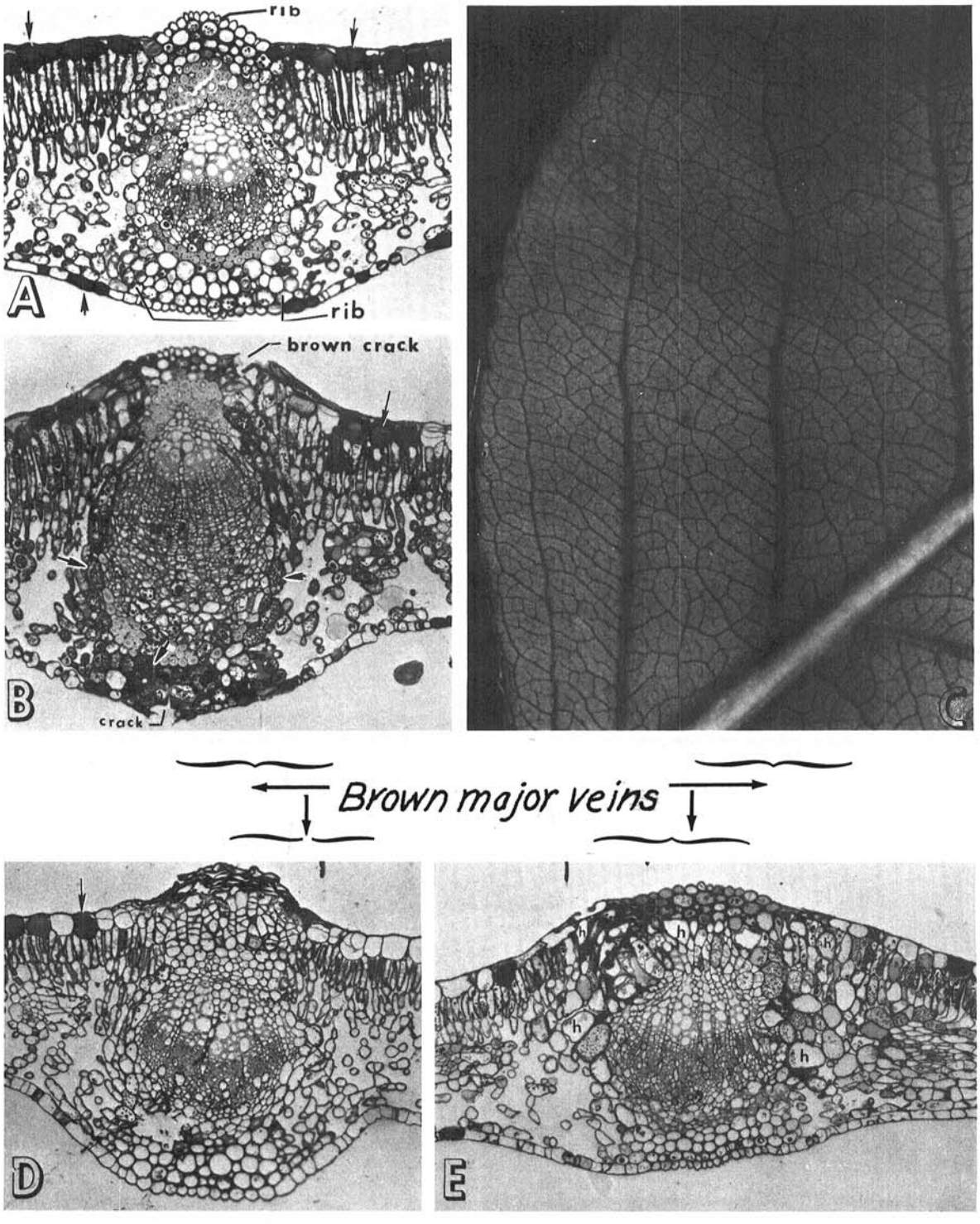
Adaxial bundle-sheath extensions of some of the larger minor veinlets also became brown, but a hand lens was needed to see the discoloration during its early stages. Cells in the sheath extensions were affected by severe hypertrophy or necrosis. The abaxial bundle-sheath extensions of minor veins were infrequently discolored.

When leaves were about 5 mo old, brown bands appeared along one or both sides of the adaxial ribs of the framework veins [Fig. 2-(A and D)]. The discoloration occurred in the thin-walled, mesophyll cells that bordered the ribs (Fig. 1-B and 2-D). Deeper within the leaf on one or both sides of affected veins, the bundle sheath and adjacent mesophyll cells were filled with polyphenolic bodies and the tissue was brittle. The lamina easily broke away from the midvein through diseased tissues [Fig. 2-(A and D)]. Browning along framework veins most often appeared on leaves that earlier escaped browning of abaxial ribs of major veins. On the under side of very old leaves, occasional large minor veins were brown where they anastomosed with grosser veins (Fig. 2-B), but this type of discoloration also appeared in 8-mo-old leaves of noninfected trees.

Abaxial browning of minor veinlets as in Magness.—The onset of browning of abaxial bundle-sheath extensions of veinlets on Magness leaves occurred 2 to 2.5 mo after dormant, defoliated Magness trees from the cold room were brought to the greenhouse. Browning of veinlets first appeared on short segments of minor veinlets, or at veinlet branchings. Gradually, browning became more extensive and localized areas of the veinal network were affected including streaks on the major veins [Fig. 3-(A, B, and C)]; later, a large portion of the network became brown. Leaves with early stages of abaxial veinal browning were slightly chlorotic, and their margins curled downward. With a magnifying lens,

browning also was seen on the adaxial surface of veinlets; with time it became visible to the unaided eye because palisade cells impinging on the vein's bundle-sheath extensions also turned brown. As veinal networks became extensively discolored, the leaves became bronzed and brittle, and abscised prematurely. Diseased trees were stunted.

The brown-veinlet symptom was most distinctive shortly after it appeared when leaves were young and green and when pathosis was closely confined to the veins



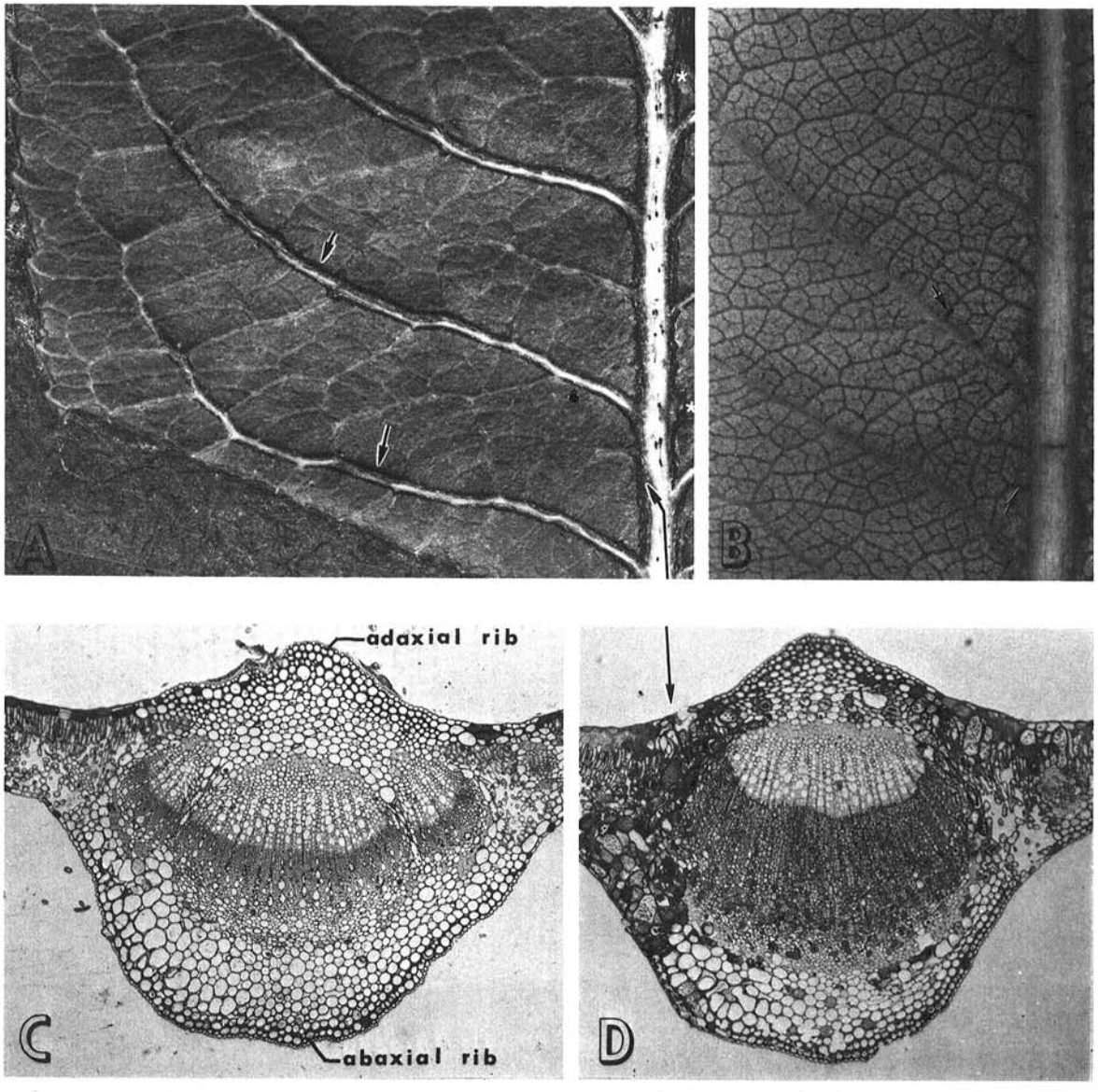


Fig. 2-(A to D). Advanced pear-decline symptoms in 6-mo-old Variolosa leaves. **A)** Adaxial surface of leaf with brown discoloration along the sides of ribs of the midvein and major lateral veins (arrows). When leaf was flattened for photography, lamina broke through pathological tissues on right side of midvein (stars). **B)** Abaxial surface of a similar leaf. Some first order veinlets were brown where they joined the major veins and the midvein (arrows). Similar browning was observed in 8-month-old healthy leaves. **C)** Cross section of midvein of leaf from a control plant. Vascular bundles from lateral veins were anastomosing with bundle of midvein. **D)** Cross section from pear decline leaf showing discoloration on the left side of the midvein where pathosis consisted of hypertrophy of parenchyma and dark staining due to accumulation of tannin. Excessive phloem was formed. (Magnifications: A and B × 5; C and D × 50).

Fig. 1-(A to E). Major lateral veins of healthy (A) and pear-decline-diseased (B-E) pear leaves. In A, B, and D, the innermost wall of many epidermal cells was thickened and nearly filled the cell (arrows) (× 140). **A)** Healthy Variolosa. **B)** Seven-month-old leaf of Variolosa with pear decline. Excessive phloem was formed and xylem differentiation was suppressed. Sheath parenchyma cells and others contained excessive starch and polyphenols (black on white arrows). Cracks occurred in upper and lower epidermises. Hyperplasia and hypertrophy occurred in the abaxial rib and in palisade cells abutting the adaxial rib. **C)** Variolosa leaf with early browning of major veins (× 5). (Compare with Figure 2-B). **D)** Major veins of Variolosa leaf that was apparently injured by miticide when leaf was immature. Wound periderm was formed. Phloem appears normal. **E)** Leaf of seedling from open pollinated *Prunus serotina* 'Chojuro' that had a nontransmissible disorder resembling vein yellows: Phloem was normal; bundle-sheath cells were hypertrophied (h); palisade parenchyma cells that abut the adaxial rib were hypertrophic and/or lacked developed chloroplasts. Necrotic cells just to left of rib were brown.

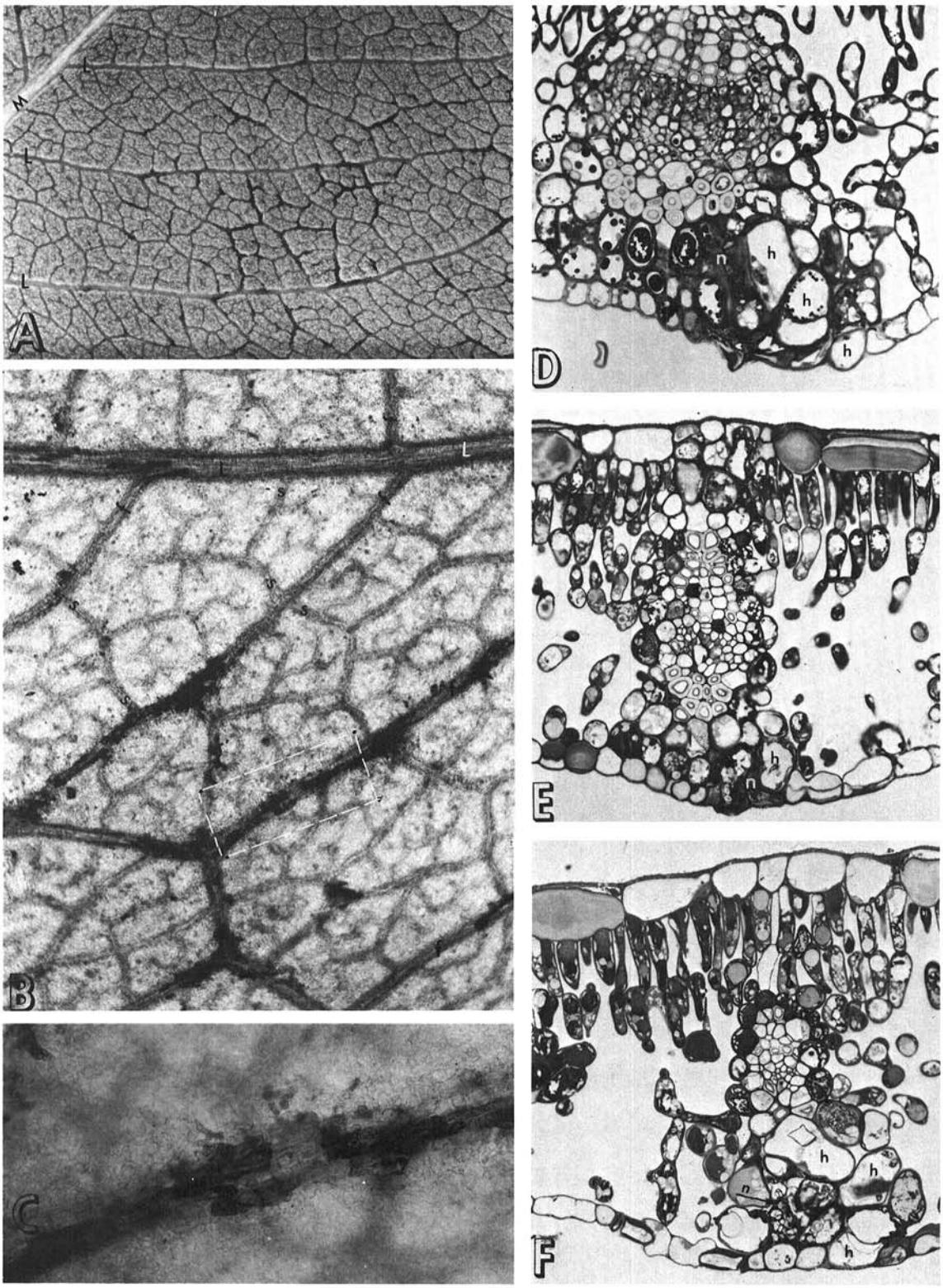


Fig. 3-(A to F). Brown veinlets in leaves of Magness infected with pear decline. **A)** Abaxial surface of leaf three months after being taken from the cold room. Brown discolorations occur in veinlets and in major veins where anastomosed with brown veinlets (affected veinlets photograph darkly). M = Midvein; L = Major lateral vein (×5). **B)** Leaf cleared with dimethyl sulfoxide in a steam bath, dehydrated in ethanol, cleared in clove oil and mounted in balsam. Pathological cells in dark veins contained oxidized tannins. L = One of the dichotomous branches of a major lateral vein; f = first-order minor veinlets; s = second-order veinlets (×28). **C)** Higher magnification of area boxed in faintly with broken lines in B (×100). **D)** Section of major vein showing lesion involving abaxial rib and epidermis. Sieve tubes with MLO were rare; but, in a finer minor veinlet, in the same section (not shown), narrow sieve tubes were filled with MLO. **E and F)** First-order and second-order veinlets, respectively. In both sections there are lesions in the abaxial bundle-sheath extensions and the epidermises; a few MLO occurred in the sieve tubes of both veins. h = hypertrophy; n = necrosis (D - F ×250).

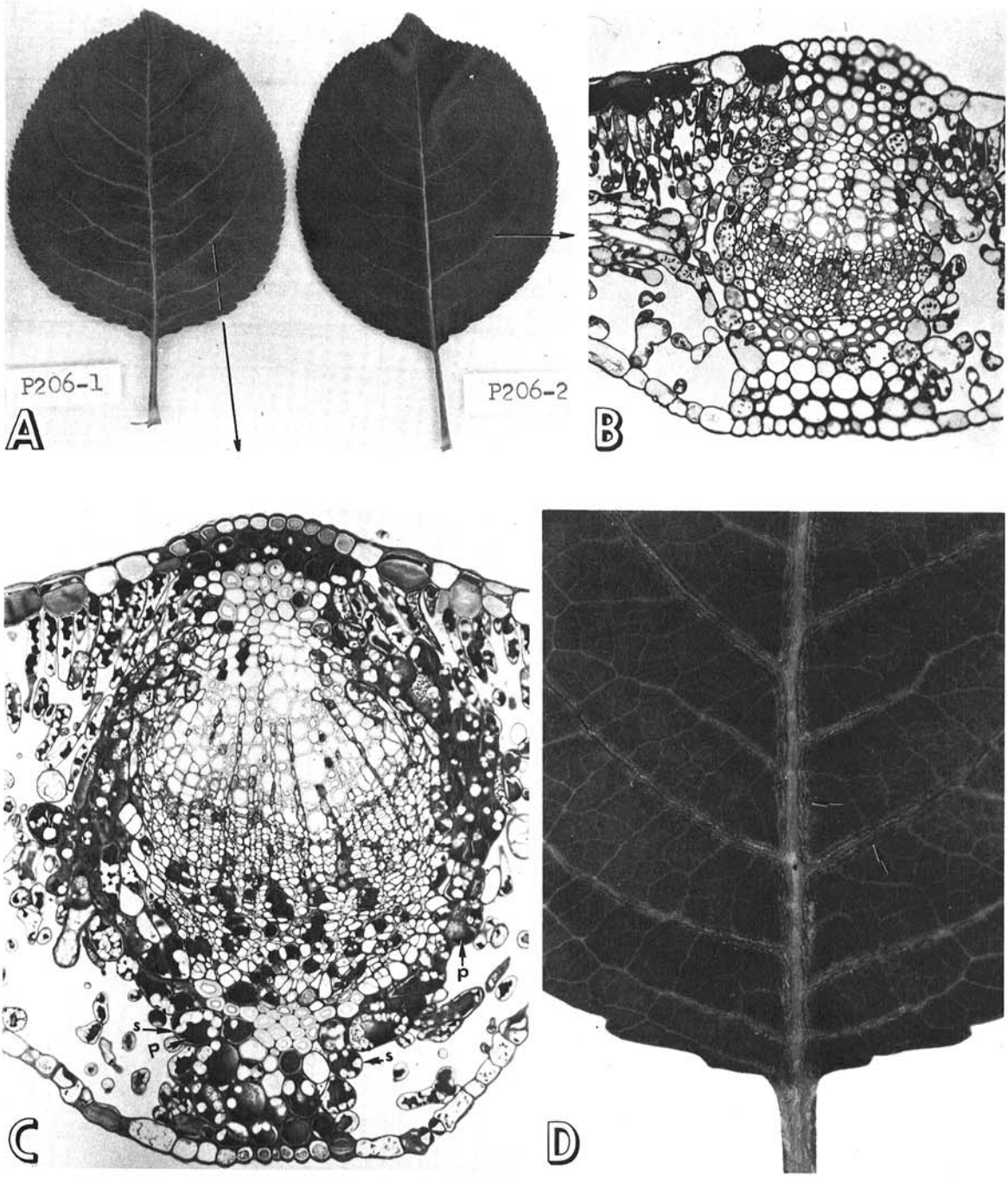
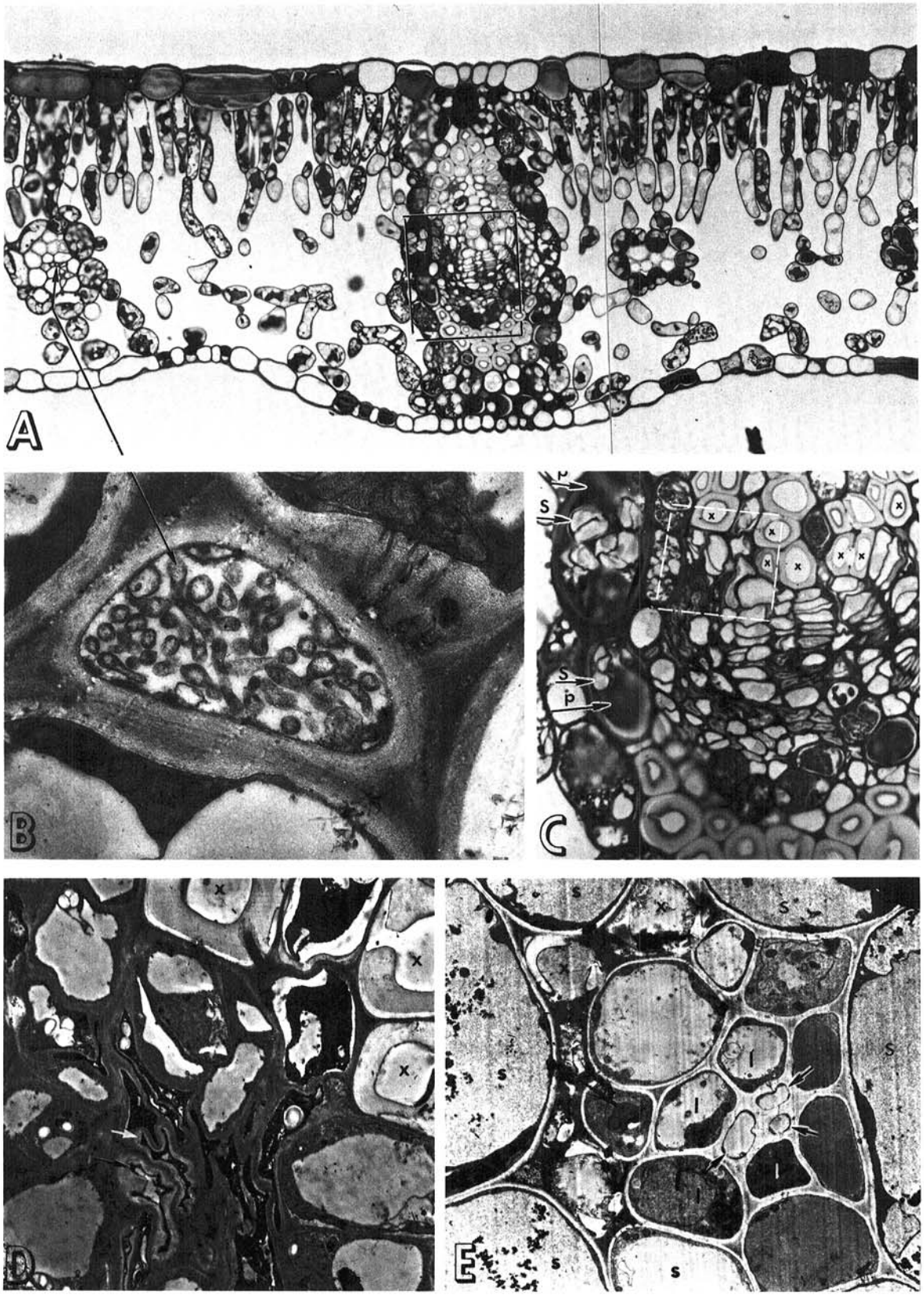


Fig. 4-(A to D). Leaves and cross section of major leaf veins of pear-decline-diseased and healthy Precocious. **A)** Adaxial surfaces of leaves nine weeks after tree was taken from the cold room. The diseased leaf (on left) is slightly chlorotic and the framework veins have become prominent (xl). **B - C).** Major veins from healthy and diseased trees ten weeks from the cold room (×220). Source trees were the same as for the leaves shown in A, and symptoms were similar. In **C**, the phloem is composed of a mixture of necrotic and normal sieve tubes; excessive phloem was formed. Large starch granules and polyphenolics have accumulated in the bundle sheath and adjacent mesophyll (arrows labeled S and P). There were occasional MLO in sieve tubes of the vein, while in adjacent fine minor veinlets (not shown) MLO were abundant. In the palisade cells that encroached on the adaxial rib, chloroplasts were disrupted by large starch granules and they lacked normal structural features and apparently chlorophyll. Evidently the cells lacked green color as do the rib cells and the ribs appear to be broadened as in A. In B and C, the ribs were equal in size, but the phloem of C was enlarged. **D)** Adaxial view of a leaf five months after inoculation. Wound periderms have formed in the mesophyll cells abutting the ribs (arrows) (×4).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[Fig. 3-(A through F)]. In old leaves, the inevitable effects of injuries and of aging made it difficult to distinguish symptoms of disease.

In Magness leaves, the pathological aberrations that were manifested as brown veinlets occurred in the bundle sheath, its extension, and the epidermis. Hypertrophy was severe but hyperplasia and necrosis were less prevalent [Fig. 3-(D, E, and F)]. Some affected cells became suberized; and in others there were brown oxidized polyphenols. Thus, a brown color was imparted to the lesion portion of the veinlets.

Comice was another pear cultivar with abaxial browning of minor veinlets. Cells of the abaxial bundle-sheath extensions were hypertrophied, and the pathosis was confined to somewhat smaller veinlets than in Magness. Sometimes the discolored veinlets could be seen best with transmitted light.

Yellow bands that later became brown along the adaxial ribs of the framework veins of Precocious.—By the time trees of Precocious had been back in the greenhouse from the cold room for 2 mo, leaves on them were slightly chlorotic and flaccid (Fig. 4-A). There was a degeneration of chloroplasts in mesophyll cells bordering the adaxial ribs of framework veins. As a result, the color of affected mesophyll was the same as the ribs, and the ribs appeared to be broadened. The rib tissues contained excessive polyphenols (compare Fig. 4-B and 4-C). A few weeks later, affected mesophyll cells became brown, and cracks occurred in the discolored tissue (Fig. 4-D). Simultaneously with the development of veinal symptoms, opposing halves of the leaves rolled upward parallel to the midveins, and the texture of leaves became leathery and later brittle. The abaxial portions of the major veins enlarged, and sometimes the ribs became brown.

Precocious is an excellent indicator for pear decline because leaf symptoms occur early and on the upper leaf surface, where they are easily seen. Severely diseased leaves do not abscise as with Magness, thus giving an extended period for observations.

Distribution of mycoplasmalike organisms in leaves.—Generally, MLO were abundant in the narrow sieve tubes of the finer minor veinlets of the three hosts examined [Fig. 5-(A and B)]. In framework veins, few sieve tubes contained MLO and sieve tubes containing MLO were often in primary phloem that laid peripherally to the secondary phloem, or in phloem of smaller veins recently anastomosed with the framework veins. Anastomosis of veins is shown in Fig. 2-C. The MLO content of veins shown in Figs. 3-D, E, F; 4-C; and 5-B is reported in the figure legends. The MLO content was determined from alternate ultrathin sections. In sections of a leaf sample from Comice, MLO were observed in minor veins, but not in the major vein.

Sieve-tube necrosis in the secondary phloem and other phenomena that occurred subsequently.—In Variolosa, sieve-tube necrosis occurred in the secondary phloem of major veins. The necrosis was followed by hyperactivity of the cambium, excessive phloem formation, accumulation of starch and polyphenols, and ultimately cambial disorganization (compare Fig. 1-A with 1-B) (20).

In Magness, sieve-tube necrosis in secondary phloem apparently occurred later and less abundantly than in Variolosa. Excessive phloem did not form. For instance, in Fig. 3-D a major vein is shown with a lesion in the abaxial rib, yet only occasional sieve tubes were necrotic. In other samples of major veins, necrosis was extensive, but cambial hyperactivity was lacking.

In Precocious, sieve-tube necrosis and replacement phloem formation occurred abundantly [Fig. 4-(B, and C); and 5-(A, C, and D)], and enlargement of major veins was so voluminous on their abaxial sides that it was visible to the unaided eye.

After 3-4 mo in the greenhouse, leaves of all hosts became leathery and eventually brittle. They tended to discolor brown.

Formation and oxidation of polyphenols.—When freehand section of living leaves were placed in the formaldehyde/ferrous sulfate solution, polyphenols precipitated and reacted with the iron to form black compounds. The glutaraldehyde/osmic acid fixing procedure for electron microscopy also caused precipitation; and polyphenols stained with both the methylene blue/Azure II/basic fuchsin used for light microscopy and the uranyl acetate/lead citrate for electron microscopy, but not specifically. In young healthy veins, there were small globules or granules containing polyphenols in the vacuoles of parenchyma cells. As the leaves matured, globules became fewer but larger; in some cells, they filled or nearly filled the central vacuole. In diseased leaves (Fig. 5-A and C), polyphenols were more abundant than in healthy leaves (compare Fig. 1-A with 1-B; 2-C with 2-D; 4-B with 4-C), and some of the bodies became brown apparently by oxidation. Chatter marks from sectioning indicated that the bodies were hard. Brown oxidized polyphenols occurred in parenchyma cells of the bundle sheath, of the palisade and spongy mesophylls, and of the phloem. They also were prevalent in cells near breaks in the epidermis that resulted when veins enlarged by excessive phloem production or by hypertrophy of subepidermal cells (Fig.

Fig. 5-(A to E). Cross section of leaves from healthy and pear-decline-diseased Precocious trees. **A)** Diseased. In the fine minor veinlet at left there are a few xylem elements and below them a few narrow sieve tubes (arrow) that contain MLO (not visible. See E for an electron micrograph of a healthy veinlet of similar size. The first-order veinlet at the center of the section was from near its point of anastomosis with a yellowed major vein; on the left, the cambial zone became disorganized and sieve tubes were necrotic (see C and D). Bundle-sheath cells of all veinlets are filled with starch and polyphenolic material. The small veinlet to the right of the first-order veinlet contained only xylem elements (×280). **B)** Electron micrograph of a cross section of a sieve tube with MLO from a fine veinlet like the one at left in A (arrows) (×15,000). **C)** Photomicrograph of the boxed-in area in A taken with an oil immersion lens (×860). Sieve tubes are necrotic and collapsed. On the left, the cambial cells and their derivatives also were necrotic. Starch granules and polyphenolic material are indicated by arrows labeled S and P. **D)** Electron micrograph of boxed-in area shown in C (× 300). **E)** Electron micrograph of a fine minor veinlet from a noninfected plant (×3,000). In D and E, arrows = sieve tubes; I = intermediary cell; s = bundle-sheath cell; x = xylem element.

1-B). Cells at the breaks also became suberized as indicated by staining with Sudan IV.

Brown veins induced by factors other than pear decline.—An apparent miticide-induced browning of adaxial ribs of major veins appeared simultaneously on leaves of trees in two experiments only 7-wk after trees had been removed from the cold room. Both pear-decline-infected and control trees of Variolosa, Magness, and a seedling-line of Chojuro were affected. Apparently the browning was not that associated with pear decline because it occurred adaxially rather than abaxially and in leaves that were too young to show pear decline symptoms. Observations with a hand lens revealed that affected portions of veins were within disk-shaped patches of spray residue. Cross sections showed that ribs had been injured while still in a primordial state and that wound periderm formation had occurred (Fig. 1-D). Characteristics of pear decline such as sieve-tube necrosis, accumulation of starch and polyphenols, and hyperactive phloem production were absent.

In a nontransmissible vein-banding and -browning disorder of a pear propagated from a seedling of *Pyrus serotina* 'Chojuro' (O. P.) (17), two types of symptoms appeared. For one type, yellow vein banding of the adaxial ribs of the major veins and of the larger minor vein occurred; and later, in the yellowed tissue, streaks of brown appeared sporadically. In the other type, tiny asteroid spots were formed, singly or in clusters, as a result of discoloration associated with complexes of fine minor veinlets. The latter symptoms resembled a transmissible form of vein yellows reported by Canova (5). Severity of symptoms varied; some trees were severely affected, some were not affected, and still others had only a few leaves per shoot with vein yellowing and browning. A chloroplast deficiency in and hypertrophy of palisade parenchyma cells adjacent to the ribs apparently caused the vein banding. Also contributing was hypertrophy of bundle-sheath cells (Fig. 1-E). Sometimes when hypertrophy was massive it imparted more of a clearing than yellowing to veins especially when deficient chloroplasts later became green. Veins with this banding and browning disorder differed from those with pear decline; the phloem was normal and the leaves were not leathery or brittle. Branches of Magness grafted into the affected trees were symptomless; and, the disorder was not transmitted to Anjou pears by grafting.

DISCUSSION

Before MLO were discovered in plants, several yellows-type diseases whose causal agents are graft transmissible were reported to have sieve-tube necrosis as a significant part of their histopathlogy; yet when MLO were reported subsequently to be associated with the diseases, the MLO usually were illustrated in sieve tubes that appeared normal (4, 18). This suggested absence of detrimental effects of MLO on sieve tubes. In the pear hosts examined, MLO occurred abundantly in the sieve tubes of the finer minor veins, moderately often in phloem of larger minor veins, and infrequently in sieve tubes of secondary phloem. The MLO had no apparent maleffects on narrow sieve tubes of fine minor veins (Fig. 5-B), but secondary sieve tubes were necrotic even in the absence of MLO (10, 20). Presumably, MLO produce metabolic products, and furthermore, the MLO might induce the metabolically-active intermediary cells of the phloem in fine minor veins to produce some translocatable metabolites not normally formed (7). Toxic metabolites produced by MLO or intermediary cells could move basipetally in the phloem and along the way diffuse laterally from the sieve tubes into bundle sheaths and contiguous tissues and cause the pathoses found in them. They also could cause the necrosis of sieve tubes that was found in the secondary phloem of framework veins.

To produce a grafted tree that shows wilt and/or decline symptoms, graft partners must be from two groups of pears with different characteristics. On the Pacific coast, the tops of pear-decline-susceptible pear trees are varieties of *P. communis*. In the greenhouse, some of them show brown minor veinlets as with Comice (17, 21). Rootstocks used with trees susceptible to decline include the Japanese pear, *P. serotina*, Quince (*Cydonia oblonga*), and Variolosa (1, 3, 8, 17). When these hosts are grown as cuttings or as scions on rootstocks, leaf major veins show browning of abaxial ribs and necrosis of secondary sieve tubes as herein reported for Variolosa. In orchard trees affected by decline, sieve-tube necrosis occurs below the bud union as if induced by a toxin being translocated basipetally, and copious replacement phloem may form (1, 16). Necrosis of secondary sieve tubes in both leaves and tree trunks, may be produced by the same hypothetical toxin.

Precocious, the promising new indicator host, has disease characteristics more like the Variolosa than the Magness host type. Precocious differed from the Variolosa type host primarily in the early and prolific appearance of pathoses and symptoms; but like the Variolosa group, there was necrosis of secondary sieve tubes and excessive phloem formation. The early browning along the adaxial ribs of framework veins in Precocious resembles the late browning found in Variolosa (compare Fig. 2-A with 4-D). Browning that sometimes occurs on abaxial ribs of major veins of Precocious is also like that typically found on Variolosa.

The brown veins exhibited by the indicator hosts are more specific for the pear decline disease than are the decline and/or wilt symptoms exhibited by grafted trees susceptible to pear decline. The indicator hosts are therefore valuable for pear decline studies. In only two instances did false brown veins appear in the indicator hosts, and in these cases the symptomatic and histopathologic characteristics were distinguished from pear decline. In both instances, the false brown veins also appeared in control trees.

This study of disease development gives insight on the mode of parasitism of three indicator hosts by the pear decline MLO. It was proposed that toxic metabolites were produced by the MLO or the host and translocated from the fine minor veins to secondary sieve tubes where they caused necrosis and also to bundle sheaths and other vein-associated tissues where they caused pathosis.

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