Farinas Simmt et al: Field Resistance of American Sycamore ‘Davis’ to Canker Pathogens

Field Resistance of American Sycamore ‘Davis’ to Canker Pathogens

By Dr. Coralie Farinas Simmt, Dr. Davis Sydnor, Elizabeth L. White, Alexis Wooten, Dr. Francesca Peduto Hand, and Dr. Pierluigi (Enrico) Bonello

Abstract. American sycamores (Platanus occidentalis L.) are found in many ecosystems and planted in urban landscapes worldwide. The trees are highly susceptible to anthracnose and canker pathogens, causing leaf blight and branch dieback. On The Ohio State University campus in Columbus, Ohio, an American sycamore was observed to thrive among many symptomatic sycamores. The healthy tree, subsequently protected as cultivar ‘Davis,’ was vegetatively propagated and tested for field resistance to natural infection of canker pathogens compared to the wildtype. Incidence and severity of leaf necrosis, incidence of dieback, and tree death were evaluated for 2 consecutive seasons. The incidence of leaf necrosis was disconnected from the incidence of dieback and tree mortality, as little to no leaves were produced on the wildtype trees. By the end of the second season, 7 out of 12 wildtype trees were dead, while all 12 ‘Davis’ trees were alive. Several canker pathogens were recovered from both ‘Davis’ and the wildtype, including Apiognomonia platani and Diaporthe eres. The latter had not been previously reported on American sycamore. Pathogenicity tests confirmed that D. eres is indeed pathogenic on sycamores and also that ‘Davis’ is significantly more resistant than wildtype to canker development and should be preferred over the wildtype in the urban landscape.

Keywords. Anthracnose; Apiognomonia platani; Canker; Diaporthe eres; Disease Resistance; Sycamore.

INTRODUCTION
Platanus occidentalis L., American sycamore, is an endemic, deciduous tree species with primary distribution in Eastern North America. The trees play an important ecological role as dominant riparian species but are also a valuable timber resource (Sullivan 1994). Thanks to the striking white bark that develops higher on the bole as they age, they are also extensively used as ornamental trees in landscapes worldwide. However, this tree species is highly susceptible to disfiguration by anthracnose, leaf spot, and canker diseases. Thus, any American sycamore resistance to these ailments is highly valuable.

The causal agent of sycamore anthracnose, the ascomycete Apiognomonia platani (Lév.) L. Lombard comb. nov. (2021)[syn. Apiognomonia veneta, Discula platani] was first described on P. occidentalis in 1976 (Milne and Hudson 1987). Other species, such as Platanus hybrida Brot. or London plane (Platanus × acerifolia), are mostly planted in urban areas and are highly susceptible to anthracnose as well (Milne and Hudson 1987). The main symptoms of anthracnose include slow leaf development in the spring, leaf blight with characteristic angular-shaped necrotic lesions along the veins, stem, and twig cankers (twig blight), and irregular branching patterns due to terminal bud death (bud blight). Despite the impact that the disease has on sycamores, studying the pathogen-host system has proven challenging. Various authors have reported having difficulties infecting London planes reliably with conidia and ascospores of A. platani (Milne and Hudson 1987). In their experiment, Milne and Hudson (1987) report that only 10% of young and wounded leaves developed symptoms. To overcome this challenge, other studies relied on natural infection and achieved up to 80% infection rate (Milne and Hudson 1987).

In addition to anthracnose, several causal agents of leaf spot and canker diseases, such as Diaporthe eres Nitschke, D. medusina (Fr.) Sacc, and Diaporthe scabra Nitschke, have also been reported on London planes (Wehmeyer 1933; Grasso et al. 2012). However, Gomes et al. (2013) report that D. scabra isolated from London plane was misidentified and
should be *Diaporthe ambigua* Nitschke. *Diaporthe* is a very complex genus that is still undergoing taxonomic elucidation. Indeed, morphologically similar isolates can be genetically distinct (i.e., cryptic species), while certain species are pathogenic in some hosts and endophytic in others (Gomes et al. 2013). Because of this complexity, molecular identification of *Diaporthe* species warrants the use of a multi-locus phylogenetic approach (Udayanga et al. 2012; Gomes et al. 2013; Udayanga et al. 2014). Furthermore, Udayanga et al. (2014) developed specific primers for the DNA-(apurinic or apyrimidinic site) lyase (Apn2) gene to help identify *Diaporthe* species.

Developing tree resistance is a key strategy to protect trees against established or emerging pathogens. Tools to develop resistance range from conventional breeding and use of molecular markers to comparative genomics and metabolomics (Naidoo et al. 2019). One way to introduce new resistance genes to the gene pool is to identify naturally resistant individuals.

The Ohio State University campus in Columbus, Ohio, is home to more than 200 American sycamores (The Ohio State University Tree & Plant App 2023). In prior observations over many years, we identified a *P. occidentalis* individual that was consistently asymptomatic or showing significantly fewer symptoms of anthracnose and canker diseases than conspecifics in the immediate vicinity, suggesting that that individual may be resistant, rather than an escape. We, therefore, proceeded to vegetatively propagate the tree of interest and protected the cultivar under the name ‘Davis’ with the objective to assess the field resistance of *P. occidentalis* ‘Davis’, relative to wildtype, to canker pathogens under conditions of natural disease pressure.

**MATERIALS AND METHODS**

**Outdoor Trials**

Two trials were established on the Columbus campus of The Ohio State University on April 27, 2018, at bud break, by planting 6 4-year-old ‘Davis’ ramets, an independent member of a clone, and 6 4-year-old ramets taken from a mixture of wildtype genets (for a total of 12 ‘Davis’ and 12 wildtype ramets), under each of 2 mature American sycamores that had displayed evident anthracnose and canker symptoms for the past several years. Before the trial began, the ramets were maintained in 3-gallon pots filled with Metro-Mix® 360 growth medium coated with one tablespoon of Osmocote® 14-14-14 slow-release fertilizer. Plants were maintained in a greenhouse at a temperature of 24 °C and hand watered as needed until they reached about 1 m in height. In each trial, ramets were transplanted around the mature American sycamore tree, 1 m from the trunk, alternating between wildtype and ‘‘Davis’. The 2 trials (40.004129, −83.027528 and 40.005008, −83.027657) were protected with fences and excluded from the University’s routine groundskeeping activities throughout the experimental period. The trees were hand irrigated as needed for about 4 months post-transplant, until established.

Incidence and severity of leaf necrosis were assessed weekly or biweekly on each plant over 2 consecutive seasons from the time symptoms first appeared in the trial (i.e., July 2018 and May 2019) until the end of August. Disease incidence was expressed as percentage of symptomatic leaves on each plant, while disease severity was expressed as percentage of total foliage area with symptoms. Photos of each leaf showing necrotic symptoms were taken and uploaded to the American Phytopathological Society (APS) Assess software (Lamari 2002) to calculate the percent area of necrotic tissue.

Because severe dieback symptoms were observed on the wildtype ramets in late-June 2019, incidence of dieback was also evaluated every 2 weeks until the end of August 2019 by recording the percentage of symptomatic branches on each plant (number of branches showing dieback/total number of branches × 100).

**Fungal Isolation and Characterization**

At the end of each season (August 2018 and 2019), symptomatic tissues (i.e., necrotic leaf lesions and twig cankers) were collected from each plant and subject to pathogen isolation procedures in the laboratory. Leaves were surface disinfected in a 2% sodium hypochlorite (NaOCl) solution for 1 minute, then rinsed 3 times in sterile Milli-Q® water (Millipore Corporation, Billerica, MA, USA). Twigs were surface disinfected by uniformly spraying them with 95% ethanol followed by flaming, and then longitudinally sectioned using a sterile scalpel to expose the vascular tissue. Following disinfection, the transition zone between necrotic and healthy tissue of either leaf or vascular tissue was plated on potato dextrose agar (PDA)(Difco Laboratories, Sparks, MD, USA) amended with 1.5% streptomycin sulfate and 1%
tetrazycline hydrochloride (Fisher Scientific, Fair Lawn, NJ, USA). Plates were incubated at 25 °C under constant fluorescent white light for up to 10 days. Plates were checked daily, and any observed fungal growth was sub-cultured onto new PDA. Single spore cultures of each isolate were obtained by streaking spores onto new PDA plates and selecting one isolated germinating spore.

Total genomic DNA was extracted from pure cultures of isolates grown on PDA at 25 °C for 7 days using a Chelex extraction method (Walsh et al. 1991). All isolates were first characterized to genus by amplifying the internal transcribed spacer (ITS) (White et al. 1990) region 1 (complete) and 2 (partial) and elongation factor (EF1-α)(Carbone and Kohn 1999) gene fragments. Subsequently, to identify isolates down to species, the calmodulin (CAL)(Carbone and Kohn 1999) locus was used to identify Apiognomonia species, while DNA-(apurinic or apyrimidinic site) lyase (Apn2)(Udayanga et al. 2014) and tubulin (TUB)(Glass and Donaldson 1995) were used for Diaporthe sp. All primers and polymerase chain reaction (PCR) conditions are summarized in Table 1.

PCR products were purified using ExoSAP-IT™ (Affymetrix, Inc., Santa Clara, CA, USA) according to manufacturer’s instructions, and both strands were sequenced at the Genomics Shared Resource at The Ohio State University Comprehensive Cancer Center using an ABI Prism 3730 DNA analyzer (Applied Biosystems, Foster City, CA, USA). Sequences were assembled, trimmed, and aligned in MEGA X v.10.2.5 (Kumar et al. 2018) and single nucleotide polymorphisms (SNPs) were identified. After manual inspection of the chromatograph peaks, consensus sequences were deposited in the NCBI GenBank database (see Results for accession numbers).

**Pathogenicity Tests**

*D. eres* has never been reported on *P. occidentalis*, hence, pathogenicity tests were conducted with select isolates of *D. eres* retrieved from twig cankers. On 2021 June 8, 9 wildtype and 19 ‘Davis’ potted trees from the same populations that were used for the field trials, were subject to artificial inoculations inside a

Table 1. Amplified loci, primers, polymerase chain reaction (PCR) conditions, and references for the molecular characterization of *Apiognomonia platani* and *Diaporthe eres* isolates from this study. Apn2 (DNA-[apurinic or apyrimidinic site] lyase); TUB (β-tubulin); EF1-α (elongation factor); ITS (internal transcribed spacer); CAL (calmodulin).

<table>
<thead>
<tr>
<th>Locus</th>
<th>Primers</th>
<th>PCR conditions</th>
<th>Product size (base pairs)</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apn2</td>
<td>GCMATGTTTYGAMATYCTGGGAG</td>
<td>95 °C 1 min; (95 °C 30 s, 54 °C 30 s, 72 °C 1 min) × 40 cycles; 72 °C 10 min</td>
<td>700</td>
<td>Adapted from Udayanga et al. 2014</td>
</tr>
<tr>
<td></td>
<td>apn2fw2: CTGGTCTCCCCAGAGGTGAAC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>apn2rw2: CTTGGTCTCCCCAGAGGTGAAC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TUB</td>
<td>GGTAACCAATCGGTGCTGTCCTTC</td>
<td>94 °C 2 min; (94 °C 30 s, 55 °C 1 min, 72 °C 1 min) × 40 cycles; 72 °C 3 min</td>
<td>450</td>
<td>Adapted from Glass et al. 1995</td>
</tr>
<tr>
<td></td>
<td>Bt2a: ACCCTCAGTGTAGTACCCCTG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bt2b: ACCCTCAGTGTAGTACCCCTG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF1-α</td>
<td>CATCGAGAAGTCCGAGAAGG</td>
<td>95 °C 1 min; (94 °C 30 s, 58 °C 30 s, 72 °C 1 min) × 35 cycles; 72 °C 10 min</td>
<td>350</td>
<td>Adapted from Carbone and Kohn 1999</td>
</tr>
<tr>
<td></td>
<td>EF1-986R: TACTTGAAGGAACCCTTACC</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ITS</td>
<td>ITS4: TCCCGGATTATGATATGC</td>
<td>94 °C 2 min; (94 °C 30 s, 58 °C 1 min, 72 °C 1 min) × 40 cycles; 72 °C 3 min</td>
<td>500</td>
<td>Adapted from White et al. 1990</td>
</tr>
<tr>
<td></td>
<td>ITS5: GGAAGTAAAGTCGTAAACAAGG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAL</td>
<td>CAL-228F: GAGTTCAAGGAGGCGTCCTCCTC</td>
<td>94 °C 2 min; (94 °C 30 s, 58 °C 1 min, 72 °C 1 min) × 35 cycles; 72 °C 3 min</td>
<td>500</td>
<td>Adapted from Carbone and Kohn 1999</td>
</tr>
<tr>
<td></td>
<td>CAL-737R: CATCTTTCTGGCCATCATGG</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
research greenhouse. The unbalanced design was due to availability of plant material. Plugs of actively growing D. eres mycelium on PDA-amended or PDA-only (mock) cultures were pre-cut using a 5-mm diameter cork borer in a laminar flow hood. The trunk of each tree was then gently wiped down with 70% ethanol. The same 5-mm cork borer was flame sterilized and used to remove 3 disks of outer bark and secondary phloem, down to the xylem, at about 35, 40, and 45 cm above the soil line. The openings at 35 and 45 cm were inoculated first, with the PDA only (mock), followed by a single inoculation with the putative pathogen at 40 cm. The true inoculation was thus flanked by mock inoculations both above and below. Each inoculation site was then wrapped with parafilm. Two weeks after inoculation, the parafilm was removed and the bark was carefully scraped off at each site to reveal and measure the full lesion.

**Statistical Analyses**

All statistical analyses were conducted using the R package agricolae v. 1.2-8 (De Mendiburu 2017) in RStudio v.3.4.2 (R Core Team 2017). For the outdoor trials, 12 experimental units (6 ‘Davis’ and 6 wildtype, fixed effect) per location were randomized under a mature tree. The relative area under the disease progress curve (rAUDPC)(Madden et al. 2007) was calculated from disease incidence and severity data to account for the different trial periods. rAUDPC data were then analyzed by fixed-effects model ANOVA and means were separated using the Tukey HSD test (α < 0.05). The treatments were blocked by location (fixed effect), and the 2 years of the trial were analyzed separately.

Significance of differences in lesion lengths between wildtype and ‘Davis’ in the greenhouse pathogenicity tests was analyzed using a one-tailed t-test with α = 0.05, after removal of 3 clear outliers, including 1 wildtype and 2 ‘Davis.’ The outliers were instances in which no lesion was produced upon inoculation and were, therefore, considered failed inoculations.

**RESULTS**

**Outdoor Trials**

The incidence of leaves showing necrotic lesions in ‘Davis’ was not significantly different from the wildtype in 2018 (P = 0.99) but was significantly higher (mean rAUDPC = 38.81, 16.14, respectively) in 2019 (P < 0.001)(Table 2, Figure 1). On the other hand, the severity of leaf symptoms in ‘Davis’ (mean rAUDPC = 1.24) was significantly lower than the wildtype (mean rAUDPC = 1.99) in 2018 (P = 0.047) but was not significantly different in 2019 (P = 0.44)(Table 2).

Incidence of dieback symptoms was much lower in ‘Davis’ (mean rAUDPC = 19.75) than in the wildtype (mean rAUDPC = 66.33, P < 0.001)(Table 3). At the end of the trials (August 2019), 7 wildtype trees out of 12 were dead, whereas no P. occidentalis ‘Davis’ tree died (Figure 2).

**Pathogen Collection and Characterization**

Throughout the 2 years of the study, 2 isolates of A. platani were recovered from the wildtype (OHS3 and OHS4). To assess diversity among the isolates, we used ITS (99% identity and coverage with KX776436), CAL (100% identity and coverage with KX811102), and EF1-α (100% identity and coverage with GU353958). No nucleotide differences were

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**Table 2. Measurements of incidence and severity of leaf necrosis (recorded in 2018 and 2019) that were used to compare sycamore (Platanus occidentalis) wildtype and ‘Davis’ trees.**

<table>
<thead>
<tr>
<th>Year</th>
<th>P. occidentalis</th>
<th>Incidence (range %)</th>
<th>Mean rAUDPC of incidence</th>
<th>P-value</th>
<th>Severity (range %)</th>
<th>Mean rAUDPC of severity</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>2018</td>
<td>wildtype</td>
<td>22-54</td>
<td>15.19</td>
<td>0.99</td>
<td>3-17</td>
<td>1.99</td>
<td>a</td>
</tr>
<tr>
<td></td>
<td>‘Davis’</td>
<td>16-93</td>
<td>15.14</td>
<td>a</td>
<td>2-8</td>
<td>1.24</td>
<td>b</td>
</tr>
<tr>
<td>2019</td>
<td>wildtype</td>
<td>16-100</td>
<td>16.14</td>
<td>&lt; 0.001</td>
<td>20-32</td>
<td>0.41</td>
<td>A</td>
</tr>
<tr>
<td></td>
<td>‘Davis’</td>
<td>38-81</td>
<td>38.81</td>
<td>A</td>
<td>6-44</td>
<td>0.5</td>
<td>A</td>
</tr>
</tbody>
</table>

*Range of disease incidence and severity values were recorded at the end of the trial in August of each year.

rAUDPC = relative area under the disease progress curve. Columns represent the AUDPC values divided by the total number of days between the first and the last assessment.

Columns not connected by the same letter are statistically different according to Tukey Honest Significant Difference test (α = 0.05).
The EF1-α (100% identity and coverage with KU557619) and TUB (100% identity and coverage with MK941319) loci did not evidence any SNPs (NCBI accessions ON045366-69 and ON045357-60 respectively). These results indicate that there is some diversity among the *D. eres* isolates recovered from this study.

### Pathogenicity Tests

None of the mock inoculations developed any lesions. Lesions on ‘Davis’ were 32% smaller than on wildtype observed among *A. platani* isolates. All sequences were deposited in the National Center for Biotechnology Information (NCBI) GenBank database under accession No. ON008480-1, ON045364-5, and ON045355-6, respectively.

Additionally, 4 isolates of *D. eres* were recovered from *P. occidentalis* ‘Davis’ (OHR1 and OHR2) and the wildtype (OHS1 and OHS2). Apn2 (99% identity and 100% coverage with KJ380918) and ITS (99% identity and 100% coverage with MK942658) were the most informative loci to assess diversity among the isolates, revealing 5 and 4 SNPs respectively (NCBI accessions ON045361-3 and ON008476-9). The EF1-α (100% identity and coverage with KU557619) and TUB (100% identity and coverage with MK941319) loci did not evidence any SNPs (NCBI accessions ON045366-69 and ON045357-60 respectively). These results indicate that there is some diversity among the *D. eres* isolates recovered from this study.

### Table 3. Measurements of incidence of dieback (recorded in 2019) that were used to compare sycamore (*Platanus occidentalis*) wildtype and ‘Davis’ trees.

<table>
<thead>
<tr>
<th><em>P. occidentalis</em></th>
<th>Incidence (range %)</th>
<th>Mean rAUDPC of incidence</th>
<th><em>P</em>-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>wildtype</td>
<td>32-100</td>
<td>66.33</td>
<td>a</td>
</tr>
<tr>
<td>‘Davis’</td>
<td>5-47</td>
<td>19.75</td>
<td>b</td>
</tr>
</tbody>
</table>

*Range of disease incidence values were recorded at the end of the trial in August 2019.

rAUDPC = relative area under the disease progress curve. Columns represent the AUDPC values divided by the total number of days between the first and the last assessment.

Columns not connected by the same letter are statistically different according to Tukey Honest Significant Difference test (*a* = 0.05).
sycamore and this difference was significant ($t_{23} = 1.76$, $P < 0.038$)(Figure 3). Three inoculations did not develop disease symptoms and were hence removed from statistical analysis to compare lesion sizes between wildtype and ‘Davis’ trees.

**DISCUSSION**

In our experiments, we relied on the availability of natural inoculum and on natural infection to assess overall field disease resistance of ‘Davis’ sycamore to canker pathogens compared to wildtype sycamore. This method was preferred because several previous artificial inoculation experiments, using a variety of methods, with *A. platani* in the greenhouse had failed to induce symptom development on either *P. occidentalis* ‘Davis’ or the wildtype.

During the first year of trial (2018), ‘Davis’ showed either less or the same amount of foliar symptoms as the wildtype, depending on trial location. However, during the second year (2019), severe tree mortality was observed on the wildtype. The incidence of leaf necrosis was disconnected from the incidence of dieback and tree mortality, as little to no leaves were produced on the wildtype trees. Indeed, by the end of data assessment in August 2019, 7 out of 12 wildtype trees were dead, while all 12 ‘Davis’ trees were alive.

Cankers were observed on all trees, and *A. platani* and *D. eres* were isolated in various frequencies. To the best of our knowledge, *D. eres* has not been previously reported on *P. occidentalis* (anecdotal report of *D. eres* on *Platanus*) (Wehmeyer 1933). However, it is a pathogen known to cause dieback and cankers on many host trees (Gomes et al. 2013). Our pathogenicity test confirmed that *D. eres* is indeed pathogenic on sycamore and also that ‘Davis’ is significantly more resistant than wildtype to canker development.
CONCLUSIONS
In this study, we evaluated the field resistance of *P. occidentalis* ‘Davis,’ relative to wildtype, to *A. platani* and *D. eres*, under conditions of natural inoculum availability. After 2 years of trial, we found that more than 50% of wildtype trees had died, while all ‘Davis’ trees were still alive. Although the trial was conducted at only one location and with a small number of *A. platani* and *D. eres* isolates, results in both years of the study were unequivocal. It is certainly possible that such resistance may not be expressed equally at different locales, times, and to different pathogenic isolates, but the quantitative resistance reported here is, by its very nature, likely to hold under a variety of circumstances. On this basis, we suggest that *P. occidentalis* ‘Davis’ should be preferred in urban landscapes over the wildtype. At the very least, given its promise, *P. occidentalis* ‘Davis’ could be tested more widely for validation under a variety of conditions.

In the future, additional research looking at genetic differences between *P. occidentalis* ‘Davis’ and the wildtype could help identify resistance markers, including by way of chemometric analysis of infrared absorbance/reflectance signals via statistical or machine learning-based classification (Conrad et al. 2014; Villari et al. 2018; Mukrimin et al. 2019; Conrad et al. 2020; Fearer et al. 2022). Such markers could be exploited to fast-forward breeding programs aiming at incorporating canker and dieback disease resistance into sycamore trees.

LITERATURE CITED


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Conflicts of Interest:  
Drs. Farinas Simmt, Sydnor, Peduto Hand, and Bonello report filing an invention disclosure with The Ohio State University technology commercialization office.

Résumé. Les platanes occidentaux (Platanus occidentalis L.) sont présents dans de nombreux écosystèmes et plantés dans les milieux urbains du monde entier. Ces arbres sont très susceptibles à l’anthracose et aux agents pathogènes chancreux qui provoquent la brûlure des feuilles et le dépérissement des branches. Sur le campus de l’université Ohio State à Columbus, Ohio, un platane occidental a été observé comme prospérant parmi de nombreux sycomores symptomatiques. L’arbre sain, considéré par la suite sous le nom de cultivar ‘Davis’, a été multiplié par voie végétative et testé pour sa résistance à l’infection naturelle par des agents pathogènes chancreux par rapport au phénotype. L’incidence et la sévérité des nécroses foliaires, l’incidence du dépérissement et la mortalité des arbres ont été évaluées pendant deux saisons consécutives. L’incidence de la nécrose foliaire était sans rapport avec l’incidence du dépérissement et la mortalité des arbres, car les arbres phénotypes ne produisaient que peu ou pas de feuilles. À la fin de la deuxième saison, 7 des 12 arbres phénotypes étaient morts, tandis que les 12 arbres ‘Davis’ étaient vivants. Plusieurs agents pathogènes du chancre ont été retrouvés à la fois sur ‘Davis’ et sur les phénotypes, notamment Apiognomonia platani et Diaporthe eres. Ce dernier n’avait pas été signalé auparavant sur le platane occidental. Les tests de pathogénicité ont confirmé que D. eres est effectivement pathogène sur les platanes et que ‘Davis’ est significativement plus résistant que le phénotype au développement du chancre et devrait être préféré au phénotype en milieu urbain.


Resumen. Los sicomoros americanos (Platanus occidentalis L.) se encuentran en muchos ecosistemas y se plantan en paisajes urbanos de todo el mundo. Los árboles son altamente susceptibles a la antracnosis y los patógenos del chancro, causando el tizón de las hojas y la muerte regresiva de las ramas. En el campus de la Universidad Estatal de Ohio en Columbus, Ohio, se observó que un sicomoro americano prosperaba entre muchos sicomoros sintomáticos. El árbol sano, posteriormente protegido como cultivar ‘Davis’, se propagó vegetativamente y se probó la resistencia en campo a la infección natural de patógenos cancros en comparación con el tipo silvestre. La incidencia y la gravedad de la necrosis foliar, la incidencia de muerte regresiva y la muerte del árbol se evaluaron durante 2 temporadas consecutivas. La incidencia de necrosis foliar se desconectó de la incidencia de muerte regresiva y mortalidad de árboles, ya que se produjeron pocas o ninguna hoja en los árboles de tipo silvestre. Al final de la segunda temporada, 7 de los 12 árboles salvajes estaban muertos, mientras que los 12 árboles ‘Davis’ estaban vivos. Se recuperaron varios patógenos del cancro tanto de ‘Davis’ como del tipo salvaje, incluidos Apiognomonia platani y Diaporthe eres. Este último no había sido reportado previamente en sicomoro americano. Las pruebas de patogenicidad confirmaron que D. eres es realmente patógeno en sicomoros y también que ‘Davis’ es significativamente más resistente que el tipo silvestre al desarrollo de chancros y debe ser preferido sobre el tipo silvestre en el paisaje urbano.