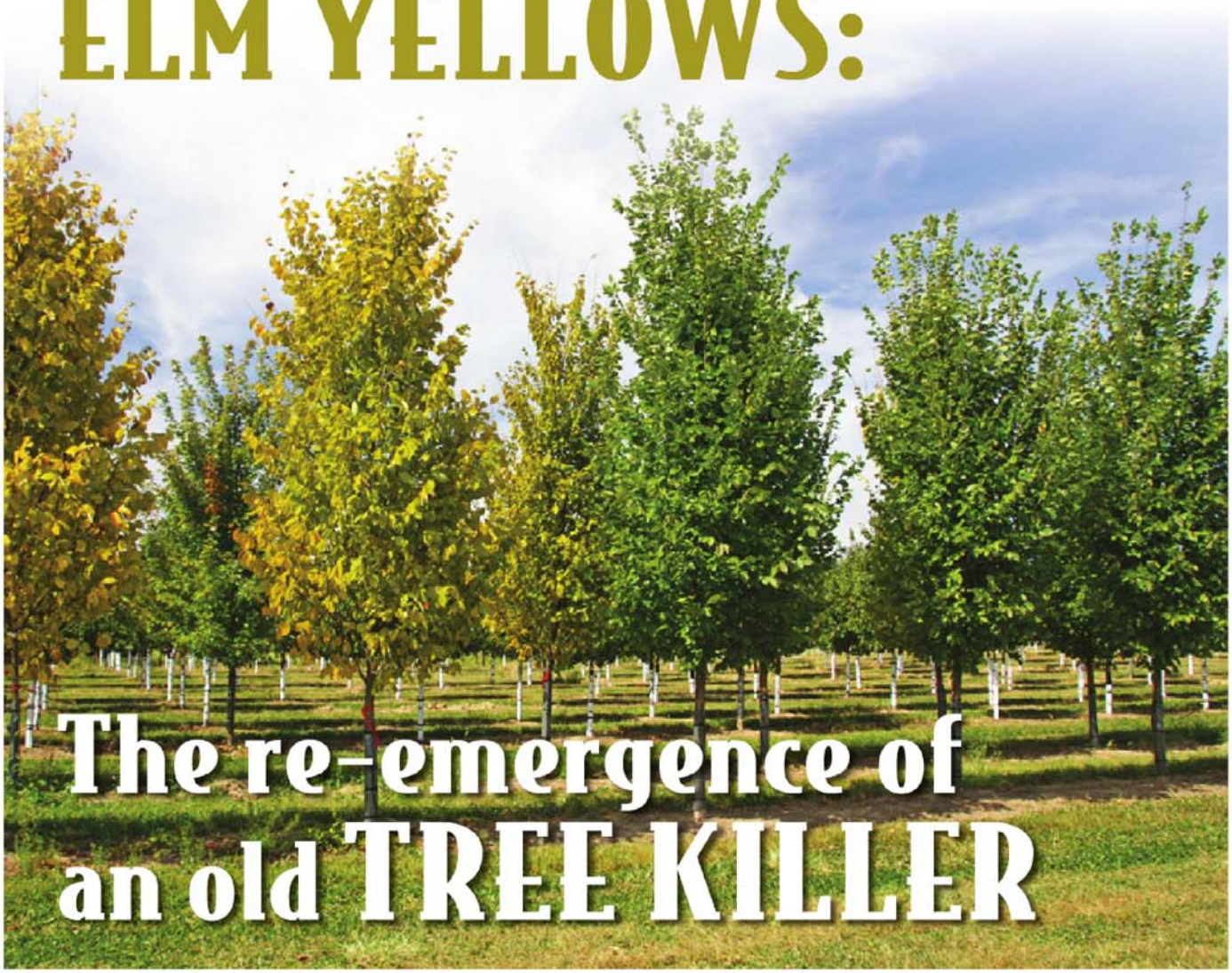
ELM YELLOWS:



The re-emergence of an old TREE KILLER

All images courtesy Joe Boggs

By Joe Boggs and Francesca Peduto Hand, PhD

The rise and fall of American elms (*Ulmus americana*) in urban forests is well-known to arborists. The story is often used as a cautionary tale illustrating the elevated risk posed by plant monocultures to support devastating pest and disease outbreaks; in this case, the over-use of a single tree species in urban plantings. Conversely, the American elm story is also used to demonstrate the value of plant diversity.

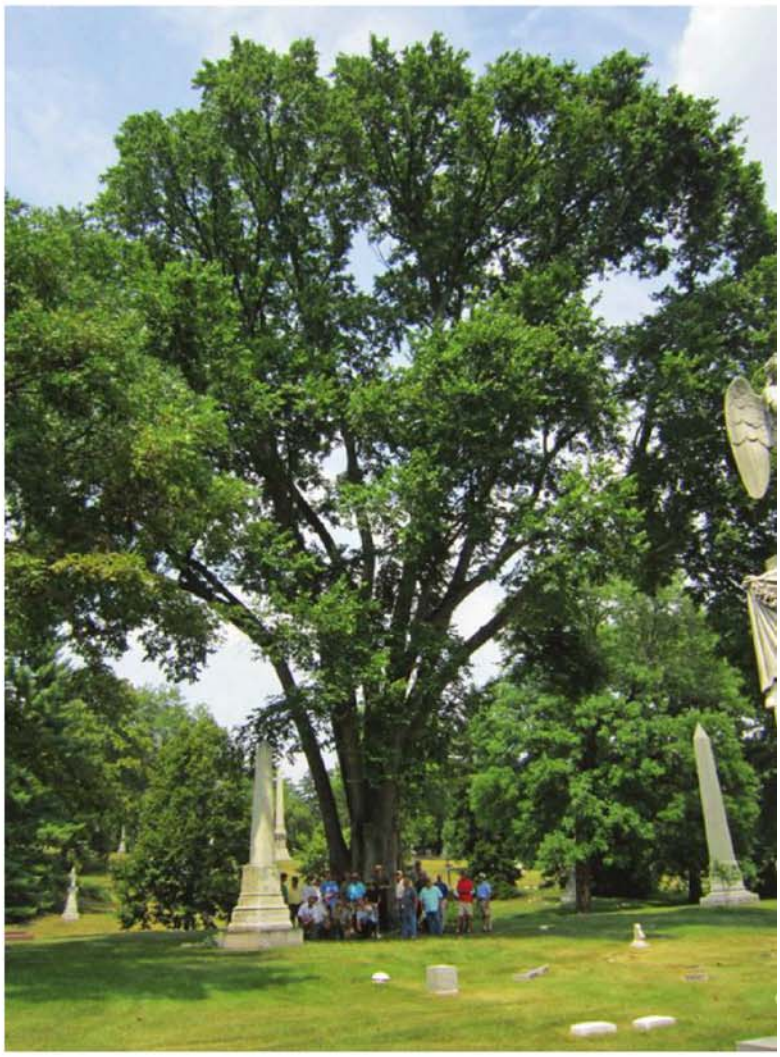
Dense, dark green foliage coupled with fast growth and a stately vase-shaped silhouette were prized characteristics that inspired the journey of this North American native from forests to city streets. The rise of American elms to become 'Canada's street tree' was further enhanced by the ability of this species to cope with a wide array of human-induced urban abuses. The pinnacle of American elm's arching trajectory as an urban tree occurred when Dutch Elm Disease (DED) was discovered in North America.

The downward spiral continued for many decades until resistant varieties and cultivars were discovered or developed. Now, American elms are making a rapid comeback; however, while these elms are resistant to DED, they are not resistant to another tree killer—elm yellows (EY).

A brief history of American Elms and DED

DED was first diagnosed on American elms in Cleveland, Ohio, in the late '20s. The disease first appeared in eastern Canada in the '40s and gradually spread westward into Ontario (1967), Manitoba (1975), and Saskatchewan (1981). DED arrived in North America from Europe; however, it was not native to Europe.

DED was first detected in Europe in 1910 when native European elms began succumbing to a mysterious malady. The causal agent was not known until 1921 when a fungus (*Ophiostoma ulmi*) associated with the elm die-off was isolated and identified by two Dutch plant pathologists, Dr. Bea Schwarz and Dr. Christine Buisman, who were working in the Netherlands at the University of Utrecht; thus, the 'Dutch' in DED. It is now known the originally identified fungus, which produces a slow death, has been largely supplanted in North America and Europe by a closely related, but much more aggressive species, *O. novo-ulmi.* It is also generally accepted that DED originated in Asia, which accounts for the resistance of most Asian elms to the disease.



American elm at Spring Grove Cemetery and Arboretum, Cincinnati, Ohio.

Research on elm resistance to DED started in Europe as soon as the causal agent of the disease was identified. Indeed, Buisman spent the remaining years of her short career researching DED, developing methods to test resistance to the fungus, and working with others to identify resistant trees. The first resistant elm selection was named (posthumously) in her honour in 1936; *Ulmus* 'Christine Buisman' is now referred to as *Ulmus procera* 'Christine Buisman.' One of the first elm cultivars found to be resistant to DED in North America was *Ulmus americana*

Emergence holes of European elm bark beetle on Valley Forge American elm.

Leaf yellowing from elm yellows versus normal leaf colour.

'Princeton.' The cultivar was actually first selected in 1922 by Princeton Nurseries in Kingston, NJ, for its landscape value; its resistance to DED was not discovered until years later, however. There is now a wide array of elms, both cultivars of American elm and hybrids between this elm and other elms, including Asian species, that are resistant to the DED fungus. Consequently, the trajectory of elms in urban landscapes is once again on the rise.

However, it is important to remember these elms are resistant to the fungus, but not to the three species of bark beetles that are responsible for spreading the fungus from tree to tree. These include: the native elm bark beetle (*Hylurgopinus rufipes*); the European elm bark beetle (*Scolytus multistriatus*); and the banded elm bark beetle (*S. schevyrewi*). In fact, as with most bark beetles, they are highly attracted to stressed trees. Place an elm that is resistant to DED under extreme stress and the tree may become riddled with bark beetle galleries and exit holes; however, the ultimate death of the tree will be from stress, not from beetles or the DED fungus.

A background on elm yellows

Elm yellows (EY) presents a similar problem; resistance to DED does not impart resistance to EY. While much has been learned about DED, the entire story behind EY remained elusive for many years. Even the discovery of the causal agent was a relatively recent event, and the taxonomy has yet to be resolved; for now the pathogen behind EY is referred to as '*Candidatus* Phytoplasma ulmi.' Much remains unknown about this pathogen including its origins.

This does not mean the disease was not recognized early on during the urbanization of

Leaf yellowing (note the lack of leaf wilting).

American elms, however. Symptoms that are now considered to be associated with EY were noted on American elms in the early 1900s.

EY is a tree-killing disease caused by a micro-organism belonging to a group of plant pathogens called phytoplasmas. These single-celled organisms were not known to exist prior to 1967 and until their discovery, EY was thought to be caused by a virus. Indeed, there are still many references in older literature to a virus causing EY. Even after its discovery, phytoplasmas remained enigmatic to microbiologists and plant pathologists. Early on, these organisms were found to share many characteristics with mycoplasmas, which belong to a class of bacteria known as Mollicutes. These extremely small bacteria lack a cell wall; 'mollis-' is Latin for 'soft,' and '-cutis' means 'skin' (*e.g.* 'cuticle'). So, phytoplasmas were once referred to as mycoplasma-like organisms (MLOs); however, mycoplasmas were most strongly associated with diseases that occur in animals such as atypical pneumonia (walking pneumonia) in humans.

The name phytoplasma was eventually applied to Mollicutes that shared several traits:

- Phytoplasmas only affect plants (= phyto-); they all produce plant diseases. Two notable examples are ash yellows and aster yellows;
- They only infect phloem tissue. In fact, EY was originally called elm phloem necrosis;
- Depending on the host, they produce a wide range of symptoms including, leaf discolouration (yellowing), production of leaf-like structures in place of flowers (Phyllody); flowers are green instead of normal floral colours; and abnormal stem growths such as rosettes, witches' brooms, etc. In fact, the symptoms can be easily confused with diseases caused by other pathogens, particularly viruses;

Cutting bark to expose the phloem.

- All phytoplasmas are spread from plant to plant by insects using their piercing/sucking mouthparts to feed on the phloem (*e.g.* leafhoppers, planthoppers, treehoppers, and froghoppers [= spittlebugs]). Indeed, while the literature generally refers to the white-banded elm leafminer (*Scaphoideus luteolus*) as the primary culprit responsible for spreading the EY phytoplasma, recent research has implicated other insect vectors, including the meadow spittlebug (*Philaenus spumarius*), the spittlebug (*Lepyronia quadrangularis*), the leafhopper (*Allygus atomarius*), and another leafhopper in the genus, Latalus; and
- Phytoplasmas also spread from infected plants to healthy plants by root-to-root grafts.

Unlike plant pathogenic fungi, thus far phytoplasmas have not been successfully cultured in a laboratory. This makes identifying these organisms more challenging

Discoloured phloem.

compared to fungi, or even some other bacteria, which cause plant diseases. However, phytoplasmas can be detected using other methods, including Enzyme-Linked Immunosorbent Assay (ELISA) and Polymerase Chain Reaction (PCR) (DNA analysis). Of course, this means a positive confirmation of EY must be made by a plant diagnostic laboratory capable of conducting these diagnostic tests.

Diagnosing EY in the field

The name elm yellows clearly describes the colour of the leaves on infected trees. Typically, new leaves become fully expanded and deep green. However, sometime in mid-to-late summer, the entire canopy rapidly turns an intense shade of yellow. The colour-change occurs without the leaves first

wilting; the yellowed leaves appear otherwise normal. The chronology of the leaf yellowing is different from leaf chlorosis caused by nutrient deficiencies. Typically, nutrient deficiency symptoms appear early in the season with new leaves appearing chlorotic. Eventually, infected trees will defoliate with the yellowed leaves dropping in late-summer to early fall.

The disease was once called 'elm phloem necrosis,' which captures the essence of the infection since the phytoplasma targets and destroys the phloem; the inner phloem becomes yellowish-brown to caramel coloured. The discolouration is generally confined to the lower portion of the trunk and the lower branches because the phytoplasma first migrates to the roots causing a rapid and substantial dieback of the fine feeder roots, then the main roots.

In essence, there is a bottom-up pattern to the infection. The discoloured phloem can be exposed on the lower branches by carefully whittling away the bark. On a cautionary note, however, elm phloem tissue will naturally become discoloured by oxidation when exposed to the air. The oxidation occurs in minutes, and mimics the discolouration caused by EY, so samples showing phloem necrosis that is suspected to be caused by EY must be fresh.

An interesting chemical reaction that produces methyl salicylate (oil of wintergreen) occurs in the phloem tissue that is colonized by the EY phytoplasma. This provides another diagnostic indicator

Early fall leaf drop.

of EY on American elm. The so-called scratch-and-sniff method of detecting the wintergreen scent involves cutting a section of bark to the white wood near the base of the main stem and placing the sample in a sealed jar. While the wintergreen scent is usually very faint at first, it becomes easily detectable after the sample has been held in the jar for approximately one to two hours.

Death from EY occurs quickly. In fact, trees that appear perfectly healthy with normal twig elongation and leaf expansion early in the growing season are often dead by the end of the season. There are no effective treatments and susceptible trees can become infected regardless of their overall health.

Arborists making a field identification of EY must differentiate between this disease and other vascular diseases of elms including *Verticillium* wilt (VW) as well as DED. Both of these diseases are caused by fungi which target the xylem; they clog the xylem shutting off the water supply to the leaves and stems.

Infected versus non-infected stems.

Oxidation discolouration on a non-infected elm.

Adding bark section to a jar to detect wintergreen scent.

Bark section held in a closed jar for one to two hours.

Leaves rapidly turn from yellow to brown and eventually become wilted.

If DED or VW disrupts water flow to newly elongating stems, the stems may curl at the tips to form a shepherd's crook. This symptom does not occur with EY. Further, both DED and VW tend to occur on sections of the tree; often only a few branches are affected at first. Although death of the entire tree may occur quickly, it most often requires years for infected trees to succumb to DED or VW.

While spraying elms to protect against the spread of the DED fungus by bark beetles has been used effectively in the past, making insecticide applications to kill the insects that spread the EY phytoplasma is problematic. First, there has been no research on the overall effectiveness of this management approach, so attempting to

Single season growth rate on an infected elm.

Yellow, wilting leaves on an infected tree next to a non-infected tree.

prevent the disease by killing the vectors would be a shot in the dark. Second, the number of insect species involved means application timing would need to be matched to varying life cycles including differences in numbers of insect generations. Finally, there is simply no way of knowing whether or not the distance between healthy and infected elms is beyond the flight ranges of the insect vectors. This means applications would need to be made preventatively, year after year.

The most effective method for controlling EY is to quickly remove and destroy infected trees. This reduces localized reservoirs of the phytoplasma pathogen, making new infections less likely.

Thankfully, EY does not appear to be following the same trajectory as DED relative to presenting a widespread threat to elms. Thus far, the disease has appeared as sporadic localized outbreaks; however, given the dire prognosis and drastic response required for control of EY, a field diagnosis should never serve as the final word on elm yellows. Stem samples should be sent to a plant diagnostic clinic for confirmation of a suspected infection.

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