Genes for Parasite-Specific, Nonhost Resistance in *Populus*

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ABSTRACT

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Nonhost resistance is hypothesized to protect plants in a nonspecific manner. For highly specialized parasites, this hypothesis applies not only to distantly related plants but also to resistant congeners of the host species. Congeners of *Populus* spp. were hybridized to create two inter-specific hybrid poplar pedigrees (i.e., *Populus trichocarpa* × *P. deltoides* and *P. trichocarpa* × *P. maximowiczii*). The pedigrees were planted in a randomized, replicated “common garden” on Vancouver Island so that they were exposed to parasites of the native *P. trichocarpa*. Monogenic

and oligogenic resistance to two ascomycetous, parasitic fungi (i.e., *Venturia inopina* and a *Taphrina* sp.) segregated in a parasite-specific manner in each pedigree. However, these resistance genes were not inherited from the native host, *P. trichocarpa*. Instead, resistance was inherited from the allopatric, nonhost congeners, *P. deltoides* (eastern cottonwood) and *P. maximowiczii* (Japanese poplar). Thus, we found that major genes condition parasite-specific, nonhost resistance, as has been true in earlier studies of this kind with additional parasites of *Populus* spp. The selective force responsible for evolutionary maintenance of such genes is unknown.

Additional keywords: host range.

In a series of articles and reviews (11–15), Heath has hypothesized that nonhost resistance is “basic” or parasite nonspecific. In contrast, major genes for resistance are known to be parasite-specific; they are thought to co-evolve with genes for virulence in gene-for-gene arms races within host and corresponding parasite populations (10). It is presumably only when a parasite overcomes basic, nonhost resistance that a coevolutionary arms race ensues. The initial, extreme susceptibility is thought to spark many generations of selection in both host and parasite. Repeated cycles of such selection ultimately result in parasite-specific, gene-for-gene interactions.

Differential, host–parasite specificity is the most basic indicator of gene-for-gene interactions in natural populations, even in the absence of evidence of pairs of corresponding resistance and avirulence genes. A summary of a study of four natural host–parasite associations (2) involving herbaceous plants has led to the view that spatial and temporal scale must be an explicit part of attempts to understand differential specificity. Theoretical models of natural, gene-for-gene coevolution often have been based on panmictic, single-population assumptions that do not reflect geographic scale and metapopulation dynamics (3). However, still unstudied among natural host–parasite associations are those that involve congeneric plants in which well-developed barriers of reproductive isolation are lacking. Incomplete barriers and occasional or local hybridization often characterize genera of long-lived and woody plants (31), yet the implications for models of metapopulation gene-for-gene coevolution are unknown.

In *Populus*, there are many examples of parasite-specific, nonhost or “exapted” resistance (23,24,26,27). Interspecific and even intersectional hybridization also are common in *Populus* spp. (1,8), and its natural hybrid zones have become model laboratories in which susceptibility and resistance to parasites and other symbionts are studied (34). Hybrid populations may filter gene intro-

gression between species (21), although subsequent selection for resistance to a parasite of a congener is impossible without exposure to that parasite.

Gene-for-gene interactions are known for *Populus* spp. in the case of *Melampsora* leaf rust (27). *Populus trichocarpa* and *P. deltoides* each are associated with a species of *Melampsora* (i.e., *Melampsora occidentalis* and *M. medusae*, respectively) and their wild distributions in western and eastern North America overlap slightly. *P. trichocarpa* possesses at least two major genes for rust resistance, *Mmd1* and *Mxc3* (27,32). However, these cannot be detected by the native *M. occidentalis* in that the latter evidently lacks the avirulence alleles that correspond to these resistance genes. Instead, *Mmd1* and *Mxc3* are detected by avirulence genes in *M. medusae* and *Melampsora* × *columbiana*, respectively. *P. deltoides* itself possesses at least two major genes for resistance, *Mxc1* and *Mxc2*, that are matched only by avirulence genes in some pathotypes of *Melampsora* × *columbiana* (27). The latter is the hybrid of *M. medusae* and *M. occidentalis* (28). Hybridization of both host and parasite congeners in the paired, continentally distributed systems have uniquely allowed differentially specific, gene-for-gene interactions to be detected. However, the evolutionary basis for these genes for resistance to *Melampsora* spp. is problematic. At least *Mmd1* and *Mxc3* specifically govern nonhost resistance to the parasite of a congener, *M. medusae*, and the basis for such selection is unknown.

P. trichocarpa is an undomesticated tree species that hosts fungal parasites other than *Melampsora* spp. Early studies have shown that the same pattern of major genes for parasite-specific, nonhost resistance prevails. For example, the resistance of hybrids of *P. trichocarpa* and *P. deltoides* to ascomycetous pathogens such as *Glomerella cingulata* (29) and *Mycosphaerella populicola* (24) was due to genes or quantitative trait loci (QTL), respectively, inherited from the nonhost, *P. deltoides*. In pursuit of generality, the genetic basis of resistance to additional, ascomycetous parasites was the focus of the present study. Interspecific hybridization was deliberately employed to create two three-generation pedigrees in which segregation for nonhost resistance could be analyzed.

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MATERIALS AND METHODS

The pedigrees. The *P. trichocarpa* × *P. deltoides* pedigree comprises three generations: (i) 93-968 (*P. trichocarpa* ♀) × Ill-129 (*P. deltoides* ♂); (ii) the F₁ generation, of which 21 clones were in this field experiment, including the parents of the F₂, 53-246 and 53-242; and (iii) the F₂, of which 103 clones were included in the field experiment. This *P. trichocarpa* × *P. deltoides* pedigree also included reciprocal backcross progenies: (i) the F₁ female clone, 53-246, was crossed with Ill-129 to produce the backcross family 342 (TD×D), of which 32 clones were included; and (ii) the F₁ male clone, 53-239, was crossed with 93-968 to produce the backcross family 353 (T×TD), of which 52 clones were included.

The *P. trichocarpa* × *P. maximowiczii* pedigree also comprised three generations: (i) 8-1023 (*P. trichocarpa* ♀) × MAX1016 (*P. maximowiczii* ♂) and 8-1038 (*P. trichocarpa* ♀) × MAX103 (*P. maximowiczii* ♂); (ii) the two F₁ clones (271-286, a ♀, and 272-99, a ♂) that resulted from the above crosses and that were crossed to produce the F₂; and (iii) the F₂ family, of which 72 clones were included in the field experiment.

In addition, the following nonpedigree clones were included: 15 clones of each of *P. trichocarpa*, *P. deltoides*, and *P. maximowiczii*. The latter three sets included the abovementioned parental clones in addition to unrelated clones of each of the three species, and the species sets were used to check on the generality of resistance of nonhost congeners. In each of three, replicated blocks, each clone was represented by a two-ramet plot for a total of six ramets of each clone. Clones were randomized within each block.

The field experiment. As previously described (29), the site was an alluvial floodplain of the Salmon River 60 km north of Campbell River on Vancouver Island (latitude 50°12'N, longitude of 125°48', and elevation <50 m) where *P. trichocarpa* is the only native, wild representative of the two sections of *Populus* involved in this study: Tacamahaca and Aigeiros. Specifically, the field experiment was on Armishaw Road, south of the town of Sayward. Summers are moist and cool. Previous field experiments (29) established expectations for basic disease patterns for the new research, including the expectation that the parasites would take 4 or 5 years to become uniformly distributed throughout the new experiment on all susceptible individuals. Segregation for resistance to the three parasites thus was observed in the spring (*Venturia* and *Taphrina* spp.) and fall (*Linospora* spp.) of 2001 and 2002, although earlier data also were recorded. A fence kept out large mammalian herbivores, and cultivation controlled competition.

Parasites. Three fungal parasites were considered. *Venturia inopina* Newcombe only recently was distinguished from its European congener, *V. populina* (Vuill.) Fabric. (25). The former parasitizes *P. trichocarpa* whereas the latter occurs in the Pacific Northwest on its European host, *P. nigra* cv. *italica*. Young leaves and shoots of *P. trichocarpa* are blighted in the spring by *V. inopina*, resulting in necrotic lesions.

The second, ascomycetous parasite is a native species of *Taphrina*, adapted to *P. trichocarpa* and its hybrids. Critical studies of *T. populina* (Fr.:Fr.) Fr. and *T. populi-salicis* mix are unavailable; therefore, the *Taphrina* sp. of this study is simply referred to as a *Taphrina* sp. that causes a leaf blister in spring.

Fig. 1. Observed segregation for resistance to two diseases in a *Populus trichocarpa* × *P. deltoides* pedigree. Data were taken during the fifth and sixth years of trees growing in a randomized, replicated planting within the geographic range of *P. trichocarpa*. Recessive genes for resistance to both diseases were inherited from the allopatric, nonhost *P. deltoides*.

Typically, yellow asci of *Taphrina* spp. develop on the concave surface of the blister.

Linospora tetraspora G. E. Thompson is also a foliar parasite of *P. trichocarpa* that develops later in the season than *V. inopina* or the *Taphrina* sp. Black fruiting bodies of the fungus speckle the light-brown lesions. Symptoms of the three parasites are easily distinguished, and microscopic confirmation of symptom-based diagnosis also is simple.

Assessment of resistance. Individuals (clones) were assessed as either resistant or susceptible in the fifth and sixth years of growth. During the fifth year, clones were recorded as resistant if, during any year up to that point, none of their ramets had been judged susceptible to a particular parasite. The sixth year was used to confirm the segregation patterns of the fifth year. Any diagnosable symptoms on any ramet of a particular clone at any time in the fifth or sixth years resulted in a classification of "susceptible".

RESULTS

Resistance to *V. inopina* in the *P. trichocarpa* × *P. deltoides* pedigree. All 15 clones of the native host, *P. trichocarpa*, were susceptible to *V. inopina*, whereas the 10 surviving clones of the original 15 of the nonhost congener, *P. deltoides*, were resistant in both the fifth and sixth years. Resistance to *V. inopina* in the *P. trichocarpa* × *P. deltoides* pedigree did not segregate in the F₁, because all 21 clones were susceptible (Fig. 1). Of the original F₂ progeny of 103 clones, 67 survived with full sets of ramets into the fifth and sixth years. Even though the F₁ parents of the F₂ were both susceptible, there was segregation in the F₂ that suggested a single, recessive gene for resistance (50 clones were susceptible and 17 were resistant). The latter observed ratio does not deviate significantly from the 3:1 ratio ($\chi^2 = 0.005$; $P = 0.94$)

expected for a recessive gene. Of the 50 susceptible clones, 28 displayed 100% incidence, in that all ramets were symptomatic in both the fifth and sixth years of growth. Incidence and disease severity varied among ramets and years for the other 22 susceptible clones. Of the 36 clones that did not survive in full into the fifth and sixth years, all data suggested segregation in line with the 3:1 ratio of the 67 survivors. In the T×TD backcross progeny, 49 of 52 clones survived in full and all were susceptible, as would be expected for a cross between the dominant homozygote for susceptibility (*P. trichocarpa* female 93-968) and a susceptible F₁ heterozygote (53-239). In contrast, survival in the TD×D backcross progeny was poor (19 of the original 32). Other than noting that there was segregation, in that 7 clones were susceptible and 12 resistant, the expected 1:1 ratio (susceptible F₁ heterozygote [53-246] × resistant homozygote [*P. deltoides* male ILL-129]) was not tested.

Resistance to *V. inopina* in the *P. trichocarpa* × *P. maximowiczii* pedigree. As already stated, all 15 clones of the native host, *P. trichocarpa*, were susceptible to *V. inopina*. In this case, all 15 clones of the nonhost congener, *P. maximowiczii*, survived and all were resistant in both the fifth and sixth years. Both F₁ clones, parents of the F₂, were resistant (Fig. 2). These results were expected, given the previous study (23) in which 32/32 and 36/36 clones of *P. maximowiczii* and *P. trichocarpa* × *P. maximowiczii* F₁ hybrids, respectively, were resistant to *V. inopina*. In this study, of the original F₂ progeny of 72 clones, 64 survived with full sets of ramets into the fifth and sixth years. Of these, 61 were resistant but 3 were susceptible. Observed segregation in the F₂ thus fits a dihybrid ratio of 15:1 ($\chi^2 = 0.267$; $P = 0.605$). In other words, when a *P. trichocarpa* × *P. maximowiczii* F₂ individual possesses a dominant allele at either of two, independent loci, it is resistant. Only individuals that are homozygous recessive at both loci appear to be susceptible.

Fig. 2. Observed segregation for resistance to two diseases in a *Populus trichocarpa* × *P. maximowiczii* pedigree. Data were taken during the fifth and sixth years of trees growing in a randomized, replicated planting within the geographic range of *P. trichocarpa*. Dominant genes for resistance to both diseases were inherited from the allopatric, nonhost *P. maximowiczii*.

Resistance to a *Taphrina* sp. in the *P. trichocarpa* × *P. deltoides* pedigree. Again, the entire set of clones of *P. trichocarpa* was susceptible, whereas the nonhost, congeneric clones of *P. deltoides* were resistant. All 21 F₁ clones were susceptible, but the F₂ segregated (i.e., 45 susceptible and 22 resistant individuals) (Fig. 1). As with resistance to *V. inopina* in the *P. trichocarpa* × *P. deltoides* pedigree, the latter observed ratio corresponds to the 3:1 ratio ($\chi^2 = 2.2$; $P = 0.14$) expected of a recessive gene for resistance. Similarly, the 49 clones of the T×TD backcross progeny were susceptible, as would be expected for a cross between the dominant homozygote for susceptibility (*P. trichocarpa* female 93-968) and a susceptible F₁ heterozygote (53-239). Both susceptible and resistant clones were observed in the TD×D backcross progeny as expected; however, again, survival was poor as noted above.

Resistance to a *Taphrina* sp. in the *P. trichocarpa* × *P. maximowiczii* pedigree. The resistance of the nonhost congener, *P. maximowiczii*, again contrasted qualitatively with the susceptibility of *P. trichocarpa*. One of the F₁ parents was susceptible (the female clone 271-286), whereas the other was resistant (the male clone 272-99). Of the 64 surviving F₂ clones, 39 were susceptible and 25 resistant. This observed ratio was tested against the 1:1 ratio expected for a dominant gene in a cross between a susceptible recessive homozygote and a resistant heterozygote ($\chi^2 = 3.06$; $P = 0.08$). In this case, a dominant gene inherited from a heterozygous *P. maximowiczii* is the simplest explanation for resistance.

Resistance to *L. tetraspora* in both the *P. trichocarpa* × *P. deltoides* and *P. trichocarpa* × *P. maximowiczii* pedigrees. No segregation was observed in the two pedigrees because all clones were at least somewhat susceptible, varying in quantitative extent. However, the expected resistance of the two nonhost congeners was confirmed.

Linkage of the two recessive genes for resistance to *V. inopina* and a *Taphrina* sp. in the *P. trichocarpa* × *P. deltoides* pedigree. Contingency testing for linkage of these two recessive genes indicated that they are linked ($\chi^2 = 4.1$; $P = 0.04$).

DISCUSSION

Resistance to parasites of allopatric congeners is not inherently surprising. Although examples such as chestnut blight suggest generalized susceptibility to parasites of congeners, resistance is implied in “enemy release” (16), whereby exotic plants are not only unaffected by parasites left behind in the continent of origin, but also are resistant to parasites of congeners of their host in the new land. Recent studies have suggested that exotic plants become invasive in part because they are resistant to root pathogens that limit the growth of related, native plants (4,17). Nonhost, gene-for-gene interactions have even been demonstrated between a New World plant (i.e., maize) that possesses an *R* gene that matches an avirulence gene in an Old World pathogen (i.e., a bacterial pathogen of rice) (36). Finally, resistance to parasites of congeners would appear simply to be a given because specialized parasites possess narrow host ranges.

But why are host ranges narrow? Findings here imply that the reported genes (Table 1) limit the host ranges of *V. inopina* and

the *Taphrina* sp. that parasitize *P. trichocarpa*. It could be argued that resistance in *Populus* is anomalous and that only broader investigations of more plant taxa with confirmation of the *Populus* pattern will allow for generalization. However, the use of resistance genes of wild relatives in crop breeding (20) suggest that the genus *Populus* is not anomalous. Of course, only congeneric plants that can be hybridized would be candidates for broader investigations of major genes for nonhost resistance.

Could the existence of parasite-specific, major genes for nonhost resistance depend on selective filtering through natural hybrid zones (21)? Filtering of this sort has been possible for millions of years (7) where the ranges of *P. deltoides* and *P. trichocarpa* overlap and these North American congeners hybridize. However, hybrid-zone filtering could not account for the parasite-specific, nonhost resistance displayed in this study by the Asian poplar, *P. maximowiczii*. This hypothesis would not, in any case, account for post-zone maintenance of genes for resistance in the absence of selection exerted by the corresponding parasite.

P. deltoides and *P. maximowiczii* are susceptible to diseases caused by *Venturia* and *Taphrina* spp. within their own native ranges. *P. deltoides* is known to be susceptible to its own host-adapted populations of *Taphrina* (19) and *Venturia* (9,22); the same is likely to be true for *P. maximowiczii* (22,35). Earlier studies of resistance to *G. cingulata* (24) and *M. populicola* (26), in which genes or QTL were inherited from the nonhost (i.e., *P. deltoides*), also potentially could be explained in terms of selection due to sympatric, host-adapted populations. However, evidence to date does not favor this hypothesis. For example, there was no phenotypic evidence for a major gene for resistance to *Melampsora occidentalis* in *P. trichocarpa* populations in common gardens (6), nor was there in an inoculation study (28). If the *Mmd1* and *Mxc3* genes were retained in *P. trichocarpa* through selection for resistance to *M. occidentalis*, then at least some resistant clones would have been detected in these studies. Similarly, in the study reported here, the 15 clones of *P. trichocarpa* all were susceptible to three ascomycetous parasites (i.e., *V. inopina*, a *Taphrina* sp., and *L. tetraspora*) in a common garden within its native range. Predictions supported by this study and its earlier counterparts include: (i) outside its native range, *P. trichocarpa* would be resistant to populations of *Venturia*, *Taphrina*, and *Linospora* spp. to which the local species of *Populus* would be susceptible; and (ii) interspecific hybrids of the local species and *P. trichocarpa* would express resistance conferred by genes inherited from *P. trichocarpa* (even though this study provided no evidence that *P. trichocarpa* possesses genes for resistance to its own parasite populations).

Major genes for parasite-specific or pathotype-specific resistance in crop plants usually are assumed to have resulted solely from coevolutionary arms races (5,18,30,33), rather than from domestication-associated hybridization in which genes for resistance were inadvertently or deliberately obtained from wild relatives. The arms-race paradigm provides a compelling example of natural selection and, thus, an easily accepted explanation for major genes for resistance. In contrast, the genes reported here and in earlier studies of resistance to parasites of *Populus* spp. cannot be explained by simple, coevolutionary arms races. In particular, the selective force responsible for the evolutionary maintenance of such genes is not yet known.

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TABLE 1. Inheritance of parasite-specific, nonhost resistance to parasites of *Populus trichocarpa* on Vancouver Island^a

Parasite	Nonhost resistance inherited from	
	<i>P. deltoides</i>	<i>P. maximowiczii</i>
<i>Venturia inopina</i>	Single recessive gene	Two dominant genes
<i>Taphrina</i> sp.	Single recessive gene	Single dominant gene

^a Contingency testing for linkage of the two recessive genes inherited from *P. deltoides*: $\chi^2 = 4.1$ ($P = 0.04$).

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