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ORIGINAL ARTICLE OPEN ACCESS

Vulnerable Juniper Populations Show Adaptive Potential in the Face of a Highly Damaging Invasive Tree Pathogen

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Received: 5 January 2026 | **Revised:** 7 May 2026 | **Accepted:** 18 May 2026

Keywords: GxG interaction | heritability | host resistance | invasive pathogens | Juniper | *Juniperus communis* | natural selection | *Phytophthora austrocedri*

ABSTRACT

Invasive tree pathogens pose a significant and increasing threat to natural ecosystems. The outcome of these novel host-pathogen interactions depends largely on the presence and nature of resistance in host populations, which will govern the host's potential to respond through natural selection and adaptation to the new threat. This study assessed the adaptive potential of juniper (*Juniperus communis*) in the face of the invasive tree pathogen *Phytophthora austrocedri* through two inoculation experiments: an excised-shoot trial using six different *P. austrocedri* isolates and a progeny-provenance trial that inoculated whole trees with a single highly virulent isolate. We found evidence for both qualitative and quantitative resistance in juniper populations, with lesion length (quantitative resistance) showing moderate to high heritability and lesion development (qualitative resistance) showing very high heritability. There was a significant genotype-by-genotype interaction between pathogen isolate and host genotype, lowering the estimate of heritability to moderate values when calculated across six different isolates. Finally, we found evidence that *P. austrocedri* is imposing natural selection on juniper populations, with individuals originating from highly exposed populations having a lower predicted probability of developing a lesion. Based on the results of this study, we recommend that the most effective management strategy for vulnerable UK juniper populations is to promote natural regeneration within populations, making use of existing genetic diversity in resistance within natural populations without risking the introduction of new *P. austrocedri* genotypes through the planting of nursery-grown juniper.

1 | Introduction

Invasive tree pathogens are a major threat to natural, managed and urban forests, causing substantial economic losses and significant ecosystem disruption (Budde et al. 2016; Lovett et al. 2016; D'Amato et al. 2023). Introduced pathogens often cause far greater damage than their native counterparts, due to differences in the fundamental evolutionary and ecological processes underlying host-pathogen dynamics (Parker and Gilbert 2004; Ennos 2015). Native pathosystems are typically the dynamically stable outcome of long-term co-evolutionary interactions, and are characterised by polymorphisms in disease-related traits (Brown and Tellier 2011; Kahlon and Stam 2021). These polymorphisms are maintained through processes such as negative frequency-dependent selection, spatial and temporal heterogeneity in disease pressure, and the fitness costs associated with resistance and virulence (Burdon and Thrall 2009; Brown and Tellier 2011). In contrast, in novel interactions between a non-native pathogen and a previously unexposed host, the lack of a shared co-evolutionary relationship means that alleles that confer resistance are not expected to be segregating at appreciable frequencies in the population (Ennos 2015). As a result, host populations may exhibit near-universal susceptibility, leading to widespread mortality. At worst, invasive pathogens can

cause catastrophic collapse of host populations across their entire range (e.g., Kinloch 2003; Harwood et al. 2011; Rigling and Prospero 2018).

Some initial resistance to a novel pathogen could, however, already be present within unexposed host populations at low frequencies, arising either by chance through random mutation or due to prior exposure to a pathogen with a similar mode of action, resulting in a degree of preadaptation (Parker and Gilbert 2004). This may be particularly true in tree species, which often have large effective population sizes and maintain high levels of standing genetic variation (Petit and Hampe 2006; Kremer et al. 2025). As the pathogen spreads through the population, any resistance alleles will experience strong positive selection, which could result in the host responding to the new threat through adaptation (Cavers and Cottrell 2015). However, whether such adaptation through natural selection occurs will depend on multiple factors, including the initial frequency of resistant genotypes in the host population, the heritability of resistance, and the durability of resistance.

The extent to which resistance to a pathogen is due to heritable genetic variation is crucial for determining the host's adaptive potential (Telford et al. 2015). The heritability of a trait—defined as the proportion of total variation that is explained by additive genetic variance (Falconer and Mackay 1996)—is a fundamental measure in predicting evolutionary response, as it governs the efficacy of natural or artificial selection (Roff 1997). The heritability of resistance will be influenced by the genetic basis of the trait, as well as by the stability of resistance across different environments and pathogen genotypes. High levels of heritability in disease resistance will allow more efficient selection and will thus increase the adaptive potential of the host.

The nature of disease resistance in plants can be divided into qualitative (complete) resistance and quantitative (incomplete) resistance (Agrios 2005). Qualitative resistance is often attributed to a single gene of large effect (R gene), while quantitative resistance is generally thought to arise from the action of many small-effect genes, which combine to produce continuous phenotypic variation (St. Clair 2010; Corwin and Kliebenstein 2017). Because resistance controlled by a single gene is expected to be less robust to pathogen evolution than resistance governed by multiple genes, qualitative resistance is predicted to be less durable than quantitative resistance (McDonald and Linde 2002; Brown 2015). However, some have argued that a strict separation into qualitative vs. quantitative is too simplistic, and that resistance is better seen as a continuum (Poland et al. 2009). In fact, while there are cases in which quantitative resistance has been shown to have a basis distinct from major-gene-associated qualitative resistance, in other cases they may share the same underlying mechanism (Kamoun et al. 1999; Poland et al. 2009; Corwin and Kliebenstein 2017). Consequently, phenotypic patterns of resistance alone may provide limited insight into the underlying genetic architecture of the trait, and hence its potential durability.

The outcome of novel plant-pathogen interactions ultimately depends on the presence and nature of resistance within the host population. Host resistance has been successfully exploited in large-scale tree breeding programmes (Sniezko and Dana Nelson 2022) that have artificially selected resistant genotypes, particularly in commercially important tree species (Sniezko et al. 2014, 2019). In non-commercial tree species, the resources for a large-scale breeding programme may not be available; however, knowledge of host resistance and adaptive potential is essential for informing conservation and management strategies designed to mitigate the impact of invasive pathogens.

Juniper (*Juniperus communis*) is an evergreen, dioecious conifer (Cupressaceae) that plays an important ecological role in UK landscapes as one of only three native conifer species (Johnson and More 2015). Although juniper remains geographically widespread in the UK, it is considered vulnerable due to forming small, isolated populations with low levels of natural regeneration (Thomas et al. 2007; Broome et al. 2017). Consequently, *J. communis* is listed as a priority species for conservation (BRIG 2007). Juniper is now under further threat from the invasive oomycete pathogen *Phytophthora austrocedri* (Green et al. 2014). First detected in the UK in 2010 (Green et al. 2012), *P. austrocedri* is causing extensive mortality in juniper populations across northern England and Scotland (Green et al. 2014). Elsewhere, *P. austrocedri* is also the causal agent of widespread dieback and mortality of Chilean cedar (*Austrocedrus chilensis*) in the Patagonian Andean forests of Argentina (Greslebin et al. 2007; Greslebin and Hansen 2010). The pathogen is thought to be invasive in both the UK and Argentina, but its origin is still unknown (Henricot et al. 2017). Previous work has shown there is evidence of resistance to *P. austrocedri* in some juniper individuals (Green et al. 2020); however, the nature of the resistance and the extent to which it is due to heritable genetic variation that could allow adaptation is not known.

This study aims to assess the adaptive potential of UK juniper populations in response to the threat posed by *P. austrocedri* by addressing the following questions: (1) To what extent is resistance to *P. austrocedri* a heritable genetic trait in juniper? (2) What is the nature of disease resistance (qualitative vs. quantitative) in juniper? and (3) Is there evidence that natural selection is already operating in juniper populations as a response to *P. austrocedri*? Addressing these questions will provide the foundation for evidence-based management and conservation of this vulnerable native tree.

2 | Methods

2.1 | Excised Shoot Inoculation Trial

An initial excised shoot-based inoculation trial was carried out in order to (i) screen *P. austrocedri* isolates to assess relative virulence and (ii) get an estimate of broad-sense heritability of juniper host resistance by making use of within-genotype replication. Six *P. austrocedri* isolates were chosen for screening, representing different populations and isolated in different years (Table S1). Older isolates had recently been passed through the host to restore virulence.

We collected 190 shoots from 10 juniper individuals from an isolated population that remains free of any symptoms of *P. austrocedri* (Lammermuir Hills, Scotland; Table S2), allowing for three replicates per isolate/genotype combination and one negative

control per juniper genotype. We processed the shoots to ensure all were approximately equal in length (35–40 cm), and dipped cut ends in wax to prevent desiccation. Excised shoots were inoculated as described in Green et al. (2020). Briefly, mycelial plugs (4 mm diameter) were taken from the edge of *P. austrocedri* cultures actively growing on V8 agar. A sterile cork borer (4 mm diameter) was used to remove a piece of bark in the middle of the shoot, and the mycelial plug was placed on the exposed cambium layer. The area was covered with cotton wool thoroughly moistened with sterilised distilled water, before wrapping with parafilm and tin foil. Negative controls were inoculated in the same way with plugs taken from a sterile V8 agar plate. Shoots were processed over 3 days, with each day forming one block and with one replicate of each genotype/isolate combination inoculated per day in a fully randomised block design. Shoots were harvested 5 weeks after inoculation and lesion length measured by careful removal of the outer bark to expose the lesion underneath (Figure S1). Shoot width at the point of inoculation was also recorded.

2.2 | Progeny-Provenance Whole-Tree Inoculation Trial

Open-pollinated seed was collected from 54 mother trees from 13 natural populations across the UK (Table S2) in autumn 2015. Seed was germinated as described in Baker et al. (2025). Trees were grown in a common environment in greenhouse conditions in a randomised block design, with each block containing one member of a family, and potted up as needed. The experiment was carried out in autumn and winter 2023/2024 when the trees were approximately 6 years old. At the time of inoculation, median tree height was 90 cm and all trees were in 3 L pots. Most families were represented by six individuals per maternal tree (half-siblings or full-siblings), but the loss of some of these trees meant that a few families were only represented by four or five individuals (Table S2). The trial included a total of 306 trees, plus six positive and six negative controls.

Trees were inoculated at the stem base following the mycelial plug method described for whole plants in Green et al. (2020) using only one *P. austrocedri* isolate (GA7; Table S1). Mycelial plugs (4 mm diameter) were taken from the edge of *P. austrocedri* cultures actively growing on V8 agar. A sterile cork borer (4 mm diameter) was used to remove a piece of bark at the base of the stem, and the mycelial plug was placed on the exposed cambium layer. The area was covered with cotton wool thoroughly moistened with sterilised distilled water, before wrapping with parafilm and tin foil. Inoculations were carried out in six fully randomised blocks, with one member of each family present in each block, along with a positive and negative control. Controls were trees of a genotype known to be highly susceptible (clone 'JC' in Green et al. 2020) that were inoculated either as described above with a mycelial plug (positive control) or as described above but instead using a sterile plug of V8 agar (negative control).

The trial was conducted over a 5-month period, as only two blocks could be accommodated concurrently in the greenhouse. Blocks one and two were inoculated in October 2023, blocks three and four were inoculated in December 2023, and blocks five and six were inoculated in January 2024. The greenhouse temperature was maintained at 15°C, and temperature and humidity were recorded using data loggers (Figure S2). However, neither humidity nor day length was controlled. Trees were kept well watered throughout the experiment. Six plugs from each colony used for the inoculations were plated on V8 agar to confirm all produced actively growing colonies in greenhouse conditions. Trees were harvested 5 weeks post-inoculation. Bark surrounding the inoculation point was carefully removed using a scalpel to expose the underlying lesion (Figure S3). Lesion length and width were recorded, along with tree height and stem width at the point of inoculation.

To confirm lesion formation was due to *P. austrocedri*, re-isolation was attempted from a randomly chosen subset of 56 trees across all blocks by plating small pieces of phloem tissue taken from the leading edge of the lesion on *Phytophthora* selective medium (synthetic mucor agar; Brasier et al. 2005). Resulting colonies were identified as *P. austrocedri* based on its distinctive coralloid hyphae (Greslebin et al. 2007). *Phytophthora austrocedri* infection was also confirmed by qPCR in a random subset of 30 trees (five trees per block). Tissue was collected from the edge of lesions and freeze dried prior to DNA extraction using the DNeasy Plant Pro Kit (Qiagen) following the manufacturer's instructions. The presence of *P. austrocedri* DNA in lesion tissue was confirmed using qPCR assays following the protocol described in Mulholland et al. (2013).

2.3 | Data Analysis and Estimation of Heritability

2.3.1 | Excised Shoot Inoculation Trial Analysis

As lesion length showed a strong positive skew in distribution, the measurements were log-transformed for subsequent analysis using linear mixed-effects models. Log lesion length was modelled using the R package lme4 (Bates et al. 2015), with *Tree* and *Isolate* fitted as random effects and *Block* fitted as a fixed effect. *Shoot width* (mm) was initially included as a covariate, but was subsequently removed from the model due to no evidence of any effect ($\beta = -0.02$ [−0.06–0.02], $p = 0.25$), and the simpler model without shoot width providing a better fit (lower AIC and BIC scores). Confidence intervals around estimated parameters and ratios of variances were generated using parametric bootstrapping. Significance of individual model terms fitted as random effects was tested using likelihood ratio tests of nested models.

A second model with an added *Tree:Isolate* interaction term was also fitted to get a measure of broad-sense heritability within isolates (see below). The interaction term was excluded from other analyses due to its poor performance in parametric bootstrapping, suggesting an overly complex random effect structure for the sample size.

2.3.2 | Estimation of Broad-Sense Heritability

Broad-sense heritability (H^2), the proportion of phenotypic variance explained by genotypes, was estimated using two approaches. First, H^2 was estimated across all *P. austrocedri* isolates (using the model without a *Tree:Isolate* interaction term) by using the variance among juniper trees (V_{tree}), isolates ($V_{isolate}$), blocks (V_{block}) and residual variance (V_{res}) as follows:

$$H^2 = V_G / V_P = V_{\text{tree}} / \left(V_{\text{tree}} + V_{\text{isolate}} + V_{\text{block}} + V_{\text{res}}\right)$$

Second, H^2 was estimated for the case when inoculated with the same *P. austrocedri* isolate (using the model with a *Tree:Isolate* interaction term) by excluding V_{isolate} from the denominator and adding the *Tree:Isolate* interaction term ($V_{t:i}$) to the numerator as follows:

$$H^2 = V_G / V_P = \left(V_{\text{tree}} + V_{t:i}\right) / \left(V_{\text{tree}} + V_{\text{t:i}} + V_{\text{block}} + V_{\text{res}}\right)$$

The amount of variation explained by fixed effects was extracted from the model using the R package insight (Lüdecke et al. 2019).

2.3.3 | Progeny-Provenance Whole Tree Inoculation Trial Analysis

Lesion length showed a bimodal distribution, with a group of trees showing no lesion or very little lesion development and a larger group of trees showing an approximately normally distributed lesion length centred around a larger mean (Figure 1). In order to analyse these data, lesion development was separated into two response variables: (1) presence/absence of lesion development and (2) length of lesion if developed. In all trees, the inoculation process itself resulted in discoloration, even if there was no subsequent lesion development (even negative controls had a discoloured "lesion" that was 4–6 mm long; see Figure S3). This means the threshold for classifying a tree as having lesion development cannot be the absence of any discoloration. Therefore, a lesion was classified as "development present" if lesion length was more than 15 mm. This threshold is unavoidably somewhat arbitrary but was chosen to best fit the distribution of the data (Figure 1): there was a large spike of trees with lesions below this size, while the remaining trees exhibited a distribution of lesion lengths that could reasonably be modelled using a normal distribution. Moreover, the analysis was not sensitive to the exact threshold, as rerunning the analysis based on different thresholds (from 10 to 20 mm) had only small effects on the estimated parameter values and would have no impact on their interpretation (Table S3).

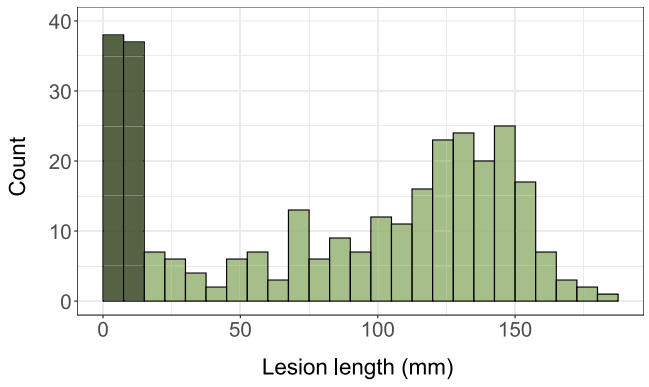

FIGURE 1 | Histogram showing the bimodal distribution of lesion lengths in the progeny-provenance whole tree trial. Dark green bars represent the trees that were classified as "lesion development absent" (lesion length ≤ 15 mm), while the light green bars represent trees that were classified as "lesion development present" (lesion length > 15 mm).

The two traits (presence/absence of lesion development and lesion length when present) were analysed using multivariate generalised linear mixed models implemented in the Bayesian R package MCMCglmm (Hadfield 2010). Presence/absence of lesion development was modelled for all data using the threshold model, which assumes there is a latent liability trait that underlies the binary trait of interest and uses a probit link function (Falconer and Mackay 1996; De Villemereuil et al. 2016). Lesion development occurs when a certain threshold in the latent trait is exceeded. For the subset of trees that developed a lesion, lesion length was modelled assuming a normal distribution. *Family*, *Population* and *Block* were fitted as random effects. We investigated whether tree size had an effect on lesion development by fitting *Stem width* (mm) or *Tree height* (cm) as covariates, then controlled for tree size by fitting *Tree height* as a random slope allowed to vary by population.

Priors were defined following the recommendations in Hadfield (2019). Parameter expanded priors were used for (co)variances of random effects (specification: $V = 1$, nu = 2, alpha. mu = 0, alpha. V = 1000), an inverse Wishart prior was used for the residual variance of lesion length (specification: $V = 1$, nu = 0.002) and default diffuse normal priors with large variances were used for fixed effects. The residual variance of the binary trait (lesion development present/absent) was fixed at 1. We set the MCMC thinning interval to 100 after a burn-in period of 30,000, and let the chain run for enough iterations to obtain a minimum effective sampling size > 2000 for all parameters. Convergence was assessed by visual inspection of trace plots and by checking that autocorrelation between successive stored samples was < 0.1.

2.3.4 | Estimation of Narrow-Sense Heritability

Narrow-sense heritability (h^2) is the proportion of total phenotypic variance explained by additive genetic variance. In a half-sibling family design, additive genetic variance is equal to four times the amount of variation explained by family, whereas in a full-sibling design it is equal to twice the amount of variation explained by family, although this latter estimate would be inflated due to shared dominance effects (Falconer and Mackay 1996). The open-pollination design of this trial means our families are likely to include a mixture of both half-siblings and full-siblings. We therefore approximated a roughly equal proportion of full- and half-siblings, and multiplied the variance attributed to family by three to get an estimate of additive genetic variance. Narrow-sense heritability was then calculated using estimates of variance attributed to *Family* (V_{fam}), *Population* (V_{pop}), *Tree_height:Population* (V_{hgt}), *Block* (V_{block}) and residual variance (V_{res}) as follows:

$$h^2 = V_a / V_p = 3 \times V_{\text{fam}} / \left(V_{\text{fam}} + V_{\text{pop}} + V_{\text{hgt}} + V_{\text{block}} + V_{\text{res}}\right)$$

We also calculated the proportion of total variation explained by *Population*, *Block* and *Tree_height:Population* and report this as the intraclass correlation coefficient (ICC).

For the binary threshold trait (lesion presence/absence), h^2 was estimated on both the latent trait scale and the observed data scale. Heritability on the latent scale can be interpreted as the heritability of a theoretical latent trait that underlies the

susceptibility to developing the lesion, whereas heritability on the observed data scale refers to the heritability of the probability of developing a lesion. Quantitative genetic parameters on the observed data scale were extracted from the model using the R package QGglmm (De Villemereuil et al. 2016).

To investigate the effect of previous exposure at the population source site to *P. austrocedri*, a separate model was fitted that included level of exposure of the population as a fixed effect. Populations were classified as either High Exposure (*P. austrocedri* presence confirmed and substantial mortality already occurred) or Low Exposure (no evidence of mortality associated with *P. austrocedri*), based on data from Green et al. (2014) and Donald et al. (2021) (Table S2).

3 | Results

3.1 | Excised Shoot Inoculation Trial

3.1.1 | Variation in Virulence Among *Phytophthora austrocedri* Isolates

Phytophthora austrocedri isolates varied significantly in the size of lesions produced (LRT $\chi^2_{(1)} = 28.3$, $p < 0.001$; Figure 2). The estimated variance in log lesion length associated with pathogen isolate was 0.020 (0.0016–0.052), which accounted for 14.1% (1.1%–33.9%) of total variance in lesion length.

Of the six isolates used in the trial, only two isolates (GA10-SG and GA7) were able to form at least small lesions on all juniper genotypes tested (Figure 2). The same two isolates also had positive conditional modes, indicating that their predicted log lesion size was larger than the average prediction across all isolates (Figure S4). These results suggest isolates GA10-SG and GA7 are more virulent than the other four isolates and would be good candidates for use in future inoculation trials. We chose isolate GA7 for further work for practical reasons, as its rate of growth when cultured in laboratory conditions is considerably faster than GA10-SG.

3.1.2 | Broad-Sense Heritability of Disease Resistance in Juniper

Juniper shoots showed a range of lesion lengths, from little/no lesion development through to large lesions, with the largest lesion measuring 176 mm (Figure 2). There was significant variation between juniper genotypes in log lesion length (LRT $\chi^2_{(1)} = 66.7$, $p < 0.001$), with the estimated variance in log lesion

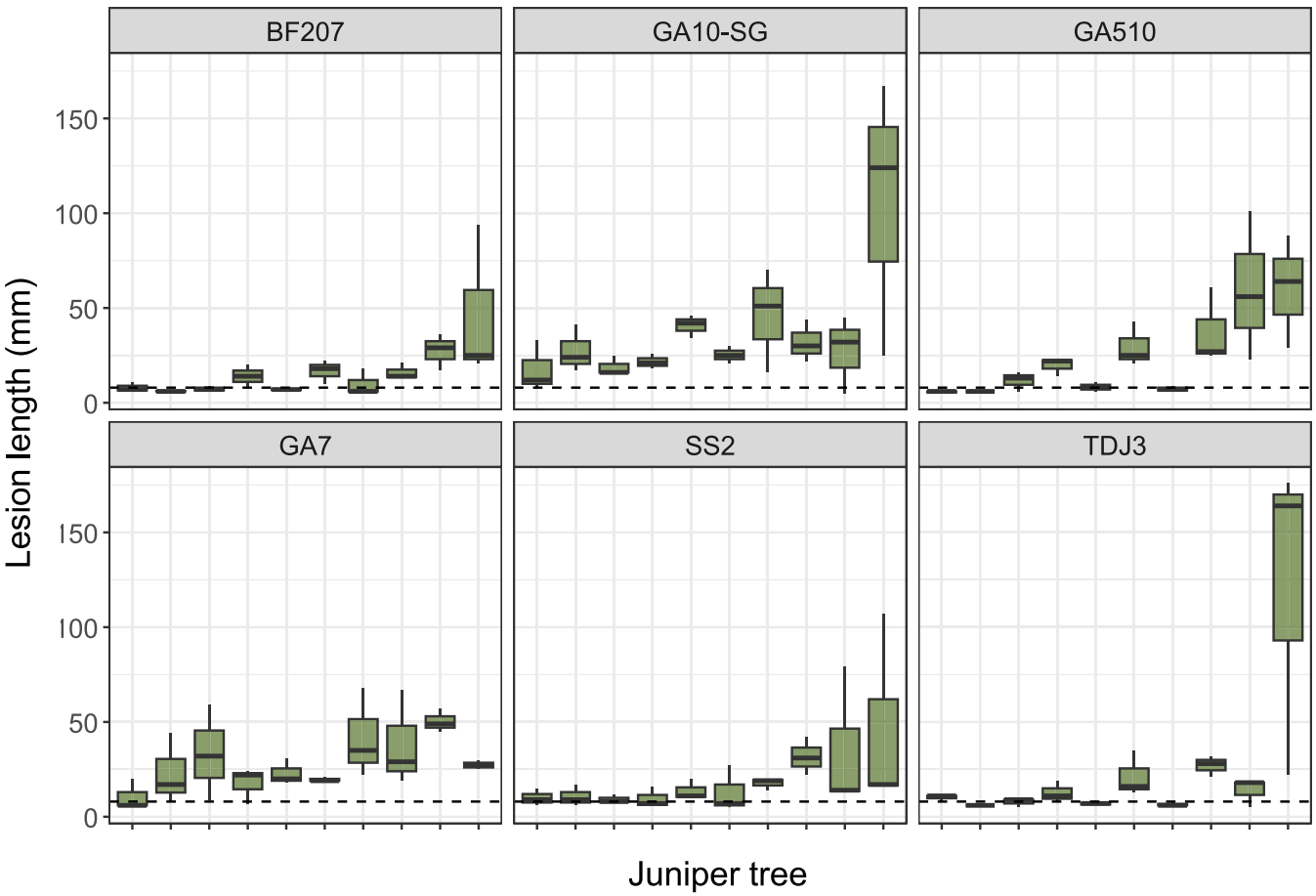

FIGURE 2 | Results of the excised shoot trial showing the distribution of lesion lengths for each *Phytophthora austrocedri* isolate and juniper tree combination, with each panel representing a different pathogen isolate. Dashed line indicates the size of the discoloration on the negative controls that was due to the inoculation process itself. Lesion sizes overlapping the dashed line were therefore visually indistinguishable from the negative controls.

associated with tree genotype being 0.048 (0.013–0.11). Broad-sense heritability of lesion length across all *P. austrocedri* isolates was estimated as 0.35 (0.13–0.57; Table 1).

When added to the model, the *Tree:Isolate* interaction term explained a significant amount of variation (LRT $\chi^2_{(1)} = 11.4$, $p < 0.001$). However, the parametric bootstrapping performed poorly on individual estimates for this model, suggesting insufficient data for the complex random effect structure. Nevertheless, the joint proportion of variation explained by both the *Tree* and *Tree:Isolate* terms was more stable, and the broad-sense heritability of lesion length when individuals were infected by the same *P. austrocedri* isolate could therefore be estimated as 0.54 (0.33–0.71; Table 1).

3.2 | Progeny-Provenance Trial

There was considerable variation between juniper trees in lesion size 5 weeks after inoculation, from little/no lesion development through to large lesions (Figure 3), with the largest lesion being 182 mm long. Of the 306 trees included in the trial, 231 (75.5%) were classified as "lesion present" (lesion length > 15 mm) while 75 trees (24.5%) were classified as "lesion absent" (lesion length ≤ 15 mm). Mean lesion length for "lesion present" trees was 112 mm, and median lesion length was 123 mm. Mean lesion length for "lesion absent" trees was 8 mm and the median was 7 mm.

Negative controls did not develop lesions, and all positive controls developed large lesions (all ≥ 98 mm; mean

TABLE 1 | Estimates of different kinds of heritability of resistance to *Phytophthora austrocedri* in juniper. For the excised shoot trial, the estimates were generated by maximum likelihood and the parentheses show 95% confidence intervals generated by parametric bootstrapping. For the progeny-provenance trial, estimates are medians of the posterior distribution and parentheses show 95% credible intervals.

Trial description	Type of heritability	Pathogen isolates	Trait	Estimate of heritability
Excised shoot with six isolates	Broad-sense (H^2)	Across isolates	Log lesion length	0.35 (0.13–0.57)
		Within isolates		0.54 (0.33–0.71)
Progeny-provenance whole tree	Narrow-sense (h^2)	Single isolate	Lesion length	0.44 (0.08–0.86)
			Lesion presence/absence (latent scale)	0.92 (0.24–1.60)
			Lesion presence/absence (observed scale)	0.43 (0.10–0.80)

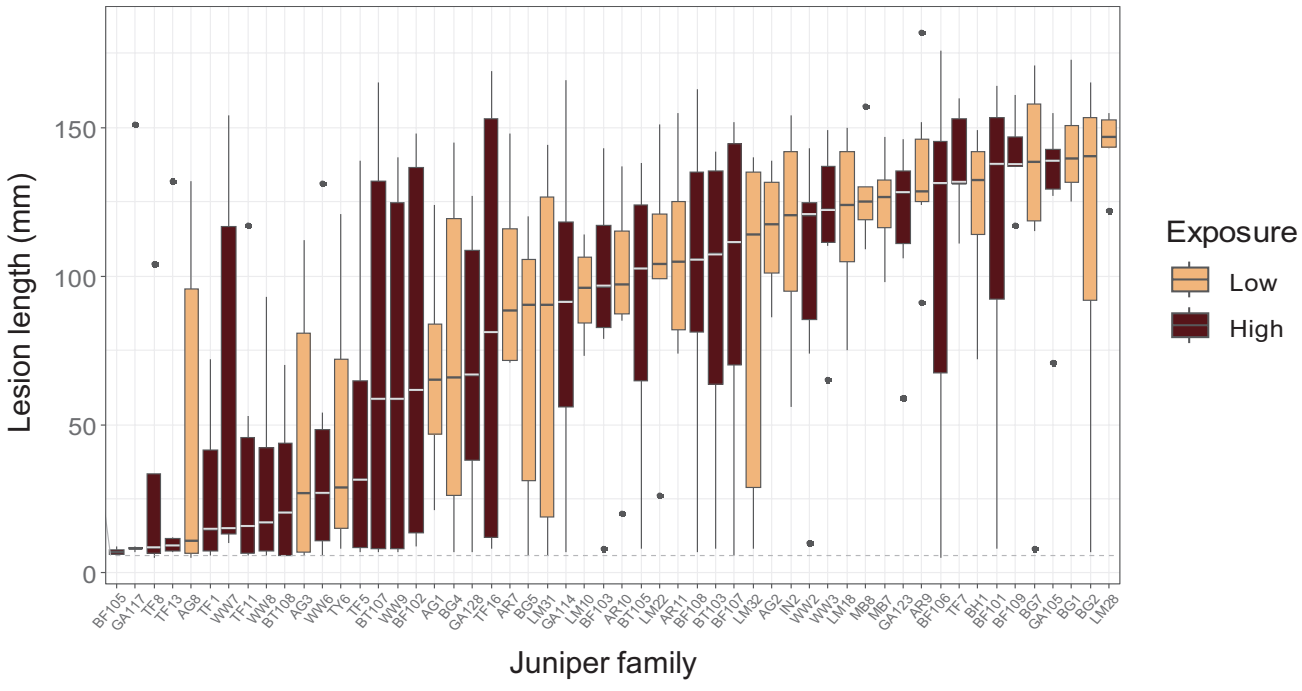

FIGURE 3 | Results of the whole tree progeny-provenance trial showing the distribution of lesion size by family. Boxplots are colour-coded according to the level of previous exposure to *Phytophthora austrocedri* in the population: High exposure to *P. austrocedri*, meaning extensive mortality has already taken place at the population site, or low exposure to *P. austrocedri*, meaning no evidence of mortality associated with the pathogen. Dashed line indicates the size of the discoloration on the negative controls that is due to the inoculation process itself.

122.5 ± 7.4 mm). *Phytophthora austrocedri* is notoriously difficult to isolate even from active infections; however, a subset of re-isolation attempts (29/56 = 52%) were successful. Furthermore, qPCR assays confirmed the presence of *P. austrocedri* DNA in all of the subset of 30 inoculated trees tested (Table S4).

3.2.1 | Effect of Tree Size

There was no evidence that stem width (mm) had an effect on either the probability of lesion development ($\beta = 0.0004$ [−0.068–0.061], $p = 0.99$) or the length of lesion (mm) if present ($\beta = -0.26$ [−1.9–1.4], $p = 0.74$), and its inclusion increased the model's DIC, suggesting a poorer fit. Stem width was therefore excluded from the model. Similarly, there was no evidence that tree height (cm) affected the probability of lesion development ($\beta = -0.001$ [−0.002–0.0004], $p = 0.28$). However, there was a significant positive association between tree height and lesion length ($\beta = 0.31$ [0.07–0.57], $p = 0.014$).

There was no significant variation in tree height between families after accounting for population and block (LRT $\chi^2_{(1)} = 0.19$, $p = 0.66$); however, populations did vary significantly in tree height (LRT $\chi^2_{(1)} = 31.9$, $p < 0.001$). This difference is most likely driven by the inclusion in the trial of juniper trees from Arran, which belong to a different subspecies (*Juniperus communis ssp. nana*) that has a small, procumbent growth form (Thomas et al. 2007). Because of these clear differences in growth form between populations, the effect of tree height was allowed to vary between populations by fitting a random slopes model.

3.2.2 | Estimates of Intraclass Correlation Coefficients and Narrow-Sense Heritability

The estimates of the variance components and ICC for all random effects included in the model are shown in Table 2. The posterior distributions of both *Block* and *Population* showed strongly skewed distributions bordering zero, which is reflected in how the confidence intervals for these parameter estimations approach zero (Table 2). Because estimates of variances cannot be negative, this can be interpreted as a lack of evidence of *Block* or *Population* explaining a significant amount of variation in the probability of lesion development or in lesion length if present. In contrast, the posterior distributions of *Family* were clearly removed from zero, suggesting *Family* does explain a significant amount of variation in both the probability of lesion development and lesion length.

Estimates of h^2 were significantly above zero for both traits: 0.44 (0.08–0.86) for lesion length and 0.92 (0.24–1.6) for lesion development presence on the latent trait scale (Table 1). The estimate of h^2 for the probability of developing a lesion (the observed data scale) was 0.43 (0.10–0.80).

3.2.3 | Genetic Covariance Between Traits

The best estimate for within-family covariance between the probability of lesion development (on the latent scale) and the length of lesion if present was positive (6.1 [−1.4–15.1]). However, as the 95% credible intervals overlap zero, we cannot be confident in this positive association between the two traits. Similarly, the genetic correlation between the two traits was estimated to be 0.50 (−0.03–0.97). Although this suggests there may be a positive genetic correlation between the probability of developing a lesion and the length of lesion if present, there is considerable uncertainty in this estimate, as the confidence intervals are very wide and overlap zero.

3.2.4 | Effect of Previous Exposure to *Phytophthora austrocedri*

There was a significant difference in the probability of lesion development between populations with high levels of previous exposure to *P. austrocedri* versus populations with no evidence of exposure to *P. austrocedri* ($\beta = 1.0$ [0.23–1.9], $p = 0.017$). However, there was no difference between high exposure versus low exposure populations in the length of lesion when present ($\beta = 5.8$ [−9.1–22.9], $p = 0.44$). After marginalizing all random effects, the predicted probability of developing a lesion was over 85% in the case of low exposure populations, and just 65% in the case of high exposure populations (Figure 4). Conversely, predicted lesion length did not vary significantly according to the level of previous exposure to the pathogen in the source population (Figure 4).

TABLE 2 | Results of the model estimating variance components and intraclass correlation coefficients (ICC) in the progeny-provenance trial. Estimates are the medians of the posterior distributions. Parentheses show the 95% credible intervals. Estimates for lesion presence/absence are on the latent scale.

	Lesion presence/absence		Lesion length	
	Variance	**ICC**	**Variance**	**ICC**
Family	0.58 (0.14–1.30)	0.30 (0.08–0.52)	234 (33–519)	0.15 (0.03–0.28)
Population	0.30 (6×10^{-8}–2.0)	0.16 (2×10^{-8}–0.55)	22 (3×10^{-6}–200)	0.014 (2×10^{-9}–0.11)
Tree_height:Pop	10^{-5} (10^{-11}–10^{-4})	6×10^{-6} (3×10^{-12}–5×10^{-5})	0.006 (2×10^{-9}–0.04)	4×10^{-6} (10^{-12}–2×10^{-5})
Block	0.02 (10^{-8}–0.29)	0.01 (6×10^{-9}–0.13)	64 (4×10^{-5}–398)	0.04 (3×10^{-8}–0.21)
Residual	1	NA	1261 (967–1501)	NA

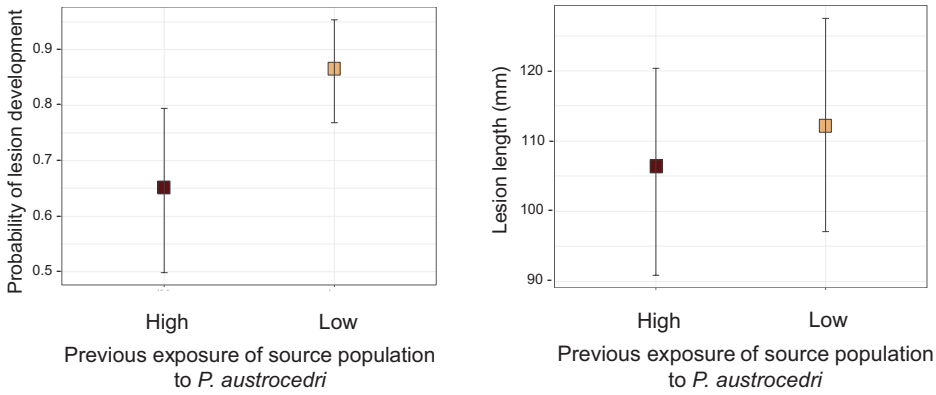

FIGURE 4 | Predicted values for the probability of lesion development on the observed data scale (left) and lesion length (right), depending on the level of previous exposure to *Phytophthora austrocedri* in the population. High exposure was defined as extensive mortality having already taken place at the population site; low exposure was defined as there being no evidence of mortality associated with the pathogen. Predictions were made after marginalizing all random effects. There was a significant difference in the probability of lesion development between high exposure and low exposure populations, but no difference in lesion length if present.

4 | Discussion

4.1 | Evidence for Both Qualitative and Quantitative Resistance

The distribution of lesion lengths across all individuals suggests both qualitative and quantitative resistance to *P. austrocedri* may be present in UK juniper populations (Figure 1). While most trees developed lesions, variation in lesion length indicated that some susceptible trees were able to slow down the rate of pathogen spread to some extent (quantitative or incomplete resistance). Meanwhile, a subset of trees showed little or no lesion development, suggesting they were able to prevent pathogen establishment altogether (qualitative or complete resistance).

The presence of both qualitative and quantitative resistance has also been reported in the closely related pathosystem of *P. lateralis* and Port-Orford-cedar (*Chamaecyparis lawsoniana*; Sniezko et al. 2020). In that study, qualitative resistance was proposed to result from a single major gene, based on Mendelian segregation patterns in progeny survival (Sniezko et al. 2020). However, in our case, the number of progeny per family was insufficient to test for Mendelian segregation patterns.

Some inferences could potentially be made from the positive within-family covariance between the two resistance traits, which could indicate that the probability of lesion development shares an underlying genetic basis with length of lesion when present. However, a positive covariance between resistance traits could also indicate co-occurrence of these traits in genotypes for other reasons; for example, selection imposed by *P. austrocedri* could increase the frequency of both types of resistance in the same families. Furthermore, the 95% credible intervals of the estimated covariance overlap with zero, meaning we cannot be confident in the positive association between the two resistance traits.

Overall, while the results presented here show a distinction between qualitative vs. quantitative resistant phenotypes in juniper, further work is needed before any conclusions can be drawn about the underlying genetic architecture of resistance in this pathosystem.

4.2 | Heritability of Resistance

The best estimate of narrow-sense heritability for lesion length was moderate to high (0.44), while the best estimate for lesion presence (on the latent scale) was very high (0.92). To put these values in context, Graham et al. (2018) reviewed genetic variation in *Phytophthora* resistance across multiple tree pathosystems and reported heritability estimates ranging from 0.34 to 0.9 for resistance to pathogens affecting roots and shoots. More recently, the heritability of survival in eastern white pine inoculated with *P. cinnamomi* was estimated at 0.44 (Frampton et al. 2018), and the heritability of survival of alder inoculated with either *P. uniformis* or *P.* × *alni* was estimated to be 0.32 and 0.1, respectively (Redondo et al. 2020).

The estimate of heritability of lesion presence on the observed data scale reflects the heritability of the probability of developing a lesion. This estimate was lower than the estimate on the latent scale, which is always expected to be the case (De Villemereuil et al. 2016). Although the transformation onto the observed data scale provides insight into how much variation in the phenotypic (observed) trait is attributable to additive genetic variance, this estimate is likely to be a poor predictor of response to selection (De Villemereuil et al. 2016). By contrast, the latent-scale estimate of heritability can be used for evolutionary predictions (De Villemereuil et al. 2016), and is therefore a more relevant parameter when assessing juniper's adaptive potential.

Several caveats should be considered when interpreting the estimates of heritability of resistance traits presented here. Firstly, the experiments were conducted under partially controlled glasshouse conditions, which is likely to substantially reduce environmental variation compared to natural settings. Because heritability reflects the proportion of total phenotypic variance attributable to genetic factors, any additional variation in phenotypic resistance caused by variation in the environment would

result in lower heritability estimates if measured in natural populations. Secondly, heritability in this study was estimated across multiple populations, whereas in natural systems it is typically within-population heritability that is most relevant for predicting evolutionary response to selection. This distinction is important because differences among populations can inflate estimates of genetic variance and, consequently, heritability (Falconer and Mackay 1996).

Finally, the 95% credible intervals around the heritability estimates were relatively wide, indicating there is considerable uncertainty surrounding these estimates. This reflects the complexity of the random effects structure relative to the available sample sizes; precise estimates of quantitative genetic parameters generally require thousands rather than hundreds of individuals. Nevertheless, it is worth noting that the estimates of heritability of lesion length in the two separate inoculation trials (when estimated for a single isolate) were similar, providing an independent corroboration of the result.

Ultimately, many of these limitations are inherent to studying ecologically important but experimentally challenging tree-pathogen systems. The absence of a reliable, scalable inoculation method and the long generation times of woody hosts such as juniper impose practical constraints that make large-scale quantitative genetic studies difficult. However, despite these challenges, the primary aim of this study was not to obtain highly precise estimates of quantitative genetic parameters, but rather to determine whether resistance to *P. austrocedri* that had previously been observed in juniper (Green et al. 2020) has a heritable genetic basis. The results presented here provide strong evidence that it does.

4.3 | Genotype-By-Genotype Interaction Between Host and Pathogen

In addition to *P. austrocedri* isolates varying in virulence across juniper genotypes, there was also a significant genotype-by-genotype (GxG) interaction between pathogen isolate and host genotype. For example, isolate TDJ3 failed to produce lesions on many of the more resistant genotypes, but caused the largest lesions on a particularly susceptible genotype (Figure 2). Such GxG interactions are expected under the gene-for-gene model of R-gene-mediated resistance (Flor 1971; Dodds 2023), but they have also been reported in cases of partial, quantitative resistance (e.g., Flier et al. 2003; Darvishzadeh et al. 2007).

An important consequence of the significant GxG interaction between isolate and genotype is a reduction in the estimate of heritability of resistance when measured across multiple pathogen isolates. Frampton et al. (2018) found that the interaction between *P. cinnamomi* isolate and pine host family was so strong that estimated heritability dropped from 0.44–0.57 to almost zero when calculated across just two pathogen isolates. The effect in the present study was less extreme, with still moderate levels of heritability maintained across six *P. austrocedri* isolates (Table 1). Nevertheless, this still suggests juniper may be less able to respond to *P. austrocedri* through adaptation if multiple pathogen genotypes are present within one host population.

4.4 | Evidence for Natural Selection in Juniper Populations

The difference in predicted probability of developing a lesion between juniper populations that have been highly exposed to *P. austrocedri* and those with no evidence of exposure (Figure 4) is likely a result of extensive mortality in exposed populations causing a shift in the relative frequency of resistant genotypes due to the removal of more susceptible genotypes. Thus, this is evidence that the invasive *P. austrocedri* is imposing rapid natural selection on juniper populations. This is an important result that suggests that, despite being previously naïve to *P. austrocedri*, juniper populations are demonstrating resilience through standing genetic variation in resistance that is effective enough to significantly increase their probability of survival in natural populations.

Evidence of natural selection in response to an invasive tree pathogen is rare, but has been reported in other systems. For example, Redondo et al. (2020) found significant differences in the frequency of resistance between exposed and unexposed alder populations in the case of *P. uniformis*, although not in the case of *P.* × *alni* (Redondo et al. 2020). Heritability of resistance in the latter case was low, which may explain the lack of evidence of natural selection. Several studies have also reported natural selection to be occurring in ash trees in response to *Hymenoscyphus fraxineus*, the causal agent of ash dieback, showing increased reproductive success in more resistant ash trees compared to more susceptible ash trees (Semizer-Cuming et al. 2019, 2021) and an age-related shift in frequency of multiple alleles putatively associated with resistance (Stocks et al. 2019; Metheringham et al. 2025).

By contrast, no significant difference in predicted values between exposed and unexposed populations was seen in the case of lesion length (Figure 4). There are several possible explanations for this. Firstly, variation in lesion length detected over a 5-week inoculation trial may have little relevance to long-term survival on timescales relevant to juniper. This would mean that, although some juniper genotypes appear to have some degree of quantitative resistance by slowing down the progression of *P. austrocedri*, they do still succumb to the pathogen, resulting in no measurable increase in the relative frequency of genotypes with more quantitative resistance. Alternatively, the lack of evidence of selection on lesion length could reflect differences in the temporal dynamics between the two resistance traits, as the lower heritability of lesion length could result in slower, less efficient selection. In this case, quantitative resistance would still be expected to increase in frequency over time in populations exposed to *P. austrocedri*.

4.5 | Implications for the Conservation of Vulnerable Juniper Populations in the Face of *Phytophthora austrocedri*

Based on the results presented here, we suggest the best management strategy for the conservation of juniper in the UK is to promote and enhance natural regeneration within each population, giving juniper populations the best chance to respond to the threat posed by *P. austrocedri* through natural selection

and adaptation. Unfortunately, rates of natural regeneration are known to be very low in many UK juniper populations, and developing reliable methods to enhance regeneration remains a major challenge (Thomas et al. 2007; Broome et al. 2017). Despite these practical difficulties, we make this recommendation for the following reasons:

1. Resistant genotypes are present in most populations. Resistant juniper individuals were detected in nearly all populations where more than two families were sampled, indicating that most populations possess standing genetic variation in disease resistance without the need for introducing resistant genotypes from other sources. The only exception was juniper sampled from the Isle of Arran, which belongs to a different subspecies (*J. communis ssp. nana*) and which, based on the results of this study, may be universally susceptible. Fortunately, *P. austrocedri* has not yet been detected on Arran, and every effort should be made to prevent its introduction.
2. Both qualitative and quantitative resistance are present and heritable. Juniper populations contain heritable genetic variation for both qualitative and quantitative resistance, providing the raw materials necessary for adaptation to *P. austrocedri*. Although further work is required to determine the genetic architecture underlying these resistance phenotypes, the coexistence of both types of resistance may also enhance its durability and robustness against pathogen evolution (McDonald and Linde 2002; Brown 2015; Sniezko and Liu 2023).
3. There is evidence that natural selection is occurring. The significant difference in predicted probability of developing a lesion between populations based on levels of prior exposure to *P. austrocedri* suggests natural selection is already acting in exposed populations, and that resistance is effective at increasing the probability of host survival in natural field conditions.
4. Not all *P. austrocedri* genotypes are equal. Not only do *P. austrocedri* isolates vary in virulence, but there is also a significant GxG interaction between host and pathogen that reduces estimates of heritability of resistance when calculated across multiple pathogen isolates. This means that it is important not to transport *P. austrocedri* within and between sites, even in exposed populations, as juniper genotypes resistant to one genotype of *P. austrocedri* may be less resistant or even fully susceptible to a different pathogen genotype. *Phytophthora austrocedri* is soil- or waterborne, and therefore has limited natural dispersal capacity (Riddell et al. 2020). However, many *Phytophthora* species, including *P. austrocedri*, are frequently detected in nurseries and nursery-grown plants (Parke et al. 2014; Prigigallo et al. 2015; Green et al. 2025). Active planting of nursery-grown resistant juniper genotypes therefore carries a real risk of introducing *P. austrocedri* into unexposed populations or introducing new *P. austrocedri* genotypes into already-affected populations (Donald et al. 2021).

In summary, the conservation of juniper in the UK will be best served by promoting natural regeneration within populations while limiting the spread of *P. austrocedri* between populations, giving juniper the greatest opportunity to respond to this invasive tree pathogen through natural selection and adaptation.

Acknowledgements

The authors would like to thank Annika Perry for her help in establishing the juniper resources used in this study and Heather Dun for her assistance during the inoculation trials.

Funding

This work was supported by a NERC Doctoral Training Partnership grant (NE/S007407/1) and the Science and Innovation Strategy (SIS), which is jointly funded by the Forestry Commission, Defra, Scottish Forestry and the Welsh Government.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are openly available on Zenodo at https://doi.org/10.5281/zenodo.19128776.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** Representative pictures showing range of lesion sizes underneath the bark in juniper shoots inoculated with *Phytophthora austrocedri* 5 weeks post-inoculation. Length of lesion in each case is indicated in the bottom right of the picture. **Figure S2:** Temperature (above) and relative humidity (below) in the greenhouse over the period of the whole-tree inoculation trial. **Figure S3:** Representative pictures of different degrees of lesion development. Length of lesion in each case is indicated in the bottom right of the picture. **Figure S4:** Conditional modes for the six *Phytophthora austrocedri* isolates used in the excised shoot inoculation trial, showing the difference between the predicted response for each isolate relative to the average predicted response across all isolates. **Table S1:** Location and date of isolation for the *Phytophthora austrocedri* isolates used in the inoculation trials. **Table S2:** Location of the population, number of families and individuals included in the seed-grown progeny-provenance trial, and whether the population was classified as high exposure to *Phytophthora austrocedri* (extensive mortality already taken place) or low exposure to *P. austrocedri* (no evidence of mortality associated with the pathogen). **Table S3:** Parameter estimates when using a cutoff of 10, 12, 15, 18 or 20 mm in lesion length for defining lesion development as "present" or "absent". **Table S4:** Results of qPCR assays on DNA extractions from phloem/cambium tissue.