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Ash Yellows and Lilac Witches'-Broom: Phytoplasmal Diseases of Concern in Forestry and Horticulture

Ash yellows (AshY) and lilac witches'-broom (LWB) are diseases of *Fraxinus* and *Syringa* species, respectively, believed to be caused by phytoplasmas (formerly called mycoplasmalike organisms). AshY and LWB in highly susceptible taxa are characterized by slow growth, progressive loss of vitality, dieback, and premature death (Figs. 1 and 2). These diseases are considered together in this article because evidence indicates they are caused by the same pathogen. Knowledge of AshY and LWB has accumulated primarily since 1983, but enough is now known for these diseases to serve together as a model for tentative understanding of lesser known phytoplasmal diseases of trees and shrubs in forests and horticultural landscapes. Objectives of this article are to summarize information about AshY and LWB, relate this information to knowledge of other phytoplasmal diseases of perennial plants, and identify needs for further research.

Fraxinus and *Syringa* are closely related genera in the *Oleaceae*. Some ash species in Europe and North America are commercially important for wood products, including furniture, tool handles, and baseball bats; and some species are planted in horticultural landscapes (Fig. 2A). Three of the 12 North American species, white ash (*F. americana*), green ash (*F. pennsylvanica*), and velvet ash (*F. velutina*), receive emphasis in this article. Lilacs are flowering shrubs or small trees grown in temperate regions for their fragrant and attractive floral displays in spring. Lilac and ash taxa are generally graft-compatible, and some cultivars of lilac have been grown on ash rootstocks.

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History and Distribution of AshY and LWB

AshY, known only in North America, was discovered in the 1960s during research on a decline syndrome of white ash called "ash dieback." C. R. Hibben noticed witches'-brooms on occasional declining ash trees in New York State and, with B. Wolanski, performed electron microscopy that revealed pleomorphic membrane-bound bodies in phloem sieve tubes of the brooms (18). These bodies were like those of the phloem-limited microorganisms discovered by Doi et al. (5), now called phytoplasmas (33) (Fig. 3). Hibben transmitted them by dodder to periwinkle (*Catharanthus roseus*), resulting in symptoms typical of yellows-type diseases (*yellows* for similarity to aster yellows): chlorosis, suppressed flowering, reduced leaf size, dwarfing, shoot proliferation, and ultimately, wilting and death. The organisms in the brooms were not suspected to be a major cause of ash decline, however, because most declining trees lacked brooms. Moreover, techniques suitable for assaying large numbers of declining versus healthy ash for phytoplasmas were not available. The capability for such surveys would await development of the DAPI fluorescence technique (34) and Dienes' stain (4). AshY was named and more fully described and its association with decline of white ash shown in 1983 to 1990 (23,24,42). In this article, the term *decline* refers to progressive loss of vitality, regardless of cause, and *dieback* in absence of quotation marks refers only to dead apical parts or the process of their dying.

"Ash dieback" was believed caused primarily by drought stress (16,31,36,45).

Fig. 1. White ash with ash yellows on an old-field site in New York State. Diseased trees display dieback or thin crowns with light green foliage.

In current understanding, drought-induced decline of white ash occurs on sites where soil water is intermittently depleted (48), whereas AshY occurs on diverse sites (22,42,44). A hypothesis linking AshY and drought stress in white ash is that AshY phytoplasmas, by causing diminished root function (6), predispose the trees to more severe or prolonged stress than that experienced by healthy trees during drought. This hypothesis is supported by observations that growth recovery of phytoplasma-infected white ash after years of drought was incomplete compared to that of healthy trees (13).

LWB (Fig. 4) was first reported in the 1950s (1) and was more fully described three decades later (14,15). Lilac collections in several arboreta have been damaged by LWB, and some have been decimated as diseased plants were removed in attempts to diminish the threat to remaining plants. The disease is also common in home and institutional landscapes. Hibben

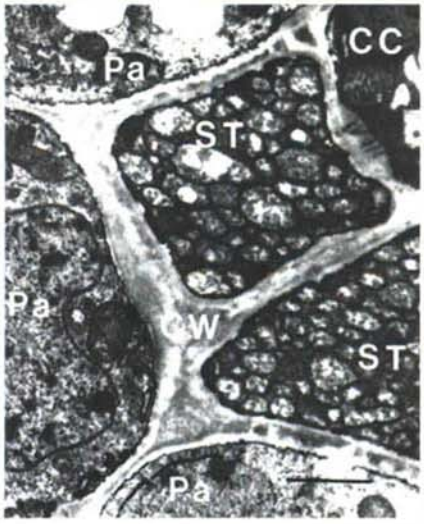

Fig. 3. Transmission electron micrograph of a transverse section of lilac leaf midrib containing a dense population of phytoplasmas in phloem sieve tubes. CW = cell wall, Pa = parenchyma, ST = sieve tube, CC = companion cell. Bar = 2 µm.

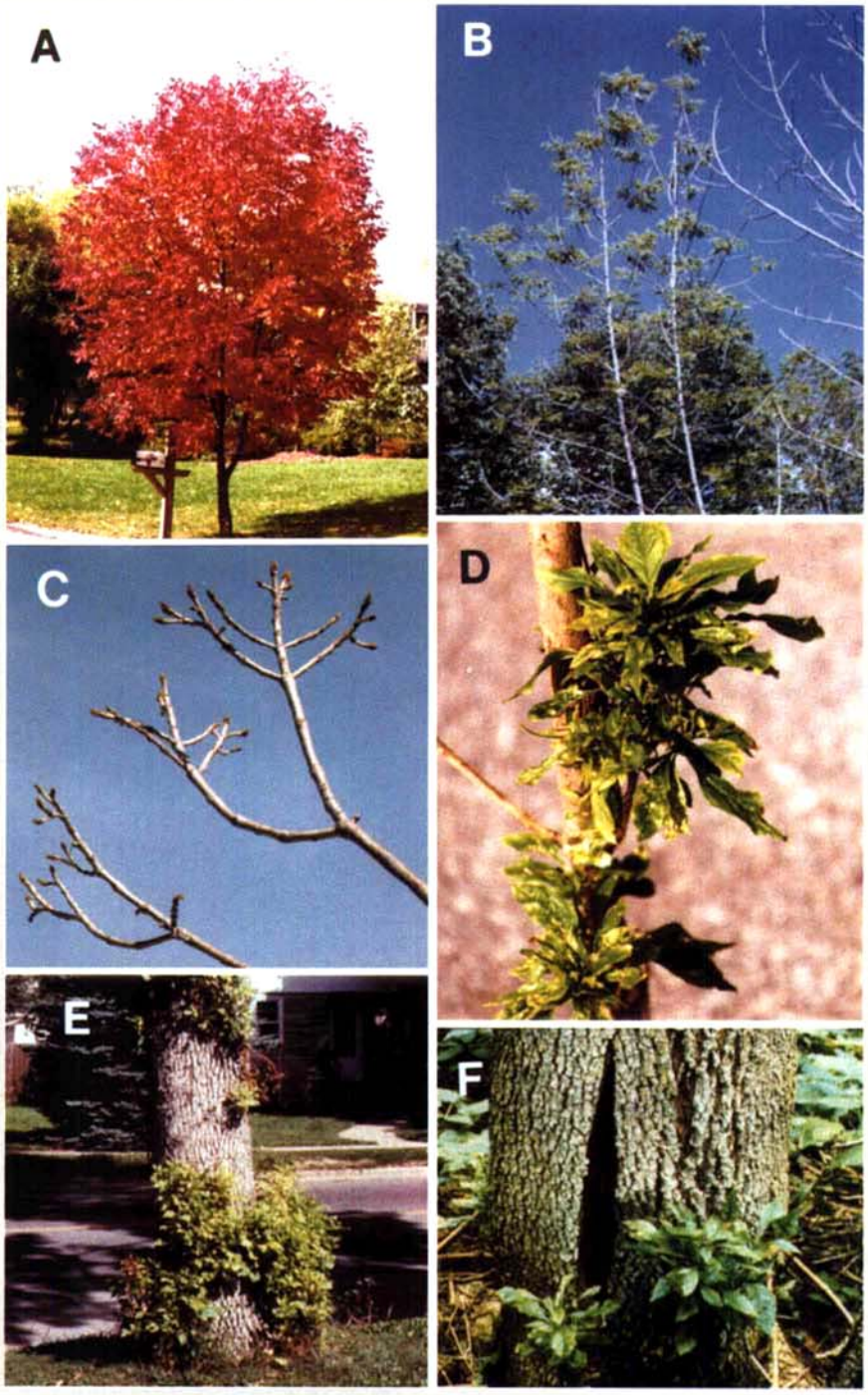

Fig. 2. (A) Healthy white ash cv. Autumn Applause in autumn color. (B–F) Symptoms of ash yellows. (B) Tufted foliage on slowly growing twigs of white ash. (C) Deliquescent branching on white ash. (D) Witches'-brooms with simple leaves on a white ash sapling. (E) Witches'-brooms on a green ash. (F) Witches'-brooms and split bark caused by frost damage to the cambium of a white ash.

Fig. 4. Lilac witches'-broom on lilac cv. Royalty. (A) Tufted foliage and dieback. (B) Dense brooms near the root collar.

and coworkers (14) noticed coincidental presence of AshY and LWB in several arboreta and suspected the two diseases had a common cause. This suspicion proved accurate, as AshY and LWB phytoplasmas caused indistinguishable symptoms in ash and lilac after reciprocal transmission by grafting and in the experimental host periwinkle after transmission by dodder. Also, AshY-specific DNA probes hybridized with DNA from lilacs infected with LWB (11,17), and a monoclonal antibody selected for its ability to react only with phytoplasmas in the AshY group reacted with phytoplasmas in lilacs as well as those in ash (11). Thus, AshY phytoplasmas cause LWB. The currently known host range of AshY phytoplasmas in nature includes 12 ash species and 19 lilac species (14,17; unpublished data). These are: *Fraxinus americana*, *F. angustifolia*, *F. bungeana*, *F. excelsior*, *F. nigra*, *F. latifolia*, *F. ornus*, *F. pennsylvanica*, *F. potamophila*, *F. profunda*, *F. quadrangulata*, *F. velutina*, *Syringa* ×*diversifolia*, *S.* ×*henryi*, *S.* ×*josiflexa*, *S. josikaea*, *S. julianae*, *S. komarowii*, *S. laciniata*, *S. meyeri*, *S. microphylla*, *S.* ×*nanceiana*, *S. oblata*, *S. patula*, *S.* ×*persica*, *S.* ×*prestoniae*, *S. sweginzowii*, *S. tomentella*, *S. villosa*, *S. vulgaris*, and *S. yunnanensis*. Many infraspecific ash and lilac taxa and interspecific hybrids are also recorded hosts. Experimental hosts include *Cuscuta* spp. (dodder), *Daucus carota* (carrot), *Trifolium pratense* (red clover), and *Catharanthus roseus* (periwinkle) (37).

AshY now occurs in at least 19 contiguous states and two Canadian provinces from Montana and Nebraska eastward to New England, and also in scattered southwestern localities (40,42,47; Fig. 5). Both naturally seeded and planted ash are affected. AshY incidence greater than 50% has been found in some white ash populations in northeastern states and in a velvet ash (*F. velutina*) population in Utah (38,40). Incidence rates of 3 to 27% were detected in green ash shade trees in Iowa and Wisconsin cities (9). The disease is rare in shade-tree nurseries, presumably because of unsuitable habitats for vectors. An outbreak in one midwestern nursery was caused by grafting with diseased scion material (G. L. Worf, *personal communication*). The range of LWB is less well-known than that of AshY but extends at least from Wisconsin to Massachusetts (14). These diseases have not been reported from Pacific Coast states, but susceptibility of the two most common ash species there, Oregon ash (*F. latifolia*) and velvet ash, is known from disease occurrence elsewhere (17,42).

Symptoms

Full descriptions of AshY and LWB symptoms and lists of susceptible plants are available elsewhere (14,15,24,37). In brief, both of these diseases cause slow apical and radial growth, diminished apical dominance or deliquescent branching (Fig. 2C), suppressed root development (Fig. 6), precocious flowering and/or shoot growth, and witches'-brooms (Figs. 2D–F and 4). Subnormal greenness and foliar deformities are common, and chlorosis occurs in occasional plants. Highly susceptible plants commonly sustain dieback of branches and roots, produce brooms and stunted deliquescent branches, and die prematurely (14,24). White ash produces abnormally short, bushy roots (Fig. 6) or sustains rootlet necrosis that in small plants may lead to sudden wilting and death (6). Histological symptoms of AshY or LWB, more readily observed in roots than in twigs, include autofluorescent sieve tubes and pathological sclerenchyma (6) (Fig. 7B). Ash and lilac infected by phytoplasmas are abnormally sensitive to freezing. Cambial damage to white ash (Fig. 2F) and unusual winter kill in lilac collections have been observed (14,24).

Witches'-brooms are diagnostic for AshY and LWB, but other symptoms are only indicative, because environmental factors or other pathogens or pests could cause them. In midwestern states, green ash with AshY and with dieback commonly occur coincidentally, but no consistent relationship between dieback and phytoplasmal infection has been detected (9,22). A similar situation occurs in velvet ash in Zion National Park, Utah (40). Diagnosis of AshY or LWB in plants that lack brooms usually requires laboratory tests, discussed in a later section of this article.

Impact on Host Growth and Survival

The impact of AshY on growth has been studied in three species: white, green, and velvet ash. Growth of phytoplasma-infected white ash and green ash usually diminishes progressively (Fig. 8) (7,39), but velvet ash tolerates infection with only slight growth loss (39; unpublished data). Tree-to-tree variation in growth loss occurs in inoculated white ash and green ash (unpublished data) and has been observed

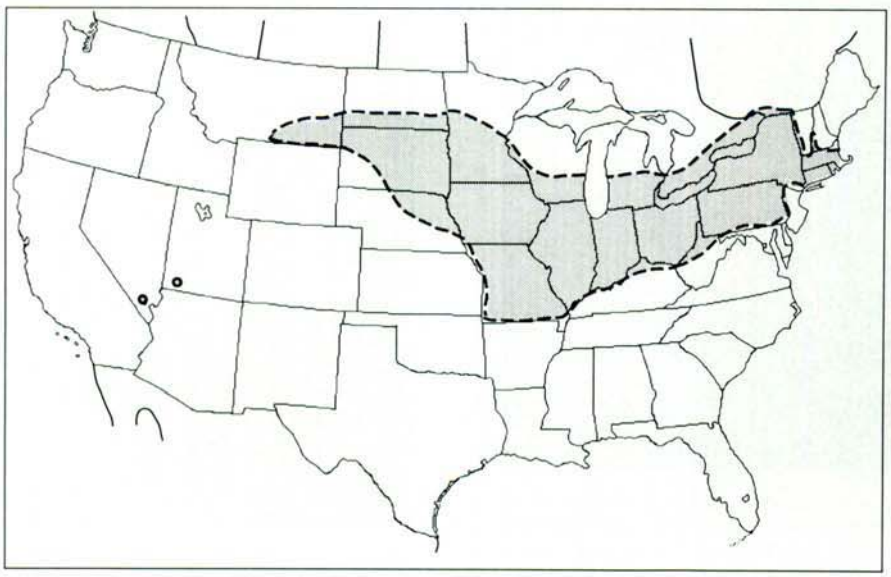

Fig. 5. Geographic distribution of ash yellows as known in 1995.

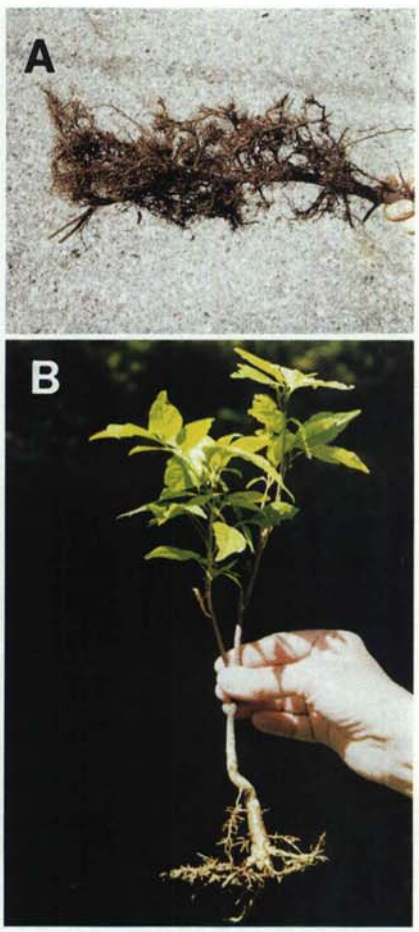

Fig. 6. Abnormally short, tapered, and bushy roots of white ash affected by ash yellows. (A) Root from a mature tree. (B) Roots of a naturally diseased seedling.

in naturally infected trees (39). Severely affected trees produce growth rings less than 1 mm wide (24,39) (Fig. 9), whereas other diseased individuals grow at moderate rates for many years.

In some areas, AshY limits production of ash wood for commercial uses, and both AshY and LWB reduce the longevity and amenity value of host plants. AshY acts as a thinning agent in dense young stands, because diseased trees lose the ability to compete effectively for growing space and site resources, become overtopped by faster growing neighbors, and die from suppression if not from direct effects of the disease (38,44). In stands of mixed species, this thinning facilitates conversion to nonsusceptible species. In stands dominated by ash, however, progressive increase of AshY diminishes productivity.

Phytoplasmas have often been detected in healthy-appearing lilacs or ash, and some diseased ash appear normal, or nearly so, for many years (38). Tolerance of infection is one of several possible explanations for these observations. Alternative possibilities are that plants may have been observed during early stages of infection, or they may have harbored phytoplasma strains having low virulence.

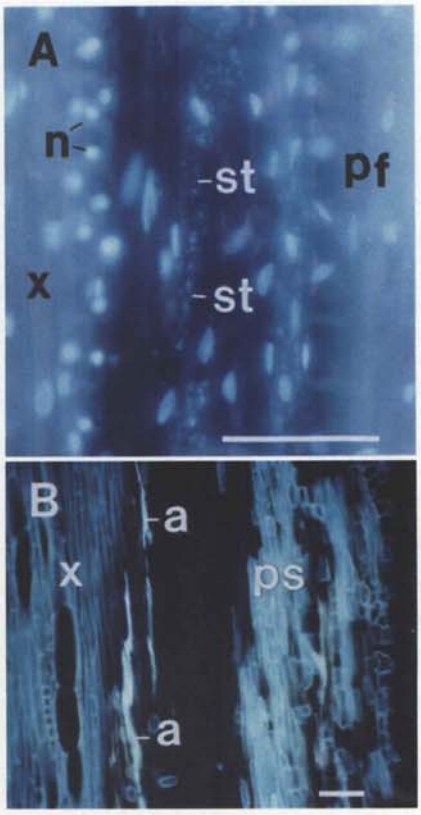

Fig. 7. Longitudinal sections of unstained roots of ash yellows–affected white ash viewed with a fluorescence microscope configured for the DAPI test. (A) Section treated with DAPI. Luminous objects (n) are plant nuclei. Fluorescent specks in phloem sieve tubes (st) are phytoplasmal DNA. x = xylem, pf = phloem fibers. Bar = 50 µm. (B) Untreated section showing disease-associated anatomical features: autofluorescent sieve tubes (a) and pathological sclerenchyma (ps). Bar = 100 µm.

The Pathogen

Phytoplasma concept. The change in terminology from *mycoplasmalike organism* to *phytoplasma* in 1994 (33) reflected new knowledge about plant-inhabiting pleomorphic mollicutes (prokaryotes in the class *Mollicutes*). These organisms were, until recently, considered to be allied with mycoplasmas (*Mycoplasma* and related genera) on the basis of morphological and ultrastructural similarity revealed in electron micrographs. Both phytoplasmas and mycoplasmas are unicellular and lack cell walls; their cells are delimited only by a membrane. Strong circumstantial evidence for pathogenicity of phytoplasmas has been presented by numerous researchers, but up to now these organisms cannot be grown in pure culture, and proof of their pathogenicity remains incomplete.

Studies of DNA homology in the highly conserved genes encoding ribosomal RNA and ribosomal protein have shown that phytoplasmas comprise a coherent set distinct from all other prokaryotes (12,35). Their closest known relatives are in the genus *Acholeplasma.* Several groups of phytoplasmas have been differentiated on the basis of nucleotide sequence variation in 16S ribosomal RNA genes (12,28,32,35). This differentiation is supported by sequence homology in ribosomal protein genes and in random parts of the phytoplasma genome (12,19). Thus, the stage is set for classification of phytoplasmas. Until these organisms can be cultivated and characterized apart from their hosts, they may be referred to *Candidatus* status (27). "*Candidatus* Phytoplasma" will accommodate all putative species of plant-inhabiting pleomorphic mollicutes. Approximately 12 *Candidatus* species will be proposed initially (Table 1), and more will probably follow. The classification will be based primarily on genome size and phylogeny deduced from nucleotide sequence of 16S ribosomal RNA genes. Secondary characteristics will include plant and insect host range where known. Phytoplasma strains causing AshY and LWB will be assigned to one species, because all such strains studied to date are closely related to one another and distinct from other phytoplasmas (3,11,12,32).

Phytoplasma detection and identification. Ash and lilac phytoplasmas, in common with many others, are detectable microscopically in phloem by means of Dienes' stain (4,15,24) or the DAPI test (34,41). The latter is preferred because of its greater accuracy. DAPI (4′,6-diamidino-2-phenylindole·2HCl) binds to DNA and causes it to fluoresce under UV. When longitudinal sections of twigs, petioles, or small roots, treated with DAPI, are examined with a fluorescence microscope, phytoplasmal DNA appears as blue-white fluorescent specks or aggregations in sieve tubes (Fig. 7A), whereas normal sieve tubes remain dark. The DAPI test is nonspecific, as DNA of any organism fluoresces under the test conditions.

Detection and identification are possible by several DNA-based techniques (Fig. 10), among which those involving the polymerase chain reaction (PCR) have become popular because of high sensitivity. PCR is preferred in situations where the concentration of phytoplasmas in a host may be very low. AshY phytoplasmas and some others can be simultaneously detected and identified using PCR primers

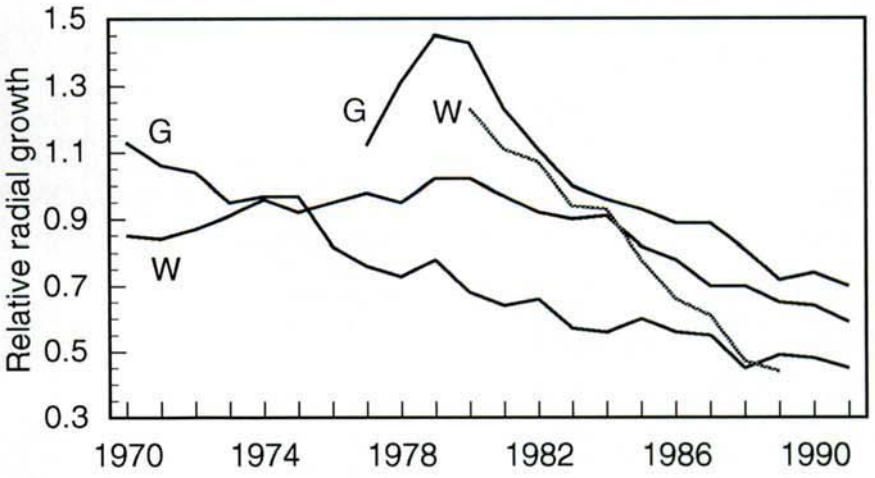

Fig. 8. Decline in relative radial growth of trees that contracted ash yellows in two populations of white ash (W) and two of green ash (G) in New York State over periods of 10 to 22 years. Average growth rates of tree groups in which phytoplasmas were detected with DAPI in 1989 or 1991 are plotted as proportions of the average growth rates of healthy trees in the same populations. The year when each tree became infected is unknown. Relative growth rates >1 in the early years of three of the records reflect the tendency for the largest trees in young populations to be the first infected. Figure adapted from reference 39.

that permit amplification of DNA sequences possessed only by strains belonging to particular groups (2,20; J. P. Prince, *unpublished*). A one-step detection–identification using a group-specific primer pair is useful to confirm preliminary diagnosis or suspected presence of particular phytoplasmas but may miss unrelated phytoplasmas or mixed infections. Nonspecific detection followed by identification of detected strain(s) may be accomplished by several means, one of which involves use of PCR primers based on sequences in the 16S ribosomal RNA gene that are common to all phytoplasmas but do not occur in plants (21). A DNA segment of characteristic size is amplified from any phytoplasma. The organism can then be identified by using this initially amplified DNA segment as a template for further PCRs using primers that amplify DNA from only particular phytoplasmas. These primers are based on nucleotide sequences between the positions of the first primer pair on the 16S rDNA (20). As an alternative, the initial PCR product can be subjected to restriction fragment length polymorphism (RFLP) analysis, in which the amplified segment is digested with certain restriction endonucleases and the fragments are separated by gel electrophoresis. Phytoplasmas in different groups have different RFLP profiles (Fig. 11). AshY phytoplasmas can be distinguished from others by RFLP analysis of 16S rDNA with the restriction enzyme *Alu*I (11,21). AshY phytoplasmas can also be identified by means of DNA hybridizations using probes that hybridize to group-specific sequences (3,11).

Fig. 9. Transverse view of the stem of a small, declining white ash affected by ash yellows. Annual xylem rings during the most recent 10 years (approximately) consist of only earlywood vessels and associated parenchyma. The arrow is positioned at the beginning of the third ring formed after growth slowed. Bar = 5 mm.

Immunological tests have been used to identify certain phytoplasmas, including those associated with AshY and LWB (11). However, these tests, including the recently developed immuno-capture PCR (30), are less useful than PCR-based techniques alone when phytoplasmal identity is initially unknown.

Epidemiology

AshY occurs most often and causes the most damage in regions where wooded and open areas are intermixed (42). It is less common in areas that are primarily forested, and then tends to be associated with stand openings and other exposed sites (44). Vector habitat preference could explain this pattern of occurrence.

AshY phytoplasmas apparently have a limited host range in nature. Searches for these organisms in plants other than ash on sites of abundant AshY were unsuccessful, although phytoplasmas belonging to several other groups were found (10,11). The natural plant host range may be determined primarily by vector feeding preferences.

Rates of AshY increase in young white ash populations were studied in New York State, where single-year increases on particular sites varied between 0 and 10% of the initial population per year (38) (Fig. 12). These rates, while considerably lower than those reported for epidemics of elm yellows, lethal yellowing of palms, and X disease of cherries, would be sufficient for AshY to interfere with stand productivity. The gradual increase of disease over a wide range of disease incidence, as shown in Figure 12, indicates the likelihood of sustained disease increase on conducive sites.

Table 1. Phytoplasmal disease groups based on relatedness of pathogens

Typical disease	Phylogenetic subclade[a] and corresponding 16S rRNA group[b] of pathogen		Diseases with related phytoplasmas[c]
Aster yellows	i[a]	I[b]	Apricot chlorotic leaf roll; bigbud and stolbur of nightshade, pepper, tomato; blueberry stunt; chrysanthemum yellows; dogwood stunt; grapevine yellows; Molière's disease of cherry; mulberry dwarf; onion yellows; paulownia witches'-broom; peach decline; periwinkle little leaf; phyllody of clover, hydrangea, safflower; plum leptonecrosis; sandal spike; tomato yellows; virescence of columbine, *Diplotaxis,* evening primrose, hydrangea, larkspur, periwinkle, *Plantago,* primrose, rape
Apple proliferation	ii	X	European stone fruit yellows, black alder witches'-broom, pear decline, oak decline
Peanut witches'-broom	iii	II	Red bird cactus witches'-broom, sweet potato witches'-broom
X disease of *Prunus* spp.	iv	III	Almond brown line; blueberry witches'-broom; clover yellow edge; goldenrod yellows; grapevine yellows; milkweed yellows; peach yellow leafroll; peach yellows; pear decline; pecan bunch; spirea stunt; X diseases of cherry, peach, prune; tsuwabuki witches'-broom; walnut witches'-broom
Rice yellow dwarf	v	XI	Sugarcane whiteleaf, Bermudagrass whiteleaf
Pigeon pea witches'-broom	vi	IX	
Lethal yellowing of palms	vii	IV	"Lethal disease" of palms (in Africa)
Ash yellows	viii	VII	Lilac witches'-broom, eggplant little leaf (32)
Potato witches'-broom	ix	VI	Clover proliferation, tomato bigbud, virescence of periwinkle
Elm yellows	x	V	Alder decline, flavescence dorée of grapevine, hemp-dogbane yellows, *Rubus* stunt
Loofah witches'-broom	xi	VIII	
Lime witches'-broom[d]			Faba bean phyllody, sunn hemp witches'-broom, sesame phyllody, *Cleome viscosa* phyllody (32)

[a] Subclade numerals (lower case) are those used by Gundersen et al. (12).
[b] Group numerals (upper case) are those used by Lee et al. (21).
[c] Some diseases are listed with more than one group, because more than one kind of phytoplasma has been associated with them.
[d] The *Candidatus* name "*Candidatus* Phytoplasma aurantifolia" has been proposed for the phytoplasma associated with lime witches'-broom (49).

Alate vectors of AshY are implicated by epidemiological data. In populations of green ash and white ash saplings, those with branches above the prevailing vegetation were the first infected (39). Spatial analyses revealed no significant differences in AshY incidence in crowded versus scattered white ash saplings and small trees in pure stands, and plants that became infected during a 4-year observation period were not located closer to previously diseased trees than to previously healthy ones. New infections were more frequent in saplings with crowns exposed to the sky than in those with crowns not exposed (38). These observations are consistent with the hypothesis that AshY phytoplasmas are transmitted by alate insects. The possibility of transmission through natural root grafts has not been studied directly but was not indicated by spatial analyses of disease increase.

The vectors of AshY and LWB are still unknown. Leafhoppers and planthoppers are the predominant vectors of phytoplasmas (29,46). Field-collected *Paraphlepsius irroratus* (a leafhopper) and *Philaenus spumarius* (meadow spittlebug) transmitted phytoplasmas to caged ash seedlings (25). The identities of the phytoplasmas were unknown, however, and laboratory colonies of these insect species did not transmit AshY phytoplasmas under controlled conditions (37). Leafhoppers collected on sites of AshY occurrence in New York State represent nine genera with species known to be phytoplasma vectors (unpublished data). Thus, multiple species must be evaluated for a possible role in the spread of AshY.

Long-distance spread of AshY phytoplasmas could occur via windborne vectors and the shipment of diseased plants. Leafhopper vectors of other mollicutes, carried aloft on prevailing winds and then deposited, have been blamed for disease outbreaks or scattered occurrences hundreds of kilometers from the previously known ranges of the diseases (8). Diseased lilacs were shipped among lilac collections before LWB became well-known (14). AshY apparently spreads from wild sources to planted trees, as diseased green ash street trees are common in midwestern municipalities (9).

Long-distance transport of phytoplasma-infected plants can be important if insects capable of vectoring the pathogens inhabit the locality where diseased plants are sent. No information on this point exists for AshY phytoplasmas. Both ash and lilac should be considered in regulatory attempts to restrict AshY phytoplasmas to North America, and other genera of *Oleaceae* should be tested for possible susceptibility to AshY phytoplasmas.

Need and Prospects for Control

AshY and LWB are incurable at present, and no measures for direct protection of individual plants have been devised. Disease control in lilacs and ash shade trees must therefore be based primarily on disease avoidance or use of tolerant plants. Management of ash in forests and woodlots where AshY or ash decline is promi-

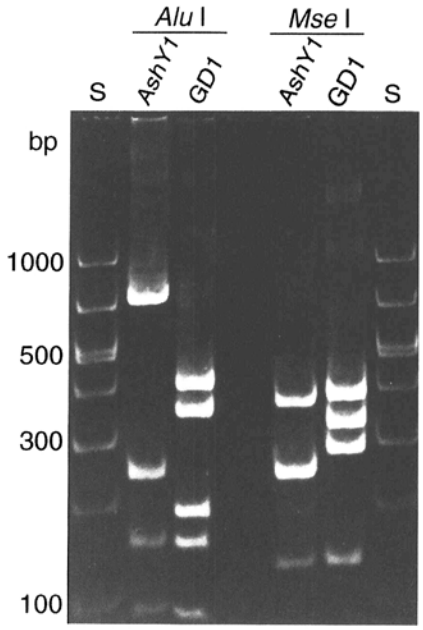

Fig. 11. Differentiation of phytoplasmas based on restriction fragment length polymorphisms in 16S rDNA. Ribosomal DNA was amplified by polymerase chain reaction using universal primers 16SF2/R2 (21) and digested with restriction endonucleases *Alu*I and *Mse*I. DNA bands were separated by electrophoresis in 5% acrylamide gel and visualized with ethidium bromide on a UV transilluminator. S = size standards, bp = base pairs, AshY1 = a strain of ash yellows phytoplasma, GD1 = a phytoplasma strain associated with stunt disease of gray dogwood (*Cornus racemosa*).

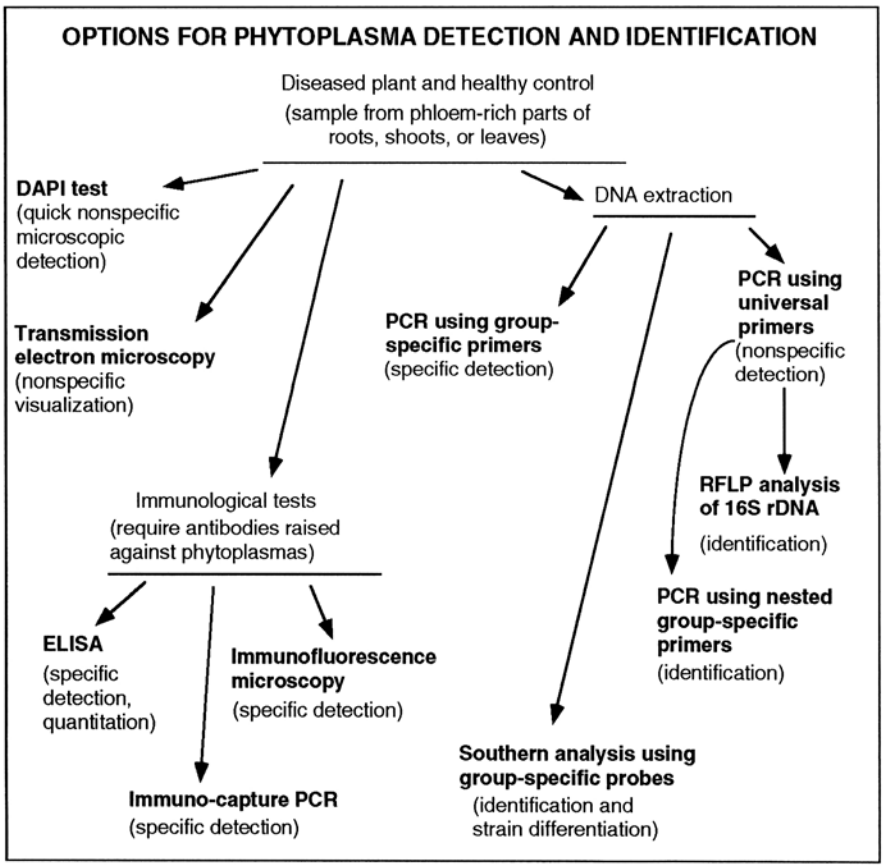

Fig. 10. Relationships among procedures used for detection and identification of phytoplasmas.

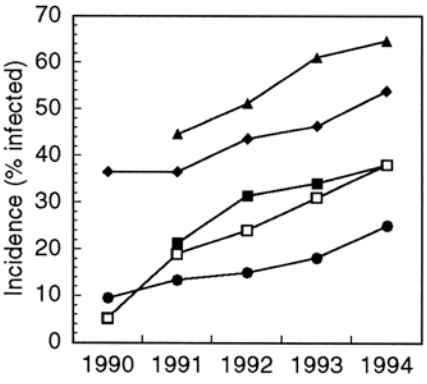

Fig. 12. Cumulative ash yellows incidence over 4 to 5 years in five young white ash populations in New York State as revealed by DAPI tests and symptom assessment. Figure adapted from reference 38.

nent should emphasize reducing the proportion of ash in the tree population.

Lilacs and ash shade trees. Propagation by seed, cuttings from healthy plants, or scions from healthy plants grafted onto seedling rootstocks ensures plants initially free from phytoplasmas, because these organisms are not transmitted in seed or on implements (14,26). Seedling rootstocks are used for many ash and some lilac cultivars. For lilacs propagated on their own roots, two approaches in concert are worthwhile: propagation from stock plants known to be healthy, and sale or planting only of tolerant cultivars. Several LWB-tolerant lilac taxa exist (14). Tolerant species or genotypes might also be useful as rootstocks for ash or lilac cultivars.

AshY in shade trees would be inconsequential if it only retarded growth. Slow growth of street trees may be desirable in some circumstances. However, current knowledge does not justify an assumption that AshY is merely a growth retardant in tolerant taxa. Although this disease has not been linked to dieback of green ash in shade-tree or forest populations (9,22), saplings of this species inoculated with AshY phytoplasmas often developed chlorosis and sustained more winter dieback than did healthy plants (unpublished data). Also, incidence of dieback was higher in phytoplasma-infected than in noninfected velvet ash in Zion National Park (40).

Ash in forests and woodlots. If decline is apparent, management to reduce the proportion of ash in the stand is appropriate (43), regardless of the cause of decline. Healthy-appearing dominant or codominant ash may be retained, because such trees could be tolerant of AshY and, even if susceptible, will not decline so rapidly as to lose value before a later harvest. Individual white ash have been observed growing at moderate rates for more than 15 years while infected with AshY phytoplasmas.

Knowledge Gaps and Prospective Applications

The most important gaps in knowledge of AshY and LWB include vector identities and ecology, variation in virulence of the pathogen and tolerance of hosts, mechanisms of pathogenicity and virulence, AshY impact in shade trees, and geographic distribution of the pathogen. Once vectors are known, their biology and host and habitat preferences can be related to disease occurrence, and this knowledge can be utilized to avoid or diminish the hazard of infection. Virulence variation in phytoplasmas is essentially unstudied; yet knowledge of this subject is necessary for evaluation of host tolerance. Cultivar testing with phytoplasma strains of known virulence is needed so that recommendations for cultivar usage in areas of AshY hazard can be based on knowledge of disease reactions. The geographic distribution of AshY phytoplasmas, whether in ash or lilac, is of interest because further expansion is clearly undesirable. The risk of long-distance transport of these organisms in latently infected lilac or ash could be evaluated by means of surveys for AshY or LWB near nurseries involved in interstate or international shipment of ash or lilac materials.

Conclusion

AshY and LWB are among the few phytoplasmal tree diseases that have been studied extensively in natural plant communities and horticultural landscapes. Relationships between AshY and LWB, and similarities between these and other phytoplasmal diseases have been revealed. Information acquired up to now provides a partial basis for management of these diseases, but a fuller understanding of interactions between the phytoplasmal pathogen and its plant and insect hosts is needed for adequate control. Although knowledge of the AshY disease system is incomplete, this system can serve as a model for tentative understanding of lesser known diseases. In particular, knowledge of the variable association between AshY or LWB and decline of host plants will promote caution in interpreting limited data about phytoplasmal infection in other woody species.

Acknowledgments

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