Anatomy of Citrus Fatal Yellows: Evidence for Strains of the Causal Agent and for Double Infections with a Sunken-Vein Disease Agent

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ABSTRACT

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In orchards, lemon trees (*Citrus limon*) with alemow (*C. macrophylla*) rootstocks expressed symptoms of decline that resulted from necrosis of vascular tissues in the rootstocks. The causal agent was graft transmitted from rootstocks of declining orchard trees to alemow seedlings in the greenhouse, where serial passages of the isolates were made in alemow. A characteristic array of stem and leaf symptoms (shoot symptoms) occurred in some infected alemow seedlings, while yellowing and cessation of growth (decline) occurred in others. Both symptom types originated from a unique pathosis consisting of death and collapse of differentiating phloem, with the cambium and differentiating xylem also involved. Unaffected parenchyma cells divided, enlarged, and compressed the pathological tissue. In the resulting parenchymatous tissue, a cambium sometimes was reconstituted and formation of replacement vascular tissues ensued. Lasting remissions sometimes resulted, but relapses also occurred. The six isolates studied differed in virulence, ease of transmission, and perpetuation. Fatal yellows (FY) was difficult to study because of low rates of transmission, long incubation periods, remissions, uneven distribution of the agent in the tree, a complexity of strains, and double infections with a sunken vein (SuV) disease agent. Symptoms of SuV were: veins sunken abaxially and raised adaxially, episodes of formation of small yellow leaves, and twisting of stems. The disorder is apparently a separate entity because, during serial passages of two isolates, the FY syndrome was lost and the SuV syndrome maintained.

Fatal yellows (FY) is a graft transmissible disease of the alemow (*Citrus macrophylla* Wester) rootstocks of Eureka lemon trees (*C. limon* (L.) Burm.), which was studied in a plantation near Thermal, CA. Decline of trees that resulted from disease of the rootstocks was noted in 1973 in Blocks 10 and 11 planted in 1967 and in 1977 in Block 30 planted in 1970. Decline consisted of root deterioration, wilting, discoloration of leaves, defoliation, twig die back, and sparse growth, followed by tree death or recovery. When fatal yellows first appeared, tests for anatomical disorders such as lemon sieve-tube necrosis and Macrophylla rootstock necrosis were negative (7). A pathological condition (pathosis) in the rootstocks suggested a new disease and a causal agent was graft transmitted with root segments from affected trees into healthy citrus seedlings (4). An orchard tree in Block 10, row 37 was the first source tree for successful transmission. The agent it was carrying (hereafter, Isolate 10-37) was relatively easy to transmit and to maintain in alemow seedlings. A host range study of isolates 10-37 and 30-23 was made concurrently with the early part of this research, but none of the susceptible citrus species was a better host for experimental use than alemow (6). This paper for the first time gives illustrated descriptions of the symptoms and of the pathological conditions of tissues in diseased orchard trees and also in alemow seedlings infected with isolates from six orchard trees. Also reported are virulence, difficulty of transmission of some isolates, and the tendency toward remissions when alemow seedlings were infected with some isolates. A sunken vein syndrome is described that accompanied fatal yellows.

MATERIALS AND METHODS

Isolates were from scattered declining trees in three blocks of the plantation. Designation of isolates was by block and row number of source trees. Isolate 10-37, the first strain isolated, was moderately virulent and easy to transmit and to maintain. The 30-23 isolate was virulent and at onset caused symptoms like 10-37, but then tips of the central axis and branches died basipetally—eventually to the ground. The 30-30 and 10-55 isolates were also like 10-37, but after rest periods new shoots were often normal and seedlings recovered. The 10-33 and 11-6 isolates typically caused decline in seedlings with fatal-yellows pathosis associated with the decline.

Microtechnique. Bark was fixed in Randolph's Navashin solution (1) and sectioned frozen on an A.O. 860 sliding microtome. Sections of field trees were made radially through bud unions and transversely 5 cm above and below unions. Staining was progressive with hematoxylin and counterstaining was with lacmoid (1,2). The semithin monitor sections pictured in Figures 1D–F and 2 were from material processed for transmission electron microscopy according to a procedure previously described (5). A color test for starch was made by cutting across finger-sized roots with pruning shears and applying I_2-KI (1).

Graft transmissions. Roots about 1 cm diameter from declining orchard trees were immersed 3 min in a 5% aqueous solution of commercial bleach (6% NaOCl) and washed 2 hr in running water. Segments of the roots 6 cm long were then placed as tip and/or side grafts on trunks of alemow seedlings growing in the greenhouse. Rootstock suckers on the two declining trees in Block 30 supplemented root inoculum. After transmissions were obtained from the six orchard trees, passages of the isolates were made through alemow seedlings. These subinoculations were made by using tip and/or side grafts (Fig. 3B) or by placing buds, scions, or leaf patches (hereafter, patches) into T cuts in stems of alemow seedlings. Control trees in each experiment were grafted with healthy tissues or were ungrafted. To induce new growth in which shoot symptoms might form, plants were topped just above the grafts several weeks after inoculation and if needed at intervals thereafter. If symptoms failed to appear following these methods or if remissions occurred, trees were placed in the lathhouse or in a 10 C growth chamber for 2–3 mo, pruned, sometimes partially or completely defoliated to force buds, and returned to the greenhouse.

Greenhouse conditions. As the experiments progressed, there were changes in greenhouse (GH) assignments. In each section, temperatures were programmed for different parameters with thermostatically controlled steam radiators and evaporative coolers; although in summer temperatures were often higher than desired. Experience with experiments reported herein and in another paper (6) showed that low temperatures, short days, and shading when the sun was low slowed or prevented development of

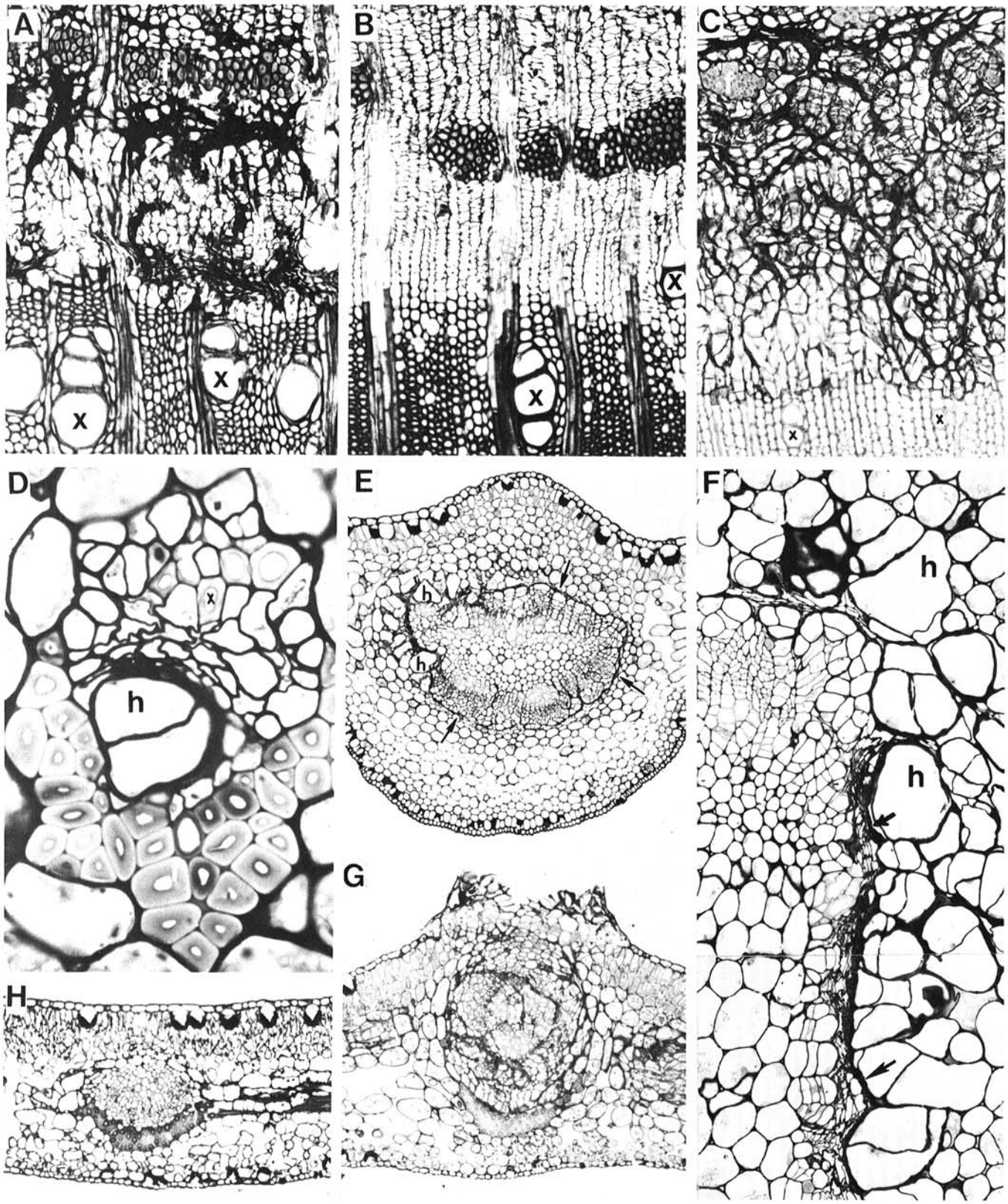


Fig. 1. Cross sections of vascular tissues of *Citrus macrophylla* infected with the 10-37 isolate except as noted. **A,** Trunk bark and wood of alemow rootstock from below bud union of a lemon tree in the orchard sampled on 29 November 1973. Cells of inner phloem, cambium, unlignified xylem and rays are variously affected by necrosis. Unaffected parenchyma cells have undergone hypertrophy and hyperplasia to form a parenchymatous tissue (×110). **B,** Control (×110). **C–H,** From greenhouse-grown seedlings. **C,** Trunk bark from dying seedling (30-23 isolate). Necrotic phloem, cambium, and unlignified xylem are compressed by proliferating parenchyma cells (×120). **D,** Cleared first order minor veinlet. Unlignified, necrotic cells are partially compressed by a large hypertrophic cell that divided once (×1,010). **E,** Midvein. At left, cambium and immediate derivatives are necrotic and compressed by hypertrophic cells. In rest of vein, earlier formed primary phloem is necrotic, but later forming secondary phloem is near normal (×100). **F,** Stem below D and E. In the leaf traces at top of figure, new vascular tissue is nearly normal, but the procambium and protophloem were earlier severely affected. Cortical cells are hypertrophied (h). In remainder of section, cambium and phloem are collapsed (arrow). Cortical cells severely hypertrophied (×260). **G,** Corky, proliferated, major-lateral vein from an old leaf (see 3G and H and 1H). Sieve tubes are necrotic on abaxial side. The proliferation of vascular tissue on lateral and adaxial sides of vein caused the surface of leaf to be pushed upward. The epidermis split open and a corky layer of cells formed (×120). **H,** Control for G. Phloem occurs only abaxially to xylem (×120). f = phleom fibers; h = hypertrophic cells; and x = xylem vessels.

symptoms. All experiments were ultimately moved to GH 11c, a warm section with a southern exposure and little shading, where monthly average temperatures at 1 p.m. varied from 25 C in December to 32 C in July, except that from 1977 to 1979 summer maximums reached 37 C on some days. From May to September GH 12b was warm enough for symptom development, but, in late autumn, winter, and early spring it was cool, partially shaded, and disease development was poor.

RESULTS

Decline of orchard trees. The leaves of declining lemon trees on alemow stocks wilted and discolored and many abscised. Fruiting and shoot growth were subnormal. A severe pathosis of the phloem, cambium, and immature xylem was sometimes encountered below the bud union (Fig. 1A, Table 1). Fibrous roots were usually lacking, while pioneer roots (3) were often rotted at their extremities. Starch was depleted in some roots, and locales of depletion appeared to be related to scattered disease activity in the root system. When trees were pulled, severed pioneer roots that were left protruding from the soil sprouted shoots and some were symptomatic.

Fatal yellows syndromes in alemow seedlings in the greenhouse. Localized destruction of the differentiating vascular tissues was fundamental to symptom formation. Seedlings became yellow and failed to make further growth (decline) when the central axis became diseased. Shoot symptoms occurred when disease first occurred in growing shoots. Decline preempted shoot symptoms if seedlings were not pruned after inoculation or if they were not ready to flush. Isolates 10-33 and 11-6 regularly caused decline (Table 1). Shoot symptoms were as follows: epinasty, clearing of veins, midvein curvature, adaxial swelling of veins, and failure of leaves to attain normal size and color (Fig. 3A–D). The major lateral veins were the first to clear during early stages of leaf development (Fig. 3E). On leaves with curvature of midveins, the portion of the leaf blade within the curvature was thin and chlorotic, and veins were clear. Some thorns were affected by a necrosis that progressed from tips to base and occasionally on into the stem. On severely affected seedlings, tips of growing shoots died.

During secondary growth, the adaxial enlargement of veins that began during primary growth continued. It resulted from copious amounts of phloem being laid down on the lateral and adaxial sides of veins where secondary thickening normally does not occur.

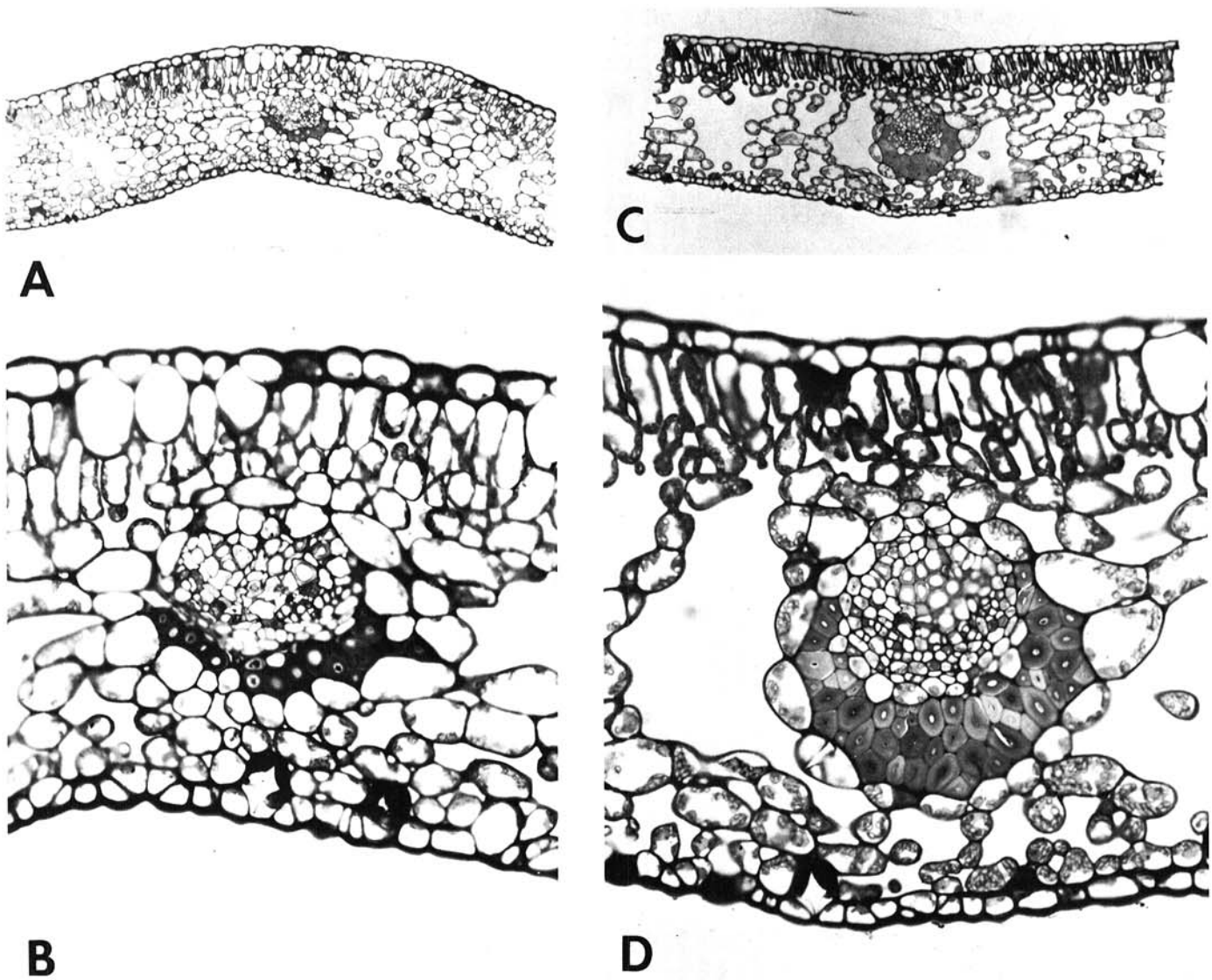


Fig. 2. Pathoses in a leaf affected by the sunken vein syndrome in *Citrus macrophylla*. **A and B,** Section of a leaf at two magnifications that had a marginal vein arched up on its adaxial side and sunken on its abaxial side (isolate 10-33). The leaf is thinner than the control (0.79 as thick). The palisade and spongy mesophyll tissues are poorly developed with smaller-than-normal air spaces. Stored starch in the plastids is deficient. The xylem was laid down in a disorderly manner and vessels are fewer and narrower than normal. The phloem and the primary phloem fibers are poorly formed. Sieve tubes are few but not necrotic. **C and D,** Two magnifications of a cross section of a healthy leaf at a marginal vein. Note the slight sinking of the adaxial surface at the vein and a slight protrusion of the abaxial surface—just the opposite from diseased. (A and C: ×120; B and D ×400).

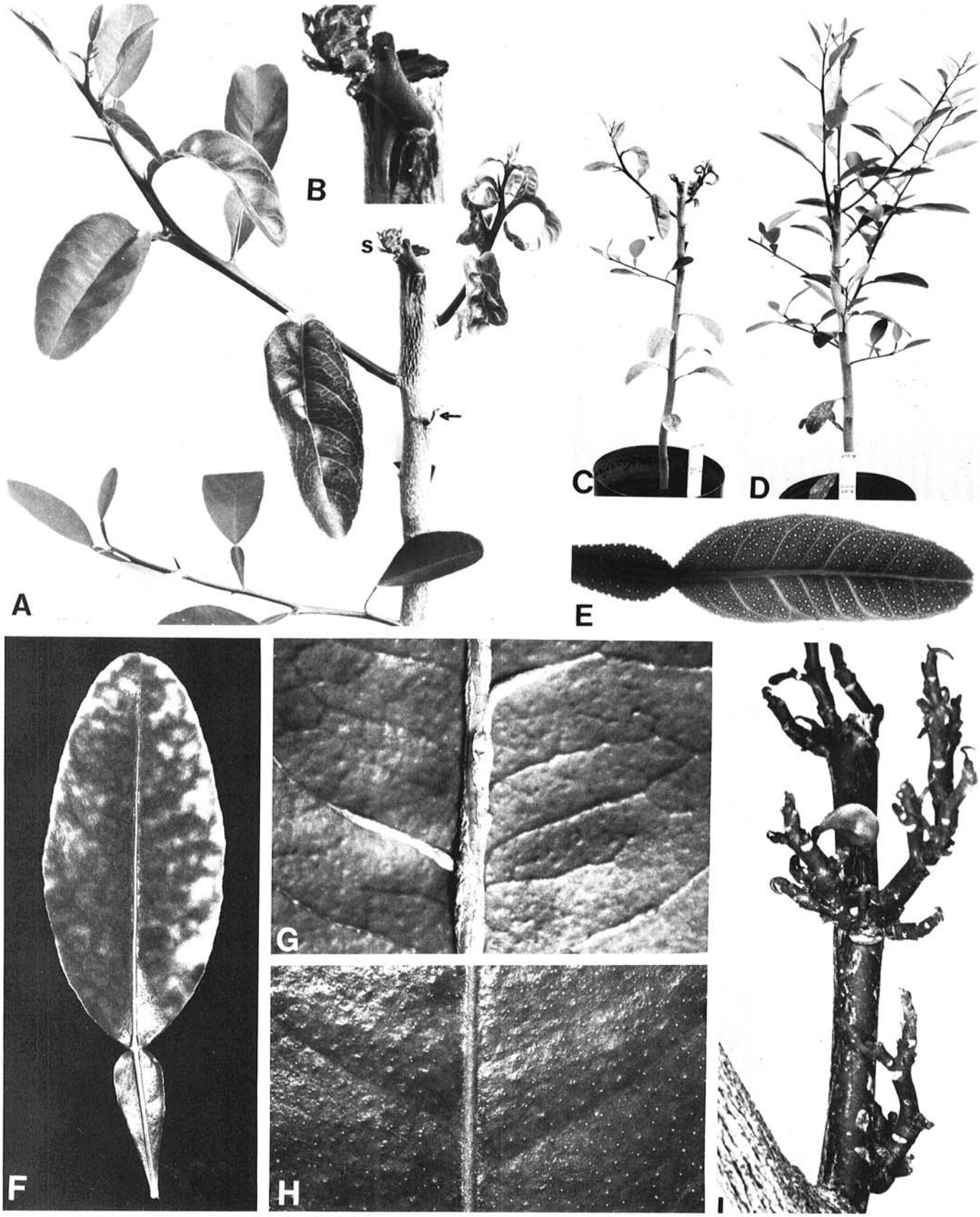


Fig. 3. Fatal yellows symptoms in *Citrus macrophylla* seedlings (10-37 isolate). **A,** 58 days after inoculation and 21 days after trunk was cut off above scion (s). Epinasty, retarded growth, and crumpled leaves occurred in shoot at upper right. Shoot below it failed to grow (arrow). Left side of tree less severely affected. Larger veins cleared in basal leaf (×0.8). **B,** Enlargement of side grafted scion shown in A. Multiple, malformed shoots grew from axillary buds on back side of scion (×2.5). **C,** Lower magnification of A (×0.16). **D,** Healthy control (×0.16). **E,** Cleared major veins in immature leaf—especially lower side of photograph (×4). **F,** Mature leaf with mottle (×0.75). **G,** Enlargement and corking of midvein and major lateral veins on adaxial surface of mature leaf (×4). **H,** Control for G (×4). **I,** Multiple aborted shoots at nodes (×2).

Eventually, the enlargement caused the epidermis to rupture; then, cork tissue formed from the underlying rib cells (Figs. 1G and H and 3G and H). Mature leaves often were blotchy (Fig. 3F). Bumpy areas sometimes formed on surfaces of stems and occasionally gum oozed. Peeling away the bark revealed that the cambial face was also bumpy. Excessive production of phloem and little xylem formation caused stems to be rubbery. On severely affected plants, tiny shoots formed one after another from whorls of buds in the axils of leaves, but the shoots soon aborted, preventing normal growth of the seedling (Fig. 3I). In seedlings inoculated while still small, development of root systems was suppressed. Fibrous roots as well as extremities of pioneer roots deteriorated. Chronically diseased seedlings were stunted and some died (Fig. 3C and D).

Decline occurred when seedlings were not pruned after inoculation and when seedlings were infected with isolates 10-33 and 11-6. Seedlings in decline eventually either died or recovered without developing fatal-yellows leaf symptoms. The phloem, cambial zone, and undifferentiated xylem in trunks and roots expressed pathosis similar to that for orchard trees and for seedlings with primary growth symptoms (compare Fig. 4A–C with 1A and C). Roots were severely underdeveloped and deteriorated. Often only a few stubs of the major lateral roots remained.

Histopathosis. Necrosis was initiated in the differentiating phloem and then the cambium and xylem mother cells became affected (Fig. 4A and B). Unaffected parenchyma cells enlarged and divided, compressing the necrotic tissues (Fig. 4C). The extent of pathosis around the circumference of stems, roots, and midveins varied from isolated patches to more or less continuous (Fig. 1E–G). Succulent stems and expanding leaves with cleared veins were sectioned for light and electron microscopy. The phloem, cambium, and immature xylem had become masses of dead cells that were compressed by drastic hypertrophy and slight hyperplasia of adjacent parenchyma cells (Fig. 1D–F). The contents of some dying cells stained darkly, but particles that might be virus or mycoplasmas were not observed with the electron microscope. Masses of large hypertrophic cells no doubt caused the cleared appearance of veins. Pathosis was valuable for characterizing fatal yellows.

The sunken vein syndrome (SuV). Sunken veins and some associated symptoms subtly and erratically accompanied the symptoms of fatal yellows (Figs. 2 and 5). Seedlings with the SuV syndrome but without FY were as vigorous as controls. Shoots would grow normally, and then occasionally a few small symptomatic leaves formed (Fig. 5E). At veins, leaf surfaces were arched up on their adaxial sides and sunken on their abaxial sides (Fig. 5A and B). Often adaxial sides of affected leaves took on a glossy yellow-green color near margins where there were complexes of sunken viens. Rugosity occurred when veins throughout a leaf were arched up (Fig. 5C). A faint vein netting that affected all of the veins—even the finest—often affected several leaves in a series (Fig. 5D). Other erractically occurring symptoms were: leaves with small leaflets but petioles of normal size, and leaves small on one side of a shoot and of normal size on the other. When citrons (*C. medica* L.) were inoculated with 10-33 void of FY, growth was retarded, epinasty and vein clearing occurred, and shoots grew in a corkscrew fashion.

The pathological condition that occurred in leaves with sunken veins resulted from hypoplastic processes. Where the leaf's abaxial surface was sunken at veins, air spaces in the spongy mesophyll were fewer and smaller than normal (compare Fig. 2A and B with 2C and D). This resulted in the sinking of the epidermal surface at veins on the abaxial side of the leaf and to the folding down of the blade on both sides of the vein. This folding contributed further to the sunken appearance of the vein on the leaf's abaxial side and to the arched up appearance on the adaxial side (Fig. 5A and B). In such a situation a vein was at the top of a crease. The bundles of primary phloem fibers were small and walls of individual fibers were not as thick as those of normal fibers, and they stained darker (Fig. 2B and D). The phloem was underdeveloped and necrosis, which characterized FY, was absent. The xylem vessels were few, narrow, and not arranged in radial rows. Chloroplasts were underdeveloped in cells of the mesophyll and bundle sheath.

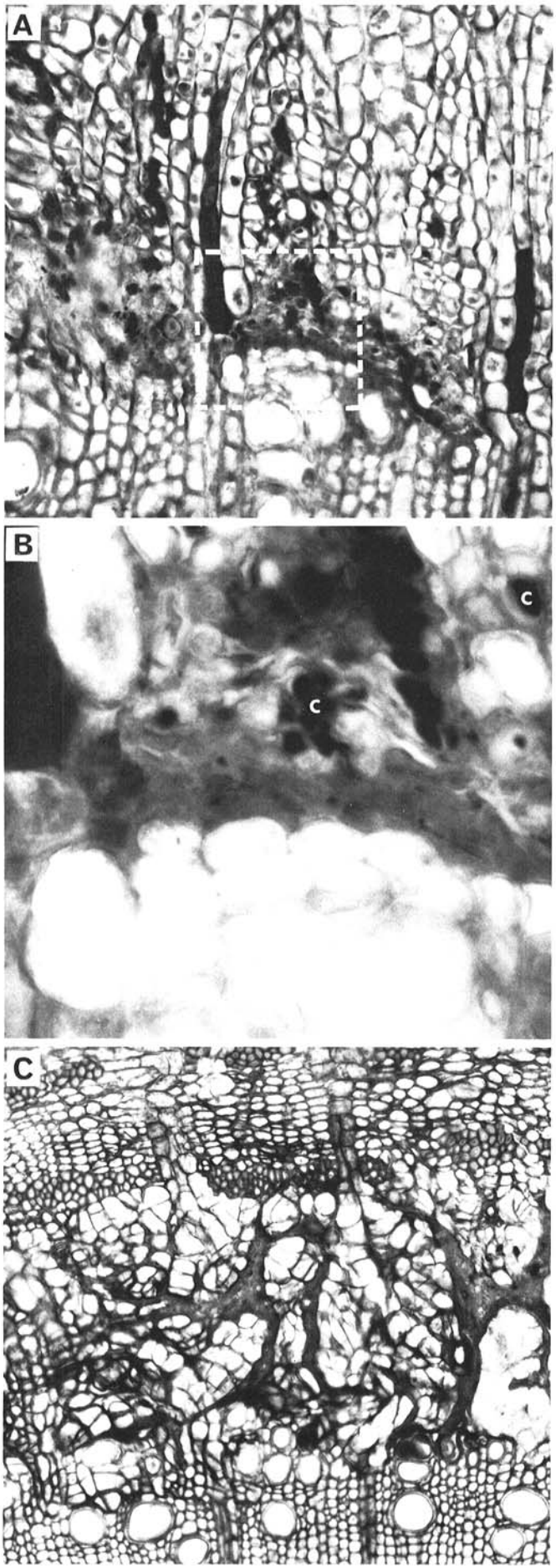
Symptoms of the SuV syndrome were not observed on alemow leaves of rootstock suckers in the orchard. However, they may have been overlooked because the SuV disease had not yet been observed at the time that orchard trees were studied.

Graft transmission of the fatal yellows and sunken vein agents. When it was determined that isolate 10-37 was graft transmissible (4), transmission attempts were made from other declining orchard

TABLE 1. Symptoms and transmissions of six different sources of the citrus fatal yellows disease

	Tree or isolate					
	10-37	30-23	30-30	10-55	10-33	11-6
Characteristics of diseased orchard trees						
Tree age (yr)	6	7	7	6	8	8
Severity of decline	severe	severe	severe	severe	chronic	severe
Pathosis below union[a]	2/2	2/2	1/3	0/2	1/10	3/4
Conditions of roots						
Fibrous roots	none	none	scarce	none	scarce	none
Pioneer roots						
Starch content	+	−	+	+	+	+
Extremities rotted	yes	yes	yes	yes	no	yes
Alemow suckers						
From trunk	no	yes[b]	yes	yes	no	no
From roots	yes[c]	yes[b]	no	yes[c]	no	no
Fate of tree	pulled	died	remission	pulled	chronic	remission
Inoculated alemow seedlings						
Leaf symptoms	yes	yes	yes	yes	no	no
Twig dieback (blighting)	some	much	some	none	some	none
Multiple shoots	some	many	some	none	none	none
Bumpy stems, corky veins	some	much	some	none	some	d
Pathosis	yes	yes	yes	yes	yes	yes
Decline and/or death	rare	rare	often	none	often	often
Sunken vein syndrome	yes	yes	...[d]	...[d]	yes	yes
Transmission	easy	easy	moderate	hard	moderate	moderate
Systemic infections	common	common	frequently	rare	decline	decline
Remissions	none	none	some	always	usual	usual

[a] Number positive/no. of samples collected.
[b] Suckers were symptomatic.
[c] Suckers only grew from roots after trees were pulled and a few were symptomatic.
[d] No record.



trees to determine whether a single disease was involved. Five new sources of fatal yellows were found. The results are summarized in Table 1. These isolates varied in virulence, in ability to persist in alemow, and in stability of symptoms. Repeated attempts over periods of years were required to obtain transmissions from some trees. Isolate 10-55 was the most difficult. Three transmission attempts with roots and symptomless-alemow-trunk suckers failed. Then, the tree was pulled and some of the roots left protruding from the soil sprouted shoots that grew into bushes. One bush was vigorous and green while another was small and discolored (deline). Transmissions were attempted from shoots of the latter, but they failed. Later the bush defoliated, and it was dug and transmission trys were made using various plant parts. Only scions from large lateral roots caused infections in alemow seedlings. Subtransmissions to other alemow seedlings were successful but then further passages failed. It may be concluded that isolate 10-55 was poorly distributed in its hosts and inherently difficult to maintain.

The following studies were performed with the 10-37 isolate. Passages were made through alemow seedlings many times, and the shortest incubation periods occurred from late April through October (Table 2). Seedlings were affected moderately, and the disease was induced systemically by pruning out normal branches, which were replaced by diseased shoots. Chronically affected seedlings lived for several years (Fig. 3C and D).

Tests were made to determine which parts of trees were infectious. Leaf patches from tender expanding leaves with cleared veins (Fig. 3E) and patches from mature symptomatic leaves transmitted equally, but the incubation period was longer for young leaf tissue (Table 2, Exp. 307). Tangential slabs with both bark and wood were cut from stems where bumps occurred and placed in T cuts. Infections occurred in two of four trees. Leaf patches with corky veins as inoculum induced infections in one of two cases.

To determine whether a fast rate of cambial activity, which is associated with elongating shoots, might enhance transmission and shorten the incubation period, 10 1.5-yr-old, 1.5-m-high, pot-bound seedlings in 4-L cans in a cool shaded greenhouse were divided into two groups. Group 1 trees were repotted into 8-L containers to promote growth and inoculated with symptomatic leaf patches when growth became vigorous. Trees in Group 2 were not repotted and were inoculated on nongrowing branches if available, otherwise on slow-growing branches. Symptoms appeared earlier and the disease distributed faster in the slow-growing Group 2 trees than in the luxuriantly growing Group 1 trees. By 133 days, all inoculated trees were showing symptoms, but even after 2 yr the Group 1 trees were growing vigorously and did not become systemically affected as did the slow-growing trees. Apparently a very active cambium does not enhance disease development.

To determine whether a decline of Eureka lemons on alemow rootstocks could be produced in the greenhouse, alemow seedlings were budded high with healthy Allen Eureka lemon buds, and the budlings were inoculated near the soil with infected alemow buds. Alemow suckers below the lemon buds developed fatal yellows

Fig. 4. Cross sections from the trunk of a fatal yellows diseased seedling that was in severe decline (10-33 isolate). **A and B,** Two magnifications of an area on the circumference of the vascular cylinder where pathosis is in an early stage of development (A: ×230; B: ×1,010). Darkly staining homogeneous material in the phloem is callose (C) stained with lacmoid on sieve plates. Its presence indicates a severe host reaction to infection. Some ray cells contain a vacuolar material that stained red and photographed darkly. Most of the thin walled cells in the cambial zone have collapsed, but a few ray, phloem, and thin-walled xylem cells are still turgid. **C,** An advanced stage of pathosis in which parenchyma cells that remained alive during earlier necrosis subsequently underwent hypertrophy and hyperplasia producing clumps of calluslike tissue that compressed the necrotic cells (×120).

symptoms, and lemon shoots that grew after topping the alemow seedlings showed symptoms of varying severity. Some lemon shoots recovered and others died.

DISCUSSION

The pathosis of fatal yellows consists of a necrosis of newly forming phloem that spreads to the cambium and immature xylem and then host reactions, consisting mainly of hypertrophy of parenchyma cells (Fig. 4). FY pathosis was useful for diagnoses and for distinguishing FY from the sunken-vein disorder, which has a pathosis unique to itself. The FY pathoses associated with orchard trees in decline and with seedlings inoculated from them were identical, confirming that FY of the orchard trees was transmitted (Figs. 1A and C and 4C). Likewise shoot symptoms and decline symptoms both stemmed from FY pathosis (Figs. 1C–G and 4A–C). Therefore, it may be concluded that both shoot and decline symptoms are parts of the same disease. On the contrary, the SuV syndrome stemmed from an hypoplastic condition that continued to transmit after the necrotic condition

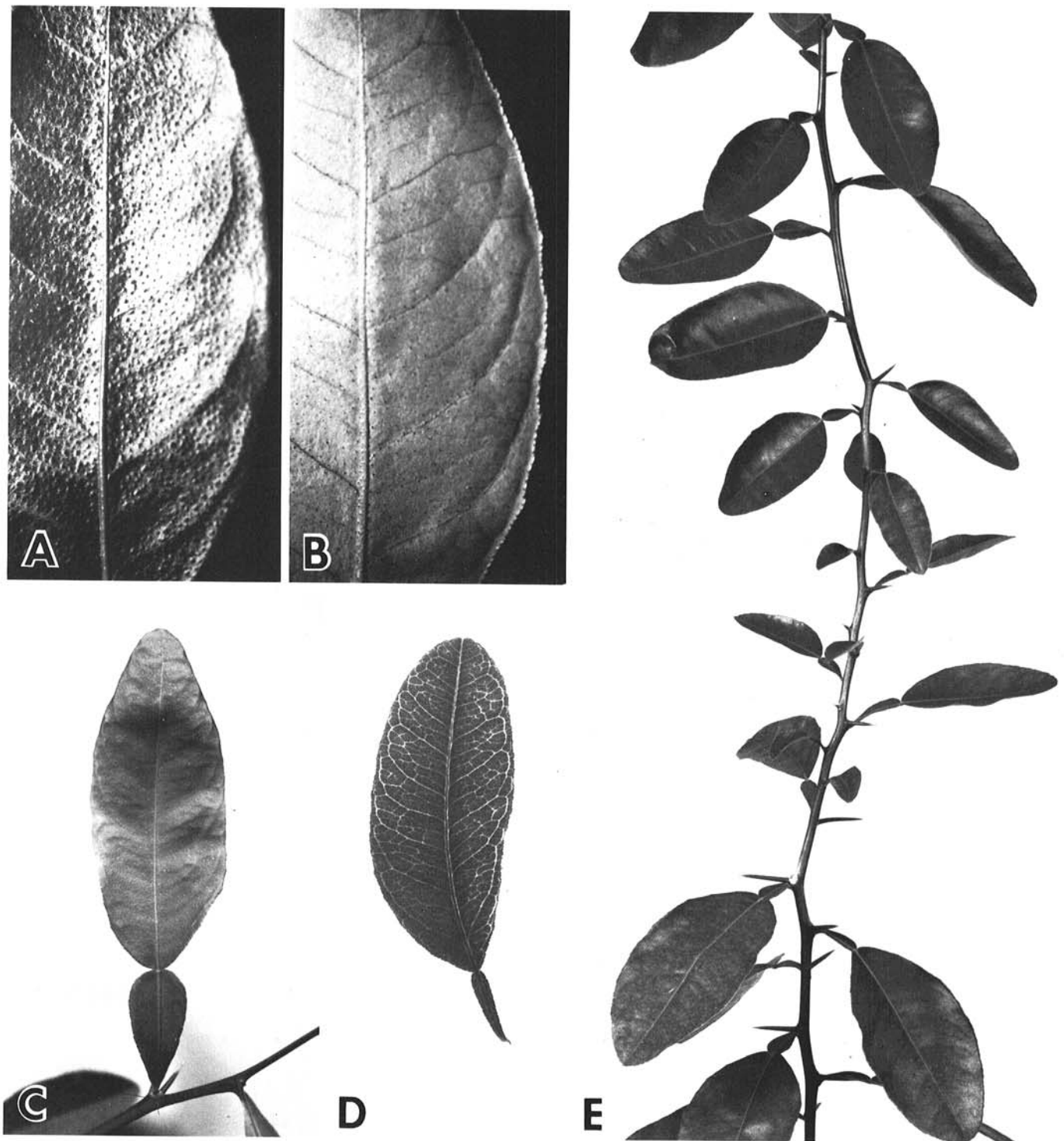


Fig. 5. Symptoms of the sunken vein syndrome in alemow, *Citrus macrophylla*. **A,** Arching up at veins on adaxial side of a leaf. Oil glands appear as small sphaerules (×2). **B,** Sunken veins on the abaxial side of a leaf. Stomata appear as small dots (×2). **C,** Rugosity resulting from many arched up veins (×1). **D,** Leaf with network of cleared veins. Clearing at right and slight curvature of the midvein may be due in part to fatal yellows (×1). **E,** Branch whose normal growth was interrupted with a series of small leaves. Leaves on one side of trunk are more affected than are those on other side. (×0.38). Isolates are: A-C, 10-33; D, 10-37; E, 11-6.

TABLE 2. Effect of season and source of inoculum on transmissions and incubation periods for the 10-37 isolate[a]

Experiment no.	Inoculation date	Inoculum	Transmission rate	Incubation periods for individual trees (days)
240	23 April 1974	2 buds, 2 patches	2/2	37,37
256	9 August 1974	3 buds	4/4	28,28,28,65
270	14 February 1975	2 buds	2/2	44,143[b]
271	17 February 1985	4 buds	4/4	29,46,67,144[b]
286	4 November 1975	2 side grafts	3/4	84,84,83
297	24 May 1976	2 side grafts	8/8	Less than 53
301	2 June 1976	1 bud, 2 patches	1/1	18
307	17–19 October 1976	5 patches (young)	3/4	71,110,151
307	17–19 October 1976	5 patches (old)	3/4	34,42,51
343	19 July 1977	1 bud, 4 patches	3/3	44,56,82
371	18 April 1978	3 young patches	0/3	...
	Reinoc. 30 May	3 additional patches	3/3	65[c]
376	16 May 1978	3 patches	2/2	21/48

[a]Trees were cut off above the inoculum at 10–15 days after inoculations except for trees in Experiment 371, which were bent into a circle.
[b]Severe pruning was required to induce new growth and symptoms.
[c]The other two trees declined without showing primary symptoms.

associated with isolates 10-33 and 11-6 ceased transmitting.

Remissions of fatal yellows could have resulted from infected cells becoming necrotic, collapsing, and finally being crushed by hypertrophy and hyperplasia of adjacent parenchyma cells. It is conjectured that the causal agent was trapped in the necrotic tissue, especially in cases with virulent isolates and strong host reactions. Remissions were characteristic for isolates 30-30, 10-55, 10-33, and 11-6. Severe pathological processes could have caused donor trees to be difficult to transmit from as in the case of tree 10-55.

Long incubation periods (Table 2) and remissions were prone to occur from mid-October to mid-April. Both phenomena appeared to be partially related to winter with its low maximum temperatures, short days, and shading associated with the low angle of the sun. Placing trees next to south facing glass walls promoted symptom development.

The erratic occurrence of pathosis in the trunks of orchard trees (Table 1) and in infected seedlings in the greenhouse and frequent failures in transmission indicate that the fatal yellows causal agent was unevenly distributed in the host.

The formation of suckers on trunks and pioneer roots of declining orchard trees apparently resulted from the localized blockages of the translocation stream. Rootstocks of citrus and pear trees commonly sucker when translocation is restricted by diseases such as Tristeza and Pear Decline (personal observations).

Trees that declined in the orchard may have become infected as seedlings before being budded to lemons, perhaps in the seed bed or nursery. The disease could have been in remission when alemow seedlings were budded, and it could have become reactivated years later in the orchard. Symptoms of FY were not found in a nearby block of mature alemow trees used as a seed source. Therefore, seed transmission seems unlikely. Lemons are hypersensitive to fatal yellows, and the disease could probably not become systemic in them (6).

Severity of disease in seedlings in the greenhouse was influenced by location of the infection at onset, virulence of the isolate, and the type of inoculation. As mentioned earlier, if disease occurred first in the central axis of nongrowing seedlings, they declined without shoot symptoms. On the other hand, trees severely pruned 1 or 2 wk after graft inoculations put out shoots with leaf and stem symptoms. Virulence of isolates was expressed in several ways. Isolate 30-23 expressed virulence by a gradual basipetal killing of the central axis of seedlings. Bumpy stems and swollen corky veins were also considered expression of virulence. Isolate 11-6 made its virulence known by sudden wilting of the large vigorous orchard tree that it infected and by a recovery that was also an expression of virulence. Isolate 11-6 killed seedlings in series of inoculation experiments as did isolates 10-33 and 30-30. These latter two isolates transmitted best with segments of the central axis shortly after decline appeared.

The pathosis of the SuV disease is quite different from the pathosis of FY. Pathoses for the SuV disease are expressions of underdevelopment, while pathoses for FY arise from necrosis of young phloem with the cambium and its immediate derivatives also becoming involved. A deficiency of xylem vessels in SuV diseased seedlings may have accounted for wilting of leaves under stress.

The sunken vein syndrome appears not to belong to the fatal yellows disease because, toward the last of a series of FY transmission experiments with the 10-33 and 11-6 isolates, the FY syndromes ceased transmitting but the SuV syndrome continued. The symptoms of the SuV syndrome had been considered a part of the FY syndrome (6), and the possibility that there might be two diseases was not recognized for a long time after transmission of the FY syndrome ceased.

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