

# 1 Demographic and genetic impacts of powdery 2 mildew in a young oak cohort

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## 14 **ABSTRACT**

15 The demographic and genetic impacts of powdery mildew on the early stages of an oak  
16 population were studied in an *ad hoc* field design with two disease exposures. This enabled a  
17 detailed phenotypic monitoring of 1,733 emerging individuals from 15 progenies over nine  
18 years, and the genotyping of 68% of them. The pathogen induced high levels of seedling  
19 mortality several years after sowing, associated with reduced growth and capacity to  
20 overwinter. The probability of juvenile survival could be predicted from mean disease severity  
21 in early years and acorn weight. Fast-growing families showed the highest survival rate under  
22 both natural and protected disease exposure. Correlatively, no equalizing effect of increased  
23 powdery mildew pressure on the relative contribution of mother trees to the next generation  
24 could be detected. Contrary to a possible trade-off hypothesis between growth and defense,  
25 family height potential was not negatively related to disease resistance across the studied oak  
26 mother trees. Overall, our results suggest that in *Quercus robur* natural populations, infection  
27 levels (related to resistance *sensu stricto*) may be less determinant than growth-related  
28 tolerance to infection for the fate of seedlings. However, an equalizing effect of powdery  
29 mildew on relative oak genotype performances cannot be excluded at later stages since such  
30 an effect was already visible on height. Average genomic diversity was not significantly  
31 affected by mortality associated with powdery mildew. However, our study brings support to

32 a deleterious effect of very low individual heterozygosity on the probability of survival across  
33 the different families. Finally, our study points to a few candidate genes for several fitness-  
34 related traits.

35

36 **Keywords:** *Erysiphe quercicola*; *Erysiphe alphitoides*; pedunculate oak; oak powdery mildew; oak  
37 regeneration; disease-diversity relationship; trade-off

39 Seedling establishment and early growth stages are crucial phases in the tree life cycle. Most forest  
40 tree species show a typical concave mortality curve, characterized by a very high juvenile mortality (*e.g.*,  
41 Harcombe 1987; Peñuelas et al 2007; Petit & Hampe 2005; Kelly 2002). Under natural conditions, both  
42 abiotic and biotic factors affect tree seedling survival, in addition to stochastic processes (*e.g.*, Shibata et  
43 al 2010; Petritan et al 2014; Martini et al 2019). Among biotic factors, many pathogens may affect seedling,  
44 and more generally juvenile survival. Seedlings and saplings are especially susceptible to pathogens due to  
45 their mostly non-woody tissues, both in roots and stems (Dominguez-Begines et al 2020; Jankowiak et al  
46 2022). For example, Augspurger (1984) reported that damping-off pathogens (soilborne fungi and  
47 oomycetes) accounted for the largest proportion of seedling deaths within the first year in several species  
48 of tropical forest trees.

49 By their negative effect on the individual fitness of their host (by definition), pathogens can strongly  
50 affect plant population demographic patterns. On the other hand, at community level, their positive role  
51 in maintaining between and within species diversity has received increasing support (Dobson & Crawley  
52 1994; Alexander 2010; Mordecai 2011; Bever et al 2015). The impact of pests and pathogens on seedlings  
53 has been extensively studied as a possible mechanism promoting tree species coexistence (maintenance  
54 of spatial diversity) in species-rich tropical forests. According to the Janzen-Connell model, species-specific  
55 herbivores and pathogens provide a frequency-dependent spacing (thus diversifying) mechanism by  
56 causing increased mortality of seedlings growing at a short distance from their mother tree (Janzen 1970;  
57 Connell 1971; Summers et al 2003). Many studies have provided support to this model both in tropical and  
58 temperate environments (Packer & Clay 2000; Bell et al 2006; Yamazaki et al 2008; Terborgh 2020),  
59 although the magnitude and generality of Janzen-Connell effects are still a matter of debate (Song et al  
60 2021). Such frequency-dependent and density-dependent processes are especially important for  
61 specialized pathogens, as in Janzen-Connell effects, or in co-evolutionary dynamics at population level  
62 (Mundt et al 2008; Parker & Gilbert 2018; Burdon & Laine 2019). Pathogens may also affect competitive  
63 interactions between genotypes, between or within species, in a non-frequency dependent or density-  
64 dependent manner, by causing a differential cost on the fitness of the competing plants (Mundt et al 2008;  
65 Creissen et al 2016). For example, foliar diseases have a debilitating effect on highly infected seedlings,  
66 which may result in a competitive disadvantage in presence of less affected neighbors (Wiener 1990;  
67 Gilbert 2002; Power & Mitchell 2004). When the competitively dominant genotypes in the absence of  
68 disease experience a greater cost to disease than less competitive genotypes in the presence of pathogens,  
69 pathogens reduce fitness differences and therefore promote plant diversity (Mordecai 2011). This occurs  
70 when the fast growing/strongest competitors are the most vulnerable to pathogens (Summers et al 2003;  
71 Bever et al 2015; Cope et al 2021). The prevailing hypothesis in the literature to explain this negative  
72 correlation is the growth-defense trade-off concept, based on the premise that defense is costly thus  
73 requires allocation of resources at the expense of growth (Monson et al 2022). Growth-defense trade-offs  
74 have been reported at inter-and intra-specific level in many groups of plants under various environments,  
75 including for tree species (Lind et al 2013; Heckman et al 2019; Kruger et al 2020; Cope et al 2021).

76 Studies on the impact of pathogens on plant populations have been extensively performed in an  
77 agricultural context, in relation to yield losses (*e.g.*, Savary et al 2019). Studies in natural systems are fewer,  
78 and mainly focused on some model systems (Burdon & Thrall 2014), *e.g.*, flax rust (Thrall et al 2012),  
79 *Arabidopsis* pathogens (Creissen et al 2016), anther smut of *Silene* (Bernasconi et al 2009), *Plantago*  
80 powdery mildew (Laine 2004; Safdari et al 2021). In this study, we aimed to characterize the impacts of  
81 powdery mildew on fitness-related traits and genetic diversity during the early life-stages of an oak cohort.  
82 Powdery mildew is one of the most important diseases on temperate oaks in Europe, in particular

83 pedunculate oak, *Quercus robur* (Mougou et al 2008; Lonsdale 2015). In Europe, powdery mildew was  
84 shown to be associated with a complex of cryptic (morphologically similar) species, of which  
85 *Erysiphe alphitoides* (Griffon & Maubl.) U. Braun & S. Takam is nowadays the most prevalent throughout  
86 Europe, often in mixture with *Erysiphe quercicola* in southern Europe S. Takam. & U. Braun and with  
87 *Erysiphe hypophylla* (Nevod.) U. Braun & Cunningt. in northern Europe (Mougou et al 2008; Desprez-  
88 Loustau et al 2018; Gross et al 2021). Demeter et al (2021) suggested that powdery mildew could be one  
89 of the major factors involved in regeneration failures in pedunculate oak throughout Europe. Seedlings and  
90 young trees, with a relatively high amount of young, succulent, fast growing tissues, are especially  
91 susceptible to disease (Pap et al 2012; Marçais & Desprez-Loustau 2014). A significant negative effect of  
92 powdery mildew on height and radial growth of oak saplings was demonstrated in comparison with  
93 controls protected by fungicide applications (Pap et al 2012; Desprez-Loustau et al 2014). Powdery  
94 mildew, as an obligate parasite, derives nutrients produced by plant photosynthesis to its own benefit  
95 thanks to specialized feeding structures (called haustoria) that penetrate into living cells of the leaf  
96 parenchyma~~Powdery mildew, as an obligate parasite, develops haustoria (specialized structures) in living~~  
97 ~~cells of the leaf parenchyma and derives nutrients produced by plant photosynthesis to its own benefit~~  
98 (Hewitt & Ayres 1976). As a consequence, several types of damage have been described: reduced net  
99 assimilation rate, reduced height and radial growth, greater susceptibility to frost (Hajji et al 2009; Marçais  
100 & Desprez-Loustau 2014; Pap et al 2014; Bert et al 2016). However, how the impacts of powdery mildew  
101 scale up at oak population level have little-rarely been explored.

102 The spatial, demographic, and genetic structure of oak populations (especially *Q. robur* and *Q. petraea*)  
103 has nevertheless received much attention owing to the ecologic, cultural and economic importance of  
104 these species in Europe (e.g., Kremer & Petit 1993; Streiff et al 1998; Gömöry et al 2001; Vakkari et al 2006;  
105 Kesić et al 2021). Overall, oak populations exhibit a high level of genetic diversity, with no significant or  
106 little differences among cohorts of different ages in the same stand~~Overall, a high genetic diversity within~~  
107 ~~oak populations is found, with no significant or little changes of its level or its differentiation among cohorts~~  
108 ~~of different ages~~ (Vranckx et al 2014a from adults to established seedlings; Gerzabek et al 2020 from  
109 emergence to 3-year old seedlings). In a natural context, the various biotic and abiotic factors affecting oak  
110 seedling recruitment can vary in space and time (e.g., Crawley & long 1995; Alberto et al 2011; Gerzabek  
111 et al 2020). The diversity and fluctuation of selective pressures acting on different genetic components  
112 have been proposed as possible explanations for the maintenance of genetic diversity in plant populations  
113 (Ennos 1983; Delph & Kelly 2014).

114 Genetic changes in plant populations under pathogen pressure have been reported in a few  
115 pathosystems~~By contrast, under conditions favoring a constant directional selection associated with the~~  
116 ~~deleterious effect of a pathogen on the susceptible genotypes, a significant change in genetic diversity of~~  
117 ~~host populations may be expected, and was reported for some pathosystems in some contexts~~ (Thrall et al  
118 2012). In this case, with alleles being selected due to their positive association with a greater resistance  
119 and/or tolerance to the disease, it may be possible to identify some of these variants using an association  
120 genetics approach. Genome Wide Association Studies (GWAS) are a powerful tool to link phenotypic  
121 variation with genetic polymorphisms, allowing the identification of the underlying biological mechanisms  
122 (Korte & Farlow 2013; Tibbs Cortes et al 2021). High quality genomic resources are now available for  
123 *Q. robur* (Lepoittevin et al 2015; Plomion et al 2018; Lang et al 2021). Both genetic variation among families  
124 (Desprez-Loustau et al 2014) and putative candidate genomic regions for oak susceptibility to powdery  
125 mildew (Bartholomé et al 2020) were previously demonstrated in independent studies ~~(Desprez-Loustau~~  
126 ~~et al 2014; Bartholomé et al 2020)~~. Together with a very high genomic diversity within oak populations and  
127 a rapid decay of linkage disequilibrium among variants across the oak genome (Lang et al 2021), these  
128 species characteristics provide an advantageous setting for performing GWAS.

129 Our general objective was to characterize the demographic and genetic impact of powdery mildew in  
130 the early stages of an oak population. We used an original experimental field design with two levels of  
131 powdery mildew exposure and a half-sib family genetic structure, that we analyzed with a large range of  
132 methods, in order to address the following questions:

133 1. How does powdery mildew affect juvenile survival? Phenotypic monitoring was carried out during  
134 the first nine years after sowing. We analyzed the effect of powdery mildew on the probability of seedling  
135 survival with various logistic regression models, and used structural equation modelling (SEM) to describe  
136 the multiple relationships between the measured phenotypic variables and survival.

137 2. Does the impact of powdery mildew, in terms of survival, vary among oak families, *i.e.*, does powdery  
138 mildew differentially affect the reproductive success of different oak mother trees? In particular, do the  
139 families performing best (*i.e.*, with greatest survival and growth) in conditions of low powdery mildew  
140 pressure also perform best in conditions of high powdery mildew pressure? We hypothesized that seedling  
141 and juvenile survival is strongly affected by early growth, this trait itself being sensitive to both maternal  
142 effects such as those due to acorn weight and average family or individual genotypic effects, but that  
143 growth could be negatively correlated with resistance to the pathogen (*i.e.*, associated with a growth-  
144 resistance trade-off).

145 3. As a consequence, does powdery mildew reduce fitness differences of mother trees, measured by  
146 the mean survival of their progenies, *i.e.*, has powdery mildew an equalizing effect? If so, is the surviving  
147 population more or less diverse in terms of family composition under high powdery mildew pressure than  
148 under low disease pressure?

149 4. Does powdery mildew impact the genetic diversity of the oak population, not only in terms of family  
150 composition? In order to address this question, a large number of emerging seedlings were genotyped at  
151 several hundred SNPs and heterozygosity statistics were calculated. The genetic diversity was then  
152 compared in the surviving and initial populations under the two contrasted disease pressures.  
153 Furthermore, we tested whether possible genetic changes could be associated with a difference in  
154 individual heterozygosity between dead and living seedlings, in agreement with the HFC (Heterozygosity-  
155 Fitness-Correlations) hypothesis. Specifically, are the surviving populations more genetically heterozygote  
156 than the initial populations, especially under high disease pressure? In order to answer such questions, a  
157 large number of emerging seedlings has been genotyped at several hundred SNPs. We tested possible  
158 genetic changes associated with a difference in individual heterozygosity between dead and living  
159 seedlings, as previously reported in one oak population under stressful conditions (Vranckx et al 2014b)?).

160 5. Finally, given our experimental setting with a known family structure, can we detect significant  
161 genetic associations between some loci and seedling survival or other related traits (growth, infection)?

162

## 163 **Methods**

### 164 **Experimental design**

165 The experimental design and trait definitions were thoroughly described in the phenotypic monitoring  
166 study during the first three ~~first~~ years (Desprez-Loustau et al 2014). Briefly, the progeny of 15 isolated (*i.e.*  
167 with no overlapping canopies) oak trees (*Q. robur*) was collected in 2008 in Cestas, France. *Q. robur* is a  
168 diploid, monoecious, wind-pollinated tree species, with a highly outcrossing breeding system; it is a light-

169 demanding species (especially at juvenile stages) with a moderate tolerance to drought, compared with  
170 other oaks such as *Q. petraea* (Eaton et al 2016). We thus considered a population made of 15 open-  
171 pollinated half-sib families. The weight of each acorn was recorded for its importance on the initial seedling  
172 developmental stage (Sánchez-Montes de Oca et al 2018). Acorns were then sown in April 2009 on a 10 x  
173 10 cm grid in a field design with 9 unit plots, each containing 296 acorns, and distributed in 3 blocks  
174 (Supplementary material Figures S1 and S2). The field design was located in the INRAE experimental  
175 domain in Pierroton, Cestas, France. The unit plots were randomly attributed to one of two powdery  
176 mildew exposures: either Natural or Protected, *i.e.*, with a protection provided by myclobutanil (Dow  
177 AgroSciences, Sophia Antipolis, France), a fungicide authorized for usage in nurseries. There were six unit  
178 plots with natural exposure (corresponding to the pooled “Medium” and “High” disease treatments  
179 described in Desprez-Loustau et al 2014, which did not differ much in seedling infection and mortality) and  
180 three with fungicide (myclobutanil, a systemic fungicide which inhibits the ergosterol biosynthesis of the  
181 fungus) application. Although fungicide application limits the level of infection, it does not completely  
182 prevent the disease on treated trees. Acorns from different families were randomly distributed among  
183 plots, with 173 acorns per family on average (minimum=118; maximum=285; Supplementary material  
184 Figure S2).

185 At the end of each growing season from 2009 to 2012, survival was noted and height was measured for  
186 each individual. In following years, survival and height were assessed in early spring, a few weeks after  
187 budburst. Tree height was defined as the height of the highest living bud (*i.e.*, with leaves). Apical bud  
188 mortality occurred in some years, *i.e.*, the upper stem and branches did not show bud burst, resulting in a  
189 negative net annual height growth (Desprez-Loustau et al 2014).

190 Powdery mildew infection was monitored and scored on several occasions each year, particularly in the  
191 first years. To characterize annual disease severity, we used data corresponding to the highest annual  
192 infection score (generally from the last assessment at the end of the season, in September or October, but  
193 sometimes earlier, depending on the year's powdery mildew epidemic dynamic). Powdery mildew infection  
194 was assessed at different times across years, especially in the first years, depending on the powdery mildew  
195 epidemics. For example, in 2011, early and severe epidemics occurred, resulting in premature defoliation.  
196 Disease monitoring was therefore stopped in July. Disease severity assessments are therefore not directly  
197 comparable between years. No assessment was done in 2014 and 2015, during which infection was low.  
198 Powdery mildew infection was estimated visually by trained observers as a percentage of total infected  
199 leaf area for each individual. From the molecular analysis of various samplings in the experimental field, in  
200 different years (206 analyses in total), it was confirmed that the most prevalent species was *E. alphitoides*  
201 (more than 90% samples). *E. quercicola* was the other species detected, albeit at a much lower prevalence.

202 A late frost occurred in spring 2013, resulting in leaf damage in some seedlings. The occurrence of such  
203 damage was recorded as present/absent for each seedling. The details of the variables used in the  
204 statistical analyses are described in the Supplementary material, Table S1.

205

## 206 **Statistical analyses**

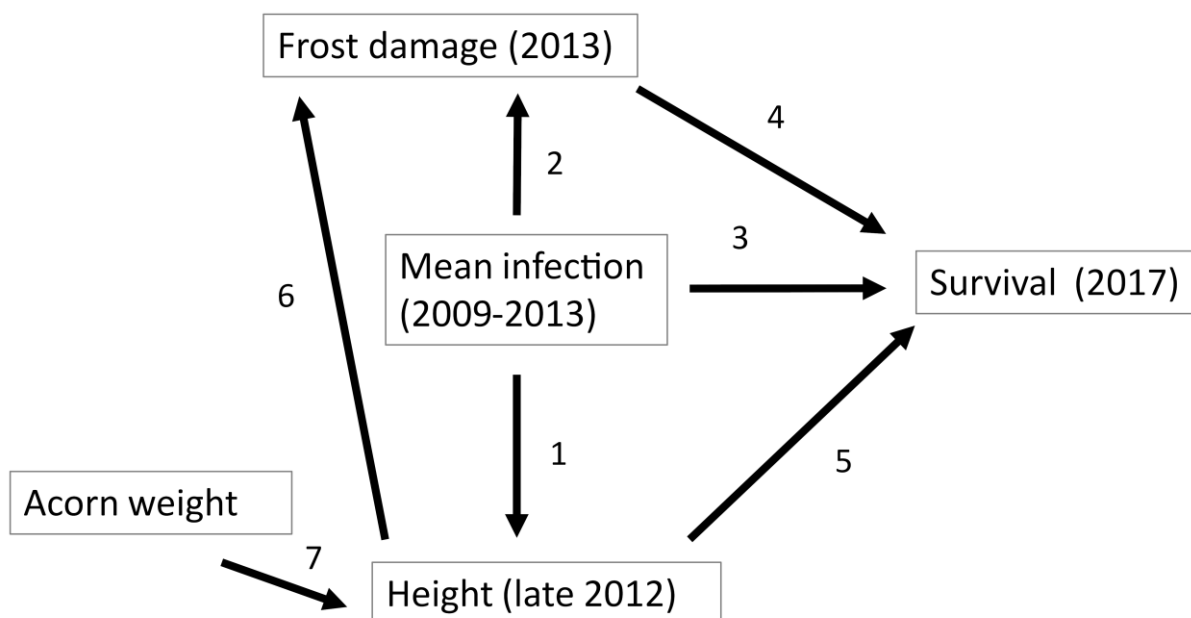
### 207 *Logistic and structural equation modelling for analysis at the individual level*

208 In order to explore the impacts of powdery mildew on survival, we used two statistical approaches.

209 First, logistic models were used to test the effects of different variables (see Supplementary material,  
210 Table S2) on tree survival (“Survival (2017)”, dead or living). In the first and simplest model, two explanatory  
211 variables of survival were included: “Acorn weight” and “Powdery mildew exposure” (Natural exposure or  
212 Protected by fungicide, Model 1). In order to further detail the powdery mildew effects, we replaced the  
213 exposure variable by a quantitative variable corresponding to the mean disease severity over the first five  
214 years (“Mean infection (2009-2013)”, Model 2). Another model was also run with seedling height at the  
215 end of the first growing season in place of acorn weight (“Height in 2009”, Model 3). The binary (*i.e.*,  
216 yes/no) variable “Frost damage (2013)” was then added to Model 1 (Model 4). Finally, a full logistic model  
217 of survival included previously studied factors (powdery mildew exposure, acorn weight and frost damage),  
218 with a family effect and an interaction effect between powdery mildew exposure and family (Model 5).

219 Second, we used Structural Equation Modelling (SEM) in order to estimate multiple and interrelated  
220 dependencies among measured phenotypic variables and survival. Based on a pre-defined causal model,  
221 the SEM method gives quantitative estimates of direct and indirect effects of several inter-correlated  
222 variables on a variable of interest, according to different “paths”. Standardized coefficients (*i.e.*,  
223 relationships expressed in terms of standard deviations) are produced by the analysis, enabling the  
224 comparison of the relative strengths of the effects of different explanatory variables, along the different  
225 paths. In addition, the total effect of each explanatory variable on the variable of interest is broken down  
226 into its direct and indirect effects, according to the specified paths. In our case, the main objective was to  
227 understand how powdery mildew affects the final survival of seedlings (in 2017). The phenotypic variables  
228 used in SEM were the mean seedling infection over the first five growing seasons (2009-2013), the height  
229 at the end of 2012 and the presence of frost damage in early 2013. These variables were selected because  
230 mortality only started in 2014 thus phenotypic data needed for the model were available for almost all  
231 seedlings. We assumed that differences in infection and height in the first five years, as well as frost  
232 damage, were important determinants of subsequent survival. Moreover, susceptibility and growth  
233 expressed during the first five years are likely correlated with susceptibility and growth in subsequent  
234 years. Taking into account previous knowledge on powdery mildew, we constructed and tested a model  
235 with three “paths” (corresponding to potentially different mechanisms) relating disease severity (“Mean  
236 infection (2009-2013)”) to survival (“Survival 2017”, Figure 1).

237



238

239

### Figure 1: SEM Model of survival.

240

241 One path was an indirect effect of powdery mildew infection on survival through an effect on height.  
242 This was based on the assumption that infection has a direct (negative) effect on growth (as reported in  
243 Bert et al 2016), thus on height (arrow "1") and that seedling height is expected to have a direct effect on  
244 survival (arrow "5"). The second path was an indirect effect of powdery mildew on survival through a direct  
245 effect on frost damage ("Frost damage 2013", arrows "2"), and a direct effect of frost damage on survival  
246 ("Survival (2017)", arrow "4"). This path is consistent with previous observations on the same field  
247 experiment (Desprez-Loustau et al 2014) or made by other authors (reviewed in Marçais & Desprez-  
248 Loustau 2014) that suggested that powdery mildew infection could affect the cold hardening process of  
249 shoots at the end of the season, resulting in greater shoot mortality during winter. A third path was a direct  
250 effect of powdery mildew infection on survival (arrow "3") which may include toxic effects of the pathogen  
251 on its host or other effects not taken into account by the other paths. Finally, we included two other effects  
252 not related to powdery mildew infection: a direct effect of height on frost damage and one of acorn weight  
253 on height (arrows "6" and "7", respectively).

254

#### 255 *Analyses at family level*

256 Since the same 15 families were tested under both powdery mildew exposures (Natural *versus*  
257 Protected due to fungicide use), their relative performance in both environments could be compared based  
258 on family mean phenotypic value. In particular, the proportion of individuals having survived in each family  
259 is an estimate of one component of the reproductive success of their mother tree under each environment.  
260 Moreover, the family mean of each trait could be considered as an estimate of the genetic value of the  
261 corresponding mother tree. The relationship between family growth potential (*i.e.*, defined as the mean  
262 progeny height in a reduced disease environment provided by the Protected exposure) and family disease  
263 resistance (inversely related to mean progeny infection scores under natural conditions) can then be  
264 analyzed.

265 In order to assess temporal changes in the relative family composition of the surviving populations  
266 under both disease exposures, we calculated a Shannon Index in each plot and year as  $H' = -\sum p_i \cdot \log_2(p_i)$ ,  
267 with  $p_i$  the proportion of each family in the population.  $H'$  can vary between a maximum value of  $\log_2(N)$ ,  
268 with  $N$  the number of groups, if all groups have the same frequency (here  $N=15$  and  $H'_{\max}=3.91$ ), and a  
269 minimum value of 0 if the population is composed of a single entity.

270 All analyses were performed with the SAS software Version 9.4 (Copyright © 2013, SAS Institute Inc.,  
271 Cary, NC, USA), in particular the Logistic, GLM and Calis (for SEM) procedures (scripts and data can be found  
272 in the zenodo repository <https://doi.org/10.5281/zenodo.7517641>).

273

#### 274 **Genetic analyses**

##### 275 *Sample collection and DNA extraction*

276 Three to six leaves were sampled on each emerged seedling at the 15-leaf stage so as not to  
277 compromise the survival of individuals. Nine 9-mm-diameter leaf discs were cut off from the dried-leaves



278 for each individual and stored at -80°C in 96-well plates until DNA extraction. DNA was extracted using the  
279 Invisorb DNA plant HTS 96 kit (Invitex, Germany). We followed the manufacturer instructions except that  
280 samples were disrupted with two 4-mm tungsten carbide beads during 2 X 1 min, at 30 Hz and that the  
281 lysis step lasted 1 hour (instead of 30 min) at 65°C. A Mixer Mill MM300 (Retsch, Germany) was used to  
282 disrupt the leaf samples. DNA was eluted in a final volume of 60 µl of elution buffer. SNP genotyping  
283 required high DNA quality and quantity. Genomic DNA samples quantity was assessed using the Quant-iT™  
284 PicoGreen® dsDNA Assay Kit (Invitrogen™) according to the manufacturer's instructions. Absence of DNA  
285 degradation was controlled on 1% agarose gel by the DNA bank platform of the Genotyping National  
286 Center, CNG (CEA-IG, Evry, France). A second genomic DNA extraction was performed for samples where  
287 concentration was lower than 45 ng.µl<sup>-1</sup>, or if the total amount of DNA was lower than 1 µg. Assignment of  
288 individuals to their half-sib families was checked using nine microsatellites (Guichoux et al 2011).

289

### 290 *SNP selection and array design*

291 The SNPs were chosen among a subset of 8,078 polymorphic SNPs from the allelic resequencing of  
292 more than 800 initial targeted genic regions within the genome of 13 *Q. robur*, using a high quality SNP  
293 database from Sanger sequence data, (Lang et al 2021;  
294 <https://github.com/garniergere/Reference.Db.SNPs.Quercus>). Two Perl scripts, *SNP\_statistic* from the  
295 SeqQual pipeline (<https://github.com/garniergere/SeqQual>) and *snp2\_illumina* (Lepoittevin et al 2010),  
296 were used to compute statistics for each SNP and to design a template file compatible with the Illumina  
297 Assay Design Tool (ADT) software respectively. Within the 13 *Q. robur* sequence data, three criteria were  
298 used to further filter SNP genotypes: a minimum depth of 8 reads, a minor allele frequency higher than 7%  
299 (~~allowing exclusion of variants found only once for the minimum of 8 diploid individuals sequenced; i.e.,~~  
300 ~~excluding singletons~~) and an Illumina ADT score greater than 0.4, which yielded 2,447 SNPs. Moreover, in  
301 the case of two SNPs within 60 bp of each other, only one was kept, following Illumina's recommendations,  
302 the chosen SNPs fulfilling the same previous quality criteria (Supplementary material, Figure S3), which  
303 yielded 1,670 SNPs. Finally, two stringent filters were added: (i) SNPs with an ADT score lower than 0.6 and  
304 with only 2 sequences for one of the alleles and (ii) SNPs with a minor allele frequency lower than 10%,  
305 with no heterozygous individuals identified and with only 2 sequences for one of the alleles. These two  
306 categories of SNPs were excluded. Finally, 1536 SNPs were included in the genotyping assay.

### 307 *SNP Genotyping*

308 The SNP genotyping experiment was performed on the subset of seedlings with the highest quality and  
309 quantity of extracted DNA, 1,185 individuals being finally retained (i.e., 71% of those that underwent DNA  
310 extraction) with 759 and 426 individuals for the Natural and Protected exposure, respectively. For each 96-  
311 well plate, we checked the quality and reproducibility of the genotyping assay with one negative control  
312 (water) and four positive controls (DNA samples of two well-known genotypes, 3P and A4, duplicated  
313 twice). We also included across plates 59 DNA samples of parents and potential parents to further test for  
314 possible Mendelian inconsistencies between parents and offsprings. A total of 30-50 ng of genomic DNA  
315 per individual was used for SNP genotyping by the INRA-EPGV group using the Illumina BeadArray platform  
316 of the Genotyping National Center, CNG (CEA-IG, Evry, France) and following the GoldenGate Assay  
317 manufacturer's protocol (Illumina Inc., San Diego, CA, USA). Three assays, over a 3-days period each, were  
318 performed to genotype 1,284 samples for the 1,536 SNPs. The protocol was similar to the one described  
319 by Hyten et al (2008), except for the number of oligonucleotides involved in a single DNA reaction, which  
320 comprised 4,608 custom oligonucleotides in the Oligo Pool Assay (OPA). Raw hybridization intensity data  
321 processing, clustering and genotype calling were performed using the genotyping module of the

322 BeadStudio/GenomeStudio package (Illumina, San Diego, CA, USA) with a GeneCall score cutoff of 0.25 to  
323 obtain valid genotypes for each individual at each SNP.

324

#### 325 *SNP quality criteria for genotyping reliability*

326 After a first genotype calling of the raw data, we assessed SNP genotype quality across individuals using  
327 the methodology proposed by Illumina (Tindall et al 2010). Briefly, 50% GC score and 10% GC score were  
328 plotted as a function of the sample call rate. Poorly performing samples were obvious outliers across many  
329 genotypes when compared to the majority. In our experiment, these outliers corresponded to samples  
330 with 50% GC score and call rate lower than (mean(50% GeneCall score)-0.0.1) and (mean(call rate)-0.015)  
331 respectively or to samples with 10% GeneCall score and call rate lower than (mean(10% GeneCall score)-  
332 0.0.15) and (mean(call rate)-0.015) respectively (Figure S4). After discarding those poor-quality samples, a  
333 new genotype calling was performed on remaining individuals using the same GeneCall score cutoff. SNP  
334 quality was further determined automatically using a call frequency greater than 0.99, a 10% GeneCall  
335 score greater than 0.6, a heterozygote frequency greater than 1%, and a very low level of inconsistencies  
336 for Parent-Child or Parent-Parent-Child testing. To avoid discarding valuable SNPs or keeping poor quality  
337 SNPs, a visual inspection of all SNPs clusters was further performed after the automatic pipeline. SNP  
338 markers that displayed either compression or unexpected clustering patterns were discarded  
339 (Supplementary material, Figure S5 and S6). A total of 819 SNPs were finally kept for further analyses  
340 (Supplementary material, Table S3).

341

#### 342 *Multi-locus individual genetic diversity*

343 Genetic diversity indices were computed for different groups of individuals: the 1185 individuals that  
344 were representative of the initial populations, and individuals that survived or not under both powdery  
345 mildew exposures (Natural and Protected). Observed and expected heterozygosities ( $H_o$  and  $H_e$ , *sensu* Nei  
346 1973), and  $F_{ST}$  indicating differentiation between the initial and the surviving populations for both  
347 exposures were estimated (Weir and Cockerham 1984), using 'adegenet' (Jombart 2008) and 'Genepop'  
348 (Rousset et al 2008) R packages. Differentiation between populations and deviation of populations from  
349 Hardy-Weinberg equilibrium were tested using 'Genepop' R package (Rousset et al 2008). Five  
350 heterozygosity statistics were estimated for each individual based on the 819 successfully genotyped SNPs,  
351 using the GENHET function in R (Coulon 2010): the proportion of heterozygous loci (PHt), two standardized  
352 heterozygosities based on the mean expected heterozygosity and on the mean observed heterozygosity  
353 ( $Hs\_exp$  and  $Hs\_obs$ , respectively), the internal relatedness (IR) and the homozygosity by locus (HL). The  
354 preliminary analyses showed that these five statistics were highly correlated (absolute Spearman's rank  
355 correlation coefficient between 0.96 and 1.00; Supplementary material, Figure S7). Therefore, only PHt  
356 was kept for further analyses. Mean estimates of PHt were compared among the group of individuals that  
357 did survive *versus* the ones that did not for each exposure (Natural and Protected) using Mann-Whitney  
358 tests. Equality of variances were also assessed using Fligner-Killeen tests among the same group of  
359 individuals (survivors *versus* dead individuals, both for Natural and Protected exposures). The mean values  
360 of PHt in Natural *versus* Protected, initial and surviving, sub-populations were compared by running a GLM  
361 model. The effect of PHt on survival was also tested by logistic regression along with four other main  
362 explanatory variables (Supplementary material, Table S2, Model 6).

363

365 The physical position of SNP markers was obtained by aligning the flanking sequence of the SNP  
366 markers (with a maximum of 100 base pairs on each side of the SNP; ~~Supplementary material, Data S1~~)  
367 using Blast (Johnson et al 2008) on the *Q. robur* genome assembly available on The Darwin Tree of Life  
368 project (accession PRJEB51283). ~~This confirmed that the 819 SNPs were scattered across the whole~~  
369 ~~genome, on all chromosomes, in 426 different genic regions (see Table S3), with an average distance~~  
370 ~~between regions of 2.11 Mb (ranging from 0.0029 to 15.3 Mb). This confirmed that the 819 SNP were~~  
371 ~~scattered across the whole genome in 426 different gene regions, consistently with the fragments that~~  
372 ~~were originally re-sequenced (see Table S3), with an average distance across chromosomes among regions~~  
373 ~~of 2.11 Mb (range 0.0029 to 15.3 Mb).~~ The associations between SNP markers and the four phenotypic  
374 traits of interest (“Mean infection (2009-2013)”, “Height in 2012”, “Acorn weight” and “Survival (2017)”)   
375 were tested using the Bayesian-information and Linkage-disequilibrium Iteratively Nested Keyway (BLINK,  
376 Huang et al 2019) in GAPIT3 R package (Wang and Zhang 2021). Default parameters were used for all  
377 analyses. This method allows to account for the different relatedness levels among individuals, building  
378 from the multilocus mixed model of Segura et al. (2012) by iteratively incorporating associated markers as  
379 covariates, but with a special optimization criterion (Tibbs Cortes et al 2021). Because of the large number  
380 of tests, a false discovery rate (FDR) analysis was used to control for false positive associations (Benjamini  
381 & Hochberg 1995), using a threshold of 0.01 for the FDR-corrected p-value. Deviation of the observed p-  
382 values from the expected values was assessed with a QQ-plot (Supplementary material, Figure S8 and S9  
383 for Natural and Protected exposures, respectively). Both powdery mildew exposures were analyzed  
384 separately.

385

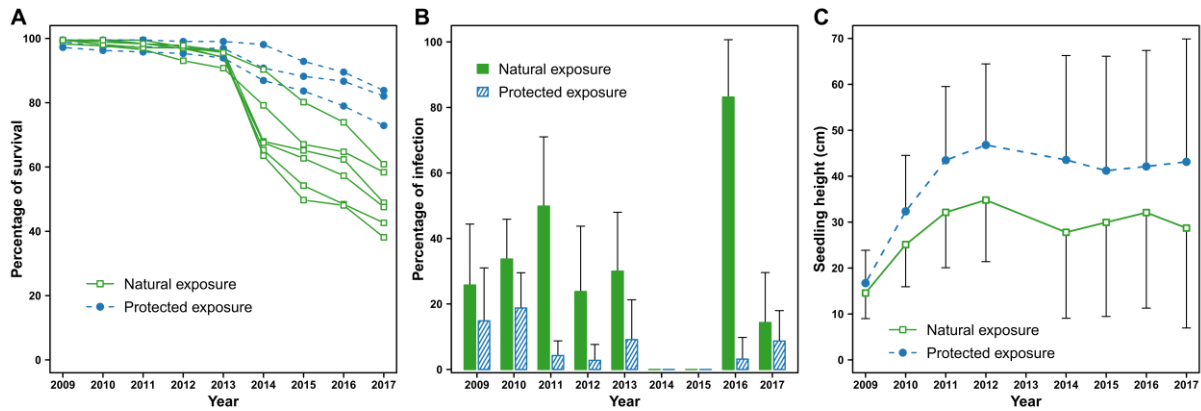
386

## Results

### 387 Seedling and juvenile survival

388 Oak survival was very high during the first five years, close to or greater than 90% in both powdery  
389 mildew exposures, *i.e.*, the protected or fungicide-treated one, and the natural or non-protected one that  
390 was submitted to natural powdery mildew infection (Figure 2A). Survival decreased in the following four  
391 years. The decrease was much steeper for non-protected trees. Mortality was observed mainly at the  
392 beginning of spring when individuals failed to flush, and not during the growing seasons. The annual  
393 mortality rate was highest in 2014 in all non-protected plots but one, compared to the fungicide-treated  
394 plots. Survival at the end of the monitoring period was 49.7% on average in plots with natural powdery  
395 mildew infection, compared to 79.5% in fungicide-protected plots. As expected for an efficient fungicide  
396 treatment, disease severity was much lower in fungicide-treated plots than in non-protected plots  
397 throughout the experiment (Figure 2B). Seedlings showed higher height in the protected plots than in the  
398 naturally infected plots, as soon as the second year (Figure 2C).

399



400

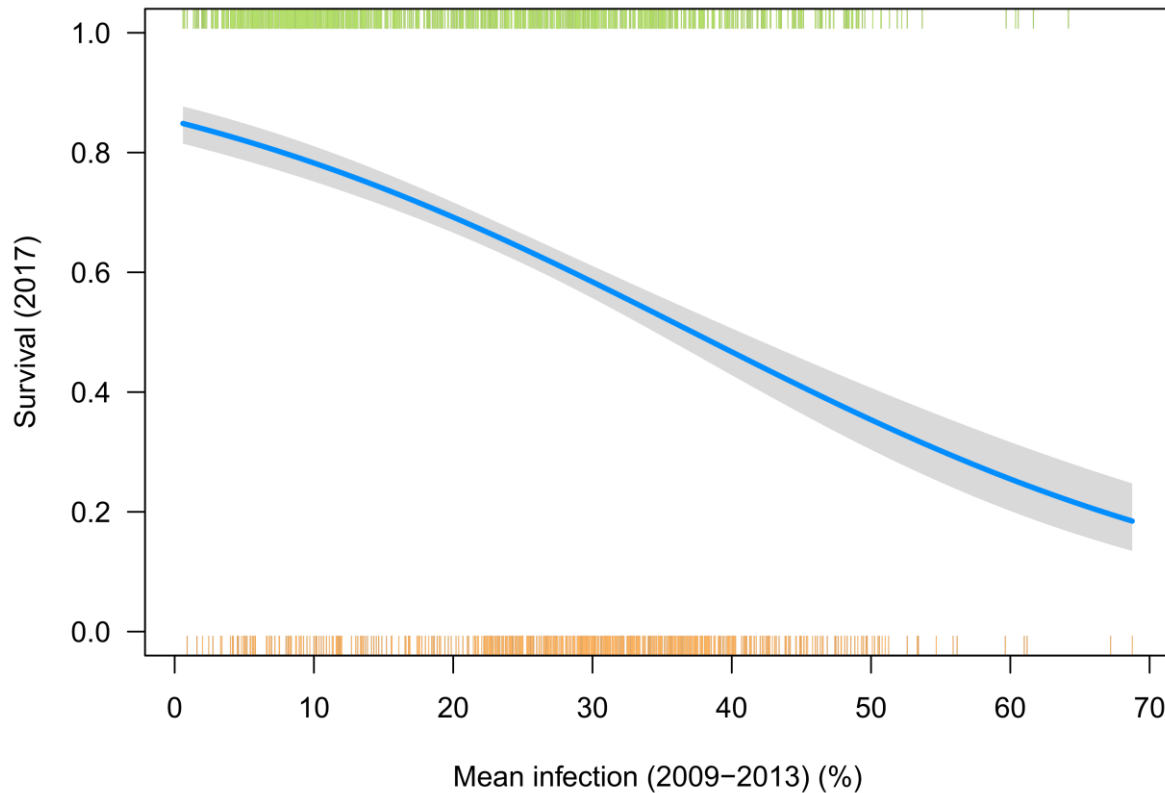
401 **Figure 2:** A. Time course of individuals' survival rate in the three protected replicates and the six natural  
 402 infection plots; B. Mean annual infection across plots under both powdery mildew exposures; C. Seedling  
 403 height across studied years in protected *versus* natural exposure plots.

404

405 The powdery mildew exposure and the acorn weight were both significant predictors of survival in the  
 406 last year of observation *i.e.*, 2017 (Wald  $\chi^2 = 4.00$ ,  $P = 0.045$  and Wald  $\chi^2 = 27.1$ ,  $P < 0.0001$ ,  
 407 respectively; Model 1 results in Supplementary material, Table S4 and Figure S10). The natural mildew  
 408 exposure was associated with a four-fold increase in the odds of mortality compared to the fungicide-  
 409 protected one, which corresponds to an odds ratio of 4.0 with a 95% confidence interval (C.I.) of 3.19 to  
 410 5.06. For the acorn weight, a 23% increase of the odds of survival per additional gram was observed on  
 411 average (odds ratio of 1.23 with a 95% C.I. of 1.14 to 1.32). The interaction effect between acorn weight  
 412 and powdery mildew exposure was not significant in the model (Supplementary material, Table S4), as well  
 413 as the block effect (not shown in the final analysis).

414 Model 2 provided a quantitative assessment of the effect of the mean percentage of infection across  
 415 the first five years, on survival (Supplementary material, Tables S2 and S5), with an estimated odds ratio of  
 416 0.954 (95% C.I. of 0.946 to 0.962). This means that each additional percent of leaf infection is expected to  
 417 reduce the odds of survival by 4.6% (Figure 3).

418



419

420 **Figure 3:** Logistic model predicting juvenile survival in year 2017, based on Mean infection between 2009  
 421 and 2013 (at mean acorn weight = 5.028 g). Distributions of the individual values of the variable “Mean  
 422 infection (2009-2013)” were displayed as orange marks at the bottom of the figure and light-green marks  
 423 at the top of the figure for dead and live trees, respectively. The grey envelop around the blue line  
 424 represents the 95% confidence interval.

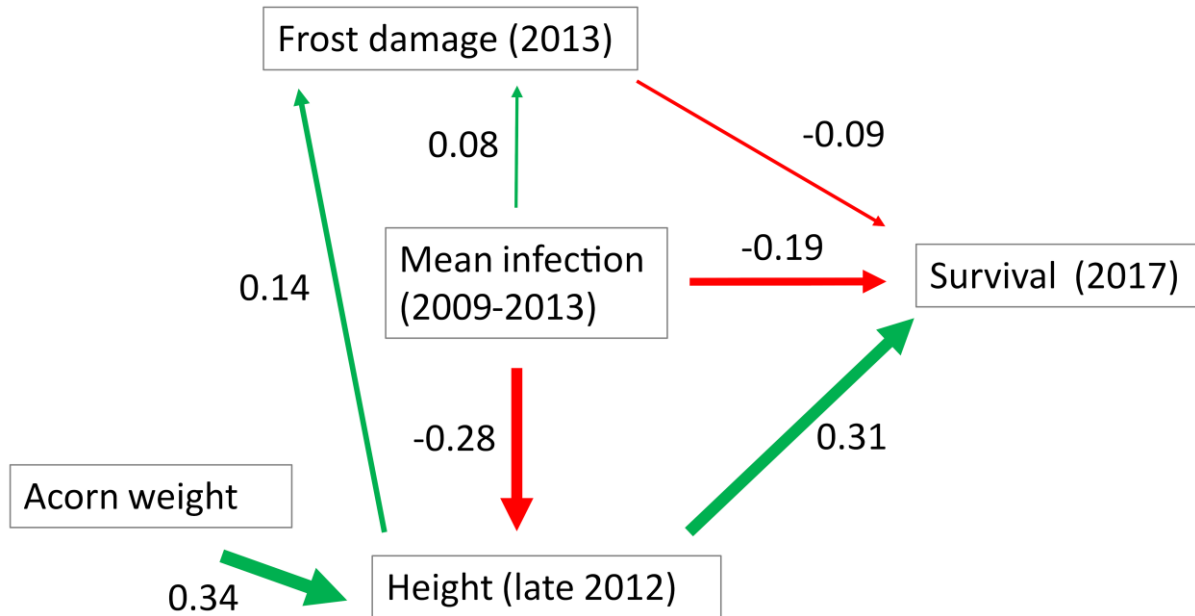
425

426 Using seedling height at the end of the first growing season (Model 3, Table S2) instead of acorn weight  
 427 as a predictor variable (Model 1, Table S2) had little influence on the results, with a very slight improvement  
 428 of the concordance of the association between predicted probabilities and observed responses (68.7%  
 429 instead of 68.1%). Thus, seedling height at the end of the first growing season was a good predictor of  
 430 survival at the end of the monitoring period (*i.e.*, eight years later), with a strong negative impact of the  
 431 “Powdery mildew exposure” at a given height (Supplementary material, Figure S11). The “Height in 2009 :  
 432 Powdery mildew exposure” interaction was not significant in this model. When “Frost damage (2013)” was  
 433 added to the logistic model (Model 4), this variable had a significant negative effect on survival (Wald  
 434 Khi2 = 5.99, P = 0.014, odds ratio = 0.804; C.I. of 0.649 to 0.996) in addition to the effects of “Acorn weight”  
 435 and the “Mean infection (2009 and 2013)” (Wald Khi2 = 17.8, P < 0.0001 and Wald Khi2 = 112.8, P < 0.0001,  
 436 respectively; Supplementary material, Figure S12).

437 The Structural Equation Model showed almost equal but opposite effects of “Height (late 2012)” and  
 438 “Mean infection (2009-2013)” onto final survival, with total standardized coefficients (not displayed on  
 439 Figure 4) of 0.30 (positive) and -0,28 (negative), respectively. The total negative effect of powdery mildew  
 440 infection corresponds to a direct negative effect of -0.19 (path 3) and indirect negative effects of -0.09. The  
 441 most important indirect effect is -0.087 (= -0.28\*0.31, according to paths 1 and 5) through “Height (late

442 2012)", the indirect effect through "Frost damage (2013)" being much less ( $0.08 \times -0.09 = -0.007$ , according  
443 to paths 2 and 4) (Figure 4). The direct contribution of "Frost damage (2013)" on final survival (path 4) was  
444 a mild negative effect (-0.09) (Figure 4).

445



446

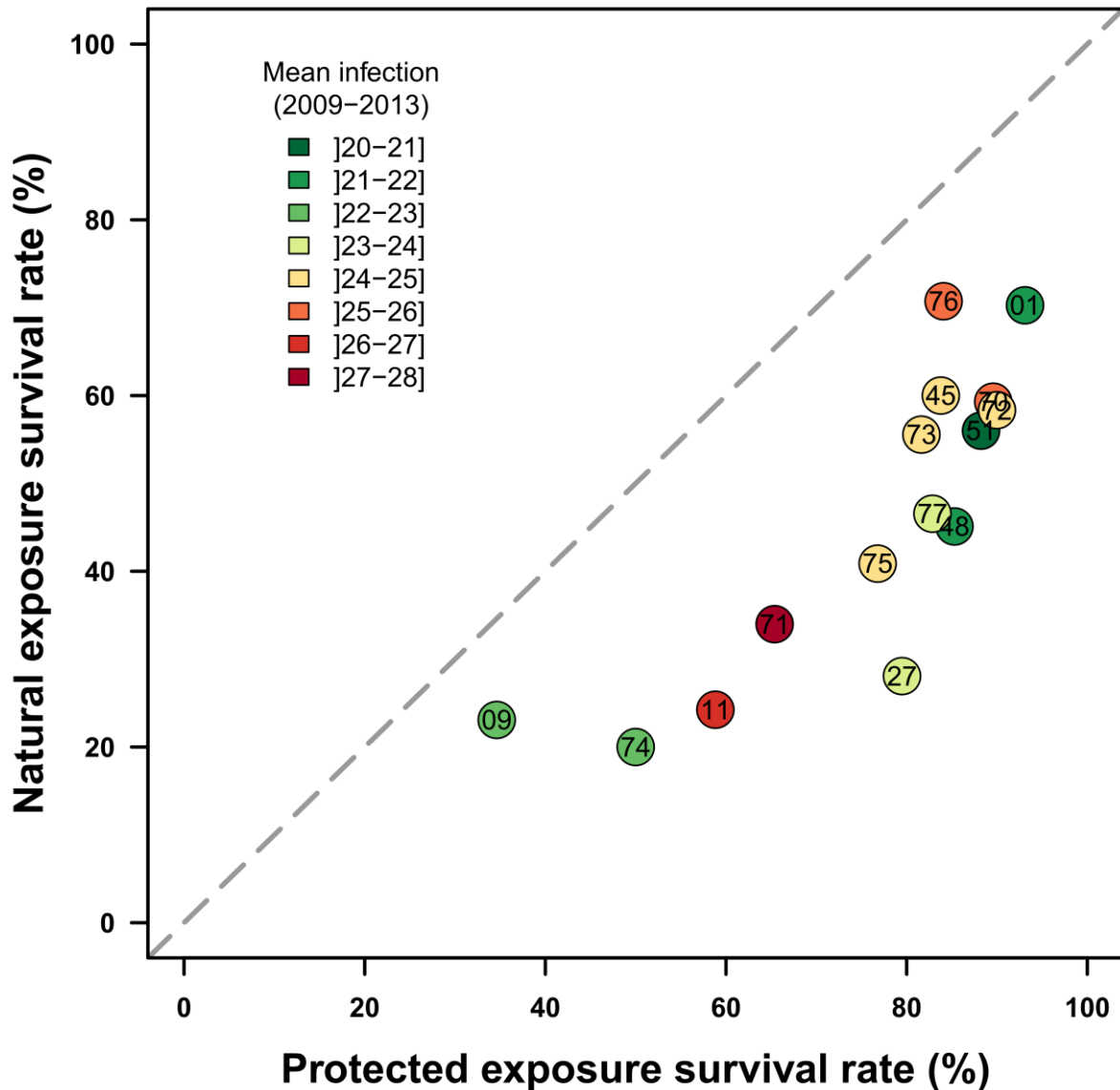
447 **Figure 4:** Results of the Structural Equation Model – Line width is proportional to the effect value.

448

#### 449 **Differential impact of powdery mildew among open-pollinated families?**

450 Average proportions of individuals having survived varied among families, ranging from 35% to 93% in  
451 the fungicide-protected plots and from 23% to 72% in the plots submitted to natural powdery mildew  
452 exposure (Figure 5).

453



454

455 **Figure 5:** Progeny survival percentages across families in Protected plots *versus* Natural powdery mildew  
 456 infected plots. Each dot corresponds to a family identified by its number. The mean powdery mildew  
 457 infection (2009-2013) for each family in the Natural infection exposure is color-coded from dark-green =  
 458 low mean powdery mildew infection (minimum=20.9) to dark-red = high mean powdery mildew infection  
 459 (maximum=27.8).

460

461 The full logistic model of survival, including previously studied factors (powdery mildew exposure,  
 462 acorn weight and frost damage), a family effect and an interaction between powdery mildew exposure and  
 463 family (Model 5) was highly significant and showed an improved concordance percent between predictions  
 464 and observations of 76.7%. All previously studied factors remained significant but the family effect further  
 465 explained the probability of survival (Wald  $\chi^2 = 126.68$ ,  $P < 0.0001$ ; Supplementary material, Table S6).

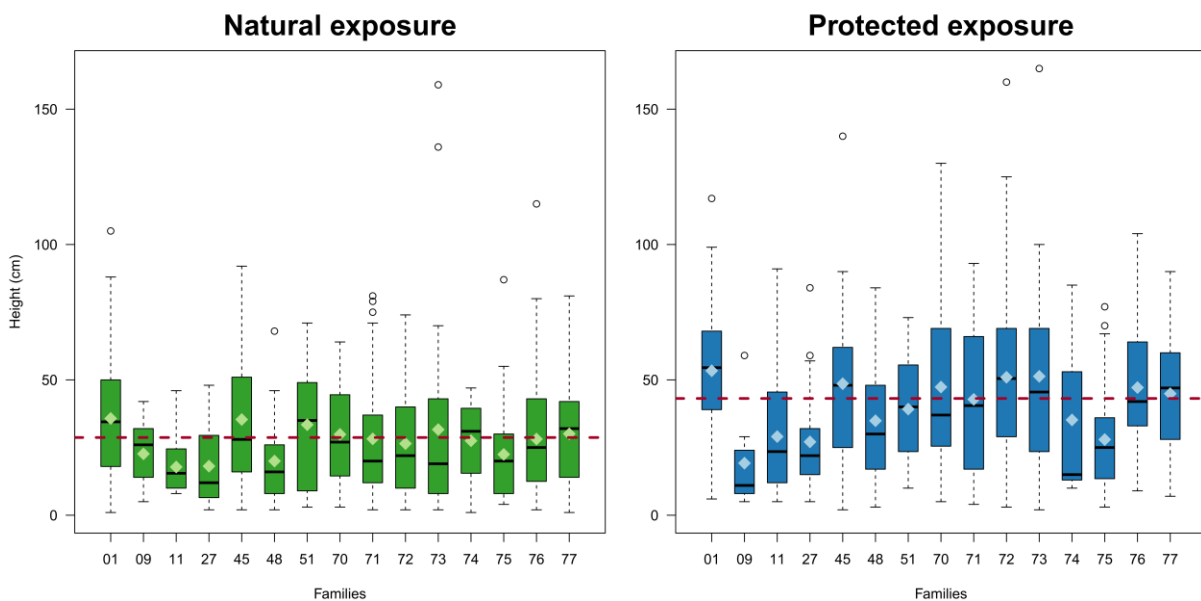
466 However, the interaction between family and powdery mildew exposure was not significant. This  
 467 means that overall, in this experiment, exposure to powdery mildew had a similar negative effect on the



468 survival of all families without any strong changes in their ranking for survival (Spearman correlation = 0.86;  
469  $P = 0.0001$ , and see Figure 5).

470 Mean family survival (percent surviving progeny in 2017) was significantly correlated with family height  
471 (*i.e.*, mean value over the progeny) from 2014 onwards in both disease exposures (*e.g.*,  $r = 0.71$  and  $0.69$   
472 with height in 2017 in fungicide-protected and powdery mildew natural exposures, respectively). In plots  
473 under the natural exposure, the relationship between family survival and family “height potential”, *i.e.*,  
474 mean height of the same family measured in protected plots in 2017, was even stronger than with realized  
475 height ( $r = 0.82$ ,  $P = 0.0002$ ). No significant correlation was observed at family level between height  
476 potential (in any year) and powdery mildew susceptibility (= mean infection observed in powdery mildew  
477 exposed plots in 2009-2013), although both variables showed a significant family effect. The height of  
478 surviving juveniles at the end of the monitoring period (in 2017) varied significantly among families: from  
479 19.2 cm to 53.4 cm in fungicide-treated plots ( $F = 5.01 - df = 14$   $P < 0.0001$ ), and from 17.8 to 35.7 in  
480 powdery mildew plots ( $F = 2.38 - df = 14$   $P < 0.0032$ ) (Figure 6).

481



482

483 **Figure 6:** Boxplot of recorded heights in 2017 in both Natural (left) and Protected (right) exposures across  
484 families. The black lines represent the median height for each family and the light-colored diamonds  
485 represent their mean height. The red dotted lines represent the overall averages of juvenile height in  
486 each exposure.

487

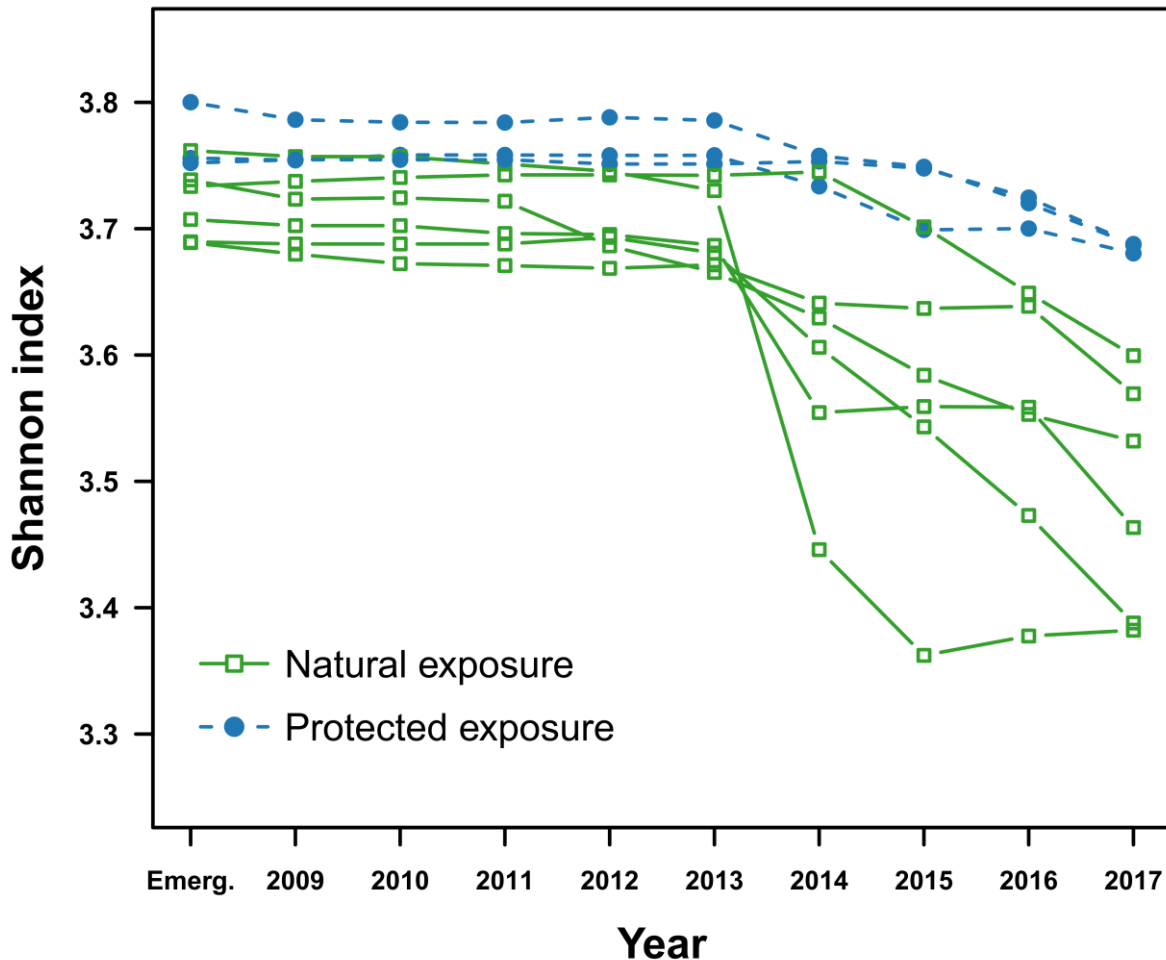
488 The between-family coefficient of variation for final height (standard deviation/mean) was lower in the  
489 natural disease exposure than in the protected by fungicide exposure (21.5 and 26.4, with standard  
490 deviations = 5.84 and 10.54, respectively) (Figure 6). Within-family variation (SD) was also reduced in the  
491 natural disease exposure compared to the protected exposure ( $t = -2.4$ ;  $P = 0.0306$ ). Mean powdery  
492 mildew infection over the first five years under the naturally exposed plots varied significantly among  
493 families from 28.5 to 35.7% ( $F = 5.28 - df = 14$   $P < 0.0001$ ).

494 The Shannon index, measuring diversity within plots in terms of family composition, remained very high  
495 in fungicide-treated plots throughout the experiment, but decreased after 2013 in all plots naturally



496 exposed to powdery mildew, as a result of increasing differences across years in the relative numbers  
497 (percent of surviving individuals) of the different families (Figure 7).

498



499

500 **Figure 7:** Temporal changes in Shannon index, used as a family-diversity index calculated from the  
501 proportional family abundances in plots under natural or protected by fungicide powdery mildew  
502 exposure. The index was calculated for each of the nine experimental plots: six under natural powdery  
503 mildew exposure (green) and three under protected by a fungicide powdery mildew exposure (blue)  
504 every year.

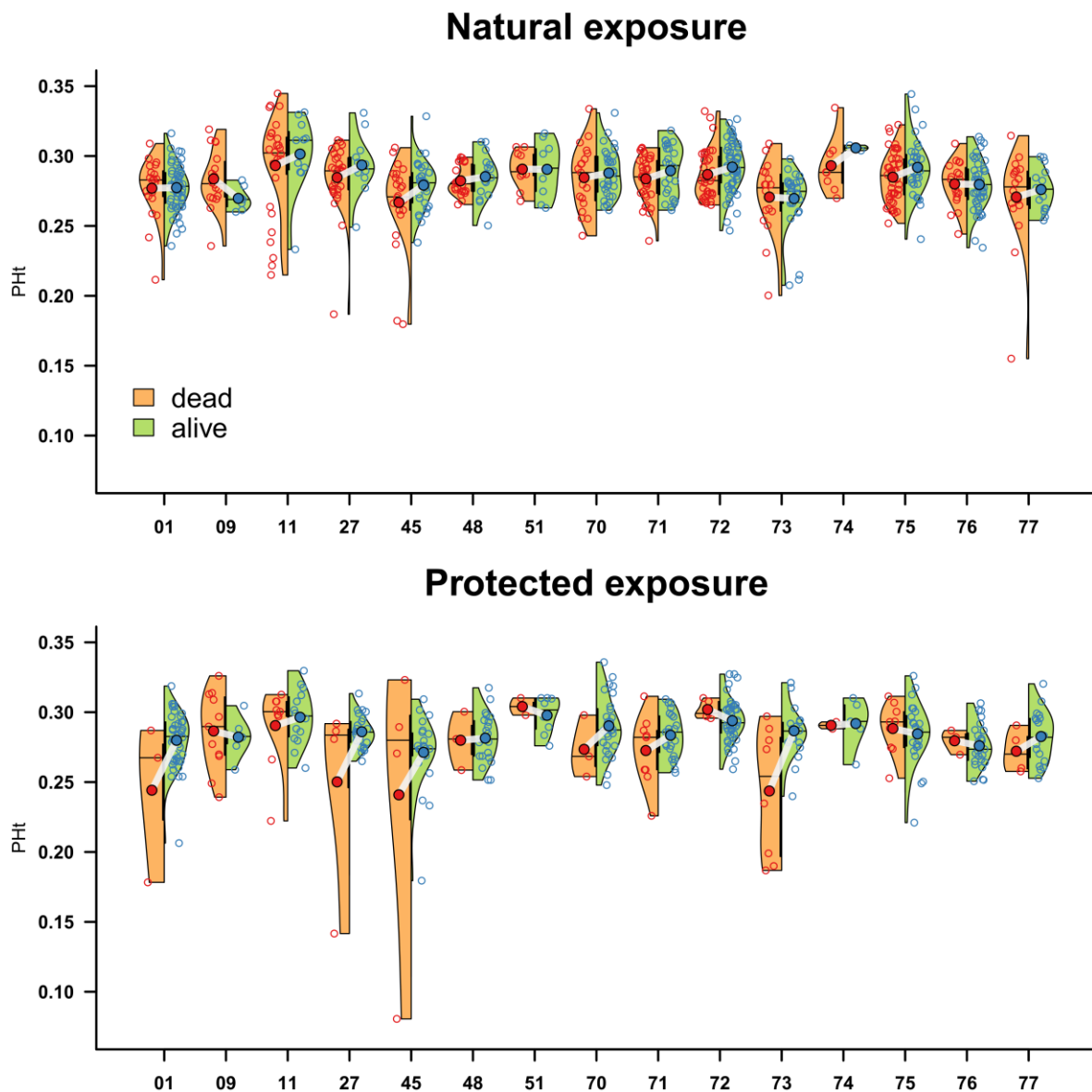
505

### 506 Multi-locus heterozygosity

507 Out of the 1,185 individuals included in the SNP genotyping experiment, 1,143 were successfully  
508 genotyped with less than 0.08% of missing data. Observed and Expected heterozygosity did not vary  
509 between initial and surviving populations in both disease exposures, with values of  $H_o$  and  $H_e$  of 0.32-0.33  
510 in all cases (Supplementary material, Figure S13). Genetic differentiation between initial and surviving  
511 populations were very low and not significant (Supplementary material, Figure S13). The distribution of the  
512 proportion of heterozygous loci (Pht, see methods) values estimated on all individuals was negatively

513 skewed: while most individuals had a PHT value in the range of 0.235-0.335, very few individuals (2.54%)  
 514 showed lower values (< 0.235, minimum 0.18) (Supplementary material, Figure S14). The mean PHT across  
 515 individuals was very similar in both disease exposures when comparing initial *versus* surviving populations:  
 516 0.283 ± 0.022 *versus* 0.284 ± 0.020 for the natural exposure; 0.283 ± 0.025 *versus* 0.285 ± 0.019 for the  
 517 protected exposure (Supplementary material, Figure S15 and Table S7). However, in both exposures and  
 518 across all families, the individuals with very low PHT were over-represented in dead seedlings (Figure 8,  
 519 Supplementary material, Figure S16). This resulted in a decrease in the variance of the PHT between  
 520 surviving and dead individuals (Khi2 = 0.567, P = 0.45 and Khi2 = 11.4, P = 0.0007 for Natural and Protected  
 521 exposure, respectively).

522



523

524 **Figure 8:** Violin plots of the PHT values for Natural (above) and Protected (below) exposures across  
 525 families. Red and blue empty (or full) circles represent the PHT values, across dead and alive individuals  
 526 (or families), respectively. Black horizontal lines delimit the median values for dead and alive individuals  
 527 across families.

528

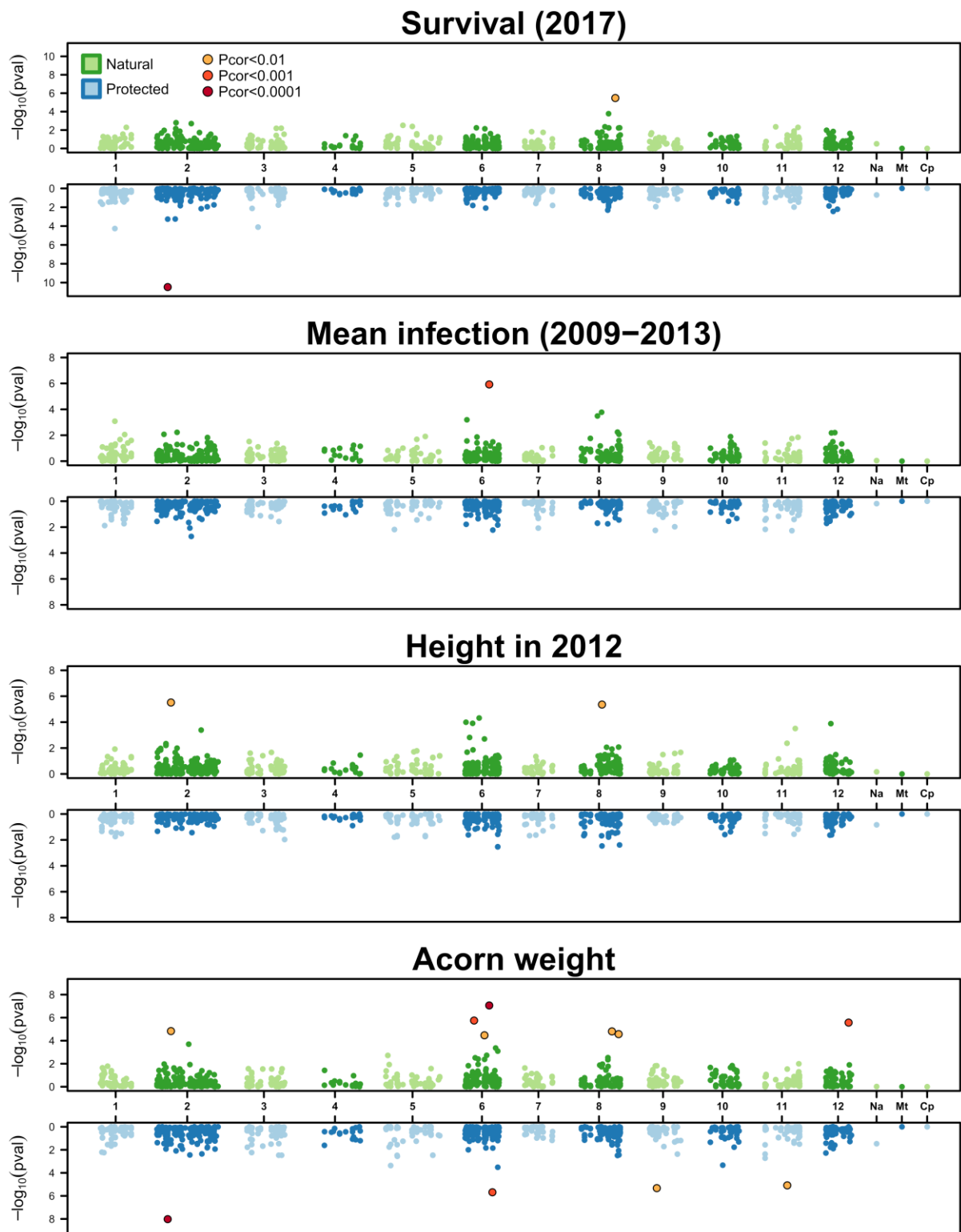
529 The logistic model of survival including PHT in addition to the exposure (Natural *versus* Protected),  
530 family, acorn weight and frost effects (Model 6, Table S2) demonstrated a significant positive effect of  
531 individual heterozygosity on survival, but the Powdery mildew exposure\*PHT interaction was not  
532 significant. This suggests that the effect of low heterozygosity was not more deleterious in naturally  
533 exposed than in protected seedlings but simply added to the negative effect of infection.

534

### 535 **Tests of genetic associations**

536 Overall, 16 significant genetic associations were found between 14 loci (SNPs) and the four phenotypic  
537 traits investigated, mostly on chromosomes 2, 6 and 8 (Figure 9). In the Natural exposure, one SNP was  
538 statistically associated with “Mean infection (2009-2013)” on chromosome 6. This SNP belonged to the  
539 same gene ~~than-as~~ another close one (distant from 336 bp) that was significantly associated to “Acorn  
540 weight” (Figure 9). One SNP located on chromosome 8 was associated with “Survival (2017)”. Two SNPs  
541 were significantly associated with “Height in 2012”, one of which was also associated with “Acorn weight”  
542 (Figure 9). In the protected exposure, a different SNP located on chromosome 2 was associated with  
543 “Survival (2017)”. This SNP was also associated with “Acorn weight”.

544



545

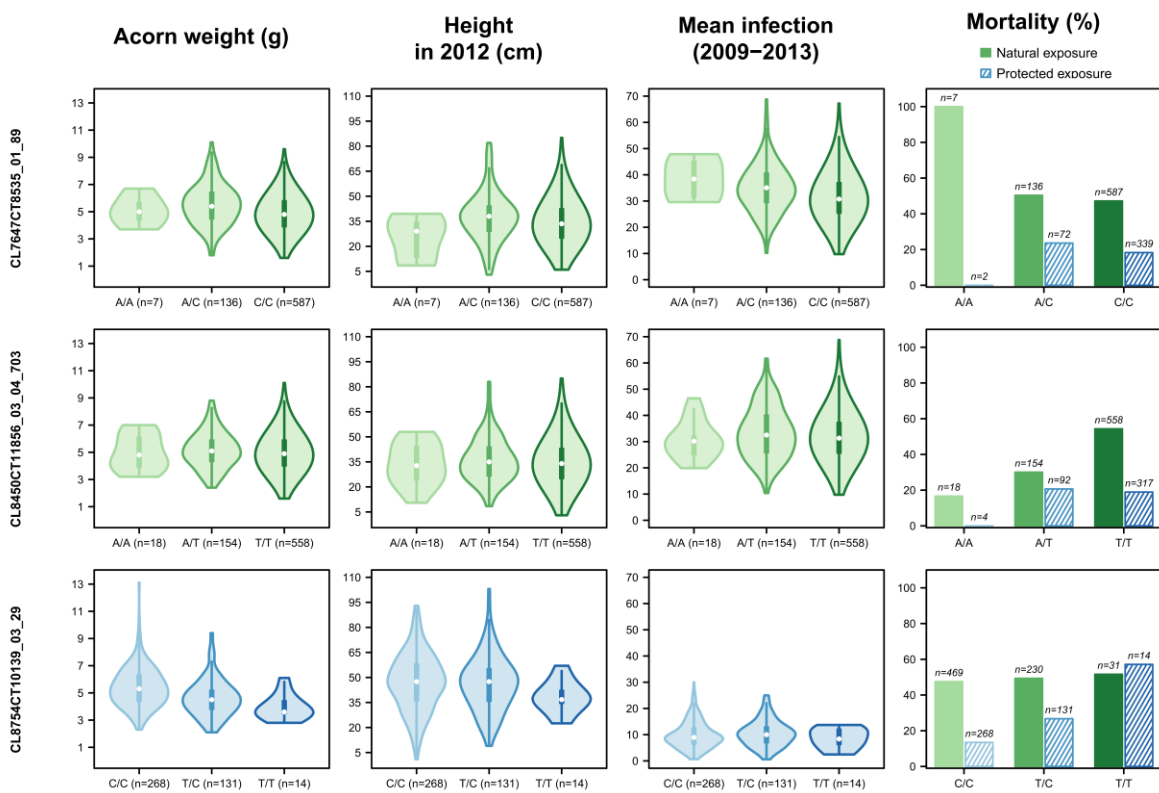
546 **Figure 9:** Manhattan plot for the Genome Wide Association Study results across the four phenotypic  
 547 traits investigated: “Survival (2017)”, “Mean infection (2009-2013)”, “Height in 2012” and “Acorn  
 548 weight”, and across both exposures (Natural in blue, Protected in green). Each dot represents a SNP. The  
 549 negative logarithm of the association  $p$ -value corrected for multiple tests is displayed on the vertical axis.  
 550 SNPs with a significant association with a trait are indicated with yellow, orange and red dots. The SNP

551 markers are ordered along the genome and grouped by chromosome. 1 to 12: chromosome number; *Na*:  
 552 unknown location; *Mt*: mitochondrial; *Cp*: chloroplast.

553

554 The SNP *CL7647CT8535\_01-89* linked to “Mean infection (2009-2013)” is located in a gene predicted  
 555 to be an ethylene response factor C3. While no significant association of this SNP was found with seedling  
 556 survival in the natural exposure, the genotypic classes with lower mortality are consistent with those  
 557 showing less infection and thus increased resistance (Figure 10, top row). “Acorn weight” and “Height in  
 558 2012” did not show any association with this marker. The SNP *CL8450CT11856\_03\_04-703* linked to  
 559 survival in the natural exposure is located in a gene coding for a putative histone H4. No differences were  
 560 observed for the three other traits among genotypic classes at this locus (Figure 10, middle row). The SNP  
 561 *CL8754CT10139\_03-29* significantly associated with the survival in the protected exposure is situated in a  
 562 gene coding for a putative pentatricopeptide repeat-containing protein. This marker was also significantly  
 563 associated with “Acorn weight”, but not with “Height in 2012” or “Mean infection (2009-2013)” traits  
 564 (Figure 10, bottom row).

565



566

567 **Figure 10:** Genotypic classes distribution and mean trait values across traits for SNPs *CL7647CT8535\_01-*  
 568 *89*, *CL8450CT11856\_03\_04-703* and *CL8754CT10139\_03-29* that were significantly associated with “Mean  
 569 infection (2009-2013)” under natural exposure, “Survival (2017)” under natural exposure and “Survival  
 570 (2017)” under protected exposure (last column), respectively. Genotypic classes are named using the  
 571 IUPAC nucleotide symbol convention: A= adenine, C=cytosine and T=thymine.

572

574 In this experimental approach, the effect of powdery mildew on oaks was monitored during nine years  
575 after acorn sowing in contrasting plots that were either under natural infection or where pathogen  
576 pressure was limited by fungicide treatments. More than one thousand individuals identified by their acorn  
577 weight and mother tree (15) were subjected to fine phenotypic monitoring across years (height growth,  
578 mildew infection and survival) and genotyped at 819 SNPs. ~~This powerful design enabled us to demonstrate  
579 various effects of powdery mildew at demographic and genetic levels in early stages of an oak cohort. This  
580 powerful design brought several significant results on the demographic and genetic impacts of powdery  
581 mildew in early stages of an oak cohort. First, we demonstrated that powdery mildew infection had a strong  
582 deleterious effect on the survival at the juvenile stage, including a direct effect and an indirect effect mainly  
583 through reduced seedling height. Also, survival varied significantly among progenies from different mother  
584 trees, but without strong changes in the ranking of progenies under low or high infection pressure.  
585 Powdery mildew infection thus did not suppress the competitive advantage of the dominant, fast growing  
586 families, and no evidence of a growth-defense trade-off was obtained. Exposure to powdery mildew  
587 pressure did not significantly affect the mean genetic diversity at population level. Finally, a few significant  
588 associations between some genetic markers and phenotypic traits were found.~~ The significance of these  
589 results and a number of related issues are discussed below.

590

### 591 1. Strong negative powdery mildew impact on juvenile survival

592 The negative impact of powdery mildew on oak regeneration was pointed out by many authors (Pap et  
593 al 2012; Marçais & Desprez-Loustau 2014; Demeter et al 2021). However, we could not find any  
594 quantitative data on pathogen-induced mortality of seedlings in forest. Our experimental approach, with  
595 a comprehensive individual monitoring of seedlings across nine years under two contrasted disease  
596 exposures, provides supporting evidence of a causal association between powdery mildew infection and  
597 mortality of seedlings, under field conditions. Mortality was indeed significantly much higher in the plots  
598 exposed to natural infection than in plots treated with fungicide. Moreover, the probability of mortality  
599 could be quantitatively related to disease severity in the previous years.

600 The high mortality rates in early stages of naturally regenerated forest stands are generally attributed  
601 to an intense competition among tree seedlings (Collet & Le Moguedec 2007). However, mortality patterns  
602 in our experiment suggest that powdery mildew effects overcame competition effects. Indeed, mortality  
603 rates remained very low in the fungicide-treated plots during the monitoring period even though plants  
604 were taller and maintained at a greater density than in plots without fungicide (where seedlings  
605 progressively died), thus at a potentially stronger competition level. Maybe the competition-related  
606 mortality (self-thinning stage) (Peet and Christensen 1987; Collet & Le Moguedec 2007) will simply be  
607 delayed in our conditions, characterized by full light availability and an initial seedling density (1 acorn per  
608 10\*10 cm) which may be lower than in some spots of natural regeneration (Diaci et al 2008; Annighöfer et  
609 al 2015; Kuehne et al 2020).

610 Infection induced mortality has been reported for other powdery mildew diseases, such as  
611 *Podosphaera plantaginis* on *Plantago lanceolata* (Laine 2004), or *Erysiphe cruciferarum* on  
612 *Alliaria petiolata* (Enright et al 2007), and rust diseases (other plant biotrophic pathogens), such as myrtle  
613 rust (Carnegie et al 2016), *Melampsora medusa* f. sp. *deltoidae* on Poplar (Newcombe et al 1994) or  
614 *Puccinia lagenophorae* on groundsel (Paul & Ayres 1986). Mortality started only five years after sowing in  
615 our experiment, which suggests cumulative and delayed effects of infection, as expected for this kind of

616 pathogen. As biotrophic parasites, powdery mildews strongly affect the carbon economy of their host  
617 plant, by direct consumption of carbon fixed by photosynthesis (through their haustoria) but also by forcing  
618 allocation of plant carbon to defense (Hückelhoven 2005, Oliva et al 2014). In addition, powdery mildew  
619 infection has a direct negative effect on net carbon assimilation by photosynthesis, as was demonstrated  
620 for *E. alphitoides* (Hewitt & Ayres 1975; Hewitt & Ayres 1976; Hajji et al 2009; Pap et al 2014). The depletion  
621 of carbon by the pathogen likely explains growth reduction. Cumulative and delayed effects of powdery  
622 mildew were previously described on radial growth in young oak trees (Bert et al 2016). Then it is  
623 reasonable to assume, although the full demonstration remains to be made (Martinez-Vilalta 2014), that  
624 severe infections, recurring in successive years, can lead to exhaustion of reserves and ultimately death  
625 (Oliva et al 2014). The structural equation model that we tested is consistent with a strong direct effect of  
626 powdery mildew infection on seedling survival, twice as important as the effect mediated by decreased  
627 height. One possible mechanism could be reduced root growth in infected plants resulting from the  
628 alteration of the carbon metabolism. In the case of oak, the development of a large root system facilitates  
629 survival when aerial parts are affected or killed (Larsen & Johnson 1998). Finally, the SEM also suggested  
630 an indirect effect of powdery mildew through frost sensitivity, in agreement with previous observations of  
631 severe shoot mortality following winter in infected seedlings (Desprez-Loustau et al 2014). The late spring  
632 frost of 2013 could have given the “coup de grâce” to already weakened seedlings. Paul & Ayres (1986)  
633 also reported that heavy infection could compromise the ability of plants to tolerate winter stress in  
634 groundsel infected by rust. Jarosz & Burdon (1992), with flax rust, noted that the main effect of disease  
635 was to reduce survivorship during the winter following infection which could lead to pathogen-generated  
636 cycles in the host population size (Susi et al 2017).

637

## 638 2. Differential impact of powdery mildew across families

639 The observations of progenies from identified mother trees allowed to assess fitness components that  
640 were either linked to growth, disease resistance, or progeny survival of the mother trees that were  
641 originally sampled. Powdery mildew infection was quite high across years in our experiment and families  
642 showed different levels of disease severity (% leaf area infected). The progenies of all 15 mother trees were  
643 negatively affected in their survival under higher disease pressure. However, the ranking of the mean family  
644 values for survival was very similar under both powdery mildew exposures.

645 In particular, progenies from most competitive mother trees (*i.e.*, with greatest progeny survival under  
646 low disease pressure, with fungicide) were also among those with greatest survival under high powdery  
647 mildew pressure. The hypothesis of changes in mother tree survival ranking related to powdery mildew  
648 exposure was therefore not supported. This hypothesis was based on the assumption of a negative  
649 relationship between resistance to powdery mildew and growth (considered as an important component  
650 of fitness at seedling stage). In our experiment, seedling survival was indeed strongly related to growth, as  
651 estimated by seedling height. However, the results suggested there was no apparent trade-off between  
652 growth and disease resistance: the families with the greatest mean height in the fungicide-treated plots  
653 (*i.e.*, representing the growth trait) did not have the highest infection rates when exposed to disease  
654 (representing the defense trait). In both disease environments, the families with highest survival rates were  
655 also those with the greatest height growth potential (assessed under fungicide treatment).

656 Some features of our experiment may explain such absence of negative correlation between growth  
657 and disease resistance. First, only 15 mother trees were sampled on a small spatial scale in one local  
658 population, thus limiting the phenotypic variation that could be observed. It has to be noted however that  
659 genomic diversity was high, in the range of values, or greater than He values reported with the same type



660 of markers (i.e. SNPs) for various *Q. robur* stands in Europe (Blanc-Jolivet et al 2021, Degen et al 2021 a and  
661 b). Trade-offs between traits (including disease resistance) may be easier to detect when considering  
662 phenotypic variation across a wider spatial range, in relation to differing selection pressures and  
663 evolutionary strategies of populations~~First, only 15 mother trees were sampled on a small spatial scale in~~  
664 ~~one local population, thus limiting the variation that could be observed. Trade-offs between traits~~  
665 ~~(including disease resistance) may be easier to detect when considering variation across a wider spatial~~  
666 ~~range, in relation to differing selection pressures and evolutionary strategies of populations~~ (McKnown et  
667 al 2014; Heckman et al 2019). In addition, the expression of growth-defense trade-offs can be context  
668 dependent (Karasov et al 2017), and it is usually stronger in environments where the level of resource  
669 acquisition is limited by shade or abiotic stresses (van Noordwijk & de Jong 1986; de Jong 1995).

670 Finally, the detection of growth-defense trade-offs may depend on the choice of the traits that are  
671 assessed. In our study, with height as the growth variable, we considered trade-off in a very general sense,  
672 encompassing processes linked to both acquisition and allocation of resources (Laskowski et al 2021).

673 Our results could also suggest that tolerance was more important than resistance to explain mean  
674 survival differences of progenies across mother oak trees under high disease pressure. Plants use different  
675 lines of defense to respond to pathogens, including resistance *sensu stricto* and tolerance (Desprez-Loustau  
676 et al 2016; Pagan & Garcia-Arenal 2020). Resistance *sensu stricto* relates to mechanisms that limit pathogen  
677 development within the plant. The variable corresponding to percent leaf area infected in our monitoring  
678 can be considered as inversely related to resistance. By contrast, tolerance relates to mechanisms with no  
679 direct effect on the pathogen but that limit the negative impact of infection on plant fitness (Jeger et al  
680 2006).

681 We previously demonstrated that mechanisms such as increased polycyclism and compensatory  
682 growth<sub>7</sub> are likely involved in the response of oak seedlings to powdery mildew (Desprez-Loustau et al  
683 2014). In our experiment, mean survival across families in plots naturally exposed to powdery mildew, *i.e.*,  
684 one component of tree fitness, was not correlated with mean leaf area infection but significantly correlated  
685 with mean progeny height in protected plots (height potential). We can thus hypothesize that such height  
686 potential is related to tolerance mechanisms. Parker and Gilbert (2018) also reported that the impact of  
687 infection (tolerance) on 17 closely related clover species was less negative on fast-growing species, possibly  
688 because of their better ability to acquire resources in the environment and compensate for damage (de  
689 Jong 1995). Moreover, some authors suggested that tolerance could be especially advantageous for long-  
690 lived species (Roy et al 2000). Although tolerance has been far less investigated than resistance, there is  
691 ample evidence of its occurrence in crops and wild plants (Pagan & Garcia-Arenal 2020), and more in-depth  
692 molecular approaches would probably be needed for unraveling the cascades of metabolic pathways  
693 behind tolerance and its correlation with growth-related traits (Monson et al 2021; Monson et al 2022).

694

### 695 **3. Increased powdery mildew pressure had no equalizing effect on the relative contribution of mother** 696 **trees to the next generation**

697 The surviving population was slightly less diverse in terms of family composition under high than under  
698 low powdery mildew pressure. This pattern may be explained by previous results showing that the  
699 advantage of the fast-growing families over the slow-growing families in terms of survival was not  
700 suppressed under pathogen exposure. On the contrary, fast-growth might be associated with higher  
701 tolerance to infection damage. Parker and Gilbert (2018) obtained very similar patterns with closely related  
702 species instead of families and suggested that greater tolerance in fast-growing species may limit rather



703 than promote species coexistence. Similarly, at infra-specific level, Mundt et al (2008) showed that the  
704 absolute fitness advantage of the more competitive genotype in absence of disease increased in the  
705 presence of disease.

706 However, differences in mean final height among families were reduced under disease pressure, as  
707 well as the variance within families. During the time frame of our experiment, this did not affect the family  
708 ranking for survival between disease exposures but maybe in a longer term, or with greater disease  
709 pressure, powdery mildew could have an equalizing effect on family and individual performances (survival).

710

#### 711 **4. Impact of powdery mildew on genetic diversity**

712 We did not observe any changes in genetic diversity (estimated by the mean  $H_e$  across a large number  
713 of SNPs) or any significant genetic differentiation (estimated by  $F_{ST}$ ) between the initial and the surviving  
714 oak populations in both exposures, with mortality rates as high as 60% in some naturally infected plots at  
715 the end of the monitoring period. Few studies have addressed the impact of disease on genetic diversity  
716 of natural plant populations contrary to animal populations (McKnight et al 2017). One of the best studied  
717 wild plant pathosystem in a long time series is the interaction between *Linum marginale* and  
718 *Melampsora lini*, which showed temporal patterns of genetic change in the host and pathogen at local  
719 scale, consistent with coevolutionary dynamics (Thrall et al 2012). However, these changes were associated  
720 with a gene-for-gene model, *i.e.*, the existence of matching genes for resistance in the host and virulence  
721 in the pathogen (Flor 1971), which is not characterized for the oak-powdery mildew interaction. One  
722 possible explanation to the lack of any observed change in our study is that the powdery mildew pressure  
723 was not strong enough to have significantly affected the very high background diversity revealed across  
724 the oak genome (Plomion et al 2018). Also, few if any powdery mildew infection causative or linked variants  
725 are probably included in our SNP sets due to a very low background linkage disequilibrium across the  
726 genome (Lang et al 2021). A similar argument can be invoked for variants involved in the genetic  
727 determinism of traits linked to growth and survival that are most probably multigenic, which means that  
728 one single allelic variant explains a very small part of the total variation (see below in section 5).

729 We also did not observe a significant increase of the mean individual heterozygosity in surviving  
730 populations compared to the initial ones, or between surviving populations between exposures. An  
731 increase of this index could have been linked to an overall Heterozygosity-fitness correlation (HFC),  
732 associated with a deleterious effect of inbreeding (Slate et al 2004). We can notice, however, that the  
733 seedlings with lowest heterozygosity values (PHt inferior to 0.235) were often dead at the end of the  
734 experiment, leading to a slight increase of this statistic at the end of the experiment for most families in  
735 both exposures. Although a slight effect of multilocus heterozygosity at SSRs was detected on growth traits  
736 in *Q. robur* (Vranckx et al 2014b, less than 5% of total variation explained), and in other species with  
737 different reproductive strategies (Cole et al 2016 in Aspen; Stilwell et al 2003 in *Castanea dentata*), a better  
738 resistance or tolerance to pathogen infection for heterozygotes has not been established for plants,  
739 contrary to animals (*e.g.*, Budischak et al 2023). Specifically, oaks have generally large populations with a  
740 low inbreeding of their seedlings (Gerzabek et al 2020), a genetic context for which HFC is not expected  
741 (Slate et al 2004). HFC may also be more easily detected in stressful environments (Mopper et al 1991, and  
742 see references above). In addition, opposite correlations between heterozygosity, growth, and  
743 mechanisms of resistance against pathogens were reported in Cole et al (2016). Antagonistic effects of  
744 heterozygosity on different biological traits could occur in oak seedlings, thus masking possible HFC. As  
745 explained above (section 2), tolerance to oak powdery mildew could be a composite process involving

746 different physiological functions that would lead to moderate optimizing selection effects on genotypes  
747 and thus the maintenance of a mean diversity level (Walsh & Lynch 2018).

748

## 749 **5. Few genetic associations identified**

750 Our design with well-characterized phenotypic traits and individuals genotyped at several hundred  
751 SNPs was suitable to test for genetic associations. Overall, in conservative statistical testing conditions,  
752 very few significant associations were observed: zero, one or two per trait for survival, mean powdery  
753 mildew infection and height, and up to seven for acorn weight, across both exposures. We did detect one  
754 SNP significantly associated with seedling survival in the natural exposure, which differed from the one  
755 detected in the disease protected exposure. This SNP was not associated with powdery mildew infection,  
756 and the allele with a beneficial effect on survival was at very low frequency. If true, this beneficial effect  
757 might be counterbalanced by pleiotropic effects, and the advantage detected in our study might be related  
758 to particular conditions. Indeed, this SNP was located in a gene coding for a putative histone H4, a type of  
759 protein involved in the structure of chromatin that has been linked to survival strategy against drought in  
760 plants (Kim et al 2017). The second SNP associated with survival but under protected exposure was located  
761 in a gene coding for a family of proteins involved in organelle biogenesis (O'Toole et al 2008).

762 Genetic association studies of resistance to plant diseases are common in pathosystems of agronomic  
763 interest (*e.g.*, maize (Zhao et al 2022, reviewed in Shrestha et al 2019), wheat (Du et al 2021) or soya bean  
764 (Wen et al 2018)). They are far less common in natural pathosystems. The genetic architecture of resistance  
765 (*sensu lato*) to powdery mildew in oak species is still poorly understood, although QTLs have been detected  
766 and some candidate genes have been suggested (Bartholomé et al 2020). The present study points to  
767 another possible candidate gene identified by the GWAS approach, a gene coding for a putative ethylene  
768 response factor C3. Interestingly, a similar type of protein has been linked to pathogen resistance in cotton  
769 (Guo et al 2016). Despite a measurable effect on tree mortality from oak powdery mildew in our  
770 experimental setup, the only SNP associated with susceptibility to powdery mildew was not associated  
771 with the survival trait. This is probably related to the complex and partly indirect nature of the effect of  
772 powdery mildew on oak mortality, as evidenced by the SEM model results.

773

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781

782

### Data, scripts and codes availability

783 Data, [supplementary data](https://doi.org/10.5281/zenodo.7517641) and scripts are available online in a Zenodo repository:  
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785

786

### Supplementary material

787 Supplementary Materials ~~and data~~ are available online: <https://doi.org/10.5281/zenodo.7931510>

788

789

### Conflict of interest disclosure

790 The authors of this preprint declare that they have no financial conflict of interest with the content of  
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792

793

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796

797

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