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Elms Revisited

By Michael Marcotrigiano

Abstract. Until Dutch elm disease (DED) was accidentally introduced into the United States around 1930, the streets in many states were lined with American elms (*Ulmus americana*). This review highlights the aftermath of DED, and updates readers on the advances in our knowledge of the pathosystem, which consists of a tree, a fungal pathogen, and an insect vector. Conventional breeding has produced new cultivars of American elm that are more disease-tolerant, although still not resistant. Suitable DED-resistant hybrid elms have been bred using species from Europe and Asia. The discovery of diploid populations of American elm may open new opportunities in elm hybridization and genome analysis. Growing knowledge of resistance mechanisms reveals a complex interaction of anatomy, physiology, environmental factors, and tree age. The beetle's role is largely understood but appears not to be a viable point of attack in the war on DED. The genome of the fungal pathogen has been sequenced, and gene expression studies are well under way. There is a renewed interest in understanding the evolution, genetics, and physiology of the DED pathogen. The genetic engineering of elms has been demonstrated but not with the specificity and vigor as has been reported for genetically engineered American chestnut. Elm yellows, caused by a phytoplasma, are still a deadly problem for elms, although outbreaks are more regional than for DED. Germplasm resources are critical to elm improvement, and the first comprehensive survey of living elm species, hybrids, and cultivars growing in America is presented in tabular form.

Key Words: American Elm; Dutch Elm Disease; Elm; Elm Bark Beetle; Elm Yellows; Germplasm Storage; *Ophiostoma novo-ulmi*; Transgenic Tree; Tree Breeding; *Ulmus*.

THE DUTCH ELM DISEASE PROBLEM BEGINS

There was a time when an ideal street tree dominated cities and towns in the United States from the East Coast to the Midwest. Thousands of streets were named for it. With its arching, graceful habit, rapid growth rate, urban tolerance, and relatively strong wood, the American elm (*Ulmus americana*) became one of, if not the most commonly planted tree for towns, commons, parks, campuses, and cities. Saplings could be extracted from the wild and placed where they were needed. It was iconic in the American landscape and played a role in American history (Campanella 2003; Figure 1). Then, around 1930, Dutch elm disease (DED), a vascular wilt disease, was accidentally introduced to the U.S.; the pathogen was likely transported on elm logs imported from Europe for veneer. It was first discovered in Ohio but was observed soon after in New York and New Jersey (May 1934). The causal organism was determined to be the fungus *Ophiostoma ulmi* (formerly *Ceratocystis ulmi*). DED is "Dutch" not because the causal organism originates from the Netherlands (the origin is likely Asia), but because the early researchers on the pathogen were seven Dutch scientists (Holmes 1993). The pathogen was not easy to contain because an insect vector, the European bark beetle, was in the U.S. By 1960, over 40 million elms had succumbed to the disease in the U.S. (Dunn 2000). Urban elms fared worse than woodland elms. The disease quickly caused "a major environmental catastrophe in North America" (Stipes 2000). The massive tree kill caused by DED is arguably the most significant event in the history of urban forestry, as it affected the way arborists would view the planting of monocultures and how the public would view street trees and their management (Watson 2012).

This review focuses on the status of elms in the U.S. with a concentration on American elms. The main goal is to bring the reader up-to-date on research and on the availability of elm germplasm in the U.S. Enough background information is presented to set the stage for the current state of research. Only cursory information is given

where recent reviews on specific components of DED are published. While elms in America are a main focus of this review, it should be noted that even before the U.S. epidemic, other elm species in Europe and the United Kingdom were under siege by DED (Heybroek 1993a; Mittempergher and Santini 2004; Santini et al. 2008; Tomlinson and Potter 2010; Santini et al. 2012). In fact, DED was a European problem at least 20 years before it reached North America (Brasier 2000). Recently, the causal fungus for DED has been reported in Japan (Masuya et al. 2010). Research in Europe is active with regard to DED as it relates to tree physiology and breeding (Santini et al. 2008; Santini et al. 2012). In Canada, the DED fungus has received a great deal of attention (e.g., Bernier et al. 2015).

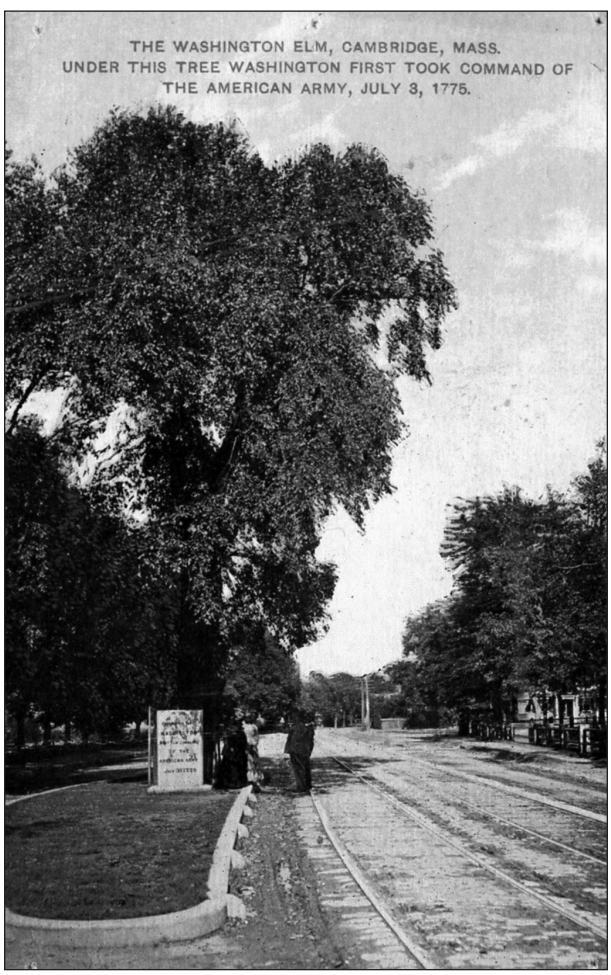

Figure 1. A photograph of the American elm (*Ulmus americana*) under which unsubstantiated claims state that George Washington first took command of the American army. It was made into a widely circulated postcard (circa 1909). This is but one example of the importance of American elms to American popular culture.

REACTION TO THE DED OUTBREAK IN THE UNITED STATES

Even though the American chestnut was decimated by a pathogenic fungus only a few years before, the reaction to the appearance of DED was slow at first and its virulence was highly underestimated both in Europe (Tomlinson and Potter 2010) and in the U.S. (Campanella 2003). In time, the impact of DED was one of the few plant-related issues that rallied the public and politicians to find a solution. Although mired in federal bureaucracy, the U.S. Department of Agriculture's Bureau of Entomology, the Works Progress Administration, the Civilian Conservation Corps, and the American Forestry Association were all enlisted to solve the problem (Campanella 2003). But the disease outpaced the funding, and in time, fueled by weather disasters that downed trees and increased vector habitat, the fight against DED became a losing battle. Then, another event, World War II, began, and with it higher priorities. It became evident that there was no chance to stop DED from changing the American landscape forever.

CONVENTIONAL BREEDING FOR ELM IMPROVEMENT

By the time the DED catastrophe took hold in the U.S., breeding programs and basic research were moving forward in the Netherlands, Italy, Spain, and to a lesser extent in Russia (Heybroek 1993b; Mittempergher and Santini 2004), using species endemic to their regions but also plagued by DED. Until the rapid spread of DED in the U.S., there was little urgency to develop new elms by trying to hybridize with other elm species. Although unusual selections arose, largely via bud sports or odd seedlings, American elm was considered a model urban tree that needed no improvement. The most current list of registered cultivar names was published over 20 years ago (Santamour and Bentz 1995). Many of these clones no longer exist, likely eliminated because they showed little tolerance to DED.

Breeders realized that the DED problem is complex, a pathosystem composed of three components: a tree, a fungus, and a fungal vector. Intangibles, like environmental stress, are also involved in disease susceptibility. Therefore, breeding elms for a specific trait might enhance one

component of tolerance, but that might never completely solve the DED problem. The term "resistance" is sometimes used when discussing DED. Resistance implies that the tree can resist infection. To date, no American elm is DED resistant. However, some are quite tolerant in that after infection they do not continue to decline, the fungus does not spread through the entire tree, and the tree has the ability to recover (Townsend 2000). Tolerance by some individuals, and the fact that young trees are often less prone to DED, assured that American elms did not disappear completely with the arrival of DED. Whether by being isolated from diseased trees and the vector, or by being more tolerant than other trees, some elms persisted. Many, however, were reduced to sprouting stumps of no aesthetic value. Others, which would eventually succumb to DED, lived long enough to produce some seed. After the initial DED outbreak, it soon became obvious that American elms were not going to become extinct, but if they would ever be a sensible choice for urban plantings, more DED-tolerant selections must be bred or isolated.

Tree breeding programs require time, commitment, space, and long-term funding. In America, the sudden demise of elms appeared to warrant these investments. That being said, only a handful of U.S. institutions attempted to tackle a very time-consuming, expensive, and complex problem. From a scientific standpoint, breeding probably commenced earlier than it should have, as so little was known about DED tolerance. Yet, there was pressure to breed new elms, since the devastation quickly mobilized urbanites, politicians, researchers, and elected officials.

Several approaches to breeding better elms were possible and were initiated. One was to breed and/or select within *Ulmus americana* by trying to isolate DED-resistant individuals. To most, this was the preferred route because the traits of American elm were unrivaled by other elms, and also because it was the American elm that was disappearing from the American landscape. A second approach would be interspecific hybridization within the genus *Ulmus* to create useful urban elms that might act as suitable substitutes for American elm. And yet another approach was to select superior individuals from within species other than *U. americana*. The latter two approaches resulted in the development of some diminutive trees that are more suitable for smaller urban spaces than American elm.

Early on, not much was known about the biology of the fungus, the insect vector, or elm defense mechanisms. More informed breeding strategies could have been made with elms if a greater understanding of DED existed at the time. With herbaceous crops, generation times are short enough for one breeder to reach an end goal relatively quickly. When American elm breeding began, there were no genetic markers nor known gene products, the mode of DED tolerance was not clear, and the generation time required an institutional, not a personal, commitment. The assumption—and it was a correct one—was that the inheritance of DED tolerance could not be coded by a single or small number of genes (Townsend 2000). Therefore, early strategies were dependent on the premise that by crossing surviving trees to each other, combining ability would result in a small subset of seedlings accumulating a larger number of "tolerance genes" than either of their parents.

In the mid-1930s, the first American elm selection program began as a cooperative venture with Cornell University and the Boyce Thompson Institute (Sinclair et al. 1974). It was extensive and long-term; 21,000 seedlings were grown out and tested. Repeated inoculations with the DED fungus occurred over a period from 1937 to 1965, with only 16 trees showing tolerance. Most of these were slow-growing and did not appear to transmit their DED tolerance in subsequent crosses and/or later contracted elm phloem necrosis (now known as elm yellows, to be discussed later in this paper). Despite the effort, none of the selections were worthy of release to the nursery industry. This lack of success discouraged funding for breeding, especially on a plant with such a long life cycle.

In the U.S., DED had marched westward by the 1950s. Reacting to widespread public concern and encouraged by state politicians, researchers at the University of Wisconsin began a research and breeding program, with student education as a focus (Guries and Smalley 1990). The program arose from a state act "to solve the Dutch elm disease problem" that was erasing landscape elms from the state. Raymond Guries, now retired

but a principal player at the time, stated (personal communication), "They [the legislators] could not have foreseen the magnitude of the problem from a scientific standpoint." The program consisted of hybridizing selected DED survivors and screening over 3,000 F1 progeny (Smalley and Guries 1993). There were 530 survivors after several years of growth and inoculation with the fungus. Continued evaluation resulted in six select trees that became known as the "American Liberty Multiclone." They were released as *Ulmus* 'American Liberty', although the "cultivar" represented six different genotypes. At the time, these selections were proposed as "acceptably tolerant" for certain uses (Guries and Smalley 1990). One of the six was eventually named and patented as 'Independence'. Releasing a multiclone seemed to be a good idea at the time as it assured that genetic diversity would persist once the trees were reintroduced (Smalley et al. 1993). In hindsight, others criticized the release of an unmarked group of trees (some of which later proved not very DED-tolerant and difficult to distinguish from one another) because it made future research on individual clones difficult, and marketing them as a group risky. In addition, there have been reports of the 'Liberty' clones being killed by elm yellows (Sinclair 2000). Although the Liberty Tree Society of the Elm Research Institute still sells them, trees of the American Liberty Multiclone have been surpassed by more recent releases that have been evaluated as more DED-tolerant (Townsend and Douglass 2001). The Wisconsin elm research program is no longer active.

Another significant American elm breeding program is at the National Arboretum of the Agricultural Research Service of the U.S. Department of Agriculture (USDA-ARS). The program has contributed three *Ulmus americana* that are adequately tolerant of DED to make them a current option for limited landscape plantings. After 20 years of research, 'New Harmony' and 'Valley Forge' were released in 1995. As with most American elms, they have a vase shape and are tolerant of air pollution and poor soil. They can be propagated by cuttings (Oakes et al. 2012). 'Jefferson', another American elm, was released in 2005 by the USDA in conjunction with the National Park Service (Hammond 2006). It was not the result of a breeding effort but had been growing for decades on the National Mall in Washington, D.C., without contracting DED.

Most recently, a few American elms found as lone survivors have been cloned and released. Southwest of Fargo, North Dakota, North Dakota State University discovered a lone survivor in a stand of American elm trees (Johnson 2014). Introduced in 2004, it was named 'Lewis and Clark' and marketed as Prairie Expedition®. It is cold hardy to USDA Hardiness Zone 3. In 2008, 'St. Croix' (Palmer 2015) and 'UASNZ' (found in New Orleans, Louisiana, and marketed as Creole Queen™) (Select Trees 2015) were released into the trade. The former is patented. The DED tolerance of 'Lewis and Clark' and 'St. Croix' is based on comparisons with wild-type American elms, but their rating among the many other DED-tolerant clones has not been published. No information on the disease resistance of 'UASNZ' is published. A noteworthy attribute for all of the above is regional adaptability, either to cold or warm climates. Perhaps inspired by these lone-survivor introductions, the University of Guelph (Guelph, Ontario, Canada) has launched a Canadian initiative to seek out, clone, and breed American elm survivors in Canada (Elm Recovery Project 2015).

The genus *Ulmus* contains about 40 recognized species (Wiegrefe et al. 1994; A.T. Whittemore, personal communication). The species are spread between Europe, Asia, and North America (Hollingsworth et al. 2000). With the advent of DED, one strategy was to attempt to move resistance from non-native species by crossing them to American elm. With the exception of American elm, all elm species are diploid ($2n = 2x = 28$), whereas American elm was, until recently, reported to be an entirely tetraploid species ($2n = 4x = 56$) (Santamour and Ware 1997). Successful hybridization (with verification) between American elm and other elms is rare (Bey 1990). Attempts at halving the chromosome number of the polyploid American elm (Lester 1971) or doubling the chromosome number of diploid elm species (Dermen and May 1966) were made in an effort to overcome the ploidy issue, but neither tactic resulted in progress in American elm breeding.

Two triploid American elms have been discovered in cultivation, which suggests that these may

have resulted from crosses between diploid and tetraploid trees. In a rather startling discovery (Whittemore and Olsen 2011), it was reported that a survey across the natural range of wild American elms indicated that over 20% of sampled American elms were diploid, not tetraploid, and in some locations the diploids overlap with tetraploid populations. While tetraploids exist throughout the natural range of American elm, the diploids were most common in the Atlantic coastal plain, Cumberland Plateau, and southern Ohio. Isolated diploids were also found in central Texas, Oklahoma, and Missouri. Since early research on American elms was centered in the northeast and upper Midwest, it is almost certain that diploids were not utilized for any American elm improvement programs. Whittemore (personal communication) thinks that the diploid and tetraploids may be cryptic species (i.e., species that appear morphologically identical but rarely, if ever, interbreed). Work using molecular markers has shown they are quite distinct genetically. Interestingly, two DED-tolerant elms that thrived on the National Mall in Washington, D.C., since the 1930s, were found to be triploid. For decades, it was assumed that the triploids (one incorrectly marketed as 'Washington' and the other correctly marketed as 'Jefferson') were unplanned interspecific hybrids between a tetraploid American elm and some diploid *Ulmus* species (Sherald et al. 1994), but a later study concluded that it was actually a triploid American elm (Pooler and Townsend 2005). When 22 cutting-propagated trees derived from the original tree were inoculated with the DED fungus, none developed systemic wilt, whereas eight of the 18 tetraploid American elms did (Sherald et al. 1994). It has not yet been investigated if this triploid could have acquired its resistance from the diploid parent.

Beginning with Asian species already known to be resistant to DED (reviewed by Smalley and Guries 2000), The Morton Arboretum in Lisle, Illinois, bred *Ulmus* species other than *Ulmus americana* to develop acceptable alternatives. Their interest in interspecific hybridization and Asian species led to the arboretum's extensive elm collection (Ware 1995). By the year 2000, The Morton Arboretum possessed 23 *Ulmus* species from China and 10 other exotic species in their collection (see Table 22.1 in Ware 2000). The goal, to reintroduce urban-tolerant elms to the nursery trade, was successful, with five unique introductions, some of which have had wider appeal and are more adapted to a variety of climates. The releases, all currently available in the trade, are 'Morton' (Accolade™), 'Morton Glossy' (Triumph™), 'Morton Stalwart' (Commendation™), 'Morton Plainsman' (Vanguard™), and 'Morton Red Tip' (Danada Charm™). Accolade received the 2012 Tree of the Year award from the Society of Municipal Arborists (Figure 2). Although the breeder, Ware, is now deceased, some of his latter hybrids remain in evaluation, but it appears that other genera (e.g., *Quercus*, *Carpinus*, and *Platanus*) are a more recent focus at The Morton Arboretum (K. Bachtell, personal communication).

The USDA-ARS/National Arboretum also bred and released to nurseries interspecific and Asian elm selections that are resistant to DED. While none have the size and silhouette of American elm, they are a viable option for cities and towns.

Figure 2. Original specimen of The Morton Arboretum, Accolade™ elm (*Ulmus davidiana* var. japonica 'Morton'), showing the general form of an American elm. Photo by Michael Marcotrigiano.

They are 'Urban' (Schreiber and Main 1976), 'Homestead' (Townsend and Masters 1984a), 'Pioneer'(Townsend and Masters 1984b), 'Pathfinder' (Spongberg 1991), 'Dynasty' (Santamour 1984), 'Prospector' (Townsend et al. 1991a), 'Frontier' (Townsend et al. 1991b), 'Ohio' (released in 1992), and 'Patriot' (Townsend et al. 1995a).

The Wisconsin program also made interspecific elm crosses. While certain hybrids performed well in DED screening, their size, form, and grace left them far behind American elm in popularity. The end result was a few cultivars that have become more popular in Europe (Eisele 2015), where some are planted extensively because Europeans "are not fixated on the American elm type" (R. Guries, personal communication). The umbrella trademark name for the Wisconsin releases is Resista®, and the cultivars are 'New Horizon' (patented in 1994), 'Rebona,' (registered in Germany in 1993), and 'Regal' (released in 1983 and sometimes available in the U.S.), all with good tolerance to DED.

One of the problems with screening elms for DED-tolerance is that testing methods have never been standardized, nor have tests proved to resemble what a tree would face in its environment (see, e.g., Tchernoff 1965; Takai and Kondo 1979). Inoculation methods vary from needle injection, drilling, slicing, and chiseling. In addition, the amount of inoculum and position on the tree has varied. It has been shown that even if beetles are used as the testing vector, their ability to infect the tree is seasonally dependent (Takai et al. 1979). Therefore, long-term field testing is vital for evaluating the disease resistance and adaptability of new elms. New clones can be useful even if they are of similar disease tolerance because they may be more regionally adapted and are therefore less DED-susceptible in a less stressful environment. Organized field trials of elms in America and Europe have been ongoing for decades and have yielded much useful information. Some testing has the intention of ranking trees according to DED tolerance (Townsend et al. 1995b; Townsend et al. 2005), while other research takes a more holistic approach and evaluates American and hybrid elms for a variety of diseases, insects, and general performance across the country (Townsend and Douglass 2004; Costello et al. 2005; Townsend et al. 2005; Jacobi et al. 2015). The French (Pinon et al. 1998), Italians (Santini et al. 2002; Santini et al. 2007; Santini et al. 2012), and Dutch (Buiteveld et al. 2015) have bred, tested, and named new interspecific elm clones that may be viable cultivars in geographic regions of the U.S. that have similar climates.

WHAT MAKES AN ELM TOLERANT OF DED?

As breeding programs were initiated to find DED tolerance, researchers also began investigating the mode of action of the fungus and the response of elms to the pathogen. In highly susceptible elms, the pathogenic fungus quickly causes occlusion of (Elgersma 1973) and embolisms in (Newbanks et al. 1983) xylem vessels and the infected branches wilt and die, with the disease spreading from the infection site throughout the tree. Root grafts can be a cause of tree-to-tree infection (Jacobi et al. 2013) and undoubtedly accelerate the spread of DED on urban streets planted as elm monocultures. Yet, the most common cause is transmission of the fungus by species of bark beetles (Brasier 2000; Webber 2000; Santini and Faccoli 2014). The fungus must enter the tree though a wound; feeding by the beetle provides that wound. The beetle carries with it spores from visits to previously infected elms, alive or dead. The fungus produces cell-wall-degrading enzymes (Bintz and Canevascini 1996; Przybyl et al. 2006), which result in cell-wall breakdown, allowing the fungal hyphae to spread more easily. The hyphae grow into xylem vessels, and eventually the fungus sporulates in a yeast-like phase.

Plants possess phenological, physical, and chemical barriers that all play a role in resistance to fungi (Li et al. 2016). It is largely accepted that resistance/tolerance of elms to *Ophiostoma* is associated with the ability of the tree to localize the pathogen (Duchesne 1993) and inhibit its growth, but how that happens has been the topic of research for decades. As with some other pathogens or wounds, the response of the tree is to attempt to compartmentalize the infected region to limit the extent of injury, which involves the formation of tyloses, cell-wall extensions that plug the xylem (Shigo and Tippett 1981). In susceptible trees, tyloses do not form quickly enough to block the prog-

ress of the fungus (D'Arcy 2000). Lignin and suberin, more resistant to fungal enzymes than cellulose, are also synthesized. A barrier wall, which remains alive, is then formed a distance from the infection (Shigo and Marx 1977).

Anatomical studies of susceptible and resistant elm species have looked for correlations between anatomical features and resistance. Early research established a statistical correlation between the length and diameter of xylem vessels and the susceptibility to DED. Vessel length and water conductivity were greater in susceptible elms (Elgersma 1970). In shorter vessels, tyloses more quickly block the vertical movement of the pathogen, thereby reducing the extent of its movement down a limb (McNabb et al. 1970). More recently, however, conclusions based on such findings have been questioned, since seasonal changes in vessel diameter and changes in vessel dimensions with branch age are not always considered and can impact the results in infection studies (Martin et al. 2013). Elms are more susceptible when the season promotes rapid vessel elongation (Santini and Faccoli 2014). Statistical correlations have been found between tree age, xylem vessel diameter, and the extent of wilting (Solla et al. 2005). It has been hypothesized that drought resistance, an environmental factor that can also cause cavitation in xylem vessels, might make a drought-resistant elm genotype less vulnerable to DED (Venturas et al. 2014). Using progeny derived from crosses between DED-susceptible and DED-resistant individuals of *Ulmus minor*, it was noted that susceptible offspring had wider and longer vessels; while xylem structure seemed to influence the spread of the pathogen, it did not prevent cavitation. After studying a series of elm hybrids with differing susceptibility, it was concluded that the structural basis of resistance to cavitation caused by the pathogen may be too multifaceted to be captured by single traits, such as vessel measurements, and therefore, more comparative work incorporating numerous hydraulic parameters is essential (Martin et al. 2013).

It would be simplistic to think that vessel anatomy alone can predict susceptibility to DED. There are DED-tolerant and DED-intolerant American elms with similar anatomy. There are species of trees with vessel anatomy similar to American elm that do not die of DED. Yet, it has been pointed out that to acquire DED the pathogen must be transmitted by a vector that is specifically attracted to elm trees (L. Bernier, personal communication). Other tree genera, therefore, may never be exposed to *Ophiostoma*. Non-host species (*Prunus pensylvanica* and *Populus balsamifera*) that were mechanically inoculated with the DED pathogen became infected, but anatomical studies showed that continuous barrier zones containing suberin and lignin developed, whereas barrier zones were discontinuous in American elm (Rioux and Ouellette 1991). The barrier zones also formed more quickly in *Prunus* and *Populus* than in American elm. Re-isolation of the pathogen from a non-host plant (Rioux and Ouellette 1989) implies that host specificity of the DED pathogen may be overstated. Bernier (personal communication) is curious as to why this lack of host specificity is not cited more often, as it suggests the possibility that DED pathogens could jump to non-elm hosts if the fungus was acquired by vectors other than elm bark beetles.

Elms are not equally susceptible to contracting DED throughout the growing season, and this may be a combination of vessel morphology and vector behavior and life cycle. In studies of elms that broke bud ("flushed") earlier than others, the early flushers were more DED resistant (Ghelardini and Santini 2009). This fact may also explain discrepancies in the ranking of elms for DED susceptibility, as the timing of resistance tests between research groups is variable, which can affect the interpretation of results or manifestation of symptoms. For example, 'American Liberty' clone 503 (Smalley et al. 1993), which displays increased DED resistance in field studies, was very sensitive to DED when inoculations were performed in controlled growth chambers after the leaves had fully expanded (Et-Touil et al. 2005).

If the cause of DED resistance were fully understood, breeding resistance into susceptible species would be more focused. As with many other pathogens, compartmentalization associated with the synthesis of lignin and suberin is a typical response of a DED-infected tree (Duchesne 1993). Lignin and suberin synthesis occur even in pathogen-infected elm tissue cultures

(Auon et al. 2009). Phytoalexins (antimicrobial substances synthesized *de novo* in plants) have been implicated in DED defense, particularly mansonone (Duchesne 1993), yet a subsequent study noted that mansonone elicitation in elm callus culture does not require the DED pathogen (Meier and Remphrey 1997), suggesting its synthesis may be a more generalized stress reaction. In one review, the extensive list (see Table 1 in Büchel et al. 2015) of chemical defense compounds isolated from elms demonstrated that a multitude of biochemicals (e.g., mansonones, cadalene derivatives, lignin, scopoletin, flavonoids) are synthesized by elms after infection and perhaps work in concert to thwart the pathogen.

Little is known about the genetic factors regulating DED resistance. It has been demonstrated that in interspecific elm hybrids, DED resistance is heritable and is linked to the amount of highly resistant *U. pumila* DNA in the hybrid. This high degree of additive resistance indicates that backcrossing strategies might be successful to move DED resistance into other backgrounds (Solla et al. 2014). Using *Ulmus minor*, an extensive genetic sequencing of the transcriptome (the entire collection of RNA sequences that allows one to determine when and where each gene is turned on or off) was performed (Perdiguero et al. 2015). By using elm genotypes with different levels of DED tolerance and exposing the trees to biotic and abiotic stress, analysis of differential gene expression between tolerant and susceptible genotypes was accomplished. By studying the upregulation of genes after inoculation of elm callus cultures with an aggressive strain of the DED fungus and performing differential screening, 53 sequences were considered upregulated, demonstrating that reaction to the pathogen causes numerous changes in gene expression in elm (Auon et al. 2010). Many genes coded for some branch in the pathway to phenylpropanoids, a broad class of biochemicals that function to elicit inducible physical or chemical barriers against infection or act as signal molecules involved in local systemic signaling for defense gene induction (Dixon et al. 2002).

It was hypothesized that the delay in the response of *Ulmus americana* cells to react to the pathogen could reflect a suboptimal coordination of defense strategies that might, in the end, fail to produce adequate resistance (Aoun et al. 2010). It had been previously reported that increases in lignin in inoculated xylem tissue occurred earlier in resistant elms than in susceptible ones (Martin et al. 2007). When tolerant and susceptible American elms were mechanically inoculated with an aggressive strain of *Ophiostoma novo-ulmi*, defense, as indicated by gene expression, occurred within 144 hours (Sherif et al. 2016). Defense molecules, such as jasmonic acid (JA) and salicylic acid, appeared to act as defense response elicitors. The tolerant elms expressed JA induction more quickly. In this, and many other studies, it appears that temporal factors (host reaction time to infection, developmental age of vessels at the time of inoculation, age of tree, etc.) are a key to determining the level of DED tolerance.

Gene expression studies could be informative in establishing which genes should be transferred or upregulated (e.g., with genetic engineering) to combat DED. A greater understanding of gene action may help researchers understand which genes play the most significant role in DED resistance.

Recently, a Spanish team demonstrated that DED-resistant and DED-susceptible clones of *Ulmus minor* reacted differently to pathogen inoculation, and the reaction to the pathogen was correlated to biochemical profiles that were analyzed after infection (Li et al. 2016). Again, the timing of the response was a significant indicator for susceptibility. After infection with the pathogen, leaf water potential and net photosynthetic rate declined, and the loss of hydraulic conductivity increased in susceptible trees. Resistant clones showed elevated levels of phenolic compounds, saturated hydrocarbons, cellulose, and hemicellulose when compared to susceptible clones. It was hypothesized that susceptible clones had a weak activation of their defense mechanisms and quickly began to display physiological parameters that could be correlated to susceptibility to DED. Quicker depletion of carbohydrate reserves were implicated in the weakened defense. Defense against DED is multifaceted. It is polygenic and temporal and includes a complex dynamic between host, vector, and pathogen. Yet, with new genetic tools, researchers are getting closer to understanding what makes an elm combat DED.

UNDERSTANDING THE ROLE OF INSECTS

The DED fungus is not transmitted as an airborne disease but rather is dependent on an insect vector. Extensive reviews of the elm bark beetle as they apply to DED have been published (Webber 2000; Santini and Faccoli 2014), so a brief treatment will suffice. Several species of bark beetles belonging to the genus *Scolytus* are the main vectors (Santini and Faccoli 2014), although *Hylurgopinus rufipes* can also transmit the disease (Bernier et al. 2015). The beetle vectors of DED carry fungal spores on the surface of their bodies. Given the lack of species specificity, it is understandable that research on the vectors of elm disease is limited, and the focus is instead placed on understanding the fungus and DED tolerance in elms. It is known that the DED fungus causes elms to release four volatile terpenes that attract elm beetles. This may increase the probability of movement to adjacent elms (McLeod et al. 2005). There is also a species of mite (*Tarsonemus crassus*) sometimes found on elm bark beetles. They carry *Ophiostoma* spores within sporothecae (pockets adapted for fungal transmission), which can increase the spore load of the beetle (Moser et al. 2010).

Stressed elms are more likely to become infected and succumb to DED, which by inference means they are more attractive to beetles. Therefore, studies are warranted to determine if other insect pests of elms are problematic and increase the probability of susceptibility to DED. Repeated defoliation can encourage woodboring insects (Miller 2000). Extensive studies indicate that the genus *Ulmus* has a plethora of insects that act as defoliators, miners, etc. (e.g., Miller 2000; Potter and Redmond 2013), and new vector species are still being discovered (Jacobi et al. 2013). Unique vectors have been recognized on elms that were introduced in the western U.S. (Lee et al. 2011). Conversely, species of elms have been evaluated for their insect resistance (e.g., Young and Hall 1986; Bosu et al. 2007; Condra et al. 2010; Potter and Redmond 2013). While few generalizations can be made, insect susceptibility within an elm species is correlated to geographic region and interactions between specific insects and different elm species. From a genetic standpoint, elm leaf beetle (*Pyrrhalta luteola*) appears to have a preference for hybrids with *Ulmus pumila* in their lineage (Miller 2000).

UNDERSTANDING THE DED FUNGI

While tree breeders bred new elms, pathologists and mycologists were trying to elucidate the biology of the DED fungus. The life cycle of fungi is brief, making genetic analysis much quicker than with trees. Since the fungus is the killer, understanding its mode of action and genetic composition is the beginning of developing a strategic plan to halt the disease. A detailed review on DED fungi has been published (Bernier et al. 2015); therefore, a brief overview and a short update will suffice.

With DED, three fungal species are known to cause the disease. All are in the genus *Ophiostoma*, a dimorphic ascomycete with a mycelium/yeast transition controlled by nutrition (Kulkarni and Nickerson 1981). The less aggressive *Ophiostoma ulmi* dominated when DED first arose (Brasier 1991). By 1940, this species was largely replaced by the more aggressive *O. novo-ulmi* (Brasier 1991). Now, two subspecies of *O. novo-ulmi* are documented (*O. novo-ulmi* and *O. novo-ulmi americana*) (Brasier and Kirk 2001). Interestingly, a third species, *O. himal-ulmi*, has been identified as a naturally occurring endophyte on elms native to the Himalayas, where the elms are largely asymptomatic. When European elms are inoculated with this third species, it is pathogenic (Brasier and Mehrotra 1995).

Ophiostoma novo-ulmi isolates have been collected from various parts of Europe and genetically analyzed. There is now evidence of hybridization between the American and Eurasian subspecies of *O. novo-ulmi*, and it has been shown that a recombination between two genes of the two subspecies of *O. novo-ulmi* can occur where the subspecies overlap in range (Konrad et al. 2002). Gene transfer is not limited within the genus. *Geosmithia* fungal species are almost always found in conjunction with *Ophiostoma*, sharing the same vectors and habitat for a significant part of their life cycle (Pepori et al. 2015). *Geosmithia* has been shown to have the cerato-ulmin gene associated with *Ophiostoma* in over 50% of the 70 strains tested, but the gene is not active in this genus (Bettini et al. 2014). That being said, the movement of this gene between fungal genera is worth consider-

ing as protocols to thwart *Ophiostoma* are developed. Clearly, fungi have the ability to evolve more quickly, and adapt to challenges, than do trees.

While several genetic loci implicated in fungal disease have been identified (e.g., Et-Touil et al. 1999), only three genes of *Ophiostoma* have been functionally analyzed. The first, cu, encodes a hydrophobin (a surface protein) known as cerato-ulmin. Early studies implied that this was a wilt toxin, and the pathogenic factor for DED had therefore been discovered (Stevenson et al. 1979). The amino acid sequence for this protein was elucidated (Yaguchi et al. 1993). However, in 1995, it was reported that isolated *Ophiostoma* mutants that failed to produce CU were as pathogenic as the CU-producing strains (Brasier et al. 1995). When a mutant of the less aggressive *Ophiostoma ulmi* was created by inserting a single copy of the *cu* gene taken from the aggressive *O. novo-ulmi*, an increase in the CU protein was detected. Nevertheless, the transformant was not more virulent. However, the overexpressor had an altered phenotype and more hydrophobic and adherent yeast-like cells. It appears that the CU protein plays a role in making the fungus more fit by protecting infectious propagules of *Ophiostoma* from desiccation and increasing their adherence to bark beetles (Temple et al. 1997). When *cu* is expressed in *O. quercus*, a nonpathogen of *Ulmus*, it influences the virulence of this normally nonaggressive species of *Ophiostoma* (Del Sorbo et al. 2000). Any gene that increases the load of yeast-like cells carried on the vector or provides an advantage during environmental stress provides an advantage for a pathogen (Temple and Horgen 2000). It has been suggested that one form of biological control would be to create a competitor for the highly pathogenic strains of *Ophiostoma novo-ulmi* by introducing strains that overproduce CU (Temple and Horgen 2000). To date, this concept has not resulted in any promising reports.

The second gene, now known as e*pg1*, is thought to play a role in the fungal colonization of xylem (Svaldi and Elgersma 1982). It codes for endopolygalacturonase, which dissolves vessel cell walls. While it was reported that aggressive isolates of *Ophiostoma* caused the release of more arabinose and xylose from cell walls of elm wood than non-aggressive strains (Svaldi and Elgersma 1982), another study using a genetically altered form of *Ophiostoma* with a targeted disruption of the e*pg1* gene suggests that e*pg1* is only partially responsible for cell wall breakdown and likely acts in concert with yet unidentified genes (Temple et al. 2009).

The third functionally analyzed gene is one related to fungal mating. With *Ophiostoma*, mating must occur between sexually compatible individuals—i.e., those having different alleles at the mating locus (Bernier et al. 2015). This mating locus (MAT1) has been used to show that interspecific gene transfer has occurred between the less virulent *O. ulmi* and *O. novo-ulmi* and to demonstrate that rapid adaptation of an invasive pathogen to new environments can occur (Paoletti et al. 2006).

Genome projects (e.g., the human genome project), while laborious and expensive, provide the most valuable genetic information for isolating functional genes of an organism. The genome of *Ophiostoma novo-ulmi* was sequenced (Forgetta et al. 2013), as was the genome of *O. ulmi* (Khoshraftar et al. 2013). Metabolic pathways were reconstructed and specific enzymes that may play a role in virulence were identified. Information such as this will be very useful if a genetic attack on DED is to be mounted. This has now begun with functional annotation research—i.e., looking at the functional characteristics of gene products, assessing the physical characteristics of genes and associated proteins, and elucidating a metabolic profile of the organism (Comeau et al. 2015). It is predicted that studies like this will allow for a better understanding of the entire pathosystem.

ELM YELLOWS: AN OLD NEW PROBLEM

Most research on elm disease has focused on DED even though elm yellows (also known as elm phloem necrosis) is more deadly. First thought to be caused by a virus and noted in the U.S. as far back as the late 1800s (Baker 1948), elm yellows is now known to be caused by a single-celled organism that belongs to a large group called phytoplasma. Phytoplasmas are bacteria-like organisms that have no cell wall, are too small to be seen with a compound microscope, and cannot be cultured *ex situ* (Pataky 1998). Although first noticed in North America, it has been proposed that the pathogen has a Eurasian origin (Sinclair 2000). The elm yellows group of pathogens is associated with disease in elm, grapevine, blackberry, cherry, peach, and others, making

it one of the most economically troubling pathogens in both North America and Europe (Lee et al. 2004). The pathogen inhabits the phloem sieve elements in stems and collapses them, causing the tree to starve (USDA Forest Service 2012). The disease is not as pandemic as DED, being more prevalent in the eastern half of the U.S. (Sinclair 2000). Elm yellows was a significant problem in the 1990s. Afterward, reports of outbreaks declined. The incidence of infection appears to be on the rise, perhaps because the urban American elm population is increasing after the introduction of DED-tolerant cultivars (Peduto-Hand et al. 2014). Unlike the case with DED, leaves on elms infected with elm yellows do not wilt and turn brown but rather suddenly turn yellow. To date, there is no practical treatment or cure.

The current taxonomic status of the elm yellows phytoplasma calls for the name '*Candidatus Phytoplasma ulmi*' (Jović et al. 2011). At least four indigenous elm species can be infected (Sinclair 2000). Some Eurasian elms appear to be tolerant or resistant (Sinclair 2000; USDA Forest Service 2012). Several experimentally infected Eurasian elms, already in the trade, did show some signs of elm yellows infection (Sinclair et al. 2000).

As with DED, the pathogen can move from tree to tree via root graft or requires insect vectors, not beetles as with DED, but leafhoppers and spittlebugs with piercing or sucking mouthparts (Peduto-Hand et al. 2014). Vectors of the phytoplasma include the leafhoppers, *Scaphoideus luteolus* and *Allygus atomarius*, and the spittlebug (*Philaenus spumarius*) (Sinclair 2000). More recently, another leafhopper genus (*Latalus* sp.) and another spittlebug species (*Lepyronia quadrangularis*) have been added to the list of vectors (Rosa et al. 2014).

Control of elm yellows consists of removing and destroying infected trees to reduce regional pathogen load. Mittempergher (2000) states that while screening for DED tolerance, testing for susceptibility to elm yellows is prudent. Genetic sequencing of the pathogen indicates that there are many subgroups within the genus that can cause the disease (Jović et al. 2011). Detecting the pathogen generally involves a DNA analysis, such as restriction fragment length polymorphism and polymerase chain reaction (Sinclair et al. 2000; Herath et al. 2010). Detection is useful for testing the resistance of different elm genotypes but not as a preventative strategy.

GENETIC ENGINEERING: A NEW ROUTE TO ELM IMPROVEMENT

Conventional methods (i.e., hybridization, screening) have significantly improved DED tolerance in American elm. Yet, the life cycle of elms is not conducive to multigenerational breeding programs, and it has taken many decades to select American elms that tolerate DED enough to make prudent use of them sensible. With advances in molecular biology, genetic and biochemical research has increased, as has knowledge of DED, its pathogen, its vector, and elm biology.

One option to combat diseases is the generation of genetically modified organisms (GMOs). With crop plants, genetic engineering for fungal resistance has been demonstrated in numerous species (reviewed by Ceasar and Ignacimuthu 2012). In this regard, analysis of American chestnut (*Castanea dentata*) research is informative to studies of American elm. According to Powell, "chestnut research has provided additional candidate genes that could be tested in elm in the future" (W.A. Powell, personal communication). American elms and American chestnuts were both devastated by an exotic fungus. In the case of chestnut, the blight is caused by the fungus *Cryphonectria parasitica*, which is a windborne pathogen that attacks the tree trunk by growing a network of mycelia that deposit oxalic acid. This eventually destroys bark and causes cankers that girdle the tree (Powell 2014). Both *Ulmus americana* and *Castanea dentata* have Asian relatives within their genus that show resistance to the respective disease, perhaps owing to their evolution with their native pathogen over countless years. Both American elm and American chestnut have undergone conventional breeding and genetic engineering. The chestnut can be bred to a blight-resistant Asian species, the Chinese chestnut (*Castanea mollissima*). The F1 hybrids are fertile and can be backcrossed repeatedly to American chestnut in an attempt to regain the American chestnut phenotype in addition to the resistance genes from Chinese chestnut. Third backcross progeny are being field tested (Pinchot et al. 2015). In contrast, it is nearly impossible to hybridize American elm with other elms. In the rare cases where a hybrid is reported, no successful backcrosses could be made (Bey 1990). The lack of a backcrossing strategy to Asian species is a great hindrance to American elm improvement.

American elm can be genetically transformed using the common bacterial vector *Agrobacterium* or by biolistics (i.e., bombardment with DNA-coated particles), has been demonstrated (reviewed by Gartland et al. 2005). Newhouse describes a method to genetically transform American elm (Newhouse et al. 2006) using tissue culture and *Agrobacterium* methodology (Bolyard and Sticklen 1993), the same general protocol used successfully to produce some GMO crop plants. It is reported that the insertion of an antimicrobial peptide, under the control of a vascular promoter from American chestnut, reduced DED symptoms (wilting and sapwood staining) after infection by *Ophiostoma novo-ulmi* (Newhouse et al. 2007). The authors admitted that the transgenic elm trees tested were too young to conclusively demonstrate stable resistance to DED. Because of limited resources, the project is now on hold (A.W. Powell, personal communication) as this lab creates and studies genetically modified American chestnut.

Progress with the genetic transformation of American chestnut is ahead of American elm (Rothrock et al. 2007; Zhang et al. 2013). Transgenic trees of American chestnut have a wheat gene introduced into them. The gene product is oxalate oxidase, which breaks down the toxic oxalic acid produced by the pathogen (Powell 2014). In transgenic American chestnut trees, the lesion length caused by the toxin was reduced to the same level as blight-resistant Chinese chestnut (Zhang et al. 2013). A much touted GMO American chestnut has been produced (Newhouse et al. 2014). Its level of blight resistance is better than wild-type chestnut but less than Chinese chestnut. Pollen from the GMO transferred resistance to the next generation. This strategy could eventually result in outcrossed seedling populations that have the resistance genes and enough background heterogeneity to maintain an acceptable level of genetic diversity in wild populations (Newhouse et al. 2014). Unlike the wheat-gene product in GMO chestnuts, the antimicrobial peptides engineered into American elms are low molecular weight proteins with extremely broad antimicrobial activities against bacteria and fungi and are not specifically targeting the DED pathogen. Approval for the release of transgenic elms would likely be more difficult than with American chestnut, where the introduced gene is more targeted to the specific pathogen.

The chestnut work already described and the regulatory issues arising from it will be informative to those planning more studies with GMO elms. In the United Kingdom, where the American elm GMO research was conducted, regulatory issues have made field testing and scaling up the release of GMO trees nearly impossible (Gartland et al. 2005). It is thought that U.S. agencies, such as the FDA, USDA, and EPA, will eventually approve the release of some GMO trees (Powell 2014).

WHAT ELM GERMPLASM IS CURRENTLY AVAILABLE?

Maintaining a diverse gene pool within *Ulmus* and within *U. americana* is necessary if elm planting is to resume in earnest, and if breeding improvements for disease resistance are to be made. Understanding the genetic composition of the numerous elm species would enhance researcher knowledge of elm disease resistance. Resistance to DED clearly exists in other species, and the discovery of diploid populations of American elm (Whittemore and Olsen 2011) will now allow many more diploid to diploid crosses to be attempted with the many diploid species that have, to date, not been used for hybridization. While reproductive barriers that are beyond ploidy may prevent interspecific hybridization with American elm (Ager and Guries 1982), genetic studies and breeding with diploids, rather than polyploids, can be advantageous (Comai 2005; Acquaah 2012).

In recent times, no list of living elm species and cultivars in America has been generated. Over two decades ago, Santamour and Bentz (1995) compiled a cultivar checklist of published names that was not intended to be a current survey of living trees. Given the efficacy of DED and the release of more attractive and resilient clones, it is likely that many *Ulmus* listed in past publications may be extinct or no longer available. For generating a list for this review, databases from germplasm storage centers, botanical gardens, and arboreta, as well as listings from commercial growers were consulted, and internet surveys were conducted to find out which elms are being grown in the U.S. The Appendix lists elms reported as living in collections and/or available

in the trade. Although the list is extensive, it may underestimate the breadth of the elm germplasm, as some smaller institutions and growers do not list their collections on the internet and/or did not respond to requests for a list of their holdings.

CONCLUDING REMARKS

In 2000, it was proclaimed that even after a half century of conventional selection and screening, there is still no DED-resistant American elm and that this is "hardly a ringing endorsement for the status quo" (Guries and Smalley 2000). Despite this proclamation, and perhaps because of the frustration at the never-ending struggle to uncover DED resistance, there appears to be a revival in the use of simple methods to reintroduce American elms. For example, researchers (Eshita et al. 2004; Hunt 2011; Slavicek and Knight 2012), nonprofits (Nature Conservancy 2016), and universities (Blanchette 2012) are promoting the idea of finding "lone survivors" or encouraging "natural selection." These strategies assume that mature elms, never prophylactically treated with fungicides and never contracting DED, are in fact genetically more tolerant than simply isolated or fortunate. It is hypothesized that some old elms may still be alive because decades of disease exposure resulted in a natural DED-screening process. Others are not so enthusiastic about a lone-survivor model for elm recovery. Apparently, it is not uncommon to find a single elm surviving by chance where many have died. Townsend (2000) estimates that only 1 in 100,000 elms shows any DED tolerance, as the thousands of survivors he has tested turned out to have escaped exposure and are not DED tolerant (reported by Becker 1996).The current, lone-survivor group of cultivars is being commercially released before any scientifically sound comparative DED-screening data is published. The press, perhaps overzealous to pronounce the return of Elm Street, has embraced the news. There is unfounded optimism concerning lone survivors because they will "bring [the urban landscape] back to its glory of tree-lined boulevards of the beautiful American Elm" (Jensen 2013). Even if these new clones are DED tolerant, there are no American elms resistant to DED, and given climate change and urban stress, any American elm can be infected by DED and decline or die. This makes the extensive use of lone-survivor clones, without the concurrent use of prophylactic fungicides, a risky proposition for those responsible for planting American elms in cities or other prominent locations where tree removal cost and landscape impact are high. Suggestions to a return to street monocultures of American elm (Jensen 2013), or any species, are ill-advised. American elms that are tolerant to DED but become infected periodically may be suited for the restoration of riparian ecosystems rather than urban plantings. Work centered in Ohio is underway to test DED-tolerant elms in the wild in the central and northeast regions of the U.S.

American elms need not disappear entirely from the streets of America. Restrained planting of tested DED-tolerant cultivars is not unreasonable, especially when trees are monitored and symptomatic trees are quickly treated or removed (Veilleux et al. 2012). Yet, there is justifiable hesitance to use tolerant rather than resistant elms in great numbers, and therefore, alternative elms are still being developed and introduced. The elegance, grace, and nostalgia of an American elm cannot be replaced by interspecific hybrids or other species that bear little resemblance to American elms. That being said, the genus *Ulmus* has much to offer. It is unlikely that the potential of other elm species would have been realized in the U.S. if it were not for the search for American elm substitutes to transfer disease resistance to American elm. Many of the interspecific hybrids are rugged urban trees deserving of a place in the American landscape. With continued breeding, the phenotype of interspecific elms may improve, and introductions with architecture closer to American elms may be developed.

Since the advent of DNA technology, much has been done to develop more accurate evolutionary relationships for angiosperms (APG III 2009). The taxonomy of the genus *Ulmus* is still being sorted out. Researchers now have genetic sequencing protocols to assist in more clearly defining phylogeny and species validity. Knowing the relationships among species will assist breeders to develop more calculated breeding strategies. While traditional breeding is essential to any elm improvement program, American chestnut and American elm genetic engineering may outpace traditional breeding methods in the race to confer to the trees complete disease

resistance to their respective pathogens. Before transgenic elms can be commercialized, however, many technical, environmental, and ethical questions need to be answered. The debate would need to take into consideration the purpose of these GMOs in comparison to agricultural crops genetically engineered for other reasons, such as herbicide resistance (Merkle et al. 2007). Will the overall stigma of GMOs prevent the introduction of GMOs of a native species like *Ulmus americana*, even if the reason for their development is attack by an invasive non-native pathogen? GMOs have always been controversial. Whether convincing or not, arguments for proceeding with the development of GMO trees exist (Strauss et al. 2001; Adams et al. 2002; Kumar et al. 2015), as does opposition (Lang 2004).

The DED problem still exists many decades after the DED first appeared. While basic research on the pathogen and elm biology is being actively pursued, many of the major players in conventional breeding are retired or deceased. Only a few research groups are still actively working on the DED pathosystem. Some of the attention to DED in the U.S. is diverted to equally troubling exotics (e.g., emerald ash borer, woolly adelgid, and Asian longhorned beetle).

There will always be American elms, but unless greater strides are made toward developing DED resistance, most American elms will not reach impressive size without regular care. Yet, it is hoped that complacency will not take over, and popular books like *The Republic of Shade* (Campanella 2003), a compelling account of American elms in American history, will continue to remind us about the importance of urban elms and the role that the American elm can continue to play in American culture. No matter how long it takes, the goal to restore the American elm as a prominent tree in the American landscape seems worthy and justified.

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Résumé. Jusqu'à ce que la maladie hollandaise de l'orme (MHO) soit introduite accidentellement aux États-Unis vers 1930, les rues de nombreux états étaient bordées d'alignements d'ormes américains (*Ulmus americana*). Cette analyse souligne les conséquences de la MHO et informe les lecteurs de la progression de nos connaissances sur les pathosystèmes, lesquels sont constitués d'un arbre, d'un pathogène fongique et d'un insecte vecteur. La reproduction conventionnelle a produit de nouveaux cultivars d'orme américain qui sont plus tolérants à la maladie, bien qu'ils ne soient pas encore résistants. Des hybrides d'ormes convenables et résistants à la MHO ont été développés en utilisant des espèces provenant d'Europe et d'Asie. La découverte de populations diploïdes d'ormes américains peut offrir de nouvelles opportunités pour l'hybridation de l'orme et l'analyse de son génome. La découverte croissante des mécanismes de résistance révèle une interaction complexe entre l'anatomie, la physiologie, les facteurs environnementaux et l'âge de l'arbre. Le rôle du scolyte est en grande partie compris, mais ne constitue pas une avenue viable dans la lutte contre la MHO. Le génome du pathogène fongique a été séquencé (ADN) et les études d'expression des gènes progressent bien. Il y a un intérêt renouvelé à comprendre l'évolution, la génétique et la physiologie de l'agent pathogène de la MHO. L'ingénierie génétique des ormes a été démontrée, mais pas avec autant de spécificité et de vigueur que ce fut le cas pour l'ingénierie génétique du châtaignier d'Amérique. La nécrose du phloème de l'orme, causée par un phytoplasme, demeure un problème implacable pour les ormes, bien que les épidémies soient plus régionales que ne l'est la MHO. Les ressources en matériel génétique sont essentielles pour l'amélioration de l'orme et la première étude complète sur les espèces, hybrides et cultivars d'orme croissant en Amérique est présentée sous forme de tableau.

Zusammenfassung. Bis die Ulmenkrankheit (DED) zufällig um 1930 in die Vereinigten Staaten eingeschleppt wurde, waren die Straßen in vielen Bundesstaaten von Amerikanischen Ulmen (*Ulmus americana*) gesäumt. Dieser Rückblick hebt die Nachwirkungen der Ulmenkrankheit hervor und informiert den Leser über die Fortschritte in unserem Wissen über das Pathosystem, welches aus einem Baum, einem fungalen Pathogen und einem Insektenvektor besteht. Konventionelle Züchtungen haben neue Kultivare der Amerikanischen Ulme hervorgebracht, die mehr krankheitstolerant, aber immer noch nicht resistent sind. Unter Verwendung von Ulmenarten aus Europa und Asien wurden brauchbare DED-resistente Ulmenhybride gezüchtet. Die Entdeckung der diploiden Populationen der Amerikanischen Ulme könnte neue Möglichkeiten in der Ulmenhybridisierung und der Genomanalyse öffnen. Wachsendes Wissen über die Resistenzmechanismen eröffnet eine komplexe Interaktion zwischen Anatomie, Physiologie, Umweltfaktoren und Baumalter. Die Rolle des Käfers ist weitgehend verstanden, aber scheint kein gangbarer Angriffspunkt bei der Bekämpfung der Ulmenkrankheit zu sein. Das Genom des fungalen Pathogens wurde sequenziert und die Studien der Genomanalyse sind derzeit im Prozeß. Es gibt erneutes Interesse am Verständnis der Evolution, Genetik und der Physiologie des Ulmenkrankheitserregers. Die genetische Entwicklung von Ulmen wurde demonstriert, aber ohne die Spezifizierung und Kraft, die für die genetisch weiterentwickelte Amerikanische Kastanie berichtet wurde. Die Ulmenvergilbung (Phoem-Nekrose, verursacht durch ein Phytplasma) ist immer noch eine tödliches Problem für Ulmen, obwohl das Auftreten mehr regional ist als bei der Ulmenkrankheit. Die Quellen von Protoplasma zur Verbesserung des Ulmengenoms sind kritisch und die erste vollständige Erhebung der lebenden Ulmenarten, -hybriden und –Kultivare, die in Amerika wachsen, sind hier in tabellarischer Form vorgestellt.

Resumen. Hasta que la enfermedad holandesa del olmo (DED) fue introducida accidentalmente en los Estados Unidos alrededor de 1930, las calles en muchos estados fueron alineadas con olmos americanos (*Ulmus americana*). Esta revisión destaca las secuelas de DED, y actualiza a los lectores sobre los avances en nuestro conocimiento del sistema patogénico, que consiste en un árbol, un patógeno fúngico y un vector de insectos. El cultivo convencional ha producido nuevos cultivares de olmo americano que son más tolerantes a las enfermedades, aunque todavía no resistentes. Los olmos híbridos resistentes a DED se han producido utilizando especies de Europa y Asia. El descubrimiento de poblaciones diploides de olmo americano puede abrir nuevas oportunidades en la hibridación del olmo y el análisis del genoma. El creciente conocimiento de los mecanismos de resistencia revela una compleja interacción entre la anatomía, la fisiología, los factores ambientales y la edad del árbol. El papel del escarabajo se entiende en gran parte, pero parece no ser un punto de ataque viable en la guerra contra el DED. El genoma del patógeno fúngico ha sido secuenciado, y los estudios de expresión génica están en marcha. Hay un interés renovado en entender la evolución, la genética y la fisiología del patógeno de DED. La ingeniería genética de olmos se ha demostrado, pero no con la especificidad y el vigor como se ha informado para el castaño americano genéticamente modificado. Los olmos amarillos, causados por un fitoplasma, siguen siendo un problema mortal para los olmos, aunque los brotes son más regionales que para el DED. Los recursos de germoplasma son críticos para la mejora del olmo y la primera encuesta exhaustiva de especies de olmos vivos, híbridos y cultivares que crecen en América se presenta en forma tabular.

Appendix I. List of *Ulmus* (*U.*) reported to be growing in the United States[z].

NAME[y]	LOCATED IN U.S.[x]
I. Elm species present in the United States (native to)	
U. alata (eastern U.S.)	ab
U. americana (eastern N. America)	ab
U. bergmanniana var. *bergmanniana* (China)	a
U. bergmanniana var. *lasiophylla* (China)	a
U. canescens (eastern and central Mediterranean)	a
U. castaneifolia (China)	a
U. changii var. *changii* (China)	a
U. changii var. *kunmingensis* (China)	c
U. chenmoui (China)	c
U. crassifolia (southeastern U.S. and Mexico)	ab
U. davidiana var. *davidiana* (Asia)	a
U. davidiana var. *japonica* (Asia)	a
U. elongata (China)	a
U. gaussenii (China)	a
U. glabra (Eurasia)	a
U. glaucescens var. *glaucescens* (China)	a
U. glaucescens var. *lasiocarpa* (China)	a
U. harbinensis (China)	a
U. ismaelis (Mexico and Central America)	c
U. laciniata (eastern Asia)	a
U. laevis (Eurasia)	a
U. lamellosa (China)	a
U. lanceifolia (Asia)	c
U. macrocarpa var. *macrocarpa* (eastern Asia)	a
U. macrocarpa var. *glabra* (China)	c
U. mexicana (Mexico and Central America)	a
U. microcarpa (China)	a
U. minor (Europe, Asia)	ab
U. parvifolia (eastern Asia)	a
U. prunifolia (China)	a
U. pseudopropinqua (China)	a
U. pumila (Asia)	ab
U. rubra (eastern N. America)	ab
U. serotina (south central U.S., northeastern Mexico)	a
U. szechuanica (China)	a
U. thomasii (eastern N. America)	a
U. uyematsui (Taiwan)	a
U. villosa (northwestern and western Himalayas)	a
U. wallichiana (Himalayas)	a
II. Interspecific hybrids with no cultivar designation	
U. × *arbuscula* (*U. glabra* × *U. pumila*)	a
U. davidiana var. *japonica* × *U. minor*	a
U. davidiana var. *japonica* × *U. pumila*	a
U. × *hollandica* (*U. glabra* × *U. minor*)	a
U. minor × *U. pumila*	a
U. 'Morton Plainsman' × *U. parvifolia*	a
U. parvifolia × *wallichiana*	a
(*U. pumila* × *U. davidiana* var. *japonica*) × *U. parvifolia*	a
U. pumila × *U. rubra*	a
U. szechuanica × *U. glaucescens* var. *glaucescens*	a

Appendix I *(continued)*

NAME[y]	LOCATED IN U.S.[x]
III. Elm cultivars	
U. alata 'Lace Parasol'	ab
U. americana 'American Liberty'	a
U. americana 'Ascendens'	a
U. americana 'Augustine'	a
U. americana 'Augustine Ascending'	a
U. americana 'Delaware'	ab
U. americana 'Fiorei'	a
U. americana 'Jackson'	a
U. americana 'Jefferson'	ab
U. americana 'JFS-Prince II' (Colonial Spirit™)	b
U. americana 'Lake City'	a
U. americana 'Lewis and Clark' (Prairie Expedition™)	ab
U. americana 'Liberty'	a
U. americana 'Lincoln'	a
U. americana 'Littleford'	a
U. americana 'Moline'	a
U. americana 'New Harmony'	ab
U. americana 'Princeton'	ab
U. americana 'St. Croix'	a
U. americana 'Survivor'	ab
U. americana 'UASNZ' (Creole Queen™)	b
U. americana 'Valley Forge'	ab
U. 'Cathedral' (*U. pumila* × *U. davidiana* var. *japonica*)	ab
U. crassifolia 'Brazos Rim'	b
U. davidiana var. japonica 'Discovery'	ab
U. davidiana var. japonica 'JFS-Bieberich' (Emerald Sunshine™)	ab
U. davidiana var. japonica 'Morton' (Accolade™)	ab
U. davidiana var. japonica 'Morton Red Tip' (Danada Charm™)	ab
U. davidiana var. japonica 'Prospector'	ab
U. davidiana var. japonica 'Validation'	a
U. 'Frontier' (*U. minor* × *U. parvifolia*)	ab
U. glabra 'Camperdownii'	ab
U. glabra 'Horizontalis'	a
U. glabra 'Lutescens'	a
U. glabra 'Nana'	a
U. glabra 'Pendula'	a
U. 'Green King' (possibly an *U. pumila* clone)	a
U. 'Hamburg' (*U. pumila* × *U. americana*)	a
U. × *hollandica* 'Bea Schwarz'	a
U. × *hollandica* 'Belgica'	a
U. × *hollandica* 'Christine Buisman'	a
U. × *hollandica* 'Commelin'	a
U. × *hollandica* 'Dampieri'	a
U. × *hollandica* 'Dampieri Aurea'	a
U. × *hollandica* 'Elegantissima'	a
U. × *hollandica* 'Groeneveld'	a
U. × *hollandica* 'Jacqueline Hillier'	ab
U. × *hollandica* 'Klemmer'	a
U. × *hollandica* 'Major'	a
U. × *hollandica* 'Modiolina'	a
U. × *hollandica* 'Pendula'	a

NAME[Y]	LOCATED IN U.S.[X]
III. Elm cultivars (continued)	
U. × *hollandica* 'Pioneer'	ab
U. × *hollandica* 'Rugosa Pendula'	a
U. × *hollandica* 'Vegeta'	a
U. × *hollandica* 'Wredei'	ab
U. 'Homestead' (*U. pumila* × *U.* 'Commelin') × (*U. pumila* 'Turkestan' × *U. minor* 'Hoersholmiensis')	a
U. 'Kansas Hybrid' (*U. pumila* × *U. americana*)	a
U. minor 'Argenteo-Variegata'	ab
U. minor 'Aurea'	ab
U. minor 'Gracilis'	a
U. minor 'Koopmannii'	a
U. minor 'Louis van Houtte'	a
U. minor 'Pendula'	a
U. minor 'Purpurea'	a
U. minor 'Sarniensis'	a
U. minor 'Umbraculifera'	a
U. minor 'Variegata'	a
U. minor 'Viminalis Aurea'	b
U. minor 'Webbiana'	a
U. minor 'Wheatley'	a
U. 'Morton Glossy' (Triumph™) (*U.* 'Morton' × *U.* 'Morton Plainsman')	ab
U. 'Morton Plainsman' (Vanguard™) (*U. pumila* × *U. davidiana* var. *japonica*)	ab
U. 'Morton Stalwart' (Commendation™) (*U. pumila* × *U. minor*) × (*U. davidiana* var. *japonica*)	ab
U. 'New Horizon' (*U. pumila* × *U. davidiana* var. *japonica*)	ab
U. parvifolia 'A. Ross Central Park' (Central Park Splendor™)	b
U. parvifolia 'Aurea'	a
U. parvifolia 'Blizzard'	a
U. parvifolia 'BSNUPF' (Everclear™)	b
U. parvifolia 'Burgundy'	b
U. parvifolia 'Catlin'	b
U. parvifolia 'Corkbark'	a
U. parvifolia 'Corticosa'	b
U. parvifolia 'D.B. Cole'	b
U. parvifolia 'Drake'	b
U. parvifolia 'Dynasty'	ab
U. parvifolia 'Easy Street'	b
U. parvifolia 'Elsmo'	ab
U. parvifolia 'Emer I' (Athena™)	b
U. parvifolia 'Emer II' (Allee™)	ab
U. parvifolia 'Emerald Prairie'	b
U. parvifolia 'Frosty'	ab
U. parvifolia 'Garden City'	a
U. parvifolia 'Glory'	a
U. parvifolia 'Golden Rey'	ab
U. parvifolia 'Hokkaido'	ab
U. parvifolia 'Hallelujah'	a
U. parvifolia 'Hibari'	a
U. parvifolia 'Hope'	b
U. parvifolia 'JFS-Barrett' (Emerald Flair™)	b
U. parvifolia 'King's Choice'	ab
U. parvifolia 'Majestic'	a
U. parvifolia 'Matthew'	ab
U. parvifolia 'Ohio'	a

Appendix I *(continued)*

NAME[y]	LOCATED IN U.S.[x]
III. Elm cultivars (continued)	
U. parvifolia 'Pathfinder'	a
U. parvifolia 'Pendens'	a
U. parvifolia 'Seiju'	ab
U. parvifolia 'Sempervirens'	b
U. parvifolia 'True Green'	b
U. parvifolia 'UPMTF' (Bosque™)	b
U. parvifolia 'Yatsubusa'	a
U. parvifolia 'Zettler' (Heritage™)	b
U. 'Patriot' (*U.* 'Urban' × *U. davidiana* var. *japonica* 'Prospector')	ab
U. pumila 'Ansaloni'	a
U. pumila 'Dropmore'	a
U. 'Regal' ((*U.* × *hollandica* 'Commelin' × (*U. minor* × *U. minor* 'Hoersholmiensis'))	ab
U. 'Rosehill' (*U. pumila* × *U. rubra*)	a
U. 'Sapporo Autumn Gold' (*U. pumila* × *U. davidiana* var. *japonica*)	a
U. 'Urban' ((*U. pumila* × (*U.* × *hollandica* 'Vegeta' × *U. minor*))	ab
U. 'Viminalis'(also *Ulmus* × *viminalis*)	a

[z] This list is divided into three sections: I, species list with native origin in parentheses; II, interspecific hybrid list; III, cultivar list. The list is likely conservative, as several inquiries to growers and nurseryman associations received no replies. The nomenclature for some species is still in debate, so some synonyms may not appear; when known, synonyms are listed in Appendix II. The valid status of elm species was determined by use of The Plant List, a collaborative venture commonly cited as an updated treatment of plant names (The Plant List. 2013. Version 1.1. Published on the Internet. Accessed December 01 2016. <www.theplantlist.org>) For species designation, conflicts with The Plant List and published literature were resolved with the help of The Flora of China. Accessed 08 November 2016. <www.efloras.org/florataxon.aspx?flora_id=2&taxon_id=10928>, and by consulting with Alan T. Whittemore (USDA-ARS National Arboretum).

[y] When the nature of a hybrid is known, it is included with the name, and the specific epithet corrected from the original publication if the name has since changed (see Appendix II). Trademark names (™) are marketing names. In cases where a trademark name is given, the cultivar name is presented before the trademark name.

[x] A location in the U.S. designation of "a" indicates a positive response by a U.S. botanical garden, arboretum, or germplasm storage facility for having this taxon listed as living in their collections; a designation of "b" indicates a commercial grower (wholesale or garden center) is growing or selling this taxon. A designation of "c" indicates that the author was unable to find a U.S. source for this valid species or botanical variety. Survey was completed in December 2015, but names were corrected until December 2016.

Appendix II. Cross-reference synonym and invalid name list in the genus *Ulmus*[z].

PUBLISHED INVALID NAME	PRESENTLY VALID NAME	REFERENCE[y]
U. americana forma *pendula*	*U. americana*	PL
U. americana var. *floridana*	*U. americana*	PL
U. androssowii	Unresolved	
U. carpinifolia	*U. minor*	GRIN
U. carpinifolia var. *cornubiensis*	*U. minor*	PL
U. carpinifolia var. *suberosa*	*U. minor*	GRIN
U. celtidea	*U. laevis*	PL
U. chinensis	*U. parvifolia*	GRIN
U. davidiana var. *mandshurica*	*U. davidiana*	PL
U. divaricata	*U. serotina*	GBIF
U. elliptica	*U. glabra*	EU
U. floridana	*U. americana*	PL
U. georgica	*U. minor*	GRIN
U. japonica	*U. davidiana* var. *japonica*	GRIN
U. laciniata var. *nikkoensis*	Unresolved	
U. × *mesocarpa*	Unresolved	PL
U. multinervosa	*U. serotina*	GBIF
U. parvifolia var. *coreana*	Unresolved	GBIF
U. procera	*U. minor*	Gil et al. (2004)
U. propinqua	*U. davidiana* var. *japonica*	GRIN
U. sieboldii	*U. parvifolia*	GRIN
U. sukaczevii	*U. glabra*	GRIN
U. taihangshanensis	*U. macrocarpa*	PL
U. tonkinensis	*U. lanceifolia*	PL
U. turkestanica	*U. pumila*	PL
U. wilsoniana	*U. davidiana* var. *japonica*	GRIN; PL

[z] Research indicated that many institutions have not updated their nomenclature, as it is common to accession plants with a "received as" designation that remains in the database. This table acts as a guide for readers who expected to see elms that do not appear in the Appendix but are unaware that, as listed elsewhere, they may exist under presently invalid names. "Unresolved" indicates the name has yet to be established to be a synonym or an accepted name.

[y] References are as follows: EU = The Information Resource for Euro-Mediterranean Plant Diversity <www.emplantbase.org/home.html>; GBIF = Global Biodiversity Information Facility <http://demo.gbif.org/species>; Gil, L., P. Fuentes-Utrilla, Á. Soto, M. T. Cervera, and C. Collada. 2004. English elm is a 2,000-year-old Roman clone. Nature 431:1053; GRIN = Germplasm Resource Information Network. <https://npgsweb.ars-grin.gov/gringlobal/taxon/taxonomysimple.aspx>; PL = The Plant List <www.theplantlist.org>.