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1 **Acclimation to high daily thermal amplitude converts a defense response regulator into**  
2 **susceptibility factor**

3

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11

12 **ABSTRACT**

13 Acclimation enables plants to adapt to immediate environmental fluctuations, supporting biodiversity  
14 and ecosystem services. However, global changes are altering conditions for plant disease outbreaks,  
15 increasing the risk of infections by pathogenic fungi and oomycetes, and often undermining plant  
16 immune responses. Understanding the molecular basis of plant acclimation is crucial for predicting  
17 climate change impacts on ecosystems and improving crop resilience. Here, we investigated how  
18 *Arabidopsis thaliana* quantitative immune responses acclimates to daily temperature fluctuations. We  
19 analyzed responses to the fungal pathogen *Sclerotinia sclerotiorum* following three acclimation  
20 regimes that reflect the distribution areas of both species. Mediterranean acclimation, characterized  
21 by broad diurnal temperature amplitudes, resulted in a loss of disease resistance in three natural *A.*  
22 *thaliana* accessions. Global gene expression analyses revealed that acclimation altered nearly half of  
23 the pathogen-responsive genes, many of which were down-regulated by inoculation and associated  
24 with disease susceptibility. Phenotypic analysis of *A. thaliana* mutants identified novel components of  
25 quantitative disease resistance following temperate acclimation. Several of these mutants were  
26 however more resistant than wild type following Mediterranean acclimation. Notably, mutant lines in  
27 the NAC42-like transcription factor did not show a loss of resistance under Mediterranean acclimation.  
28 This resistance was linked to an acclimation-mediated switch in the repertoire of NAC42-like targets  
29 differentially regulated by inoculation. These findings reveal the rewiring of immune gene regulatory  
30 networks by acclimation and suggest new strategies to maintain plant immune function in a warming  
31 climate.

32 **INTRODUCTION**

33 The ability of species to cope with rising temperatures is a crucial factor influencing range shifts  
34 and local extinctions, as their distribution and range boundaries closely align with temperature  
35 gradients. Evidence shows that plant species adapt to local environmental conditions through genetic  
36 variation (Fournier-Level et al., 2011b; Katz et al., 2021; Clauw et al., 2022) but they also exhibit  
37 phenotypic plasticity allowing individual plants to adjust rapidly their physiology to environmental  
38 variations (Valladares et al., 2014; Brancalion et al., 2018). The short-term, reversible process that  
39 allows plants to cope with immediate environmental fluctuations is often referred to as acclimation  
40 (Kleine et al., 2021). Plant acclimation help maintain the balance of natural systems, supporting  
41 biodiversity and the services that ecosystems provide, such as carbon sequestration and water  
42 regulation. With climate change modifying the distribution area of plants (Sloat et al., 2020) and

43 causing more frequent and severe weather events (Newman and Noy, 2023), knowledge of how plants  
44 acclimate can inform strategies to manage ecosystems and agriculture. In this context, crops that can  
45 acclimate effectively are more likely to maintain high yields despite stressors. A better understanding  
46 of the genetic underpinnings of plant acclimation is therefore crucial for predicting the impact of  
47 climate change on ecosystems and for improving crop resilience.

48 Acclimation distinguishes from adaptation for involving changes to the expression of the  
49 genome instead of heritable changes to genome sequences (Kleine et al., 2021). Epigenetic and  
50 transcriptional regulation mechanisms mediating somatic stress memory are important players in  
51 plant acclimation (Charng et al., 2023; Zuo et al., 2023; Hadj-Amor et al., 2024). Cold acclimation, by  
52 which decreasing temperatures enhance freezing tolerance in plants involves alterations in membrane  
53 composition, the production of cryoprotective polypeptides and solutes, the activation of cold-  
54 responsive (COR) genes regulated by C-repeat binding transcription factors (CBFs/DREB1) (Liu et al.,  
55 2019). The accumulation of heat shock proteins (HSPs) regulated by heat shock transcription factors  
56 (HSFs) and histone 3 K4 methylation play a key role in heat acclimation (Kappel et al., 2023; Nishad and  
57 Nandi, 2021). Besides transcription factors and epigenetic marks, the hormone abscisic acid (ABA) is a  
58 central mediator of the accumulation of LEA-like protective proteins, stomatal closure and  
59 downregulation of photosynthesis under drought acclimation (Sadhukhan et al., 2022). Despite recent  
60 efforts, the interplay between regulatory mechanisms, molecular and phenotypic responses to plant  
61 acclimation is elusive.

62 With changes to the climate, not only the distribution range of plants changes, but also that of  
63 their enemies. Suitable conditions for plant disease outbreaks are expected to shift in time and space  
64 leading to a global poleward movement of plant pathogen geographic niches (Bebber et al., 2013) and  
65 an increased risk of infection by pathogenic fungi and oomycetes (Chaloner et al., 2021). Fungi,  
66 especially generalists with a broad range of plant hosts, are the most widespread and most rapidly  
67 spreading pathogens, so that if current rates persist, several major food producing countries would  
68 have fully saturated pathogen distributions by 2050 (Bebber et al., 2014). A paradigmatic example of  
69 such broad host range pathogen is the white and stem mold fungus *Sclerotinia sclerotiorum*, which  
70 infects hundreds of plant species and causes significant losses to vegetable and oil crops worldwide  
71 (Navaud et al., 2018; Peltier et al., 2012; Cohen, 2023). Although climate change may alter the overlap  
72 between crops cultivation area and *S. sclerotiorum* distribution range (Mehrabi et al., 2019), pathogen  
73 strains adapted to warm temperatures have been reported (Uloth et al., 2015) and extreme  
74 temperature may promote fungal development (Lane et al., 2019; Shahoveisi et al., 2022), raising  
75 concern about *Sclerotinia* disease incidence in the future (Singh et al., 2023).

76 Plant respond to *S. sclerotiorum* by activating quantitative disease resistance (QDR), an  
77 immune response involving multiple genes of weak to moderate phenotypic effect (Roux et al., 2014;  
78 Sucher et al., 2020). Molecular players involved in QDR against *S. sclerotiorum* include immune  
79 receptors, reactive oxygen species, phytohormones such as ABA, jasmonic acid and ethylene,  
80 transcription factors and phytoalexins (Perchepied et al., 2010; Mbengue et al., 2016; Derbyshire and  
81 Raffaele, 2023). Several of these determinants contribute to multiple biological processes such as plant  
82 development and response to the abiotic environment (Corwin et al., 2016; Badet et al., 2019; Léger  
83 et al., 2022). The genetic architecture of QDR suggests that the expression of many genes involved in  
84 QDR could be modulated by environmental conditions (Hadj-Amor et al., 2024), and that climate  
85 change may alter plant QDR response to *S. sclerotiorum* at the phenotypic and molecular level.  
86 Temperature increase notably is known to frequently impair plant immune responses, including QDR  
87 (Desaint et al., 2021; Aoun et al., 2017).

88 Analyses of plant immune responses under abiotic constraints generally focus on pathogen  
89 inoculation under prolonged and stable abiotic conditions. In *A. thaliana*, immunity against the  
90 bacterial pathogen *Pseudomonas syringae* pv. *tomato* at elevated temperature can be restored by the  
91 constitutive expression of *CBP60g*, a major transcriptional regulator of plant immunity genes and  
92 salicylic acid (SA) defense hormone production, downregulated by temperature (Kim et al., 2022). This  
93 finding indicates that engineering plant transcriptional circuits can mitigate the negative effect of  
94 climate change on some plant immune responses. Whether this strategy would restore resistance  
95 against necrotrophic pathogens such as *S. sclerotiorum*, only weakly sensitive to SA-mediated defense,  
96 remains to be determined. Another promising target is the disordered protein TWA1, a temperature  
97 sensor proposed to orchestrate acclimation by integrating temperature with ABA and JA signaling  
98 (Bohn et al., 2024), which play important roles in plant defense against necrotrophs. When applied  
99 sequentially, prior abiotic signals may alter the transcriptional and metabolic response to a subsequent  
100 pathogen inoculation (Coolen et al., 2016; Garcia-Molina et al., 2020; Garcia-Molina and Pastor, 2024).  
101 In addition to mean temperature increase, climate change drives an expansion of diurnal temperature  
102 range (Zhong et al., 2023). Daily fluctuations of the environment may alter plant metabolism, growth  
103 and flowering (Burghardt et al., 2016; Deng et al., 2021; Matsubara, 2018) as well as gene regulation  
104 and invasive growth of fungal pathogens (Jallet et al., 2020; Bernard et al., 2022). Yet, how plant  
105 immunity acclimates to daily temperature fluctuations remains largely unexplored.

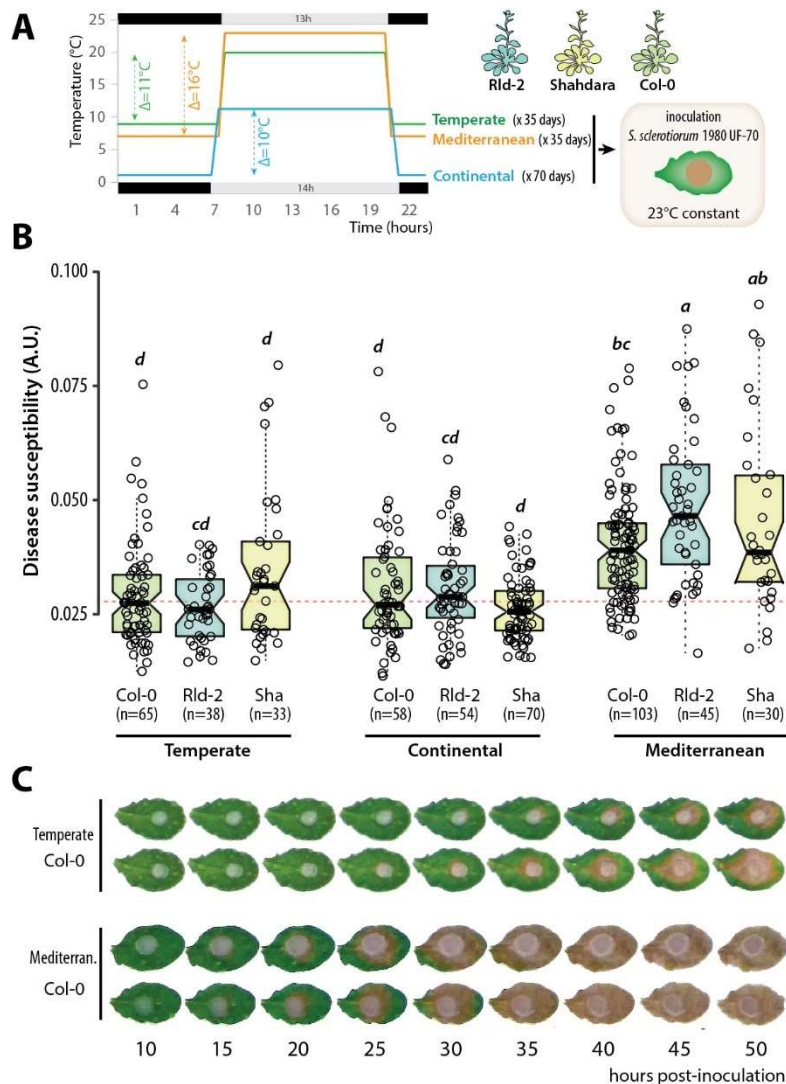
106 To fill this gap, we analyzed *A. thaliana* immune responses upon *S. sclerotiorum* inoculation  
107 following three acclimation regimes representing the distribution area of these two species.  
108 Mediterranean acclimation, characterized by a broad diurnal temperature amplitude, caused a loss of  
109 disease resistance in the three natural accessions we tested. Using global gene expression analyses,  
110 we show that acclimation alters the expression of nearly a half of pathogen-responsive genes, many  
111 of which are down-regulated by inoculation and associated with disease susceptibility. The phenotypic  
112 analysis of *A. thaliana* mutants identified novel components of QDR following temperate acclimation.  
113 Several of these mutants were however more resistant than wild type following Mediterranean  
114 acclimation. In particular, contrary to wild type, two mutant lines in the NAC42-like transcription factor  
115 showed no loss of resistance upon Mediterranean acclimation. These phenotypes associated with a  
116 switch in the repertoire of NAC42-like targets differentially regulated by inoculation according to  
117 acclimation. These findings reveal the rewiring of immune gene regulatory networks by acclimation  
118 and open new perspectives to safeguard the functioning of the plant immune system in a warming  
119 climate.

## 120 RESULTS

### 121 Mediterranean-like acclimation impairs the resistance of several *A. thaliana* accessions to *S.* 122 *sclerotiorum*

123 To determine the effect of acclimation on *A. thaliana* quantitative disease resistance (QDR), we  
124 analyzed phenotypic variation of three *A. thaliana* accessions after growth in three simulated climates.  
125 We selected accessions Col-0, Rld-2 and Shahdara (Sha) as representatives of genetic and geographical  
126 diversity of *A. thaliana* species. For acclimation, plants were grown under day length, day and night  
127 temperatures corresponding to the 30-year average for the month of April in areas with a temperate  
128 (Cfa), continental (Dfb) and Mediterranean (Csa) climates (Fig. 1A, Fig. S1). These correspond to  
129 climates in the distribution range of *A. thaliana* with major projected area variation by the end of this  
130 century (Alonso-Blanco et al., 2016; Peel et al., 2007; Cui et al., 2021). Plants were grown for 35 days  
131 under temperate and Mediterranean climate, corresponding to 13,405 and 13,930 °C.days, and for 70  
132 days under continental climate corresponding to 11,480 °C.days before inoculation with *S.*  
133 *sclerotiorum* under infection-conducive conditions. Plant susceptibility was assessed using time-

134 resolved automated phenotyping (Barbacci et al., 2020). After temperate acclimation, all accessions  
 135 appeared similarly susceptible with only a slightly lower susceptibility (-9% average) for Rld-2 and a  
 136 slightly higher susceptibility for Sha (+18% average) compared to Col-0 (**Fig. 1B, Table S1**). These  
 137 phenotypes were not significantly altered upon continental acclimation. Mediterranean acclimation  
 138 rendered all accession significantly more susceptible, with an average increase by 29% for Sha, 37%  
 139 for Col-0 and 86% for Rld-2 as compared to temperate acclimation (**Fig 1B, C**). These results show that  
 140 both genotype and acclimation affect the susceptibility of *A. thaliana* to *S. sclerotiorum* and that,  
 141 regardless of genotype, Mediterranean acclimation caused the most significant loss of resistance.



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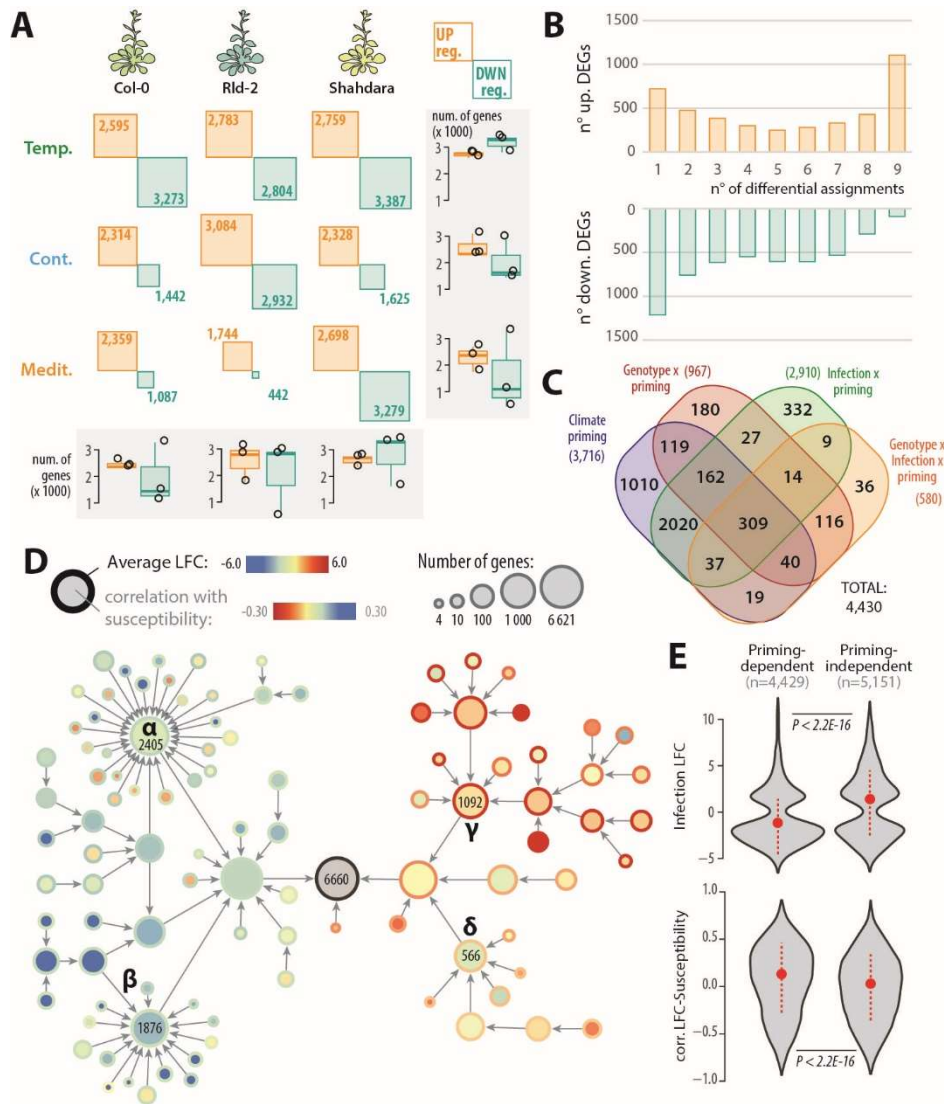
143 **Fig 1. Effect of three distinct pre-infection climate conditions (acclimation) on *A. thaliana* quantitative disease resistance**  
 144 **to *S. sclerotiorum*.** (A) Experimental design showing acclimation and inoculation phases. Daylength, day and night  
 145 temperatures typical of temperate, continental and Mediterranean climate conditions define the three acclimation  
 146 conditions used in this work. (B) Susceptibility phenotype in response to *S. sclerotiorum* infection as a function of acclimation  
 147 and genotype. Each experiment was repeated at least 3 times and the significance of the results was assessed by an ANOVA  
 148 followed by a Tukey HSD test, with significance groups labelled by letters. Boxplots show first and third quartiles (box), median  
 149 (thick line), and the most dispersed values within 1.5 times the interquartile range (whiskers). (C) Representative symptoms  
 150 of Col-0 plants between 10 and 50 hours post-inoculation by *S. sclerotiorum* on leaves harvested on plants acclimated in  
 151 temperate and Mediterranean (Mediterran.) conditions.

152 **Acclimation primarily alters the expression of infection-downregulated genes**

153 To study the molecular bases of quantitative disease resistance acclimation, we performed a global  
154 transcriptome analysis of *A. thaliana* accessions Col-0, Rld-2 and Sha grown in temperate, continental  
155 and Mediterranean climates, followed or not by *S. sclerotiorum* inoculation. To identify genes  
156 responsive to infection we performed a differential expression analysis using non-inoculated plants as  
157 reference in each of nine conditions (three climate priming, times three plant genotypes). We found  
158 17,137 nuclear-encoded genes with sufficient coverage (**Table S2**), and identified 9,580 differentially  
159 expressed genes (DEGs) upon inoculation at  $|\text{Log}_2 \text{ Fold Change}| \geq 2$  and Bonferroni-adjusted  $p$ -  
160  $\text{val} < 0.0001$  (**Fig 2A, Fig S2, Table S3**). The number of upregulated genes ranged from 1,744 (Rld-2  
161 Mediterranean acclimation) to 3,084 (Rld-2 continental acclimation), the number of downregulated  
162 genes ranged from 442 (Rld-2 Mediterranean acclimation) to 3,387 (Sha temperate acclimation). The  
163 three accessions showed a reduced number of DEGs when acclimated in conditions very divergent to  
164 the climate at their area of origin. Downregulated genes showed a relatively high degree of specificity  
165 with 1,212 genes (23%) unique to one genotype-acclimation pair and only 89 (1.7%) genes differential  
166 in all nine genotype-acclimation conditions tested (**Fig 2B**). Upregulated genes showed higher  
167 robustness with 1,105 genes (26.8%) differential in all nine genotype-acclimation conditions.

168 Next, we performed an analysis of variance on the 9,580 DEGs to determine which of the plant  
169 genotype, infection status, acclimation, and their interactions, contributed the most to expression  
170 variation for each gene. As expected, infection contributed significantly (Benjamini-Hochberg  
171 corrected  $p$ -val  $< 1E-3$ ) to the expression variance for 8,523 genes (89%). Genotype and acclimation  
172 contributed significantly to the expression variance for 3,111 and 3,716 genes (32.5% and 38.8%)  
173 respectively (**Fig 2C, Table S4**). Considering genes the expression variance of which is significantly  
174 altered by either acclimation alone or interaction between acclimation and any other factor,  
175 acclimation had an impact on the expression of 4,430 genes responsive to infection (46.2% of DEGs,  
176 **Fig S3, Table S5**).

177 To document the relationship between transcriptional response to *S. sclerotiorum* inoculation and  
178 acclimation, we built a gene co-expression hierarchical network with genes modulated by inoculation  
179 both in the differential and ANOVA analyses. For this, we used normalized read counts to calculate  
180 Spearman rank correlation coefficient for all pairwise gene comparisons across our 54 RNA-seq  
181 samples. Highly co-expressed gene pairs were grouped into hierarchical gene communities using the  
182 HiDeF algorithm (Zheng et al., 2021). 6,620 genes were included into communities of at least four  
183 genes (**Fig 2D, Data S1**). Four major top-level communities (labeled  $\alpha$  to  $\delta$  in **Fig 2D**) encompassed  
184 5,933 genes (89.6% of the network). In average, communities  $\alpha$  and  $\beta$  included genes with expression  
185 anticorrelated with resistance (putative susceptibility factors, **Table S6**), frequently acclimation  
186 dependent and downregulated upon *S. sclerotiorum* inoculation and upon heat stress. By contrast,  
187 genes from communities  $\gamma$  and  $\delta$  had expression correlated with resistance, frequently  
188 acclimation-independent, up-regulated upon *S. sclerotiorum* inoculation and heat stress (**Fig 2D, Fig**  
189 **S4**). Accordingly, the median LFC upon inoculation was -1.56 in acclimation-dependent DEGs but 0.70  
190 in acclimation independent DEGs, the median correlation between LFC and susceptibility phenotype  
191 was 0.09 in acclimation-dependent DEGs but -0.01 in acclimation independent DEGs (**Fig 2E**). Genes  
192 downregulated by infection were 64.3% and 46.7% among genes acclimation-dependent and  
193 independent respectively (1.37-fold enrichment). Reciprocally, genes upregulated by infection were  
194 35.5% and 52.6% among genes acclimation-dependent and independent respectively (1.48-fold  
195 depletion). Gene the expression of which is correlated (Pearson  $> 0.5$ ) with the susceptibility phenotype  
196 were 12.3% and 19.2% among genes acclimation-dependent and independent respectively (1.56-fold  
197 enrichment). We conclude that acclimation primarily alters the expression of genes down-regulated  
198 by infection and genes associated with disease susceptibility.



199

200 **Fig 2. Global gene expression profiling of *A. thaliana* plants inoculated by the fungal pathogen *S. sclerotiorum* following**  
 201 **temperate, continental and Mediterranean acclimation.** (A) Number of differentially expressed genes (DEGs) upregulated  
 202 (yellow) and down-regulated (blue) 48 hours post inoculation by *S. sclerotiorum* in each of three plant genotypes and three  
 203 acclimation conditions. Box plots show the distribution of DEG number per genotype (columns) and acclimation (rows), first  
 204 and third quartiles (box), median (thick line), and the most dispersed values within 1.5 times the interquartile range (whiskers)  
 205 are shown. (B) Number of up- and down-regulated DEGs according to the number of differential assignments, out of 9 tested  
 206 conditions. (C) Distribution of acclimation-dependent DEGs identified by ANOVA according to the factors explaining gene  
 207 expression variance. (D) A hierarchical network of genes mis-regulated by *S. sclerotiorum* infection identified through  
 208 differential and variance analyses. Nodes represent gene communities sized according to the number of DEGs they contain,  
 209 fill color corresponding average correlation between gene expression and plant susceptibility, border color correspond to  
 210 average LFC upon inoculation. Four major communities are labelled and their number of genes indicated. (E) Distribution of  
 211 infection LFC and correlation between LFC and susceptibility phenotype for acclimation dependent and independent DEGs.  
 212 Violin plots show a gaussian kernel, median (dot) and standard deviation (dotted lines).

### 213 Mediterranean acclimation turns some pathogen-responsive genes into susceptibility factors

214 To get insights into the role of DEGs in *A. thaliana* QDR against *S. sclerotiorum*, we first analyzed Gene  
 215 Ontologies (GO) enriched in each of the four major top-level gene communities from our hierarchical  
 216 network, relative to the rest of the network (Fig 3A, Table S7). Community  $\alpha$  was enriched in 96  
 217 biological process (BP) and 9 molecular function (MF) GO, with ‘Starch metabolism’, ‘Photosynthesis’,  
 218 ‘Translation’, ‘Primary metabolism’, ‘Chlorophyll binding’, ‘Exopeptidase activity’ and ‘Constituent of  
 219 ribosome’ among the most enriched, reflecting a general downregulation of energetic functions of the

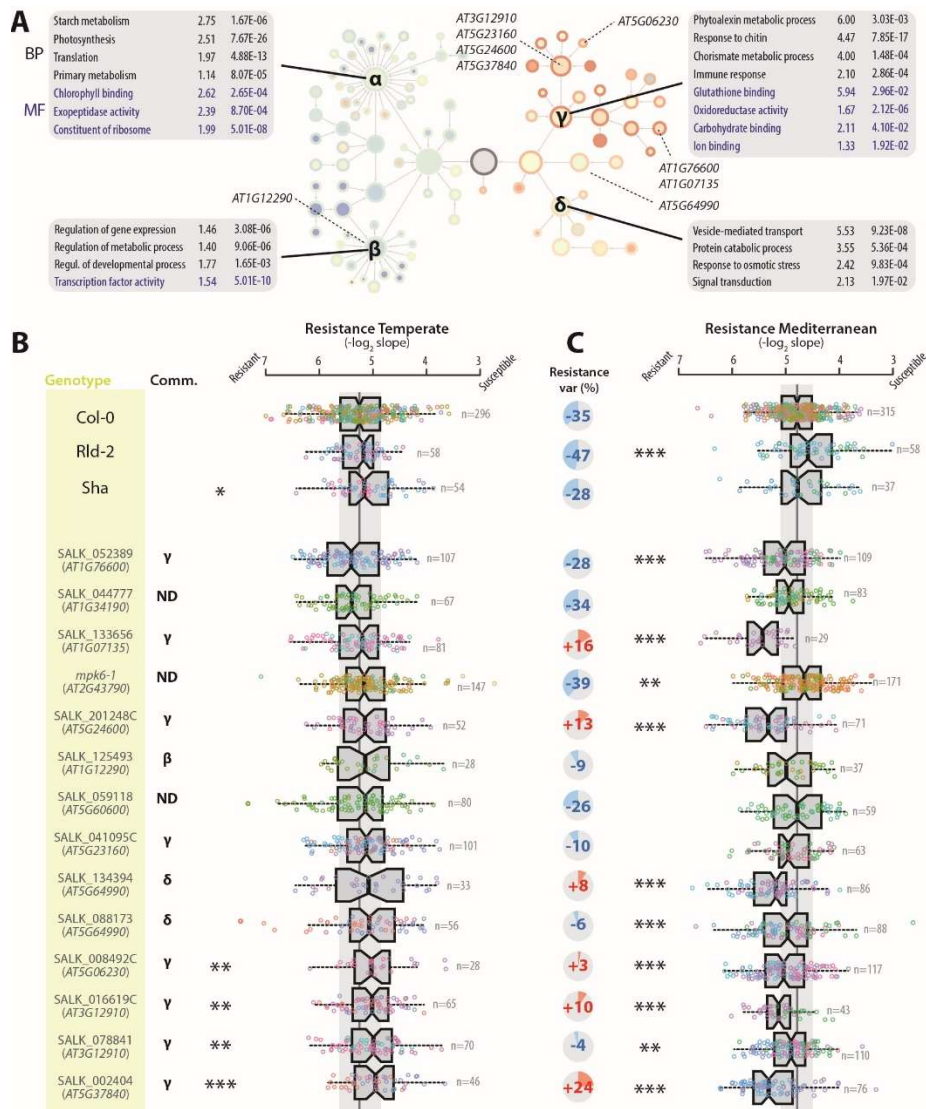
220 plant cell during infection. Community  $\beta$  was enriched in 18 BP and 4 MF GOs with ‘Regulation of gene  
221 expression’, ‘Regulation of metabolic process’, ‘Regulation of developmental process’ and  
222 ‘Transcription factor activity’ among the most enriched. Community  $\gamma$  was enriched in 68 BP and 22  
223 MF GOs, with ‘Phytoalexin metabolic process’, ‘Response to chitin’, ‘Chorismate metabolic process’,  
224 ‘Immune response’, ‘Glutathione binding’, ‘Oxidoreductase activity’, ‘Carbohydrate binding’ and ‘Ion  
225 binding’ among the most enriched, reflecting the probable involvement of genes from this community  
226 in disease resistance. Finally, community  $\delta$  was enriched in 40 BP and 10 MF GOs, with ‘Vesicle-  
227 mediated transport’, ‘Protein catabolic process’, ‘Response to osmotic stress’ and ‘Signal transduction’  
228 among the most enriched, consistent with a role in stress response.

229 To study the role of DEGs in disease resistance against *S. sclerotiorum*, we analyzed the phenotype of  
230 14 mutant lines in the Col-0 background corresponding to 11 distinct genes, with a focus on genes from  
231 community  $\gamma$  that were not previously associated with plant immunity (**Table S8**). For comparison  
232 purposes, we included mutants in one gene from community  $\beta$  (*AT1G12290*), one from community  $\delta$   
233 (*AT5G64990*) and three genes not differentially expressed in our RNA-seq experiment (*AT1G34190*,  
234 *AT2G43790* and *AT5G60600*). The natural accessions Col-0, Rld-2 and Sha were used as references.  
235 After temperate acclimation (**Fig 3B**), four mutant lines were significantly more susceptible than the  
236 Col-0 wild type, affecting genes *AT5G06230*, *AT3G12910* and *AT5G37840* from community  $\gamma$ . After  
237 Mediterranean acclimation (**Fig 3C, Table S9**), all three natural accessions were more susceptible than  
238 after temperate acclimation, consistent with our previous set of experiments (**Fig 1B**). Rld-2 was more  
239 strongly affected by Mediterranean acclimation and became significantly more susceptible than Col-0  
240 in these conditions. To our surprise, *mpk6-1* was the only mutant significantly more susceptible than  
241 Col-0 after Mediterranean acclimation. Nine mutants were more resistant than wild type after  
242 Mediterranean acclimation, covering genes *AT1G76600*, *AT1G07135*, *AT5G24600*, *AT5G06230*,  
243 *AT3G12910* and *AT5G37840* from community  $\gamma$ , *AT5G64990* and *AT5G06230* from community  $\delta$ . While  
244 natural accessions had their resistance phenotype reduced by ~37% in average after Mediterranean  
245 compared to temperate acclimation, only four mutant lines showed >25% resistance reduction,  
246 including three in genes not differentially expressed upon inoculation (*AT1G34190*, *AT2G43790* and  
247 *AT5G60600*) and one from community  $\gamma$  (*AT1G76600*). By contrast, six mutants showed increased  
248 resistance after Mediterranean compared to temperate acclimation.

249 Together these results confirm that community  $\gamma$  includes several genes contributing to resistance  
250 against *S. sclerotiorum* after temperate acclimation. Mutations in several genes from community  $\gamma$   
251 render plants more resistant than wild type after Mediterranean acclimation, indicating that they act  
252 as susceptibility factors in these conditions. Remarkably, *AT5G06230*, *AT3G12910* and *AT5G37840*  
253 would classify as resistance factors in temperate-acclimated plants but as susceptibility factors in  
254 Mediterranean-acclimated plants.

255





256

257 **Fig 3. Functional analysis of pathogen-induced genes.** (A) Gene ontology (GO) enrichment in the four major gene  
 258 communities identified based on a co-expression network of genes mis-regulated by *S. sclerotiorum* infection. Communities  
 259  $\alpha$ ,  $\beta$ ,  $\gamma$ ,  $\delta$  are labelled on the hierarchical network shown with the same layout as in Fig2D. A selection of the most enriched  
 260 biological process (BP, black) and molecular function (MF, blue) GOs are labelled, with enrichment fold and adjusted p-value  
 261 relative to *A. thaliana* genome indicated. Genes analyzed through mutant phenotyping are labeled according to their position  
 262 in the network. (B, C) Disease resistance phenotype of natural accessions and mutant plants following temperate acclimation  
 263 (B) or Mediterranean acclimation (C) and inoculated by *S. sclerotiorum*. Comm. Major community of the co-expression  
 264 network to which the gene belongs. ND, gene not differentially expressed upon *S. sclerotiorum* inoculation (not part of the  
 265 co-expression network). Pie chart in (C) indicate mean % variation of disease resistance relative to infection following  
 266 temperate acclimation. Boxplots show first and third quartiles (box), median (thick line), and the most dispersed values within  
 267 1.5 times the interquartile range (whiskers). Colors of the data points indicate independent inoculation experiments. Leaves  
 268 from n=29 to 315 plants were tested for each genotype. Significance of the difference from Col-0 wild type was assessed by  
 269 a Student's t test followed by Benjamini-Hochberg correction for multiple testing (\*\*\*)  $p < 0.01$ , \*\*  $p < 0.05$ , \*  $p < 0.1$ ).

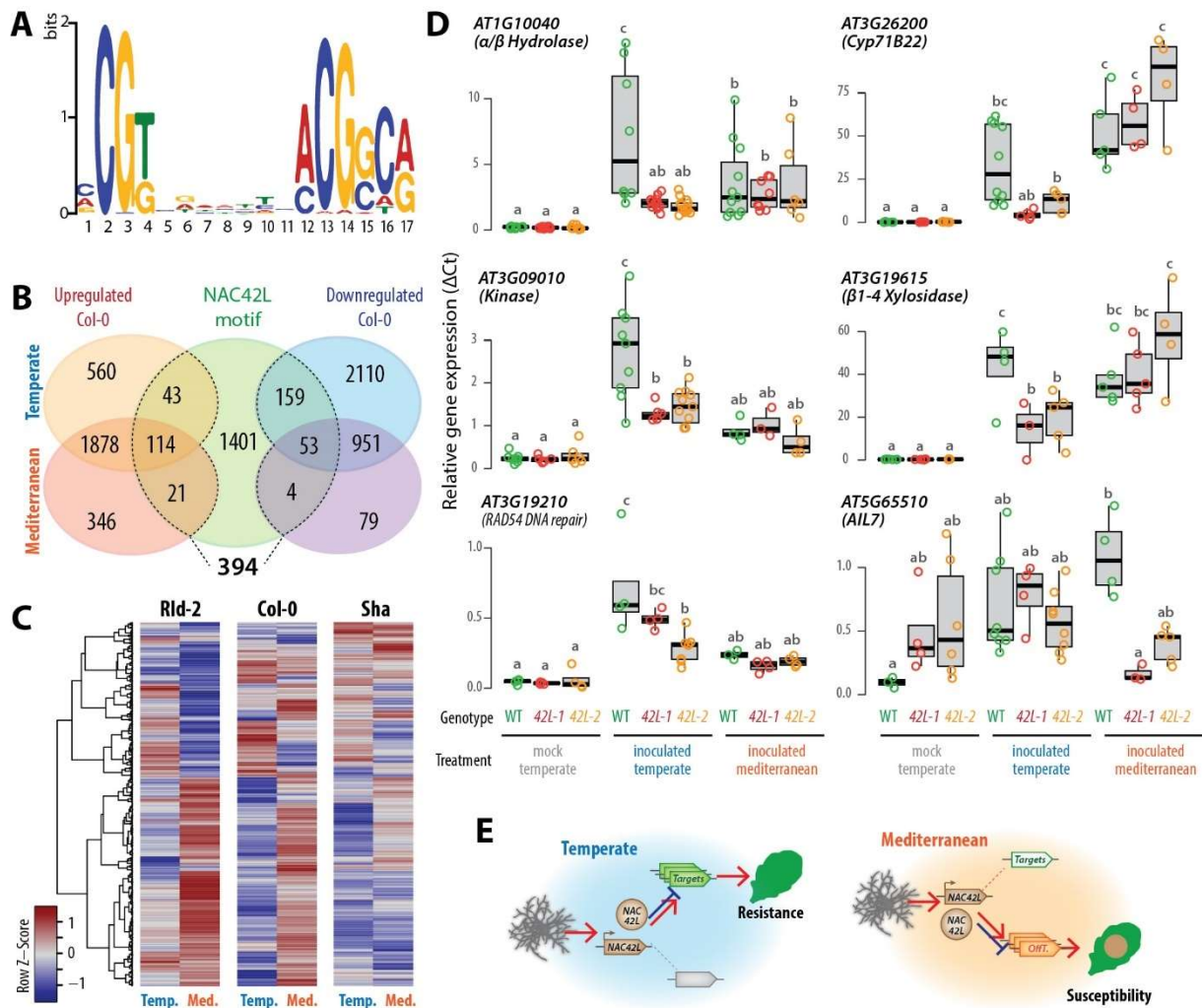
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### 271 Acclimation shifts the repertoire of NAC42-L target genes upon *S. sclerotiorum* inoculation

272 *AT3G12910* encodes a member of the NAC family of transcription factors that includes several  
 273 regulators of pathogen and abiotic stress response (Nuruzzaman et al., 2013). Its closest homolog in  
 274 *A. thaliana* genome is *NAC42/JUNGBRUNNEN1* (*AT2G43000*) (Ooka et al., 2003), we will thus refer to  
 275 *AT3G12910* as *NAC42-Like* (*NAC42-L*) hereafter. *NAC42-L* is strongly induced upon *S. sclerotiorum*

276 inoculation both in plants temperate- (LFC 7.8 p-adj. 3E-08 in Col-0) and Mediterranean-acclimated  
277 (LFC 7.5 p-adj. 8E-22 in Col-0). Yet two mutant alleles of *NAC42-L* resulted in lower disease resistance  
278 in temperate acclimated plants but enhanced disease resistance in Mediterranean acclimated plants  
279 (**Fig 3B**). To study how acclimation alters the activity of *NAC42-L* at the molecular level, we analyzed  
280 the expression of its target genes upon infection in temperate- and Mediterranean-acclimated plants.  
281 For this, we first identified targets presumably regulated by *NAC42-L* by searching for *NAC42-L* DNA  
282 binding motif determined by (O'Malley et al., 2016) in the promoter of *A. thaliana* genes. This  
283 identified 2276 potential *NAC-L* binding sites in 1795 different gene promoters, with a maximum of 5  
284 binding sites per promoter (**Table S10**). Among *NAC42-L* targets, 394 genes were DEGs upon *S.*  
285 *sclerotiorum* inoculation in temperate- or Mediterranean-acclimated Col-0 plants (**Fig 4B**). There were  
286 227 *NAC-L* targets (57.6% of *NAC42-L* target DEGs) uniquely differential following growth under one of  
287 the two climates, indicating a significant switch in the regulation of *NAC42-L* target genes upon  
288 infection according to acclimation. The effect of acclimation on the regulation of *NAC42-L* target genes  
289 upon *S. sclerotiorum* inoculation was clearly detectable in the three accessions we analyzed (**Fig 4C**).

290 To test whether the acclimation-mediated switch in *NAC42-L* targets was dependent on *NAC42-L*, we  
291 measured by quantitative RT-PCR the expression of eight of these targets in two *nac42-L* mutant lines  
292 (*42-L1* and *42-L2*) following temperate and Mediterranean acclimation (**Fig 4D**, **Table S11**, **Fig S5**). Six  
293 of these genes had an expression significantly altered by the inactivation of *NAC42-L*, supporting their  
294 position as targets of *NAC42-L* regulation. Yet for all of them, the impact of *NAC42-L* inactivation on  
295 their expression was only detected after one particular acclimation regime. Indeed, *AT1G10040*,  
296 *AT3G09010*, *AT3G26200* and *AT3G19615* were upregulated upon *S. sclerotiorum* inoculation following  
297 temperate acclimation in wild-type plants but significantly less in *42-L1* and *42-L2* plants, while the  
298 expression of these genes was similar in all three genotypes following Mediterranean acclimation.  
299 Conversely, *AT5G65510* showed a similar expression in wild type and *nac42-L* mutant lines upon  
300 inoculation following temperate acclimation, but it was significantly mis-regulated in *nac42-L* mutants  
301 following Mediterranean acclimation. Together, these results suggest that acclimation alters the  
302 contribution of *NAC42-L* to quantitative disease resistance by switching the repertoire of genes  
303 regulated by this transcription factor (**Fig 4E**).



304

305 **Fig 4. Effect of temperate and Mediterranean acclimation on the regulation of gene expression by the transcription factor**  
 306 **NAC42-L.** (A) Sequence logo of the promoter motif bound by NAC42-L according to DAP-seq data. (B) Distribution of genes  
 307 harboring NAC42-L motifs in their promoter between up- and down- regulated genes upon *S. sclerotiorum* inoculation in Col-  
 308 0 plants temperate- and Mediterranean-acclimated. (C) Relative induction of the 394 genes differentially expressed upon  
 309 inoculation harboring NAC42-L motifs in their promoter in Col-0 and Sha accessions following temperate (Temp.) and  
 310 Mediterranean (Med.) acclimation. (D) Expression of six NAC42-L predicted targets in wild type (WT) and *nac42-L* mutants  
 311 (*42-L1*, *42-L2*) in mock-treated and *S. sclerotiorum*-inoculated plants following temperate acclimation and *S. sclerotiorum*-  
 312 inoculated plants following Mediterranean acclimation. Boxplots show expression independent measurements for 3-9 plants  
 313 (dots) with first and third quartiles (box), median (thick line), and the most dispersed values within 1.5 times the interquartile  
 314 range (whiskers). Letters indicate groups of significance determined by a Tuckey HSD test following one-way ANOVA. (E)  
 315 Schematic representation of the proposed mechanism through which acclimation switches NAC42-L from a positive regulator  
 316 of disease resistance (temperate acclimation) to a negative regulator (Mediterranean acclimation). *S. sclerotiorum*  
 317 inoculation triggers the expression of NAC42-L (brown arrow) and accumulation of NAC42-L protein (brown circle) which  
 318 regulates positively (red arrow) or negatively (blue blocked arrow) target genes. Upon temperate acclimation, NAC42-L  
 319 targets (green arrow) may positively contribute to disease resistance, while upon Mediterranean acclimation, NAC42-L “off-  
 320 targets” (orange arrows) mostly promote susceptibility to pathogens.

321

## 322 DISCUSSION

323 Phenotypic plasticity, a component of acclimation, allows plant species to adjust to environmental  
 324 conditions, together with adaptation through natural selection or migration to follow conditions to  
 325 which they are adapted. Understanding the molecular mechanisms of acclimation is crucial for  
 326 predicting changes in species distributions, community composition and crop productivity under

327 climate change. In this work we show that *A. thaliana* Mediterranean acclimation is detrimental for  
328 disease resistance to the fungus *S. sclerotiorum* and converts several genes that contribute positively  
329 to quantitative immunity following temperate acclimation into susceptibility factors. Mediterranean  
330 acclimation involves a shift in the repertoire of targets of the pathogen-induced transcription factor  
331 NAC42-like that may impair the regulation of quantitative immune responses.

332 Experiments in controlled conditions have been instrumental in unraveling complex stressor  
333 interactions through tightly controlled factorial experiments. These studies emphasized that combined  
334 effects of various environmental stressors resulted in unique transcriptional changes distinct from  
335 individual stress responses (Sewelam et al., 2014; Zandalinas and Mittler, 2022). These interactions  
336 can be synergistic, where stressors amplify each other's negative effects, or antagonistic, where they  
337 dampen each other's impacts (Zarattini et al., 2021). Research on plant-pathogen interactions under  
338 abiotic constraints often relies on long-lasting stable temperature shifts, overlooking the complex  
339 acclimation processes plants undergo in response to gradual climatic shifts (Aoun et al., 2017; Desaint  
340 et al., 2021). Several studies investigated the effect of temperature acclimation by applying a stable  
341 temperature shift over a few days prior to a second stress application. For instance, growth of *A.*  
342 *thaliana* for 7 days at 4°C enhanced survival to freezing in a NPR1-dependent manner (Olate et al.,  
343 2018), and two-days growth at 30°C rendered plant more susceptible to the bacterial pathogen  
344 *Pseudomonas syringae* pv. *tomato* DC3000 when inoculation is performed either at 23°C or 30°C (Huot  
345 et al., 2017). Nevertheless, the impact of day-night temperature cycles on subsequent stress response  
346 is rarely considered. We have chosen to approximate realistic climate change scenarios by simulating  
347 30-year day and night average temperatures and photoperiods representing three climates of the  
348 Köppen-Geiger classification (Peel et al., 2007). Since the 1980s, Mediterranean climates with dry  
349 summer (Cs) have gradually replaced areas with temperate climate (Cf) (Cui et al., 2021). Predictions  
350 suggest that the Mediterranean (Csa) climate may replace a portion of the continental (Df, Dw, Ds)  
351 climates by the end of the century (Beck et al., 2018; Cui et al., 2021). Significant poleward shifts were  
352 observed for temperate (C), continental (D), and polar (E) climates with averages of 35.4, 16.2, and  
353 12.6 km.decennia<sup>-1</sup> (0.32, 0.15, and 0.11° latitude.decennia<sup>-1</sup> respectively), and are expected to  
354 accelerate in the coming decades (Chan and Wu, 2015; Cui et al., 2021). The three selected climates  
355 therefore cover a significant part of *A. thaliana* distribution area and reflect the poleward shift of  
356 climate zones and associated changes in temperature and day length. Our work revealed a significant  
357 loss of quantitative disease resistance upon Mediterranean acclimation in multiple *A. thaliana*  
358 accessions, although the daily average temperature was only 0.7°C higher under Mediterranean  
359 acclimation (average 15.67°C) than under temperate acclimation (average 14.96°C). Given the current  
360 data, we cannot determine whether the observed phenotypic differences are attributable to daytime  
361 temperatures, nighttime temperatures, the photoperiod, or a combination of these factors.  
362 Nevertheless, our findings indicate that the typical April conditions of the Mediterranean climate zone,  
363 expected to expand by the end of the century due to global warming, are detrimental to resistance  
364 against *Sclerotinia* diseases. Combined with episodes of high humidity conducive to infection, global  
365 warming may therefore increase the incidence of these plant diseases.

366 Our global transcriptome analyses indicated that the three *A. thaliana* accessions tended to show a  
367 higher number of DEGs when acclimated in conditions close to the climate at their area of origin. Col-  
368 0, originating from temperate (Cfb, **Fig. S1**) climate area (Somssich, 2019), and Rld-2, originating from  
369 continental (Dfb) climate area (Alonso-Blanco et al., 2016) has more upregulated and downregulated  
370 genes when acclimated under temperate and continental conditions respectively. Sha, originating  
371 from Mediterranean (Csa) climate (Alonso-Blanco et al., 2016) showed more down-regulated following  
372 temperate acclimation but more upregulated genes following Mediterranean acclimation. Although  
373 this did not reflect at the phenotype level, this transcriptome pattern suggests that *A. thaliana*

374 accessions adapted to their climate of origin to acclimate more efficiently, producing a stronger  
375 immune response at the molecular level. It also suggests that mapping to the Col-0 reference genome  
376 did not introduce major bias in gene expression quantification in other accessions. Adaptation to  
377 environmental change involves variations in allele frequencies within a population's gene pool over  
378 several generations, while acclimation occurs reversibly within an organism's lifestyle. Genetic  
379 variation is crucial for both plastic and adaptive potential (Fox et al., 2019). Reduced genetic variation  
380 from positive selection or limited migration can lower phenotypic plasticity. Conversely, plastic traits  
381 may become fixed or constitutively expressed through genetic assimilation (Wood et al., 2023).

382 High genetic variation in natural populations enhances their ability to withstand and adapt to new  
383 biotic and abiotic environmental changes, including climate change (Van Kleunen and Fischer, 2005;  
384 Nicotra et al., 2010). This genetic variation partly determines the capacity of plants to sense  
385 environmental changes and generate plastic responses. For instance, cis-regulatory and epigenetic  
386 variation at the *FLOWERING LOCUS C* floral repressor regulating vernalization can aid plant populations  
387 in adapting to temperature fluctuations (Hepworth et al., 2020). Yet, the role of selection and whether  
388 gene expression plasticity facilitates or hinders adaptation remains a matter of debate (Levis and  
389 Pfennig, 2016). Comparative analysis of gene expression in forest and urban populations of *Anolis*  
390 lizards showed that rapid parallel regulatory adaptation to urban heat islands primarily resulted from  
391 selection for reduced and/or reversed heat-induced plasticity, which is maladaptive in urban thermal  
392 conditions (Campbell-Staton et al., 2021). A meta-analysis of reciprocal transplant experiments  
393 indicated that adaptation to new environments only leads to genes losing their expression plasticity  
394 by genetic assimilation in rare cases (Chen and Zhang, 2024). In agreement, our results suggest that  
395 adaptation to their climate of origin maintained high expression plasticity of immunity genes in *A.*  
396 *thaliana* accessions. These insights will be valuable for assessing the adaptive potential of populations  
397 in the face of ongoing global climate change.

398 We identified three genes the inactivation of which in the Col-0 background lead to increased pathogen  
399 susceptibility following temperate acclimation but increased resistance following Mediterranean  
400 acclimation. Mutation in six other genes resulted in increased resistance following Mediterranean  
401 acclimation but no significant phenotype change following temperate acclimation. Conditionally  
402 beneficial or neutral mutations, that are deleterious in some environments but beneficial or neutral in  
403 others, have been reported in a wide range of organisms including plants (Elena and de Visser, 2003;  
404 Anderson et al., 2013). Recombinant inbred lines of the Brassicaceae plant *Boechera stricta* of diverse  
405 origin revealed that selection favored local alleles in contrasted environments, and 8.1% of the  
406 assessed markers showed evidence for conditional neutrality for the probability of flowering  
407 (Anderson et al., 2013). In the perennial grass *Panicum hallii*, an allele of the *FLOWERING LOCUS T-like*  
408 *9* locus from coastal ecotypes conferred a fitness advantage only in its local habitat but not at the  
409 inland site (Weng et al., 2022).

410 Loss of function alleles contribute to species adaptation (Olson, 1999; Xu and Guo, 2020) and have  
411 played an important role in crop domestication (Monroe et al., 2020). Naturally occurring loss of  
412 function variants are relatively rare, with an average 57 per genome in *A. thaliana* (Xu et al., 2019) and  
413 18 per genome in soybean (Torkamaneh et al., 2019) but they are found in 19% of soybean genes and  
414 66% of *A. thaliana* genes. Conditionally neutral mutations are sufficient to drive patterns of local  
415 adaptation in simulations (Mee and Yeaman, 2019) and can emerge as a compensation to deleterious  
416 mutations (Steinberg and Ostermeier, 2024; Farkas et al., 2022). Simulations of long-term evolution in  
417 changing environments produced complex gene regulatory networks with an increased rate of  
418 beneficial mutations, while a majority of mutations remain neutral (Crombach and Hogeweg, 2008).  
419 Patterns of local adaptation in *A. thaliana* (Fournier-Level et al., 2011a), the complexity of quantitative

420 immunity networks (Delplace et al., 2020) and our focus on inoculation up-regulated genes may  
421 explain the high proportion of conditionally beneficial loss-of-function we have identified. This finding  
422 suggests that targeted gene knockouts may be a promising strategy to improve climate resilience of  
423 plant immunity.

424 We identified *NAC42-L* (*AT3G12910*) as a resistance factor following temperate acclimation but a  
425 susceptibility factor following Mediterranean acclimation. Its closest homolog, *ANAC042/*  
426 *JUNGBRUNNEN1* (*AT2G43000*), was identified as a regulator of camalexin biosynthesis and positive  
427 regulator of resistance against the fungus *Alternaria brassicicola* (Saga et al., 2012), longevity (Wu et  
428 al., 2012) and tolerance to heat and drought (Ebrahimian-Motlagh et al., 2017; Shahnejat-Bushehri et  
429 al., 2012). In addition, exposure to 90min at 37°C enhanced survival of *JUB1* overexpressors to a  
430 subsequent treatment at 45°C, compared with WT and *jub1-1* knock-down seedlings (Shahnejat-  
431 Bushehri et al., 2012). Molecular changes induced in plants by heat and other environmental signals  
432 persist longer than the signals themselves and modifies subsequent responses, phenomenon referred  
433 to as somatic environment memory (SEM). In this work, pathogen inoculations were done in standard  
434 conditions, indicating that some form of SEM of previous growth conditions had influenced plant  
435 immunity. The molecular mechanisms by which SEM mediates the priming of plant-microbe  
436 interactions remain largely unknown. Our results implicated a switch in the transcriptional targets of  
437 *NAC42-L* in this process. The underlying molecular bases may include variation in trans, through  
438 changes to the composition, stoichiometry and post-transcriptional regulation of protein complexes  
439 including *NAC42-L*, or variation in cis affecting the conformation and accessibility of target gene  
440 promoter regions. Recent studies have identified chromatin state modifications as crucial components  
441 in the memory of repeated stress events in plants, particularly in response to heat, cold, and drought  
442 priming (Balazadeh, 2022; Crisp et al., 2016; Liu et al., 2022). Future investigations will aim at  
443 deciphering which molecular mechanisms mediate *NAC-L* target switch upon acclimation and what  
444 controls the duration and breadth of this switch.

445 Our results highlight rewiring of quantitative immunity gene networks as a key process in acclimation,  
446 with adverse consequence to disease resistance under warm Mediterranean-like climates. We show  
447 that acclimation can reverse the contribution of genes upregulated by pathogen inoculation to the  
448 disease resistance phenotype. We identified several mutations mitigating the negative impact of  
449 Mediterranean acclimation on disease resistance, opening perspectives for the preservation of plant  
450 immunity functions in a warming climate context.

451

## 452 MATERIALS AND METHODS

### 453 Plant material and growth conditions

454 *A. thaliana* Natural accessions and mutant lines were obtained from the Nottingham Arabidopsis Stock  
455 Center. We selected Col-0 (CS76778, 6909), Rld-2 (CS78349, 7457) and Shahdara (Sha, CS78397, 6962)  
456 as three natural accessions of *A. thaliana* originating from areas with contrasted climate conditions.  
457 Plants were grown in jiffy pots for 35 or 70 days in Percival E41-L3 and E41-L2PLT growth cabinets  
458 equipped with ultra-sonic humidifier, far-red LED clusters, closed-loop light dimming, Intellus and  
459 WeatherEZE controllers. We set day and night temperatures for each climate according to ERA5T  
460 models based on 30-year average of hourly weather simulations for daily maximum and minimum  
461 temperatures for the month of April at GPS coordinates 56.25°N, 34.19°E (Continental climate, origin  
462 of Rld-2 accession); 38.35°N, 68.48°E (Mediterranean climate, origin of Sha accession) and 38.30°N,  
463 92.30°O (Temperate climate) according to <https://www.meteoblue.com> consulted on April 2017 (**Fig**  
464 **S1**). Day temperatures were 11°C, 20°C and 23°C and night temperatures were 1°C, 9°C and 7°C for  
465 continental, temperate and Mediterranean climates respectively. Plants were grown in long day under  
466 190  $\mu\text{mol}/\text{m}^2/\text{s}$  light, with photoperiod variation between climates to represent photoperiod variability  
467 during April in the northern hemisphere, water was kept not limiting for the whole experiment at 80%  
468 relative humidity. These growth conditions were classified into Continental, Mediterranean and  
469 Temperate according to Köppen-Geiger classification (Cui et al., 2021) of climate at the corresponding  
470 GPS coordinates. Inoculations were performed on detached leaves at a constant 23°C under constant  
471 40  $\mu\text{mol}/\text{m}^2/\text{s}$  light and high humidity following the procedure described in (Barbacci et al., 2020).

### 472 Fungal strains and disease resistance phenotyping

473 0.5-cm-wide plugs of PDA agar medium containing *S. sclerotiorum* strain 1980, grown for 72 hours at  
474 20°C on 14 cm Petri dishes, were placed on the adaxial surface of detached leaves. These leaves were  
475 positioned in a Navautron system (Barbacci et al., 2020), and records were made using high-definition  
476 (HD) cameras “3MP M12 HD 2.8-12mm 1/2.5 IR 1:1.4 CCTV Lens” every 10 minutes. For each genotype,  
477 a minimum of 28 leaves were imaged from a minimum of two independent acclimation and inoculation  
478 experiments. Kinetics of *S. sclerotiorum* disease lesions were analyzed using INFEST script v1.0  
479 (<https://github.com/A02I01/INFEST>). Statistical analyses of disease phenotypes were conducted using  
480 the Tukey test or the Student t test followed by Benjamini-Hochberg correction for multiple testing in  
481 R 4.2.1. Disease susceptibility (**Fig. 1**) corresponded to the slope of disease lesion growth over time.  
482 Resistance (**Fig. 3**) corresponded to  $-\log_2$  of the slope of disease lesion growth over time.

### 483 RNA collection and sequencing

484 Total RNA was extracted from a 3mm-wide ring of leaf tissue at the edge of ~1.5cm wide disease lesions  
485 collected at 30 hours post inoculation (hpi) for temperate and mediterranean acclimation and 48 hpi  
486 for continental acclimation. The samples were harvested with a scalpel on a cool glass slide and  
487 immediately frozen in liquid nitrogen. Samples were ground with metal beads (2.5 mm) in a Retschmill  
488 apparatus (24hertz for 2x1min). RNA was extracted using the RNAlplus kit (Macherey Nagel) following  
489 the manufacturer’s instructions. A Turbo DNase treatment (Ambion) was applied to remove genomic  
490 DNA. The quality and concentrations of RNAs preparations were assessed with an Agilent Bioanalyzer  
491 using the Agilent RNA 6000 Nano kit. For the analysis of gene expression in natural accessions, libraries  
492 synthesis and sequencing was outsourced to Fasteris SA (Plan-les-Ouates, Switzerland). Libraries were  
493 sequenced as paired-end reads on an Illumina HiSeq 2500 instrument in High Output v4 mode with  
494 2x125+8 cycles on 7 lanes of HiSeq Flow Cells v4 with the HiSeq SBS Kit v4. Basecalling was performed  
495 with the HiSeq Control Software 2.2.58, RTA 1.18.64.0 and CASAVA-1.8.2. Reads QC was performed  
496 using spiked-PhiX in-lane controls yielding Q30 error rate <0.4% for all lanes. Paired-end reads were

497 trimmed and mapped to the TAIR10.0 reference genome using the RNA-seq analysis tool of the CLC  
498 Genomics Workbench 11.0.1 software (Qiagen). The following mapping parameters were used:  
499 mismatch cost 2, insertion cost 3, deletion cost 3, length fraction 0.8, similarity fraction 0.8, both  
500 strands mapping, and 10 hits maximum per read, with expression value given as total read count per  
501 gene.

## 502 **Differential expression and expression variance analyses**

503 Differential gene expression analysis was performed with the DESeq2 Bioconductor package version  
504 1.8.2 (Love et al., 2014) in R 3.4.0 in a pairwise manner using expression in uninfected plants as a  
505 reference with ~replicates + inoculation as the design formula. Genes with baseMean 0 in all  
506 differential comparisons and non-nuclear genes were discarded from further analyses. Genes with  
507  $|\text{Log}_2 \text{ Fold Change}| \geq 2$  and Bonferroni-adjusted  $p\text{-val} < 0.001$  in DESeq2 Wald test were considered  
508 significant for differential expression. For ANOVA, read counts were mean-normalized to homogenize  
509 the total number of mapped reads per sample. The ANOVA was performed on each gene using the  
510 dplyr package in R with  $\text{ReadCount} \sim \text{Genotype} * \text{Infection} * \text{Climate}$  as the model formula. P-values  
511 associated with each factor were corrected for multiple testing using the Benjamini-Hochberg  
512 procedure.

## 513 **Gene network reconstruction and analyses**

514 For gene network reconstruction, we focused on the 7,279 genes whose expression was pathogen  
515 inoculation-dependent both in the differential analysis ( $|\text{Log}_2 \text{ Fold Change}| \geq 2$  and Bonferroni-  
516 adjusted  $p\text{-val} < 0.001$ ) and in the ANOVA analysis (Benjamini-Hochberg corrected  $p\text{-val} < 1E-7$ ). Pairwise  
517 Spearman rank correlation was calculated for the expression of these genes using the rcorr function  
518 from the R package Hmisc, using the raw counts per gene from all 54 RNA-seq samples. The top 25  
519 correlated genes (Spearman  $\rho > 0.85$ ) was extracted for each gene. The top 25 expression correlations  
520 were used as edges for network reconstruction with weight  $\rho$ . Reconstruction of the hierarchical gene  
521 cluster network was performed using the Community Detection 1.12.0 plugin in Cytoscape 3.10.0,  
522 using the HiDeF algorithm with maximum resolution 45.0, consensus threshold 65, persistent  
523 threshold 6 and the Louvain algorithm. Correlation with susceptibility was the Spearman rank  
524 correlation coefficient between normalized read counts (averaged over three replicates) for each gene  
525 and the slope of disease lesion growth. Average LFC was the mean  $\log_2$  fold change over all nine  
526 genotype-acclimation modalities tested. Values for gene clusters are the mean of values for all genes  
527 in a cluster. Gene ontology enrichment were analyzed with the BinGO plugin in Cytoscape 3.10.0 using  
528 a hypergeometric test with Benjamini and Hochberg false discovery rate correction, at significance  
529 level 0.05 with *A. thaliana* whole annotation as a reference set.

## 530 **Characterization of *A. thaliana* mutant lines**

531 T-DNA insertion lines in AT1G76600 (SALK\_052389), AT1G34190 (SALK\_044777), AT1G07135  
532 (SALK\_133656), AT2G43790 (mpk6-1), AT5G24600 (SALK\_201248C), AT1G12290 (SALK\_125493),  
533 AT5G60600 (SALK\_059118), AT5G23160 (SALK\_041095C), AT5G64990 (SALK\_088173), AT5G06230  
534 (SALK\_008492C), AT3G12910 (SALK\_016619C and SALK\_078841) and AT5G37840 (SALK\_002404) in  
535 the Col-0 background were obtained from the Nottingham Arabidopsis Stock Centre. To identify  
536 homozygous insertion lines, the lines were genotyped by PCR and, if needed, self-crossed and the  
537 progeny genotyped by PCR. The disease resistance phenotype of mutant lines was analyzed as  
538 previously described in a total of 23 independent inoculation experiments each including the Col-0  
539 reference, with a minimum of 2 independent experiments for each mutant line. Primers used in all  
540 experiments are shown in **Table S12**.



## 541 **Identification of NAC42-L predicted target genes**

542 The HMM model NAC\_tnt.AT3G12910\_col\_a\_m1 for the unique DAP-seq motif bound by AT3G12910  
543 was obtained from the Plant Cistrome Database ([http://neomorph.salk.edu/dap\\_web/](http://neomorph.salk.edu/dap_web/)) (O'Malley et  
544 al., 2016). *A.thaliana* genes harboring the corresponding motif were identified using FIMO v.5.5.5  
545 (Grant et al., 2011) using the sequence 1Kbp upstream of the translation start site from the Araport11  
546 annotation with 1E-4 as p-value threshold, both strands scanning and NRDB frequencies as a  
547 background model

## 548 **Quantitative RT-PCR Analyses**

549 RNA for qRT-PCR analysis was extracted from plants 24 hours post-inoculation with *S. sclerotiorum* as  
550 for RNA-sequencing. For cDNA synthesis, 1 µg of RNA and 0.5 µL of Transcriptor reverse transcriptase  
551 (Roche) were used in a 20 µL reaction volume according to the manufacturer's protocol. The resulting  
552 cDNA diluted 1:10 served as the template for quantitative RT-PCR. qRT-PCR reactions were carried out  
553 with 5 pmol of specific oligonucleotides (**Table S12**), 2 µL of cDNA, and 3.5 µL of SYBR GREEN I in a total  
554 volume of 7 µL. Amplification reactions were performed using a LightCycler 480 (Roche Diagnostics)  
555 with the following protocol: 9 minutes at 95°C, followed by 45 cycles of 5 seconds at 95°C, 10 seconds  
556 at 65°C, and 20 seconds at 72°C. Relative gene expression was calculated as the ratio of target gene  
557 expression to the reference gene *AT2G28390* and expressed as the difference between target and  
558 reference crossing times ( $\Delta Ct$ ).

559

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568

## 569 **DATA AVAILABILITY**

570 Raw RNA-seq reads data and processed gene expression files generated in this work are available  
571 under NCBI GEO accession number GSE272240.

572

## 573 **AUTHOR CONTRIBUTIONS**

574 A.B. and S.R. designed research; Natural accessions phenotyping: M.D., J.S.; RNA-seq sampling: J.S.;  
575 RNA-seq data analysis: J.S., M.D., S.R.; Analysis of mutant lines genotype and phenotype: M.D; NAC42-  
576 L targets identification: M.D; Sampling for qRT-PCR: M.D.; Performed qRT-PCR: M.D., P.C-S, M.Z;  
577 Analyzed qRT-PCR results: M.D, P.C-S, SR; Supervised and coordinated research: A.B, S.R; Wrote the  
578 paper: M.D and S.R.

579

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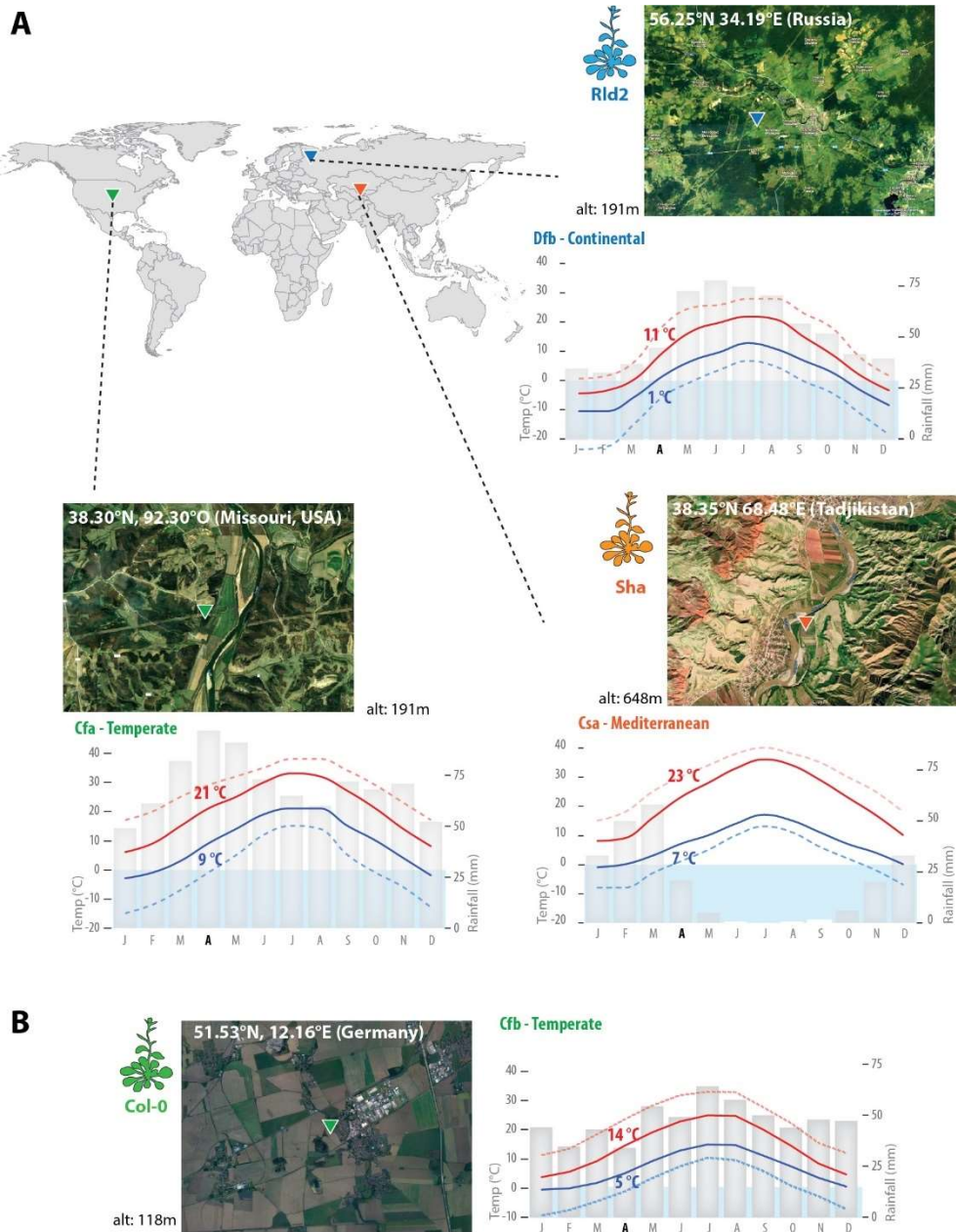
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818 SUPPLEMENTARY MATERIAL

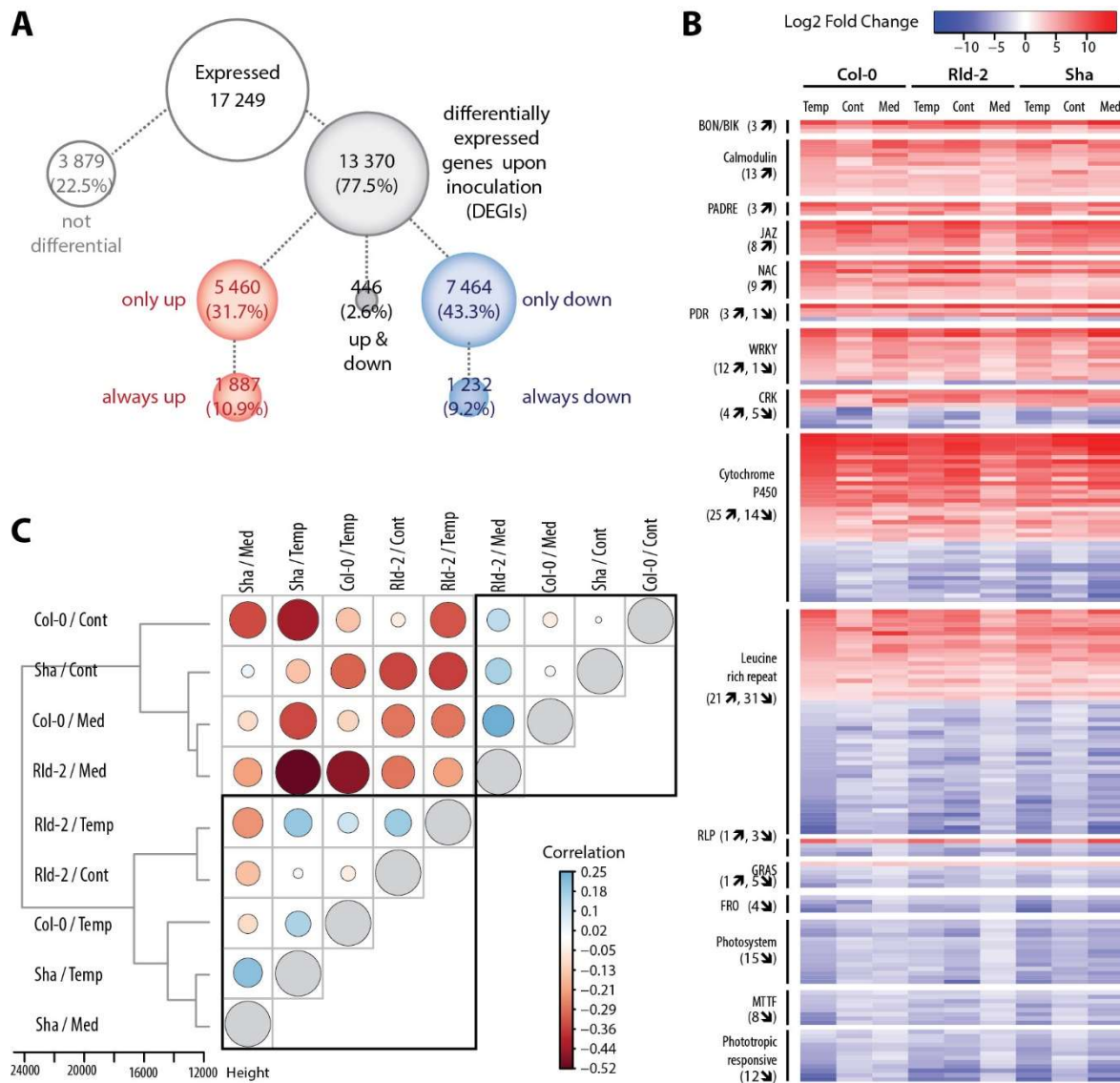


819

820 **Supplementary Figure S1. Climate data at sites in the distribution range of *A. thaliana* and *S.***  
 821 ***sclerotiorum* used for simulated climates in our experiments (A) and at the site of Col-0 accession**  
 822 **origin (B). Data come from an ERA5T model of 30-year average of hourly simulations collected from**  
 823 **meteoblue.com. Satellite views of the designated coordinates were obtained from Google Maps. Grey**  
 824 **bars show monthly precipitations in mm, red lines are mean daily maximum (plain) and hot days**  
 825 **maximum (dotted), blue lines are mean daily minimum (plain) and cold nights minimum (dotted).**  
 826 **Values for April are labelled. The corresponding Köppen-Geiger climate was obtained from climate-**  
 827 **data.org and coded as follows: Csa, hot summer Mediterranean climate; Cfa, humid subtropical**  
 828 **climate; Cfb, Temperate oceanic climate or subtropical highland climate; Dfa, Hot-summer humid**  
 829 **continental climate. Alt., altitude; Temp., Temperature.**

830





831

832 **Supplementary Figure S2. Arabidopsis genes differentially expressed upon *S. sclerotiorum***

833 **inoculation analyzed with relaxed thresholds.** Analysis of genes differentially expressed upon

834 inoculation at  $|\text{Log}_2 \text{ Fold Change}| \geq 1.5$ , adjusted  $p\text{-val} < 0.1$ , using non-inoculated plants as reference

835 in each of nine conditions (three climate priming, times three plant genotypes). **(A)** Identification of

836 13 370 differentially expressed genes (DEGs) upon inoculation. In conditions where they are

837 differential, 5 460 DEGs were upregulated only, 7 464 were down-regulated only, and 446 were either

838 up or down-regulated. We detected 1 887 DEGs upregulated in all nine conditions and 1 232 DEGs

839 down-regulated in all nine conditions, representing 10.9% and 9.2% of the expressed genes

840 respectively. These 3 119 genes are differentially expressed in a consistent manner regardless of plant

841 genotype and acclimation, they can therefore be regarded as a core transcriptome responsive to *S.*

842 *sclerotiorum* in *A. thaliana*. **(B)** Heatmap of Log2 fold change for 200 genes forming major functional

843 groups including DEGs always up and always down (numbers indicated between brackets). Major

844 functional groups in the core transcriptome included the PRR-associated *BOTRYTIS-INDUCED KINASE*

845 *1 (BIK1)*, *BONZAI1-ASSOCIATED PROTEIN (BAP) 1* and 2, members of the Calmodulin (CaM) and CaM-

846 binding, the pathogen and abiotic stress response, cadmium tolerance, disordered region-containing

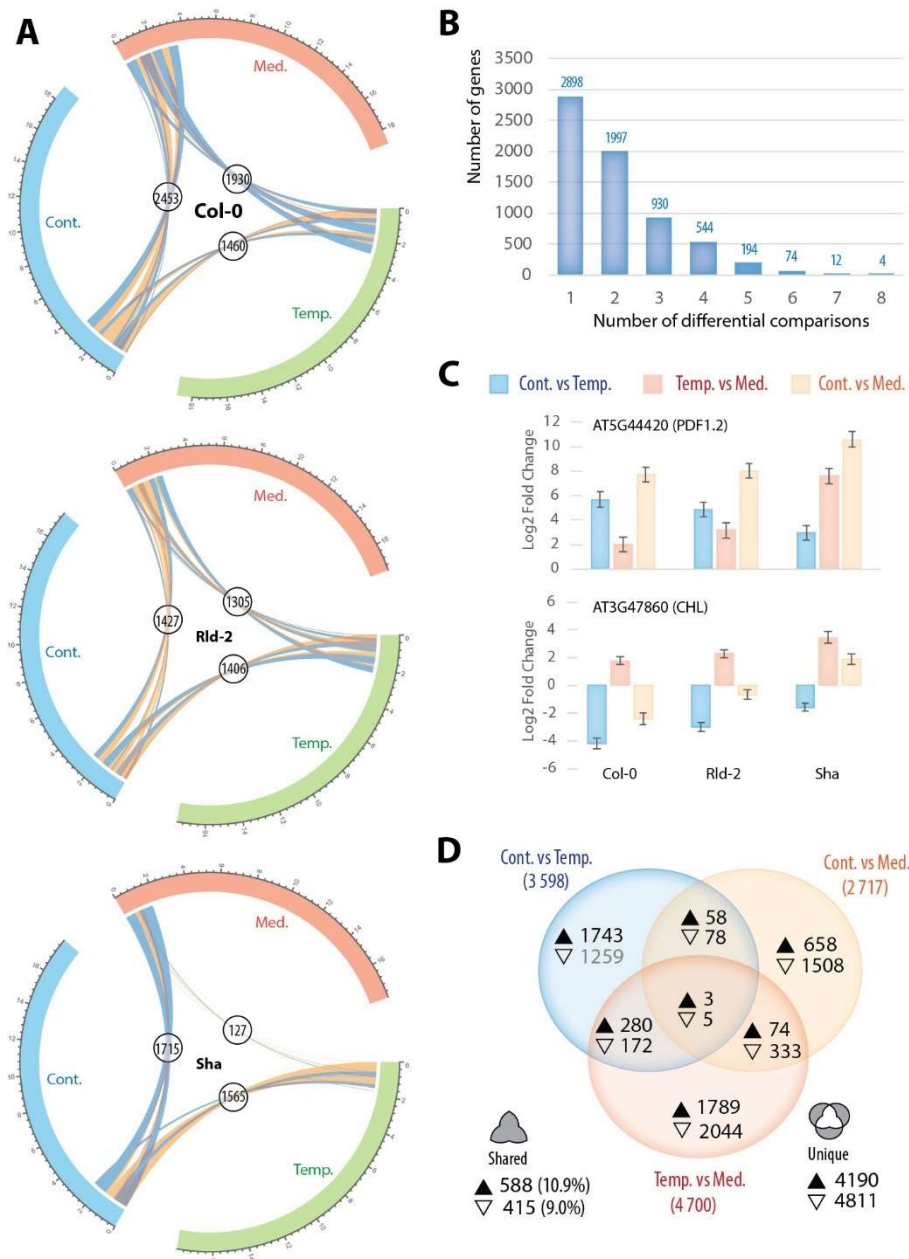
847 (PADRE), the jasmonate-zim-domain proteins (JAZ), and the NAC-domain transcription factor families,

848 with all core DEGs being upregulated by *S. sclerotiorum* inoculation. Ferric reduction oxidases (FRO),

849 Mitochondrial transcription termination factors (MTTF), Photosystems I and II, phototropin and

850 phototropic-responsive NPH3 genes showed all core DEGs downregulated. The pleiotropic drug  
851 resistance (PDR), cysteine-rich receptor-like protein kinase (CRK), WRKY and GRAS transcription factor,  
852 cytochrome P450, Leucine-rich repeat (LRR), receptor like protein (RLP) families included several core  
853 genes responsive to *S. sclerotiorum* either consistently up- or down-regulated. **(C)** Distance tree and  
854 correlation matrix showing the similarity in the 13 370 DEGs regulation across conditions. Tree based  
855 on Manhattan distance between samples and Ward clustering, correlation values shown by bubbles  
856 are Spearman rank correlations calculated using LFC for 13 370 DEGs in each condition. Transcriptomes  
857 measured after priming under temperate acclimation clustered together and with the transcriptome  
858 of Rld-2 primed under continental acclimation and that of Sha under Mediterranean acclimation. The  
859 remaining four conditions formed a second cluster. There was no clear clustering based on genotype  
860 or climates alone, suggesting a significant interaction between these factors. Cont, continental; Temp,  
861 temperate; Med, Mediterranean.

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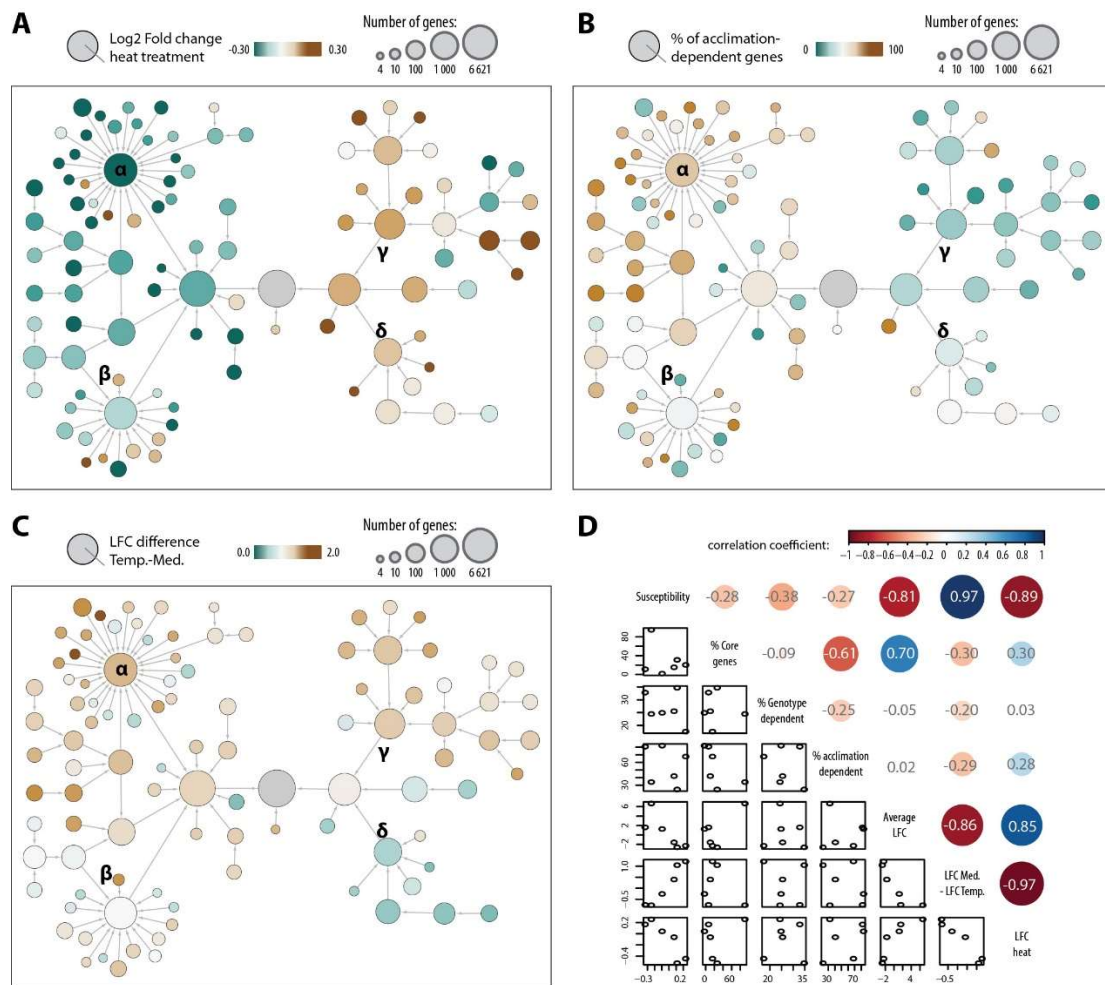


863

864 **Supplementary Figure S3. A. thaliana genes differentially expressed in pairwise acclimation**  
 865 **comparisons. (A)** Differentially expressed genes in pairwise comparisons between acclimation regimes  
 866 for *S. sclerotiorum*-inoculated samples ( $|LFC| \geq 1.5$ , adjusted  $p\text{-val} < 0.1$ ). The circos tracks show number  
 867 of expressed genes (x 1,000) with connectors representing genes differentially expressed between two  
 868 acclimation regimes (down-regulated in blue, upregulated in yellow). Labels show the sum of up- and  
 869 down-regulated genes for each acclimation comparison. The number of DEGs ranged from 127 in Sha  
 870 when comparing temperate and Mediterranean acclimation to 2,453 in Col-0 when comparing  
 871 continental and Mediterranean acclimation, representing 0.71% to 13% of expressed genes. **(B)**  
 872 Overall, 6,653 genes were differential in at least one comparison (35.2% of expressed genes). Of those,  
 873 4,895 (73.6%) were differentially expressed in one or two acclimation comparisons only, consistent  
 874 with a specific effect of acclimation on *A. thaliana* transcriptome, and supporting a significant  
 875 interaction between genotype and acclimation. **(C)** The genes most sensitive to acclimation were four  
 876 genes differentially regulated in eight out of nine comparisons. These genes encoded plant defensins  
 877 PDF1.3 (AT2G26010) and PDF1.2 (AT5G44420), the MYB transcription factor LHY (LATE ELONGATED  
 878 HYPOCOTYL, AT1G01060) and the chloroplastic lipocalin gene CHL (AT3G47860). Histograms show

879 Log<sub>2</sub> Fold change of expression for AT5G44420 and AT3G47860 in 3 acclimation comparisons for 3  
880 genotypes. Error bars show estimated standard errors for the estimated coefficients on the log<sub>2</sub> scale  
881 from DESeq2. **(D)** Venn diagram showing the distribution of DEGs for all three accessions according to  
882 the acclimation regimes compared. The full upward triangle indicates up-regulated genes, the empty  
883 downward triangle indicates down-regulated genes. For each acclimation comparison the total  
884 number of DEGs is indicated between parenthesis. The percentage of shared genes is given relative to  
885 the total number of DEGs in acclimation comparisons. Cont., continental acclimation; Med.,  
886 Mediterranean acclimation; Temp., temperate acclimation.

887

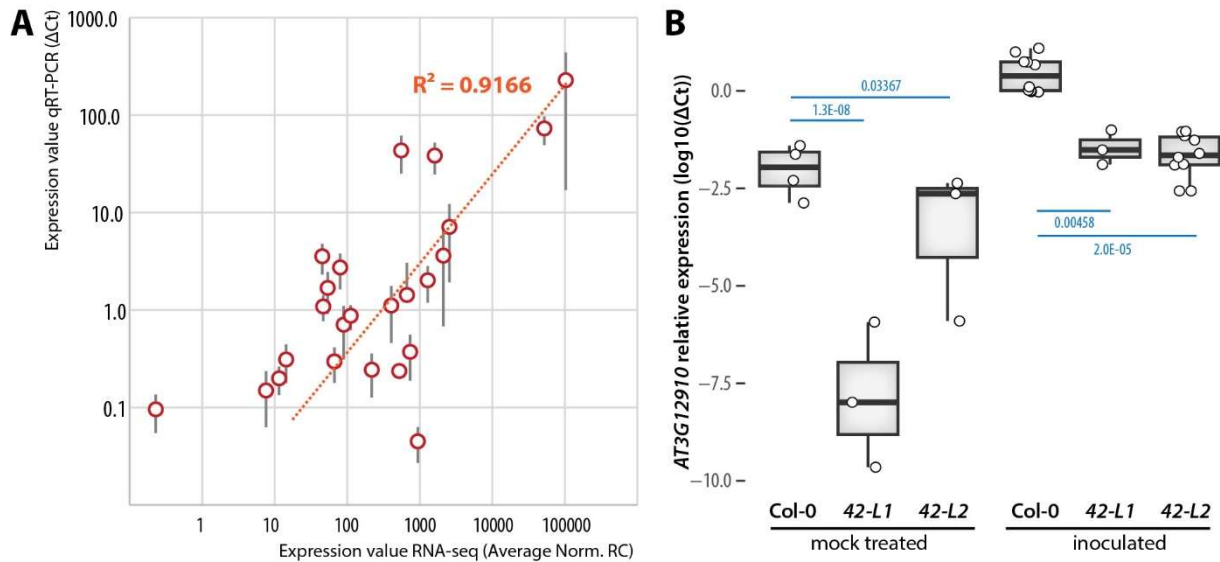


888

889 **Supplementary Figure S4. Properties of gene communities in a network of DEGs upon *S. sclerotiorum***  
 890 **inoculation. (A)** We mapped the average gene LFC upon heat treatment reported in a recent meta-  
 891 analysis (Guo *et al.* 2021). Genes from communities  $\gamma$  and  $\delta$  showed a trend for up-regulation upon  
 892 heat treatment (average LFC 0.26 and 0.17 respectively) while genes from communities  $\alpha$  and  $\beta$  were  
 893 rather down-regulated (average LFC -0.52 and -0.45 respectively). **(B)** We mapped the percentage of  
 894 gene from each community considered acclimation dependent based on our ANOVA analysis.  
 895 Communities  $\gamma$  and  $\delta$  had a low proportion of acclimation-dependent genes while community  $\alpha$  had a  
 896 majority of acclimation-dependent genes. **(C)** Considering the clear phenotypic effect of  
 897 Mediterranean acclimation, which had the highest day temperature and highest daily thermal  
 898 amplitude, we mapped the average LFC variation between plants acclimated under temperate and  
 899 Mediterranean climates. Average LFC variation was  $>1.0$  for communities  $\alpha$  and  $\gamma$  but  $<-0.7$  for  
 900 community  $\delta$ . **(D)** To summarize these analyses, we calculated the correlation between properties of  
 901 the six largest gene communities. We observed a clear correlation between association with  
 902 susceptibility phenotype and LFC variation upon temperate and Mediterranean acclimation (0.97), and  
 903 anti-correlation with average LFC upon *S. sclerotiorum* inoculation (-0.81 and -0.86). This suggested  
 904 that in our experiments, differential gene expression mostly associated with a decrease in plant  
 905 susceptibility which is strongly altered upon Mediterranean acclimation.

906 Reference: Guo, M., Liu, X., Wang, J., Jiang, Y., Yu, J., & Gao, J. (2021). Transcriptome profiling  
 907 revealed heat stress-responsive genes in *Arabidopsis* through integrated bioinformatics analysis.  
 908 *Journal of Plant Interactions*, 17(1), 85–95. <https://doi.org/10.1080/17429145.2021.2014580>

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910

911 **Supplementary Figure S5. Analysis of gene expression by quantitative RT-PCR in wild type and**  
912 **NAC42-L mutant lines. (A)** Relationship between gene expression in Col-0 determined by RNA-  
913 sequencing (X axis) and quantitative RT-PCR (Y axis). Error bars show standard error of the mean from  
914 3 to 11 independent replicates. Dots represent expression of AT1G10040, AT1G56130, AT3G09010,  
915 AT3G12910, AT3G19210, AT3G19615, AT3G26200, AT4G39950, AT5G65510 in mock-treated and *S.*  
916 *sclerotiorum* inoculated plants grown under temperate and Mediterranean acclimation. (B) Relative  
917 expression of NAC42-L (AT3G12910) in Col-0, *nac42-L1* and *nac42-L2* mutant lines determined by  
918 quantitative RT-PCR. Boxplots show expression independent measurements for 3-9 plants (dots) with  
919 first and third quartiles (box), median (thick line), and the most dispersed values within 1.5 times the  
920 interquartile range (whiskers). P-values were determined by a Student t test with Benjamini-Hochberg  
921 correction for multiple testing.

922 **Supplementary tables online**

Table	Related to	Description
S1	Fig 1B	Raw data for <i>A. thaliana</i> susceptibility phenotype in response to <i>S. sclerotiorum</i> infection as a function of acclimation and genotype
S2	Fig 2A	Normalized read counts for the 54 RNA-seq samples and identification of expressed and differentially expressed genes ('1' = YES; '0'=no)
S3	Fig 2A, 2B	Log2 fold change and p-values for genes differentially expressed (inoculated samples versus mock treated samples as a reference)
S4	Fig 2C	Analysis of variance to determine the contribution of genotype (Geno), inoculation (Inoc) and acclimation (Clim) to expression variance
S5	Fig S3	Genes differentially expressed in inoculated samples based on pairwise acclimation comparisons
S6	Fig 2D, S4	Content and representative properties of gene communities in the co-expression network of <i>A. thaliana</i> genes differentially expressed upon <i>S. sclerotiorum</i> inoculation
S7	Fig 3A	Gene ontologies enriched in major gene communities in the co-expression network of <i>A. thaliana</i> genes differentially expressed upon <i>S. sclerotiorum</i> inoculation
S8	Fig 3B, 3C	Raw data for <i>A. thaliana</i> mutant lines susceptibility phenotype in response to <i>S. sclerotiorum</i> inoculation
S9	Fig 3B, 3C	Summary statistics for disease susceptibility of natural accessions and mutant lines inoculated by <i>S. sclerotiorum</i> following temperate and Mediterranean acclimation
S10	Fig 4B	List of target sequences and genes harboring the DAP-seq motif bound by AT3G12910 identified by FIMO
S11	Fig 4D	Raw quantitative RT-PCR data for NAC42-L target genes in wild type and NAC42-L1 (SALK_016619C) and L2 (SALK_078841) mutant lines
S12	Fig3, 4	List of oligonucleotide primers used in this work

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924

925 **Supplementary Data online**

926 **Data S1.** Cytoscape session file containing the network of *A. thaliana* genes differentially expressed  
 927 upon *S. sclerotiorum* inoculation, associated metadata and hierarchical clustering network.