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Survey of *Corylus* Resistance to *Anisogramma anomala* from Different Geographic Locations

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***Abstract. Anisogramma anomala* (Peck) E. Müller is the causal agent of the disease eastern filbert blight (EFB) of hazelnuts (*Corylus* spp.). Little is known of its genetic diversity and pathogenic variation. Most sources of host resistance have been identified in the Pacific Northwest, a region outside the native range of *A. anomala* believed to have limited diversity of the fungus due to a long history of quarantine and its relatively recent inadvertent introduction. In an attempt to investigate the pathogenic variation of *A. anomala*, 12 hazelnut genotypes that showed complete resistance in Oregon were inoculated with 12 isolates collected from across its native range. At the conclusion of the study, 'Grand Traverse,' 'Ratoli', OSU 541.147, OSU 495.072, and OSU 526.041 remained free of disease. 'Closca Molla', OSU 759.007, and OSU 587.044 were infected by most isolates. 'Gasaway' was infected by the Michigan isolate, which was also the only one to infect its offspring 'Zimmerman', although the lesion lacked sporulating stromata. Interestingly, 'VR20–11', another offspring of 'Gasaway', was infected by isolates from New Jersey, Minnesota, and Michigan. The Michigan isolate also caused the only signs of infection on OSU 408.040.**

Anisogramma anomala (Peck) E. Müller is the incitant of the disease eastern filbert blight (EFB), which causes severe cankering, branch dieback, and the death of most European hazelnuts, *Corylus avellana* L. It is an obligate biotrophic pyrenomycete native to a wide geographic area east of the Rocky Mountains where it is found associated with its much more tolerant natural host, *Corylus americana* Marshall (Fuller, 1908; Weschcke, 1954; Farr et al., 1989; Johnson and Pinkerton, 2002). *Anisogramma anomala* is known to reproduce only by ascospores and has a multiyear lifecycle that requires the host plant to cycle through a dormancy period after infection to express disease symptoms (Stone et al., 1992; Pinkerton et al., 1993). EFB is believed to be the primary reason commercial hazelnut orchards were never successfully established in the eastern United States (Barss, 1921; Thompson et al., 1996). Alternatively, hazelnut production thrived in western Washington and Oregon due to being outside the native range of *A. anomala*, as well as having a climate well suited for European cultivars (Thompson et al., 1996). Currently, the top hazelnut-producing country in the world is Turkey, which generally produces 60% to 70% of the world's crop (world total was 776,890 tons in 2007). Turkey is followed by Italy, which produces around 17% of the world's total, and then the United States, which produces less than 5% (FAOStat, 2009). Ninety-nine percent of the United States hazelnut crop is produced in the Willamette Valley of Oregon (Mehlenbacher and Olsen, 1997).

The destructive nature of EFB was known and quarantine laws were established in the early 1900s to prevent its introduction into the western United States (Barss, 1921; Lagerstedt, 1979). Despite these precautions, EFB was discovered in a commercial orchard in southwest Washington in the late 1960s (Davison and Davidson, 1973). Since then, it has spread southward throughout the entire Willamette Valley of Oregon where it threatens the long-term viability of the U.S. hazelnut industry (Mehlenbacher, 2005). EFB control measures have been developed, including fungicide sprays and therapeutic pruning; however, they are expensive, yield-reducing, and not entirely effective (Johnson et al., 1996; Julian et al., 2008). Therefore, the development of cultivars with genetic resistance to the pathogen appears to be the most effective means for control (Mehlenbacher, 1994). Breeders at Oregon State University (OSU; Corvallis, OR) have been working on this objective since 1976, when the first controlled pollinations were made with the obsolete pollinizer 'Gasaway'. 'Gasaway' was shown to carry a dominant allele at a single locus that confers complete resistance to EFB (Mehlenbacher and Thompson, 1991a). Since its discovery, the 'Gasaway' source of resistance has been widely used in the OSU hazelnut breeding program, leading to the release of EFB-resistant pollinizer cultivars (Mehlenbacher and Thompson, 1991b; Mehlenbacher and Smith, 2004) and cultivars with kernel quality suitable for commercial production (Mehlenbacher et al., 2007, 2009).

Although the 'Gasaway' allele continues to provide a high level of EFB resistance in the Pacific Northwest (PNW), breeders and plant pathologists are concerned with the long-term durability of using only one source of single-gene resistance (Osterbauer, 1996; Coyne et al., 1998; Pinkerton et al., 1998; Lunde et al., 2006). Adding to this concern is the question whether the genetic diversity of *A. anomala* found in the PNW is less than that found across its native range because *A. anomala* in the PNW is believed to trace back to a single point introduction in southwest Washington (Gottwald and Cameron, 1980; Johnson et al., 1996). Based on the pathogen's wide native range and sexual reproduction, it is likely that genetic diversity and pathogenic variation exists in the species. Consequently, *A. anomala* may exist outside the PNW with the ability to overcome 'Gasaway' resistance. In an attempt to investigate this question, Osterbauer (1996) subjected trees of VR6–28, an OSU selection containing the 'Gasaway' allele, to greenhouse inoculations with *A. anomala* collected from across the eastern United States and Canada. At the conclusion of her experiment, none of the isolates were able to incite typical EFB on VR6–28. However, concern was raised when isolates from Minnesota and Ontario caused development of sunken non-sporulating lesions. While these lesions did not form the conspicuous "football-shaped" stromata typical of the fungus, their presence suggested possible variability in the pathogen. Thus, Osterbauer's findings reinforced the need to maintain quarantine regulations in the PNW and to continue the search for additional sources of genetic resistance to *A. anomala*.

Lunde et al. (2006) investigated the inheritance of EFB resistance in progeny derived from 'Zimmerman', which carries the 'Gasaway' resistance allele (Gökirmak et al., 2009). Along with reporting that 'Zimmerman' typically confers resistance to progeny in a 3 resistant:1 susceptible ratio, which is different from the 1:1 ratio observed in progeny of 'Gasaway', they found several seedlings that expressed small sunken lesions, but with no sporulation. These seedlings amplified the RAPD markers UBC152–800 and UBC268–580 that are closely linked to the 'Gasaway' allele (Mehlenbacher et al., 2004), signifying its presence. Their findings suggest that although progeny may not be "completely resistant" or "immune" as previously reported, 'Gasaway' and 'Zimmerman' still transmit a very high level of resistance to their offspring. In addition, their work suggests that further investigation is needed to elucidate the response of the 'Gasaway' allele when expressed in different genetic backgrounds.

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Since the discovery of 'Gasaway', additional sources of resistance to *A. anomala* have been identified at OSU, including several *C. avellana* cultivars and selections as well as other *Corylus* species and interspecific hybrids (Coyne et al., 1998, 2000; Lunde et al., 2000; Chen et al., 2005, 2007; Sathuvalli, 2007). However, these new sources were identified by challenging them with *A. anomala* originating in the PNW, and their response to isolates originating across the pathogen's native range is largely unknown. The objectives of this study were to develop a better understanding of *A. anomala*'s pathogenic variation and to assess the broader response of available sources of host resistance by challenging a diverse group of cultivars and selections shown to be completely resistant in Oregon with *A. anomala* isolates collected across the pathogen's native range.

Materials and Methods

Twelve hazelnut genotypes shown to express complete resistance to *A. anomala* in Oregon, comprised of six named cultivars and six OSU breeding selections (Table 1), were inoculated with *A. anomala* isolates collected across the pathogen's native range (Table 2). Five susceptible cultivars were included as controls (Table 1). In general, three replications of each genotype were challenged in 12 separate disease inoculation treatments (six in 2003 and six in 2004) and their responses were evaluated after cycling through two periods of dormancy.

Dormant rooted layers and scion wood were provided by OSU or the USDA Agricultural Research Service National Clonal Germplasm Repository (Corvallis, OR). Grafting was performed at Rutgers February through March of 2003 and 2004. Plants were potted in 3.7-L plastic containers using a peat-based planting medium (Promix® BX; Premier Horticulture, Rivière-du-Loup, Québec), top-dressed with 5 g of 5- to 6-month release fertilizer (Osmocote Plus $15N–9P_2O_5–12K_2O$ with micronutrient; The Scotts Co.), and were grown in a greenhouse maintained at 24 °C day/18 °C night. After 7 to 8 weeks, actively growing plants were moved into humidity chambers for inoculations. Following inoculations, plants remained in the greenhouse until early August, were then moved outside under shade for 3 weeks, and then were planted in the field at the Rutgers University Vegetable Research and Extension Farm (North Brunswick, NJ) for subsequent evaluations.

Each isolate consisted of populations of intact hazelnut stems containing EFB cankers collected from different geographic locations (Table 2). Stems were obtained in the winters of 2003 or 2004 and were stored at –20 °C in polyethylene bags until needed. Mixtures of several isolates were used for treatments D-04 PA2 and E-04 MA/NY to provide an adequate amount of inoculum. Ascospore suspensions were prepared as described by Johnson et al. (1994).

Individual inoculation chambers were constructed in the greenhouse for each treatment, with six performed in 2003 and six in 2004 (Table 2). Chambers consisted of 2.13 × 1.83 × 2.13 m bamboo frames covered with 4-mil (0.1-mm) polyethylene sheeting. Each contained one humidifier (Vicks Cool Mist Humidifier, Model V400; Kaz Inc., Hudson, NY) run as needed to maintain relative air humidity near 100% for the inoculation period. Replicates of each genotype were randomly distributed in each chamber. Only actively growing plants were used in the inoculations, as spores of *A. anomala* were shown to infect only newly developing tissues (Stone et al., 1992; Johnson et al., 1994). Fewer plants were available of some genotypes because plants had ceased growing before inoculation.

Ascospore suspensions were applied to newly expanding shoot tips and the most recent 8 to 10 cm of growth by spraying until runoff with a hand-held pump sprayer (approximately 10-15 mL per plant per application). Inoculations were performed in the early evening in late April or May and were performed two times for each treatment in 2003 and two or three times in 2004, based on

Table 1. Hazelnut (*Corylus*) genotypes evaluated for response to infection by *Anisogramma anomala* from different geographic locations. Resistant genotypes showed no signs or symptoms of infection by *A. anomala* in evaluations performed at Oregon State University (Corvallis, OR) at the initiation of this study. All genotypes are *C. avellana* unless otherwise noted.

Resistant genotypes	Origin and/or parentage where available
'Gasaway'	WA, USA (Mehlenbacher and Thompson, 1991a)
'VR20–11'	OR, USA; ('Barcelona' × 'Compton') × 'Gasaway' (Mehlenbacher and Thompson, 1991b)
'Zimmerman'	OR, USA; 'Barcelona' × 'Gasaway' (Lunde et al., 2006; Gökirmak et al., 2009)
'Ratoli'	Spain (Lunde et al., 2000)
'Closca Molla'	Spain (Lunde et al., 2000)
'Grand Traverse'[z]	MI, USA; 'Faroka' (*C. colurna* × *C. avellana*) × *C. avellana* (Farris, 1989; Lunde et al., 2000)
OSU 408.040	MN, USA; Weschcke seedling, OSU breeding selection (Chen et al., 2005)
OSU 759.007	Georgia; OSU breeding selection (Sathuvalli, 2007)
OSU 495.072	Russia; OSU breeding selection
OSU 541.147	OSU breeding selection; 'NY110' (*C. americana* 'Rush' × *C. avellana* 'DuChilly') × OSU 226.118
OSU 526.041	OSU breeding selection; *C. heterophylla* 'Ogyoo' (Korea) × *C. avellana*
OSU 587.044	OSU breeding selection; *C. californica* B0509 × OSU 278.113 ('Tombul Ghiaghli' × INRA H 105–28)
Susceptible genotypes	
'Ennis'	WA, USA; 'Barcelona' × 'Daviana'
'Daviana'	United Kingdom
'Barcelona'	Spain
'Montebello'	Italy
'Tonda di Giffoni'	Italy

[z]The parents of 'Grand Traverse' (self-incompatibility alleles S11 S25) are reported as 'Faroka' × 'Royal' (S1 S3) by Farris (1989). 'Faroka' is believed to be a hybrid of *C. colurna* and *C. avellana* and its phenotype is consistent with this proposed parentage. However, because neither the allele S1 nor S3 is present in 'Grand Traverse', the parentage reported by Farris is unlikely. However, the phenotype of 'Grand Traverse' does support that it is a backcross of the interspecific hybrid 'Faroka' to some unknown *C. avellana* (Lunde et al., 2000).

Table 2. Origin of *Anisogramma anomala* isolates and greenhouse inoculation dates.

Isolate ID	Isolate origin	Inoculation dates
A-03 NJ	Rutgers Fruit Research and Extension Center, Cream Ridge, NJ	5/08/03 and 5/12/03
B-03 MN1	Badgersett Research Corporation, Dayton, MN	5/08/03 and 5/12/03
C-03 MI	University of Michigan, East Lansing, MI	5/08/03 and 5/12/03
D-03 NY	Amherst, NY	5/09/03 and 5/13/03
E-03 MN2	Wykoff, MN	5/09/03 and 5/13/03
F-03 OR	Oregon State University, Corvallis, OR	5/09/03 and 5/13/03
A-04 NJ	Rutgers Fruit Research and Extension Center, Cream Ridge, NJ	4/20/04, 4/26/04, and 5/03/04
B-04 NY	Olson Tree Farms, Findley Lake, NY	4/20/04, 4/26/04, and 5/03/04
C-04 PA1	Morris Arboretum, Philadelphia, PA	4/20/04 and 4/26/04
D-04 PA2	Gettysburg and Harrisburg, PA mixed isolate populations	4/21/04, 4/27/04, and 5/03/04
E-04 MA/NY	Arnold Arboretum, Boston, MA and Amherst, NY mixed isolate populations	4/21/04 and 4/27/04
F-04 OR	Oregon State University, Corvallis, OR	4/21/04, 4/27/04, and 5/04/04

availability of inoculum (Table 2). Plants remained in the humidity chamber for 7 days following the final inoculation, with humidity levels gradually reduced over the final 4 days.

Evaluation of disease response began ≈20 months after inoculations by visual inspection of plants for presence or absence of cankers. Genotypes were considered susceptible to infection by an isolate if at least one replication developed EFB cankers. Plants that expressed sunken, nonsporulating lesions similar to those described by Osterbauer (1996) and Lunde et al. (2006) were also recorded. Canker lengths were recorded for each infected plant. Because it was reported that some *A. anomala* infections require an additional overwintering period for disease expression (Stone et al., 1992; Pinkerton et al., 1993), final evaluations were performed 32 months after inoculations. Cankers found on lateral branches were considered secondary infections and thus were not attributed to greenhouse inoculations.

Results and Discussion

At final evaluations, 'Grand Traverse', 'Ratoli', OSU 541.147, OSU 495.072, and OSU 526.041 expressed no signs or symptoms of EFB across all treatments (Table 3). 'Zimmerman' and OSU 408.040 developed sunken, incomplete lesions on one replication each, with no signs of typical EFB. The remaining five "resistant" genotypes had a total incidence of disease ranging from 2 of 36 (5.6%) plants expressing EFB for 'Gasaway' to 15 of 24 (62.5%) plants expressing EFB for 'Closca Molla' (Table 3). Nearly all trees of the susceptible control plants were infected by all isolates (Table 3). In general, average canker lengths of infected "resistant" genotypes were smaller than those of the control genotypes; however, direct comparisons between the control plants and the "resistant" genotypes cannot be made with confidence due to infected stems of most control plants dying from EFB before the 32 month evaluations (Table 3).

All isolates challenging 'Closca Molla' were able to incite EFB on at least one tree, except C-04 PA1. All isolates except D-04 PA2 incited EFB on OSU 587.044. While in 2003 the number of trees was limited for OSU 759.007 (A-03 NJ incited EFB on one of the two plants available), its presence in 2004 was more complete, and all except E-04 MA/NY incited EFB on at least one tree.

Overall, C-03 MI incited EFB on a greater number of genotypes than any other treatment, providing strong evidence that pathogenic variation exists in *A. anomala* (Table 3). In addition to causing the only sign of infection on OSU 408.040, it was the only isolate to incite a typical sporulating lesion on 'Gasaway' and sunken, nonsporulating lesions on 'Gasaway' and its offspring 'Zimmerman' (Table 3). Interestingly, isolates A-03 NJ, A-04 NJ, B-03 MN1, and C-03 MI incited EFB on at least one tree of 'VR20–11', which also carries the 'Gasaway' allele.

'Gasaway' has been exposed to EFB in the PNW for more than three decades

Table 3. Disease incidence of *Corylus* genotypes when challenged with *Anisogramma anomala* isolates collected across the pathogen's native range. Disease was scored 32 months after inoculation and the results are presented as the number of infected plants followed by the number of inoculated plants. The average canker length was recorded at 32 months for "resistant" genotypes and 20 months for control plants. Dashes represent plants not available for inoculations or plants that died in the field before evaluation.

	Isolate treatments												Total infected per genotype	Average canker length[x]
Genotype	A-03 NJ	B-03 MN1	C-03 MI	D-03 NY	E-03 MN2	F-03 OR	A-04 NJ	B-04 NY	C-04 PA1	D-04 PA2	E-04 MA/NY	F-04 OR		
Grand Traverse	0/3	0/3	–	–	–	–	0/3	0/3	0/3	0/3	0/2	0/3	0/23	0
Ratoli	0/3	0/3	0/3	–	–	–	0/3	0/3	0/3	0/3	0/3	0/3	0/27	0
OSU 541.147	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/36	0
OSU 495.072	0/3	0/3	0/3	0/1	–	–	0/3	0/3	0/3	0/3	0/3	0/3	0/28	0
OSU 526.041	0/3	–	–	–	–	–	0/3	0/3	0/3	0/3	0/2	0/3	0/20	0
OSU 408.040	0/3	0/3	1/3[z]	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	1/36[z]	25
Gasaway	0/3	0/3	2/3[y]	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	2/36[y]	40
Zimmerman	0/3	0/3	1/3[z]	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	1/36[z]	15
VR20–11	2/3	1/3	3/3	0/3	0/3	0/3	1/3	0/3	0/3	0/3	0/3	0/3	7/36	40
OSU 587.044	2/3	2/3	3/3	1/2	1/3	1/1	3/3	1/3	1/2	0/3	2/2	1/3	17/31	42
Closca Molla	1/3	3/3	3/3	–	–	–	2/3	2/3	0/3	2/3	2/3	–	15/24	49
OSU 759.007	1/2	–	–	–	–	–	2/3	2/3	1/3	2/3	0/3	2/2	10/19	68
	6/35	6/30	13/27	1/18	1/18	1/16	8/36	5/36	2/35	4/36	4/33	3/32		
Controls														
Ennis	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	36/36	103
Daviana	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	36/36	116
Barcelona	1/1	3/3	2/2	2/2	2/2	2/2	3/3	2/3	3/3	3/3	3/3	3/3	29/30	93
Montebello	3/3	3/3	3/3	3/3	3/3	3/3	3/3	3/3	1/3	3/3	3/3	3/3	34/36	92
Tonda di Giffoni	3/3	3/3	3/3	3/3	2/3	3/3	3/3	3/3	3/3	3/3	3/3	2/3	34/36	63

[z]Sunken lesion lacking fully formed stromata.

[y]EFB canker, the other had a sunken lesion lacking stromata.

[x]Average canker length of control plants was recorded at 20 months because infected branches of many control plants died before stromata developing within the canker and few continued growth the following spring. Therefore, a direct comparison between canker length of "resistant" plants at 32 months, where the branches are generally still living and the perennial canker expanding, versus those of the control plants at 20 months where most branches have died, cannot be made with confidence.

(Mehlenbacher and Thompson, 1991a). Recent selections 'VR20–11' and 'Zimmerman' carrying the 'Gasaway' allele have been exposed for a shorter period (Mehlenbacher and Thompson, 1991b; Lunde et al., 2006). Consistent with their history of testing, no trees of these genotypes were infected by isolates originating from Oregon (Table 3). Interestingly, 'VR20–11', a seedling of ('Barcelona' × 'Compton') × 'Gasaway', and 'Zimmerman', a seedling of 'Barcelona' × 'Gasaway' (Gökirmak et al., 2009), differed in their response compared with 'Gasaway'. For 'Gasaway', only one small typical lesion and one nonsporulating lesion were observed, only when exposed to the Michigan isolate. For 'Zimmerman', only one nonsporulating lesion was observed, also from the Michigan isolate. However, for 'VR20–11', seven of 36 trees showed small, sporulating lesions when exposed to isolates from New Jersey, Minnesota, and Michigan. Because greenhouse inoculation conditions were very similar between all treatments and the Oregon isolates did not incite disease, the results suggest that a genetic component of the fungus played a role in infection. These results also provide strong evidence that alleles at other loci may add or subtract from the resistance provided by the major allele from 'Gasaway'.

Cankers were observed at a higher frequency in 'Closca Molla', OSU 587.044, and OSU 759.007, than the other "resistant" genotypes (Table 3). Lunde et al. (2000) reported that 'Closca Molla' remained free of infection in two subsequent tests, but later, Chen et al. (2007) exposed potted trees under structures topped with diseased wood and observed small cankers on five of eight trees. In these tests, some trees of 'Tonda di Giffoni' (high quantitative resistance) also escaped infection, which suggests that 'Closca Molla' may also have a similar level of quantitative resistance, allowing it to escape earlier inoculations at OSU.

The expression of cankers observed on OSU 587.044 and OSU 759.007 are less clear. Both genotypes developed typical EFB on at least one tree when challenged with isolate populations originating from Oregon (Table 3). Phenotypes of all replications of these selections closely matched descriptions of those from OSU (Shawn Mehlenbacher, personal communication, 2007), confirming they are true to name. Therefore, it seems plausible that they escaped infection at OSU. However, recent evidence suggests this is not the case for OSU 759.007, as it remains free of disease in Oregon after additional evaluations, and progeny appear to be segregating for complete resistance to *A. anomala* (Sathuvalli, 2007). This information, plus the expression of typical EFB by isolate F-04 OR (two of two replicates infected), provides evidence that environmental conditions during inoculations at Rutgers may have played a strong role in infection of OSU 759.007. Unfortunately, no further information is available for OSU 587.044.

This study reinforces previous reports that *A. anomala* may require a second overwintering period of the host plant before EFB expression. The most striking development was shown by 'VR20–11'. In Dec. 2004, 20 months after the 2003 inoculations (Table 2) only 1 of 18 'VR20–11' trees developed EFB (isolate C-03 MI); however, in Dec. 2005, 32 months after inoculations, six additional plants developed cankers (Table 3). The authors are confident the cankers were the result of the greenhouse inoculations, as they developed only on 3-year-old wood of the main stem in a similar location to where cankers developed the previous year on susceptible genotypes. Additionally, three trees of OSU 587.044 and two of 'Closca Molla' did not express cankers until 32 months after being inoculated in 2003 (data not shown). These findings suggest it is important to observe trees for at least 32 months after exposure to *A. anomala*, especially when under plant quarantine situations. Interestingly, no latent cankers were observed on "resistant" genotypes inoculated in 2004.

Conclusions

To more thoroughly examine genetic variation in *A. anomala* and the durability of host resistance, a more comprehensive project must be completed. Part of this project should include generating a molecular fingerprinting method for *A. anomala* to clarify its genetic diversity and population structure. Nevertheless, this study is the first to provide evidence that pathogenic variation exists in *A. anomala*. Because the U.S. hazelnut industry will rely considerably on the single 'Gasaway' allele for protection from EFB, this variability may present a potential major vulnerability of the production system. As such, increased efforts should be made to maintain and possibly enhance restrictions on the movement of *Corylus* plant material into the PNW, as well as outside North America, where the pathogen is not present (Johnson and Pinkerton, 2002). In addition, efforts should be bolstered to identify and use additional sources of host resistance and to test these sources in multiple locations exposed to a diversity of isolates of *A. anomala* over several years when developing new cultivars for the U.S. hazelnut industry. Fortunately, this study also identified five hazelnut genotypes that appear to be resistant to a wide collection of *A. anomala* isolates, with four others only showing a trace of disease. These plants represent a diversity of genetic backgrounds, including several *Corylus* species, and most are interfertile. They may be used by breeders to pyramid multiple EFB resistance genes, which is expected to provide durable protection in the PNW and allow expansion of hazelnut plantings to the eastern United States and southern Canada, where interest in growing hazelnuts is expanding.

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