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# Unraveling the genetic architecture of the adaptive potential of *Arabidopsis thaliana* to face the bacterial pathogen *Pseudomonas syringae* in the context of global change

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1 **Unraveling the genetic architecture of the adaptive potential of *Arabidopsis***  
2 ***thaliana* to face the bacterial pathogen *Pseudomonas syringae* in the context**  
3 **of global change**

4  
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26 **ABSTRACT**

27 Phytopathogens are a continuous threat for global food production and security. Emergence or  
28 re-emergence of plant pathogens is highly dependent on the environmental conditions affecting  
29 pathogen spread and survival. Under climate change, a geographic expansion of pathogen  
30 distribution poleward has been observed, potentially resulting in disease outbreaks on crops and  
31 wild plants. Therefore, estimating the adaptive potential of plants to novel epidemics and  
32 describing its underlying genetic architecture, is a primary need to propose agricultural  
33 management strategies reducing pathogen outbreaks and to breed novel plant cultivars adapted  
34 to pathogens that might spread in novel habitats under climate change. To address this  
35 challenge, we inoculated *Pseudomonas syringae* strains isolated from *Arabidopsis thaliana*  
36 populations located in south-west of France on the highly genetically polymorphic TOU-A *A.*  
37 *thaliana* population located east-central France. While no adaptive potential was identified in  
38 response to most *P. syringae* strains, the TOU-A population displays a variable disease response  
39 to the *P. syringae* strain JACO-CL belonging to the phylogroup 7 (PG7). This strain carried a  
40 reduced T3SS characteristic of the PG7 as well as flexible genomic traits and potential novel  
41 effectors. GWA mapping on 192 TOU-A accessions inoculated with JACO-CL revealed a  
42 polygenic architecture. The main QTL region encompasses two *R* genes and the *AT5G18310*  
43 gene encoding for ubiquitin hydrolase, a target of the AvrRpt2 *P. syringae* effector. Altogether,  
44 our results pave the way for a better understanding of the genetic and molecular basis of the  
45 adaptive potential in an ecologically relevant *A. thaliana* – *P. syringae* pathosystem.

## 46 INTRODUCTION

47 Pathogens are a threat both for crops and for wild species. Yield losses resulting from  
48 pathogen attacks can be up to several tens of percent in crops (Oerke, 2006; McDonald and  
49 Stukenbrock, 2016; Savary et al., 2019), thereby threatening global food security (Ristaino et  
50 al., 2021; Savary et al., 2019). Experimental evidences indicate that naturally infected wild  
51 species plants can harbor a significant decrease in the number of offspring (Jarosz and Davelos,  
52 1995; Traw et al., 2007; Frachon & Roux 2022). A major challenge in plant breeding and  
53 ecological genomics is to identify the genetic and molecular bases of natural variation in disease  
54 resistance (Bartoli and Roux, 2017; Roux and Bergelson, 2016). The characterization of the  
55 genetic architecture of disease resistance might have enormous practical implications by  
56 increasing crop yield and quality (Deng et al., 2020; Karasov et al., 2020; Li et al., 2020), and  
57 lead to fundamental insights in the prediction of evolutionary trajectories of natural populations  
58 (Karasov et al., 2014; Roux et al., 2014). This is particularly relevant in the current climate  
59 change that is likely to favor conditions for pathogen spread and their adaptation potential in  
60 comparison with sessile plants (Bebber et al., 2014; Bergot et al., 2004; Garrett et al., 2006;  
61 Pautasso et al., 2012; Tylianakis et al., 2008). For instance, temperature elevation is expected  
62 to favor the emergence of new pathogens and to increase the severity and occurrence of  
63 epidemics (Bebber, 2015; Desaint et al., 2021; Elad and Pertot, 2014; McDonald and  
64 Stukenbrock, 2016). In addition, climate change scenarios predict an increase in epidemic  
65 severity and a northwards geographic expansion of pathogen distribution in Western Europe  
66 (Bergot et al., 2004; Evans et al., 2008; Prank et al., 2019). Accordingly, a global warming-  
67 driven movement poleward has already been observed for a large number of pathogens (Bebber  
68 et al., 2013).

69 *Pseudomonas syringe* is a ubiquitous phytopathogenic bacterium belonging to a species  
70 complex including strains with a large host range and adaptability to a wide spectrum of

71 environment outside of crop fields. Non-agricultural strains of *P. syringae* have been isolated  
72 from wild plant species (including *Arabidopsis*), snow, rain, rivers, litter etc. (Monteil et al.,  
73 2014b; Morris et al., 2013), corroborating the great adaptability of strains of the *P. syringae*  
74 complex to colonize a large range of environments. Additionally, air masses and precipitations  
75 are a way for *P. syringae* translocation over large geographic distances (Monteil et al., 2014a).  
76 *P. syringae* strains growing in icy and hostile conditions similar to cloud environment, were  
77 also shown to acquire exogenous DNA faster than other bacterial species. This suggests that *P.*  
78 *syringae* strains are able to improve their genomic content and thus their adaptability while  
79 moving in the air masses (Blanchard et al., 2017). Under these circumstances, it is not surprising  
80 that *P. syringae* outbreaks originate from hosts and habitats different than the final host where  
81 the disease is reported (Koike et al., 2017). The wide host range coupled with the great  
82 adaptability of *P. syringae* and its ability to colonize an incredibly diverse range of habitats,  
83 question the impact that the climatic change can play on the dispersal of this phytopathogen.  
84 Therefore, understanding the adaptability of *P. syringae* strains isolated from the south to  
85 colonize and infect plant populations adapted to colder conditions is a pertinent question to  
86 answer to better understand and control disease outbreaks.

87 In this study, we aimed at estimating the adaptive potential, and describing the related  
88 underlying genetic architecture, of the annual wild species *Arabidopsis thaliana* to face attacks  
89 from more southern strains of the *P. syringae* complex. Besides being a model in plant genetics  
90 and molecular biology, *A. thaliana* is above all a wild species inhabiting contrasting  
91 environments for diverse abiotic (e.g. climate, soil physico-chemical properties) and biotic (e.g.  
92 microbial communities, plant communities) factors (Bartoli et al., 2018; Brachi et al., 2013;  
93 Frachon et al., 2018; Frachon et al., 2019; Thiergart et al., 2020). In addition, disease incidence  
94 has been commonly observed in natural populations of *A. thaliana* (Barrett et al., 2009; Roux  
95 and Bergelson, 2016). For instance, a survey of 163 natural populations in south-west of France

96 reported that 72.7% of plants presented disease symptoms, with each of the two most abundant  
97 bacterial pathogens (namely the *Pseudomonas syringae* complex and *Xanthomonas campestris*)  
98 being detected in more than 90% of the populations by a metabarcoding approach (Bartoli et  
99 al., 2018).

100 More precisely, we aimed at testing the adaptive genetic potential of the TOU-A local  
101 mapping population located in Burgundy (east of France) to face the individual attack of eight  
102 *P. syringae* strains isolated from natural populations of *A. thaliana* located ~400km south-west  
103 of the TOU-A population. We chose to use this local mapping population for its several  
104 advantages. First, located under a ~350 meter long electric fence, the TOU-A population is  
105 highly polymorphic (Frachon et al., 2017). Based on the whole-genome sequences of 195 TOU-  
106 A accessions, more than 1.9 million single nucleotide polymorphisms (SNPs) were detected,  
107 only 5.6 times less than observed in a panel of 1,135 worldwide accessions (Frachon et al.,  
108 2017). Secondly, extensive genetic variation for resistance to diverse pathogens was detected  
109 when conducting experiments in controlled laboratory conditions (Debieu et al., 2016; Frachon  
110 et al., 2017; Huard-Chauveau et al., 2013) or directly in the native habitat of the TOU-A  
111 population (Roux & Frachon 2022). Thirdly, a short linkage disequilibrium (LD) decay, below  
112 3 kb, combined with a strongly reduced confounding effect of population structure allows fine-  
113 mapping of genomic regions associated with phenotypic variation (Baron et al., 2015; Brachi  
114 et al., 2013; Frachon et al., 2017; Libourel et al., 2021), which in turn leads to the cloning and  
115 functional validation of QTLs of disease resistance (Aoun et al., 2020) .

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## 120 **RESULTS & DISCUSSION**

### 121 **The adaptive genetic potential of the TOU-A population to face attacks from more** 122 **southern *P. syringae* strains is limited**

123 To estimate the adaptive genetic potential of the TOU-A population to face attacks from more  
124 southern *P. syringae* strains, we first challenged seven accessions covering the genomic space  
125 of the TOU-A population (Frachon et al., 2017) with seven *P. syringae* strains isolated from  
126 contrasting habitats from south-west of France, i.e. 0105-Psy-JACO-CL, 0106-Psy-RADE-AL,  
127 0111-Psy-RAYR-BL, 0114-Psy-NAUV-BL, 0117-Psy-NAZA-AL, 0124-Psy-SAUB-AL and  
128 0132-Psy-BAZI-AL hereafter named BAZI-AL, JACO-CL, NAZA-AL, NAUV-BL, RADE-  
129 AL, RAYR-BL and SAUB-AL (Figure 1a). In our phenotypic assay, we also included the  
130 reference *A. thaliana* accession Col-0.

131 By aligning a fragment of the *citrate synthase* (*cts*) housekeeping gene of the seven strains with  
132 reference sequences (Berge et al., 2014), a phylogenetic inference shows that four strains  
133 (JACO-CL, NAUV-BL, RADE-AL, and SAUB-AL) belong to the *P. syringae* Phylogroup  
134 (PG) 7 *clade a* (Figure 1b) and are closely related to the TA043 strain isolated from *Primula*  
135 *officinalis* (Morris et al., 2010). In the past, PG7 was known under the name of *P. viridiflava*  
136 (Bartoli et al., 2014; Parkinson et al., 2011), a phytopathogen with pectolytic activity and  
137 described to cause disease on a wide-range of crops (Conn, 1993; González et al., 2003;  
138 Goumans and Chatzaki, 1998; Morris, 1992). Re-classification of *P. viridiflava* demonstrated  
139 that this phytopathogen is defined by two *P. syringae* PGs (7 and 8), PG7 *clade a* being the  
140 most abundant in both agricultural and non-agricultural habitats (Lipps et al., 2022; Lipps and  
141 Samac, 2022). Several herbaceous plants, including *A. thaliana*, have been described as natural  
142 hosts of PG7. Accordingly, PG7 *clade a* is one of the most abundant bacterial OTU inhabiting  
143 *A. thaliana* leaves (Bartoli et al., 2018; Goss, 2004; Jackson et al., 1999). The three other strains  
144 belong to PGs 2, 4 and 13 of *P. syringae sensu stricto* (BAZI-AL, NAZA-AL and RAYR-BL)

145 (Figure 1b). We also included as a control the strain SIMO-AL, a *Pseudomonas* strain that is  
146 phylogenetically distant from the *P. syringae* complex.

147 When tested on eight local accessions of *A. thaliana* from south-west of France, very few or no  
148 symptoms were observed in response to BAZI-AL, JACO-CL, NAZA-AL and SIMO-AL,  
149 whereas strong symptoms were observed in response to the RAYR-BL strain (Bartoli et al.,  
150 2018). Milder symptoms were observed in response to the three *P. viridiflava* strains NAUV-  
151 BL, RADE-AL and SAUB-BL (Bartoli et al., 2018).

152 Similar results were observed with the seven TOU-A accessions (with or without considering  
153 Col-0) 1, 2, 3 and 4 days after inoculation (dai), with the exception of the *P. viridiflava* JACO-  
154 CL strain that was more aggressive on the TOU-A accessions than on the accessions from  
155 south-west of France (Figure 2, Table S1, Supplementary Data Set 1). The absence of  
156 aggressiveness of the BAZI-AL strain is congruent with the absence of a canonical and  
157 complete T3SS in PG2 *clade c* strains (Dillon et al., 2019). In fact, *P. syringae clade 2c* is  
158 dominated by non-pathogenic *P. syringae* strains with an atypical T3SS similarly organized to  
159 the T3SS of S-PAI (Single-Pathogenicity Island) PG7 strains (Berge et al., 2014). This indicates  
160 that PG *clade 2c* strains probably tent toward low virulence and epiphytic interactions (Hirano  
161 and Upper, 2000). Likewise, the absence of symptoms observed when inoculating the NAZA-  
162 AL belonging to the PG13 is coherent with this phylogroup including mostly non-pathogenic  
163 strains from non-plant substrates with an atypical and reduced T3SS more related to the S-PAI  
164 found in the PG7 strains (Berge et al., 2014).

165 On the other hand, the high virulence of the RAYR-B strain on diverse sets of *A. thaliana*  
166 accessions is likely mediated by the T3E HopBJ1, previously identified and functionally  
167 characterized in this strain as inducing ROS accumulation and cell death both in *Arabidopsis*  
168 and in tobacco (Fuenzalida-Valdivia et al., 2022; Zavala et al., 2022).



169 Importantly, no genetic variation was detected between the seven TOU-A accessions in  
170 response to the three *P. viridiflava* strains NAUV-BL, RADE-AL and SAUB-BL, which can  
171 induce severe symptoms (Figure 2, Table S2). This result suggests a limited genetic potential  
172 of the TOU-A population to face attacks from a non-negligible fraction of more southern strains  
173 of the *P. syringae* complex. However, we caution that the restricted number of TOU-A  
174 accessions used in this experiment may not reflect the entire range of genetic variation present  
175 within the TOU-A population.

176 On the other hand, significant genetic variation was detected between the TOU-A accessions in  
177 response to the strains JACO-CL and RAYR-BL (Figure 2, Table S2). This is in line with some  
178 studies (mainly in trees) reporting genetic variation in host plant resistance to emerging  
179 pathogens (Ahrens et al., 2019; Alcaide et al., 2020; Elvira-Recuenco et al., 2014; Kjær et al.,  
180 2012). For instance, substantial genetic variation of the common tree *Corymbia calophylla* for  
181 resistance to the leaf blight pathogen *Quambalaria pitereka* was found in Australia (Ahrens et  
182 al., 2019). To investigate the genetic architecture underlying the adaptive potential of the TOU-  
183 A population in response to more southern strains of the *P. syringae* complex, we focused on  
184 the JACO-CL strain that induces both severe symptoms on TOU-A plants in comparison to  
185 accessions from its native area and variable disease responses among TOU-A accessions  
186 (Figure 2, Table S2). As a first step, we generated a *de novo* genome sequence of the JACO-  
187 CL strain.

188

189 **The JACO-CL strain belongs to the *Pseudomonas syringae* Phylogroup 7 clade a and**  
190 **displays flexible genomic traits**

191 The genome of the JACO-CL strain was sequenced by coupling the Oxford Nanopore  
192 Technology (ONT) and the Illumina technology. The assembly is composed by two circularized

193 sequences (one chromosome and one plasmid), resulting in 6,043,462 bp for the chromosome  
194 and 42,253 bp for the plasmid. The genome annotation resulted in 5,352 CDS, 16 rRNA, 70  
195 tRNA and 2 tmRNA. Genome annotation and genomic analysis suggest genome plasticity and  
196 flexibility of the JACO-CL strain. First, we found two prophages (JACO-CL PP1 and JACO-  
197 CL PP3) displaying similarity with PHAGE\_Vibrio\_VP58.5\_NC\_027981 and  
198 PHAGE\_Salmon\_118970\_sal3\_NC\_031940 (Supplementary Data Set 2). Secondly, we  
199 identified the 42,253 bp plasmid, thereby stressing the JACO-CL genome plasticity. KEGG  
200 orthology analysis on the plasmid open reading frames (ORFs) shows that most of the plasmidic  
201 genes encode for VirB type IV secretion system (T4SS) subunits (VirB1, VirB2, VirB4, VirB5,  
202 VirB6, VirB9, VirB10, VirB11) forming the *virB* operon (Supplementary Data Set 3). The *virB*  
203 operon was described on the Ti plasmid of *Agrobacterium tumefaciens*. Structural analysis  
204 evidenced eleven ORFs coding for potential membrane-spanning regions or signal peptides  
205 (Shirasu et al., 1990). Interestingly, the *virB* operon was also observed and extensively studied  
206 in the phytopathogenic *Xanthomonas* species. For instance, the role of the plasmid in pathogen  
207 virulence has been investigated in *virB7* knockout *X. citri* mutants and showed no effect on the  
208 development of canker symptoms in citrus (Souza et al., 2011). Similar results were obtained  
209 on *X. campestris* pv. *campestris*, with the deletion of the *virB* plasmid that did not modify  
210 phytopathogen aggressiveness on Brassica crop species (He et al., 2007). Conversely, the  
211 ability of the *VirB* T4SS in killing Gram-negative bacteria has been demonstrated in both *X.*  
212 *citri* (Souza et al., 2015) and *Salmonella maltophilia* (Bayer-Santos et al., 2019). Indeed, T4SS  
213 is responsible for transferring toxic effectors into the cells of bacterial competitors. Altogether,  
214 the presence of the T4SS *virB* plasmid in JACO-CL corroborates with the hypothesis that this  
215 strain might employ T4SS effectors to compete with bacterial populations inhabiting *A.*  
216 *thaliana* phyllosphere. This might be an additional evidence of the evolutionary history of *P.*  
217 *syringae* PG7 as a widely spread and common bacterium co-evolving with *Arabidopsis*.

218 Functional studies validating the role of the *virB* T4SS will help in clarifying whether or not  
219 this operon is a crucial determinant of competitive interactions between *P. syringae* and  
220 members of *A. thaliana* microbiota. Studies on the occurrence of the T4SS *virB* operon in *P.*  
221 *syringae* populations inhabiting *A. thaliana* will also contribute to understand the role of T4SS  
222 on the evolutionary forces driving the emergence of novel highly competitive pathogens.

223

224 **The JACO-CL strain carries a Single-Pathogenicity Island (S-PAI) T3SS and hop genes**  
225 **potentially related with pathogenicity in *A. thaliana***

226 Two PG7 genotypes with distinct virulence phenotypic traits were isolated in the past from *A.*  
227 *thaliana* (Araki et al., 2006; Jakob et al., 2007). In particular, PG7 natural populations carry  
228 two structurally divergent Pathogenicity Islands (PAIs), which encode for T3SS genes and are  
229 located in different chromosomal locations. The T-PAI is organized as the classical *P. syringae*  
230 tripartite T3SS with an *hrp/hrc* gene cluster, the exchangeable effector locus (*EEL*) and the  
231 conserved effector locus (CEL). On the other hand, the S-PAI is composed of the *hrp/hrc* cluster  
232 containing the *avrE* effector and its chaperone. Interestingly, PG7 isolates only possess one of  
233 the PAI as a possible result of balancing selection on the pathogen associated with the  
234 presence/absence of specific host resistance genes (Araki et al., 2006). To investigate the type  
235 of JACO-CL PAI, we first inferred a phylogeny based on the *avrE* effector located in the JACO-  
236 CL *hrp/hrc* gene cluster and reference PG7 strains carrying the S-or T-PAI island (Figure 3a).  
237 The *avrE* is a conserved effector of the *hrp/hrc* cluster present on both S-and T-PAI strains.  
238 However, *avrE* divergently evolved in the two distinct PAIs. A phylogenetic tree shows that  
239 JACO-CL carries a S-PAI configuration and this was confirmed by genome structural analysis  
240 (Figures 3a and 3b, Supplementary Data Set 4). In addition, KEGG analysis also corroborates  
241 that T3SS is not complete in JACO-CL as expected for S-PAI strains that have a reduced T3SS

242 (Supplementary Data Set 5). More specifically, JACO-CL harbors only a *hrp/hrc* cluster  
243 containing both *avrE* and *avrF* and their relative chaperons (Figure 3b).

244 Interestingly, through genome functional annotation, we found three complete Hop effectors  
245 (HopAJ2, HopAN1 and HopJ1) located in the genome of JACO-CL but not physically related  
246 with the PAI (Supplementary Data Set 4). To investigate the possible Horizontal Gene Transfer  
247 (HGT) of the tree Hop effectors from other *P. syringae* PGs, we compared the JACO-CL alleles  
248 with those already described in *P. syringae* and available at the Pseudomonas-Plant Interaction  
249 (PPI) <http://pseudomonas-syringae.org>. We also added alleles retrieved from Genebank  
250 (BLASTP > 50% of protein coverage). Phylogenetic relationships between the Hop protein  
251 sequences indicate that both HopAJ2 and HopAN1 are highly divergent from alleles previously  
252 described in other *P. syringae* PGs (Figure 3c). This result indicates that both Hop effectors  
253 were not recently acquired through HGT from existing alleles distributed in the *P. syringae*  
254 complex. On the opposite, phylogeny based on the HopJ1 protein sequences indicates that  
255 JACO-CL HopJ1 allele is closely related to the allele found in DC3000 strain (Figure 3c) (*P.*  
256 *syringae* pv. *tomato*, PG1 clade a), thereby suggesting a possible HGT acquisition.

257 HopAJ2<sub>Pph1448a</sub> and HopAN1<sub>Pph1448a</sub> alleles were tested for translocation. Pph1448a strain (*P.*  
258 *syringae* pv. *phaseolicola*) belongs to *P. syringae* PG3. Standard Hypersensitive Response (HR)  
259 and competitive assays were performed and none of the Pph1448a candidate fusions elicited HR,  
260 thereby proving the absence of translocation of Pph1448a alleles (Macho et al., 2009). This result  
261 indicates that HopAJ2 and HopAN1 should be considered as putative effectors. However, the  
262 differences between the JACO-CL protein sequences compared to Pph1448a (Figure 3c) suggest  
263 that HopAJ2<sub>JACO-CL</sub> and HopAN1<sub>JACO-CL</sub> might be still functional. Translocation assays in tobacco  
264 and Arabidopsis are necessary to validate these putative effectors in *P. syringae* strains with a non-  
265 canonical T3SS. Up to now, the role of these Hop proteins remains speculative but opens new  
266 perspectives in the validation of novel effectors. Regarding HopJ1, its translocation was observed

267 in DC3000 but not in Pph1448a (Macho et al., 2009). However, HopJ1<sub>DC3000</sub> and HopJ1<sub>Pph1448a</sub> show  
268 N-terminal changes sufficient to explain translocation variability. On the other hand, HopJ1<sub>JACO-CL</sub>  
269 shows a high protein similarity with the HopJ1<sub>DC3000</sub>, thereby making this effector a good candidate  
270 for validation assays in *P. syringae* PG7. Investigation of the prevalence of these effectors in natural  
271 *A. thaliana* populations will also be interesting to understand their role in the evolution of  
272 pathogenicity in the *P. syringae* complex.

273

### 274 **The adaptive potential for disease resistance is mediated by a polygenic architecture**

275 To investigate the genetic architecture underlying the adaptive potential of response to the *P.*  
276 *viridiflava* strain JACO-CL, we inoculated 192 whole-genome sequenced *A. thaliana*  
277 accessions of the TOU-A population. Quantitative variation in disease resistance to the JACO-  
278 CL strain was observed between the 192 TOU-A accessions (Supplementary Data Set 6).  
279 Similar quantitative variation among TOU-A accessions was observed in response to either the  
280 bacterial pathogens *Xanthomonas campestris* pv *campestris* (*Xcc*) and *Ralstonia solanacearum*  
281 (Debieu et al., 2016; Demirjian et al., 2022; Desaint et al., 2021; Huard-Chauveau et al., 2013)  
282 or natural infections in the native habitats of the TOU-A population (Roux & Frachon 2022).

283 Highly significant genetic variation was observed at 1 dai (accession effect:  $\lambda_{LR} = 38.0$ ,  $P =$   
284  $7.07E-10$ ), 2 dai (accession effect:  $\lambda_{LR} = 70.8$ ,  $P < 1.0E-16$ ) and 3 dai (accession effect:  $\lambda_{LR} =$   
285  $64.0$ ,  $P = 1.22E-15$ ) (Figure 4), with broad-senses heritability values of 0.48, 0.58 and 0.57 at  
286 1 dai, 2 dai and 3 dai, respectively. These results suggest that a non-negligible fraction of the  
287 adaptive potential of response to the JACO-CL strain is mediated by host genetics. Similar  
288 results were obtained for the pathosystems *Fraxinus excelsior* - *Hymenoschyphus* (Kjær et al.,  
289 2012), *Pinus pinaster* – *Fusarium circinatum* (Elvira-Recuenco et al., 2014) and *C. calophylla*  
290 – *Q. pitekeria* (Ahrens et al., 2019). However, narrow-sense heritability values were smaller for

291 *F. excelsior* (~0.36) and *C. calyphylla* (~0.14) than for *Pinus pinaster* (~0.53), thereby  
292 highlighting variation among species in the adaptive genetic potential to face novel or emerging  
293 pathogens.

294 To fine map QTLs associated with quantitative disease resistance (QDR) to the JACO-CL  
295 strain, we took advantage of the Illumina genome sequencing of the 192 TOU-A accessions  
296 phenotyped in this study, which revealed a set of 1,902,592 Single Nuclear Polymorphisms  
297 (SNPs) and a linkage disequilibrium (LD) decay to  $r^2 = 0.5$  within an average of 18 bp (Frachon  
298 et al., 2017). We combined a mixed-model approach correcting for the effects of population  
299 structure with a local score approach, which allows delimiting QTL regions by accumulating  
300 single marker *p*-values obtained from the mixed-model while controlling the issue of multiple  
301 hypothesis tests (Bonhomme et al., 2019). This combined GW-LS approach was previously  
302 demonstrated to be efficient in the TOU-A population, with the fine mapping (in particular, the  
303 detection of QTLs with small effects) and functional validation of one QTL of QDR to *Xcc*  
304 (Huard-Chauveau et al., 2013) and one QTL of QDR to *R. solanacearum* (Aoun et al., 2020).  
305 QDR to the JACO-CL strain was mediated by a polygenic architecture (Figure 5), with the  
306 detection of 16 QTLs, 14 QTLs and 22 QTLs at 1 dai, 2 dai and 3 dai, respectively  
307 (Supplementary Data Set 7). All these QTLs overlap with 87 unique candidate genes  
308 (Supplementary Data Set 7). Almost half of the QTLs were common between at least two days  
309 of scoring, whereas the other QTLs were specific to one day of scoring (Figure 6), thereby  
310 suggesting that the adaptive potential of response to the JACO-CL strain is mediated by a mix  
311 of constitutive and dynamic QTLs.

312 Two constitutive QTLs overlap with candidate genes of particular interest. The first QTL  
313 correspond to 95 top SNPs (at 1 dai) located on chromosome 2 (QTL7, Figure 5b) in the vicinity  
314 or centered on the gene *RIBONUCLEASE 1 (RNS1, AT2G02990)* (Supplementary Data Set 7),  
315 which encodes a member of the ribonuclease T2 family that is involved in wound-induced

316 signaling independent of jasmonic acid (LeBrasseur et al., 2002). The second QTL corresponds  
317 to 44 top SNPs (at 3 dai) located on chromosome 5 (QTL19, Figure 5c) in the vicinity or  
318 centered in the gene *AT5G18310*, which encodes for a ubiquitin hydrolase. Interestingly, this  
319 protein is the target of the effector AvrRpt2 of *P. syringae* (Chisholm et al., 2005). A 4.04-fold  
320 variation of the expression level of *AT5G18310* in leaves was observed between 665 worldwide  
321 accessions of *A. thaliana* (Supplementary Data Set 8) (Kawakatsu et al., 2016). GWA mapping  
322 revealed that the natural variation in the expression of *AT5G18310* is controlled at the  
323 worldwide scale by one QTL located in the 5' region of *AT5G18310*, with top SNPs that were  
324 common with the top SNPs identified in response to the JACO-CL strain (Figure S1). This  
325 suggests that modification of cis-regulation of *AT5G18310* mediates genetic variation of the  
326 response to the JACO-CL strain in the TOU-A population. Estimating the expression level of  
327 *AT5G18310* in TOU-A accessions contrasted in their response to the JACO-CL strain could  
328 support this hypothesis. In addition, *AT5G18130* is located nearby a dynamic QTL (QTL20,  
329 Figure 5c) encompassing two genes (*AT5G18350* and *AT5g18360*) encoding for  
330 Toll/Interleukin-1 receptor nucleotide-binding site leucine-rich repeat (TIR-NBS-LRR)  
331 proteins. Based on the Illumina sequencing coverage depth of the QTL 20, no presence/absence  
332 (P/A) polymorphism was detected for *AT5G18360*. However, we detected a P/A polymorphism  
333 for *AT5G18350* (present in 134 accessions and absent in 58 accessions), which is line with the  
334 P/A polymorphism for *AT5G18350* identified on a set of 33 worldwide accessions by PCR  
335 amplification (Shen et al., 2006). Given the function of the candidate genes underlying these  
336 two closely located QTLs on chromosome 5, it is tempting to propose a scenario similar to the  
337 one observed between the effector AvrRpt2, the guard protein RIN4 and the *R* gene RPS2,  
338 where degradation of RIN4 proteins by the proteolytic activity of AvrRpt2 triggers defense  
339 responses *via* RPS2 (Chisholm et al., 2005). In our study, *AT5G18310* proteins might be  
340 degraded by the proteolytic activity of one the three complete Hop effectors (HopAJ2, HopAN1

341 and HopJ1), which in turn would induce defense responses *via* the *R* genes  
342 AT5G18350/AT5G18360. Further experiments testing protein-protein interactions are needed  
343 to validate this scenario.

344 Because genetic variation was found for disease resistance to the *P. syringae sensus stricto*  
345 strain RAYR-BL (Table S2), it would be interesting to test whether genetic solutions underlying  
346 the adaptive potential of response of the TOU-A population are similar between strains of *P.*  
347 *syringae sensu stricto* and *P. viriflava* originating from more southern geographic regions. It  
348 would definitely help to better understand the genetic and molecular basis of the adaptive  
349 potential in ecologically relevant *A. thaliana* – *P. syringae* pathosystems.

350

## 351 **EXPERIMENTAL PROCEDURES**

### 352 **Bacterial strains and phylogroup affiliation**

353 The eight strains considered in this study were isolated from eight natural populations of *A.*  
354 *thaliana* located south-west of France (Figure 1A) (Bartoli et al., 2018). Briefly, in spring 2015,  
355 two or three plants of each natural population of *A. thaliana* were transferred to the laboratory  
356 and leaves were aseptically homogenized and serial dilutions were plated on Trypticase Soy  
357 Agar (TSA) medium supplemented with 100 mg/L of cycloheximide. To detect *P. syringae*-  
358 like strains, isolates were screened by PCR with a *Psy* marker (Guilbaud et al., 2016). All the  
359 strains positive for the *Psy* marker were amplified with the primer set *shcA* described in (Bartoli  
360 et al., 2014), which allows discriminating among PG 7 *P. syringae* haplotypes.

361 Phylogroup/clade affiliation of the eight strains was defined based on the *P. syringae* species  
362 complex nomenclature (Berge et al., 2014). To do so, the sequences of the housekeeping gene  
363 *citrate synthase (cts)* obtained in (Bartoli et al., 2018) and the reference *cts* nucleotide sequences  
364 of *P. syringae* obtained from (Berge et al., 2014) were aligned with DAMBE version 5.1.1 (Xia,



365 2013). MEGA 7 was used to infer the phylogeny by using a maximum likelihood model (Kumar  
366 et al., 2016).

367

### 368 **Genome sequencing of the JACO-CL strain**

369 High Molecular Weight DNA of the JACO-CL strain was extracted from an overnight culture  
370 (TSA medium, 28°C) following the protocol of Mayjonade et al.  
371 ([dx.doi.org/10.17504/protocols.io.r9id94e](https://doi.org/10.17504/protocols.io.r9id94e)). An Oxford Nanopore Technology (ONT) library  
372 was prepared using the EXP-NBD103 and SQK-LSK108 kits according to the manufacturer's  
373 instructions and starting with 3 µg of 20 kb sheared DNA (Megaruptor, Diagenode) as input.  
374 This library was pooled with 11 other samples and 100 fmol of this pool was loaded and  
375 sequenced on one R9.4.1 flowcell for 72 h. The same DNA sample was used to prepare an  
376 Illumina library using the TruSeq Nano DNA HT Library Prep Kit (Illumina). This library was  
377 sequenced with 43 other samples on a HiSeq3000 instrument (2×150 bp paired-end reads)  
378 (Illumina) at the GeT-PlaGe INRAE Sequencing facility (Toulouse, France).

379 Nanopore raw signals were filtered with Guppy 2.1.3. After filtering, we obtained 52, 026  
380 filtered reads (min = 215, max = 49, 692) corresponding to 654, 999, 490 bp. The filtered reads  
381 were assembled with Canu 1.7 (Koren et al., 2017) with default parameters. Circularization of  
382 the assembled genome was performed with a home-made pipeline running a NCBI-blastn by  
383 filtering with a minimal length of the High-scoring Segment Pair (HSP) – a local alignment  
384 without gaps – of 10, 000 nt and an overhand of 5, 000 nt. Illumina sequencing yielded 1, 277,  
385 516 paired-end reads corresponding to 381, 654, 800 bp. The Nanopore assembly was error  
386 corrected by mapping the Illumina reads with BWA (version 0.7.17) and then performing two  
387 rounds of polishing with Pilon (version 1.22). Finally, we obtained two circular JACO-CL  
388 sequences, one corresponding to a plasmid (42, 253 bp) and one corresponding to the genomic

389 DNA of the strain (6, 043, 462 bp). Genomic DNA and plasmid were deposited in GenBank  
390 under Accession Numbers CP097286 and CP097286 respectively (related Bio Sample  
391 SAMN28171611 and Bio Project PRJNA836746).

392

### 393 **Genomic characterization of the JACO-CL strain**

394 The circular JACO-CL genome was annotated and analyzed with the Bacannot framework  
395 <https://bacannot.readthedocs.io/en/latest/#index--page-root> using Prokka version 1.14.5 for  
396 annotation (Seemann, 2014). KEGG KO annotation was performed with KofamScan version  
397 1.3.0 (Aramaki et al., 2020). KEGG analysis was separately run on genomic and plasmid DNA  
398 sequences and plasmid protein annotation was checked by BLASTP by searching for protein  
399 sequences showing homology with known plasmids.

400 PhiSpy and PHASTER were used for phage finding and annotation (Akhter et al., 2012; Zhou  
401 et al., 2011). T3SS effector proteins identification was determined through BLASTP search of  
402 the effector proteins reported at the Pseudomonas-Plant Interaction (PPI) [http://pseudomonas-](http://pseudomonas-syringae.org)  
403 [syringae.org](http://pseudomonas-syringae.org) database with an e-value of 1e-6. We enriched this existing effector database with  
404 T3SS effector proteins reported previously in the *P. syringae* PG7 (Bartoli et al., 2014). Only  
405 complete effectors (alignment length  $\geq$  25%) were kept in the analysis. In addition, putative  
406 effector proteins and/or proteins found in the CEL were manually blasted in GenBank for  
407 further annotation. Phylogenetic relationships for the three complete Hop proteins (HopAJ2,  
408 HopAN1 and HopJ1) found in the JACO-CL genome were estimated by comparing these Hops  
409 with alleles retrieved at the PPI database and on GenBank through BLASTP of the JACO-CL  
410 Hop sequences. Amino acid sequences of each Hop were individually aligned with MAFFT  
411 v7.271 (Kato and Standley, 2013) and a Maximum Likelihood (ML) tree was inferred with  
412 MEGA7 (Kumar et al., 2016).

## 413 **Genotype x Genotype interactions**

414 To estimate the genetic potential of the local mapping population TOU-A to face strains of the  
415 *P. syringae* complex, seven TOU-A accessions (A1-9, A1-19, A1-137, A6-18, A6-47, A6-55  
416 and A6-105) and the reference accession Col-0 were inoculated with each of the eight strains  
417 (BAZI-AL, JACO-CL, NAZA-AL, NAUV-BL, RADE-A4, RAYR-BL, SIMO-AL and SAUB-  
418 A). The reference accession Col-0 was also inoculated with the same set of eight strains. A  
419 growth chamber experiment with 192 plants was set up at the Toulouse Plant Microbe  
420 Phenotyping Platform (TPMP), with three plants inoculated for each ‘accession\*strain’  
421 combination. After a 4-day stratification treatment, plants were grown in Jiffy pots at 22 °C  
422 under 90% humidity and artificial light to provide a 9-hr photoperiod (Huard-Chauveau et al.,  
423 2013). Bacterial inoculation was conducted on 28-day-old plants using a blunt-ended syringe  
424 (Terumo® SYRINGE 1mL, SS+01T1). Four leaves per plant were entirely infiltrated with a  
425  $5.10^7$  CFU<sub>mL</sub>-1 bacterial solution. Disease symptoms were visually scored at 1, 2, 3 and 4 dai  
426 as described in (Roux et al., 2010). Each infected leaf received a score from 0 to 1, with 0  
427 corresponding to no symptoms whereas 0.5 and 1 correspond to medium and severe symptoms,  
428 respectively. These scores are determined by the presence of visible chlorosis, visible necrosis,  
429 leaf mosaic or water-soaked lesions and cell death related symptoms surrounding infection  
430 sites. We averaged the scores of the four inoculated leaves per plant.

431 For each of the six strains of the *P. syringae* complex for which symptoms were observed on  
432 plants (Supplementary Table S1), we explored genetic variation among accessions at each dai,  
433 using the following model:

$$434 \quad \text{disease index}_i = \mu_{\text{trait}} + \text{accession}_i + \varepsilon_i \quad (1)$$

435 where ‘ $\mu$ ’ is the overall phenotypic mean; ‘accession’ accounts for differences among the eight  
436 *A. thaliana* accessions; ‘ $\varepsilon$ ’ is the residual term. Inference was performed using ReML

437 estimation, using the PROC MIXED procedure in SAS v.9.4 (SAS Institute Inc., Cary, North  
438 Carolina, USA). The ‘accession’ factor was treated as a fixed effect. Because *A. thaliana* is a  
439 highly selfing species (Platt et al., 2010), Least-squares means (LS-means) obtained for each  
440 accession from model (1) correspond to the genotypic values of accessions.

441 To explore genetic variation among the eight strains, we used the following model at each dai:

$$442 \quad \text{LS-means of disease index}_i = \mu_{\text{trait}} + \text{strain}_j + \varepsilon_j \quad (1)$$

443 where ‘ $\mu$ ’ is the overall mean of LS-mean values; ‘strain’ accounts for differences among the  
444 six strains of the *P. syringae* complex for which symptoms were observed on plants; ‘ $\varepsilon$ ’ is the  
445 residual term. Inference was performed using ReML estimation, using the PROC MIXED  
446 procedure in SAS v.9.4 (SAS Institute Inc., Cary, North Carolina, USA). The ‘strain’ factor  
447 was treated as a fixed effect.

448

## 449 **Genome-Wide Association mapping of response to the JACO-CL strain**

### 450 *Experimental design and growth conditions*

451 To investigate the genetic architecture underlying the adaptive potential of response to the  
452 JACO-CL strain, we inoculated 192 whole-genome sequenced accessions of the TOU-A  
453 population. A growth chamber experiment with 1,152 plants was set up at the Toulouse Plant  
454 Microbe Phenotyping Platform (TPMP) using a randomized complete block design (RCBD)  
455 with six experimental blocks. Each block was represented by 192 plants corresponding to the  
456 192 TOU-A accessions. Growth chamber conditions, bacterial inoculation and disease scoring  
457 at 1 dai, 2 dai and 3 dai, were similar to the experiment conducted on the eight accessions, as  
458 described above.

459

460 *Statistical analysis*

461 At each dai, we explored genetic variation among the 192 TOU-A accessions using the  
462 following model:

$$463 \quad \text{disease index}_i = \mu_{\text{trait}} + \text{block}_b + \text{accession}_i + \varepsilon_{bi} \quad (3)$$

464 where ‘ $\mu$ ’ is the overall phenotypic mean; ‘block’ accounts for differences in micro-  
465 environment among the six experimental blocks; ‘accession’ accounts for differences among  
466 the 192 TOU-A *A. thaliana* accessions; ‘ $\varepsilon$ ’ is the residual term. Inference was performed using  
467 ReML estimation, using the PROC MIXED procedure in SAS v.9.4 (SAS Institute Inc., Cary,  
468 North Carolina, USA). While the ‘block’ factor was treated as a fixed effect, the ‘accession’  
469 effect was treated as a random effect. Significance of the random effect was determined by  
470 likelihood ratio tests of the model with and without this effect. Best Linear Unbiased Estimates  
471 (BLUEs) of disease index were obtained for each accession from model (3). Broad-sense  
472 heritability ( $H^2$ ) of disease index was estimated from variance component estimates for the  
473 ‘block’ and ‘acc’ terms (PROC VARCOMP procedure in SAS v.9.4)

474 *GWA mapping combined with a local score approach*

475 The effects of population structure on phenotype-genotype associations were demonstrated to  
476 be limited in the TOU-A population (Baron et al., 2015; Brachi et al., 2013)]. We nonetheless  
477 run GWA mapping using a mixed model implemented in the software EMMAX (Efficient  
478 Mixed-Model Association eXpedited) (Kang et al., 2010). To control for the effect of  
479 population structure, we included as a covariate an identity-by-state kinship matrix  $K$ . This  
480 kinship matrix was based on 1,902,592 SNPs identified among the 192 accessions of the TOU-  
481 A population (Frachon et al., 2017). Because rare alleles may increase the rate of false positives  
482 (Atwell et al., 2010; Bergelson and Roux, 2010; Brachi et al., 2010), we only considered SNPs  
483 with a minor allele relative frequency (MARF)  $> 7\%$ , resulting in 978,408 SNPs. Above this

484 MARF threshold,  $p$ -value distributions obtained from the EMMAX mixed model are not  
485 dependent on MARF values in the TOU-A population (Frachon et al., 2017).

486 In order to better characterize the genetic architecture associated with natural genetic variation  
487 in response to JACO-CL, a local score approach was applied on the set of  $p$ -values provided by  
488 EMMAX. This local score approach increases the power of detecting QTLs with small effect  
489 and narrows the size of QTL genomic regions (Bonhomme et al., 2019; Fariello et al., 2017).  
490 In this study, we used a tuning parameter  $\xi$  of 2 expressed in  $-\log_{10}$  scale. Significant phenotype-  
491 SNP associations were identified by estimating a chromosome-wide significance threshold for  
492 each chromosome (Bonhomme et al., 2019). Based on a custom script (Libourel et al., 2021),  
493 we retrieved all candidate genes underlying QTLs by selecting all genes inside the QTL regions  
494 as well as the first gene upstream and the first gene downstream of these QTL regions (Dataset  
495 7). The TAIR 10 database (<https://www.arabidopsis.org/>) was used as our reference. The  
496 number of candidate genes that were either specific to a single dai or common between several  
497 dai were illustrated by a Venn diagram using the package *jvenn* (Bardou et al., 2014).

498

#### 499 **GW-LS on the level of expression of the candidate gene *AT5G18310***

500 Data of the expression level of *AT5G18310* were retrieved from (Kawakatsu et al., 2016) for  
501 665 whole-genome sequenced worldwide accessions that grew in similar growth conditions.  
502 Based on a set of 11,769,920 SNPs (1001 Genomes Consortium. 2016), we applied the same  
503 GW-LS approach than for investigating the genetic architecture of disease index in the TOU-A  
504 population. We considered SNPs with a minor allele relative frequency (MARF)  $> 10\%$ ,  
505 resulting in 1,703,894 SNPs.

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517

518

519 **DATA AVAILABILITY STATEMENT**

520 Raw phenotypic data of disease index are available in Data Sets 1 and 6. Genomic DNA and  
521 plasmid were deposited in GenBank under Accession Numbers CP097286 and CP097286  
522 respectively (related Bio Sample SAMN28171611 and Bio Project PRJNA836746).

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799 **FIGURE LEGENDS**

800 **Figure 1. Ecological habitats and phylogenetic affiliation of the eight natural strains used**  
801 **in this study. (a)** Diversity of habitats from which the eight *Pseudomonas* strains have been  
802 isolated in south-west of France. BAZI-A: Baziège, JACO-C: Jacoy (Boussenac), NAZA-A:  
803 Saint-Pierre-de-Najac, NAUV-B : Nauviale, RADE-A : Sainte-Radegonde, RAYR-B : Rayret  
804 (Cassagnes-Bégonhès), SIMO-A : Simorre, SAUB-A : Saubens. **(b)** Phylogenetic tree  
805 (Neighbor Joining, NJ) based on the *cts* sequences (406 bp) of the 64 strains proposed by Berge  
806 *et al.*, (2014) for the rapid identification and classification of the seven strains belonging to the  
807 *Pseudomonas syringae* complex.

808 **Figure 2. Genetic variation of virulence between six belonging to the *Pseudomonas***  
809 ***syringae* complex, across eight accessions of *A. thaliana* at 1dai, 2 dai, 3 dai and 4 dai.** The  
810 four strains of *P. viridiflava* are in red while two strains of *P. syringae* are in blue. No symptoms  
811 was observed on any plant for the two other strains of *P. syringae* (BAZI-AL and SIMO-AL,  
812 Supplementary Table S1). Dots corresponds to the genotypic values of the seven TOU-A  
813 accessions (green) and the reference accession Col-0 (black).

814 **Figure 3. Type III Secretion System of the JACO-CL strain. (a)** Phylogenetic tree (Neighbor  
815 joining, NJ) based on the nucleotide sequence of the *avrE* gene (5160 bp). T-PAI and S-PAI  
816 (indicated in red) *Pseudomonas viridiflava* strains were integrated in the analysis to investigate  
817 the T3SS JACO-CL affiliation. DC3000 (PG1), B728a (PG2), 1\_6 (PG4), ES4326 (PG5),  
818 CFBP2067 (PG6) *avrE* nucleotide sequences were included in the analysis as reference strains  
819 carrying a canonical T3SS. The phylogenetic analysis demonstrates that the JACO-CL strain  
820 carries a S-PAI T3SS that is closely related to the TA043 strain isolated from *Primula officinalis*  
821 (Bartoli *et al.*, 2014). **(b)** JACO-CL *hrp/hrc* Type III locus reconstructed after genome  
822 annotation. Line lengths are proportional to protein size and protein annotation was performed  
823 both based on Bacannot pipeline and manual correction. **(c)** Phylogenetic inference (Neighbor

824 Joining, NJ) on the amino acid sequences of the predicted Hop proteins found in the JACO-CL  
825 strain. Reference protein sequences were recovered on Pseudomonas-Plant Interaction (PPI)  
826 database ([http://www.pseudomonas-syringae.org/pst\\_func\\_gen2.htm](http://www.pseudomonas-syringae.org/pst_func_gen2.htm)) and GeneBank. NJ trees  
827 show that all hops (HopAJ2, HopJ1 and HopAN1) from JACO-CL are divergent from those  
828 described previously in other *Pseudomonas syringae* pathovars.

829 **Figure 4. Genetic variation of plant response in the TOU-A local mapping population to**  
830 **the JACO-CL strain at 1dai, 2dai and 3dai.** The dots correspond to the genotypic values of  
831 the 192 accessions. Each black line corresponds to the plant response to one of the 192  
832 accessions. The red line represents the mean of disease index among the 192 accessions.

833 **Figure 5. The genetics of quantitative disease resistance to the JACO-CL strain in the**  
834 **TOU-A population. (a)** Manhattan plots comparing the GWA mapping results at 1 dai, 2 dai  
835 and 3 dai. The x-axis indicates the physical position of the 978,408 SNPs on the five  
836 chromosomes. The y-axis indicates the Lindley process scores estimated from  $-\log_{10} p$ -values  
837 from the mixed model implemented in the software EMMAX using SNPs with a minor allele  
838 relative frequency (MARF)  $> 7\%$ . **(b)** Zoom spanning a genomic region at the beginning of  
839 chromosome II from 0.1Mb to 1.2Mb. **(c)** Zoom spanning a genomic region at the beginning of  
840 chromosome V from 6Mb to 6.14Mb. The dashed line indicates the maximum of the five  
841 chromosome-wide significance thresholds. Arrows indicate the three QTLs containing  
842 candidate genes of particular interest.

843 **Figure 6. Venn diagram illustrating the number of common and specific candidate genes**  
844 **between the three days of scoring.** Colored bars indicate the number of candidate genes at  
845 each dai. The vertical stacked bar plot indicates the number of candidate genes specific to one  
846 dai (right) or shared between two or three dai.

## 847 SUPPLEMENTARY INFORMATION

848 **Table S1.** Mean ‘strain’ effect with and without considering the reference accession Col-0 at  
849 1dai, 2 dai, 3 dai and 4 dai.

850 **Table S2.** Genetic variation among accessions for each strain, with and without considering the  
851 reference accession Col-0 at 1dai, 2 dai, 3 dai and 4 dai.

852 **Figure S1.** GWA mapping results of the expression level of the candidate gene *AT5G18310*.

853

## 854 SUPPLEMENTARY DATA SETS

855 **Data Set 1.** Raw phenotypic data of disease index of seven TOU-A accessions and the reference  
856 Col-0 in response to four strains of *P. syringae* and four strains of *P. viridiflava*.

857 **Data Set 2.** Prophages found in the genome of the JACO-CL strain.

858 **Data Set 3.** pDR208 plasmid annotation based on the KEGG metabolic pathways.

859 **Data Set 4.** Type III Secretion System (T3SS) proteins predicted on the annotated genome of  
860 the JACO-CL strain.

861 **Data Set 5.** Heatmap on the KEGG metabolic pathways estimated for the JACO-CL strain.

862 **Data Set 6.** Raw phenotypic data of disease index scored on 192 TOU-A accessions in response  
863 to the JACO-CL strain.

864 **Data Set 7.** List of QTLs and associated SNPs detected for disease index at each dai.

865 **Data Set 8.** Normalized RNA-seq read counts for the candidate gene *AT5G18310* for 665  
866 worldwide accessions.

**Figure 1**

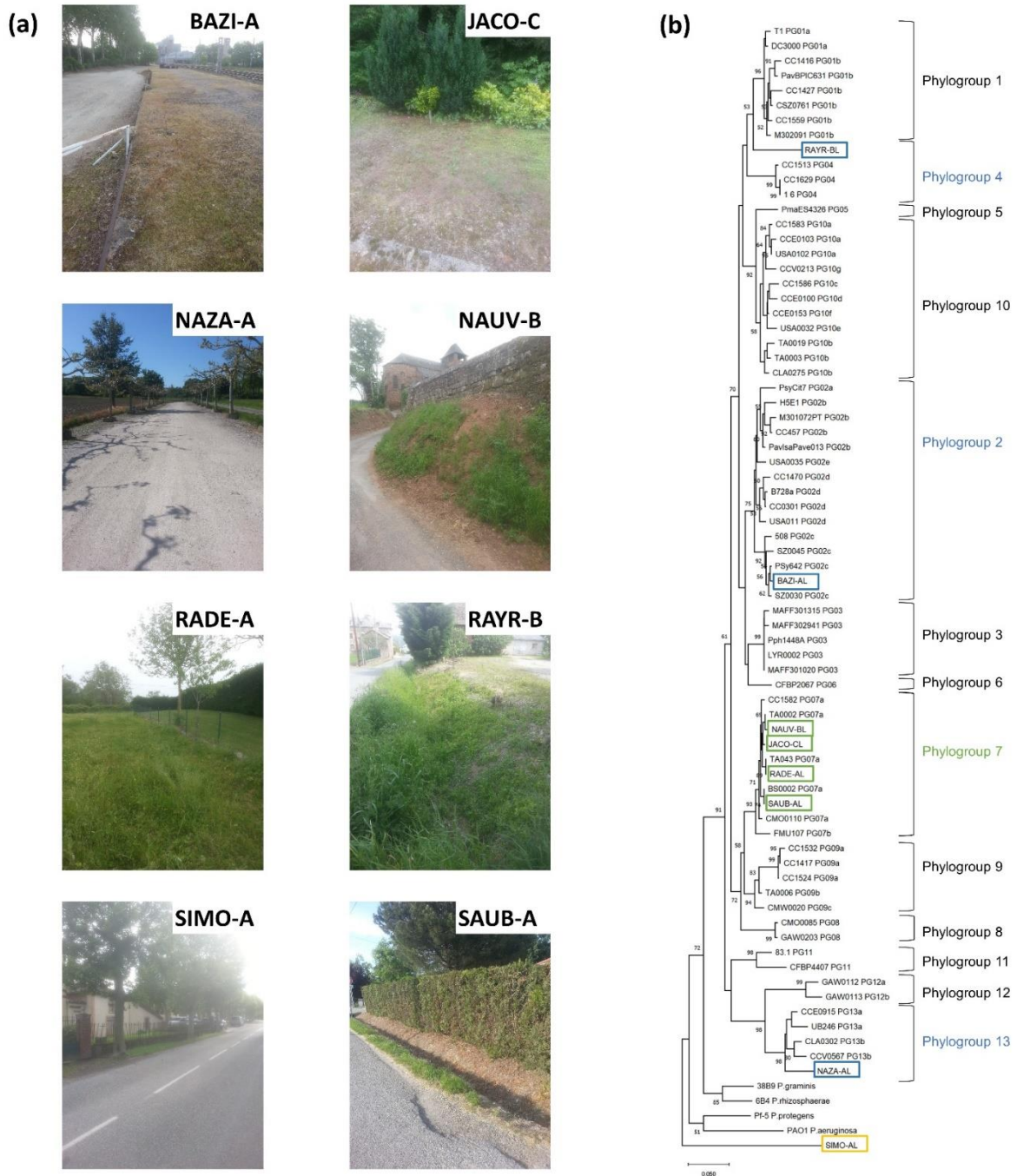
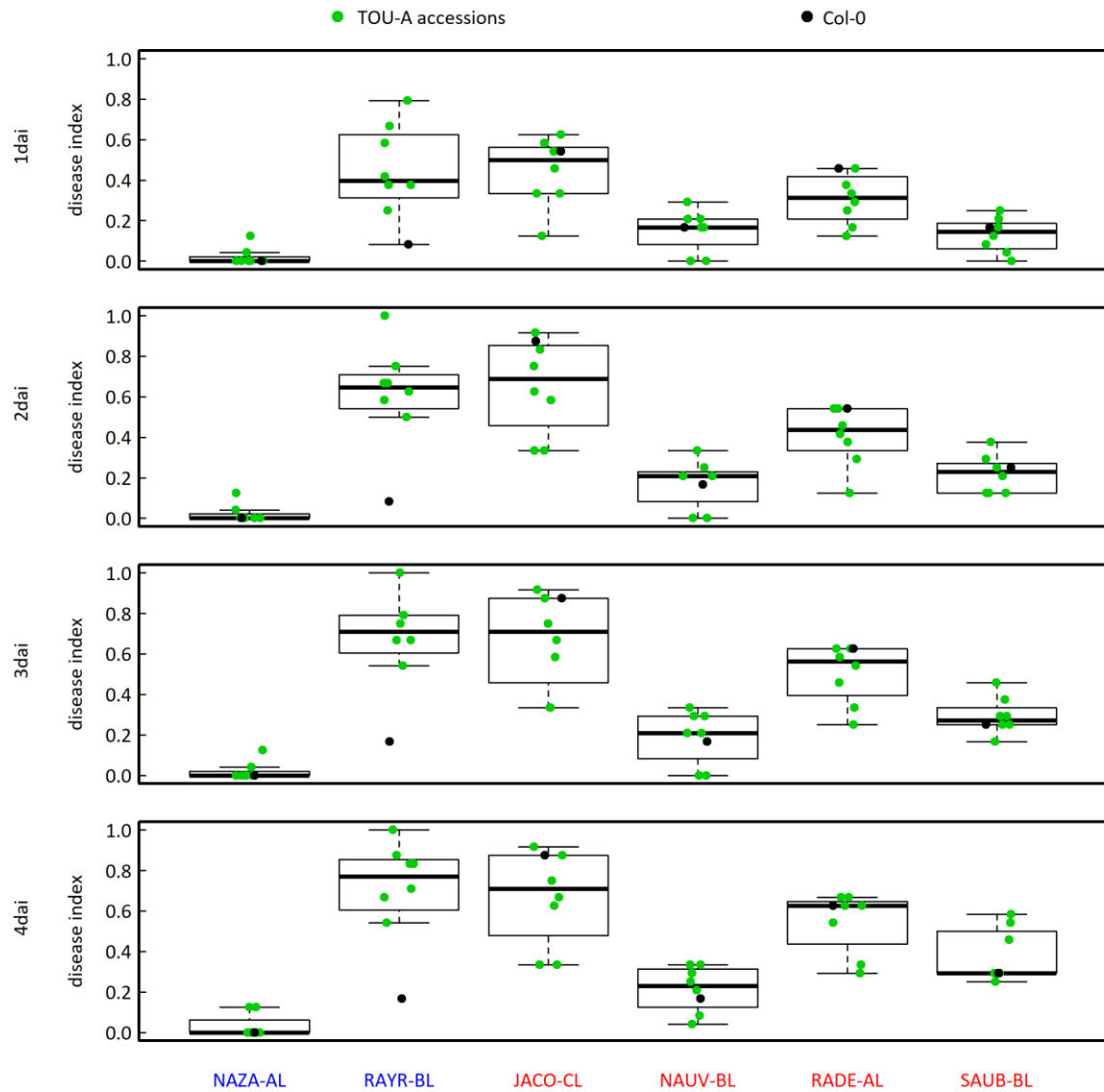
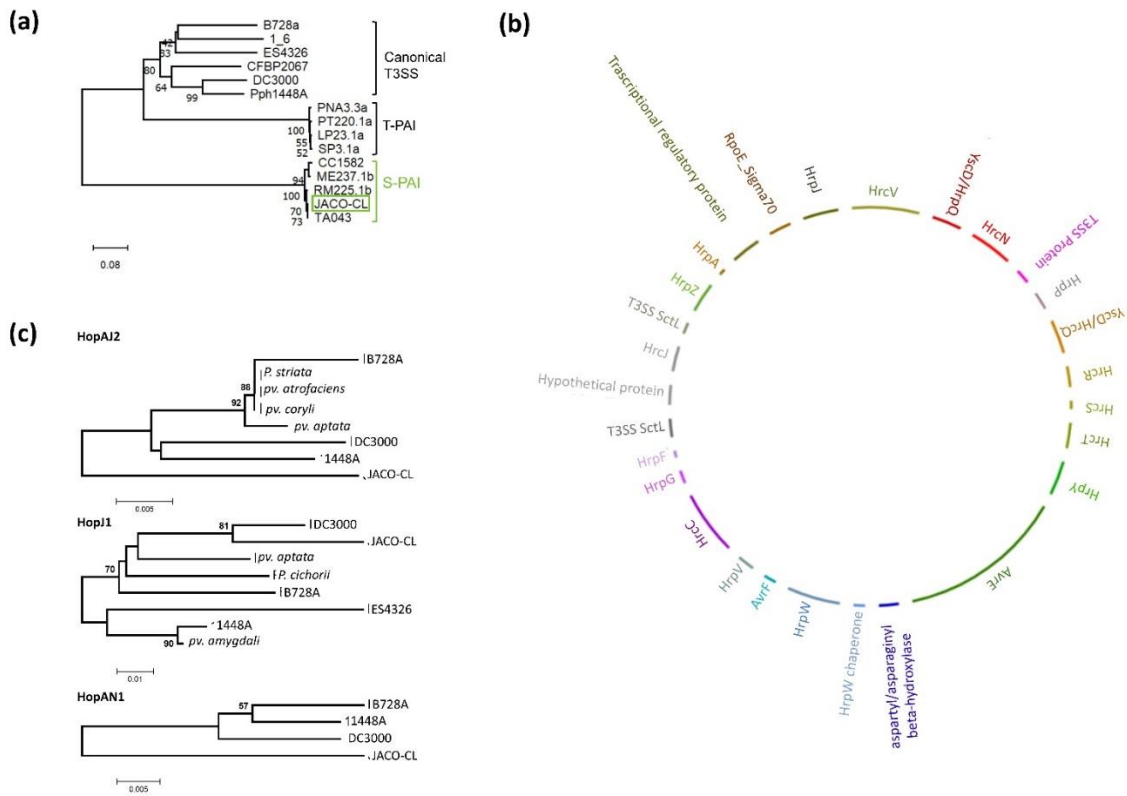


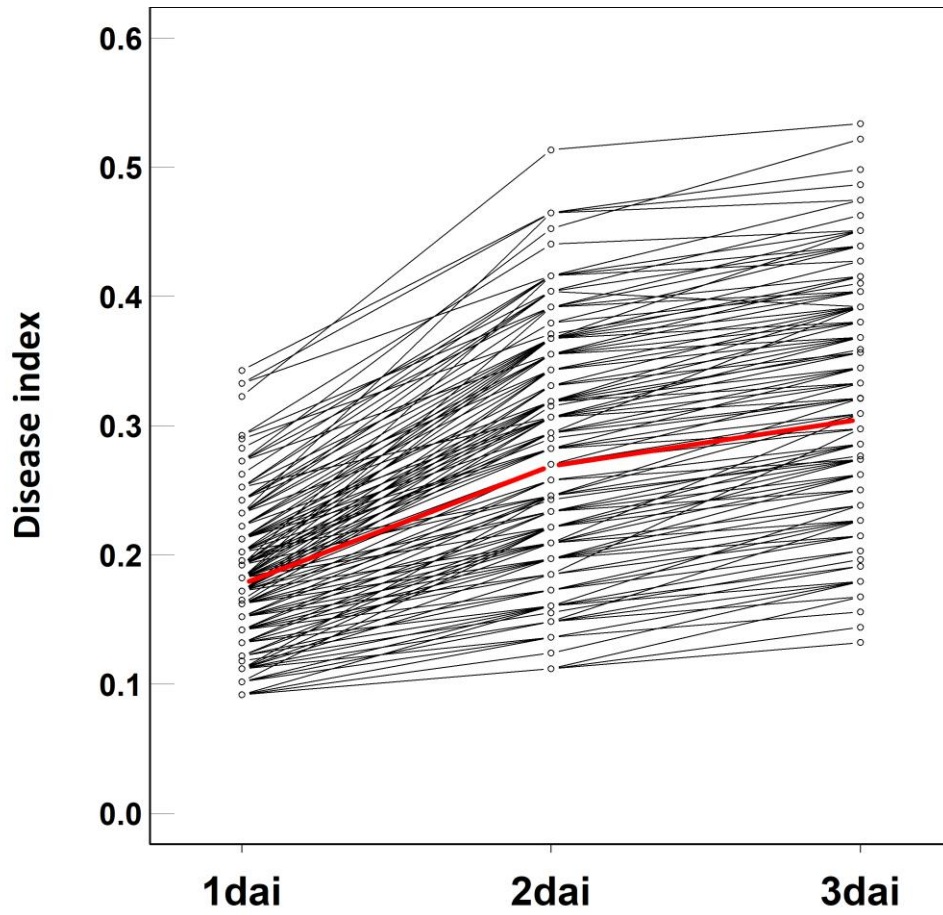
Figure 2



**Figure 3**

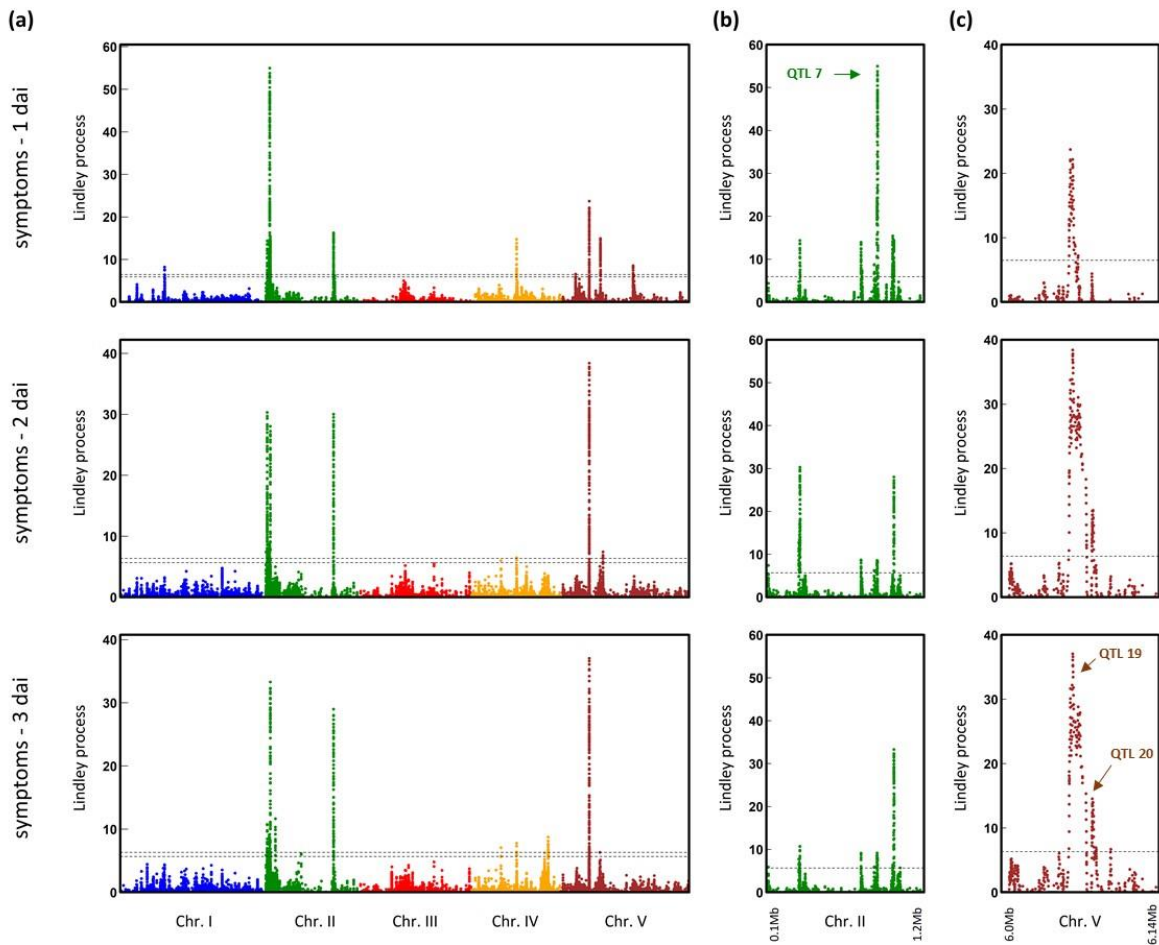


**Figure 4**





**Figure 5**



**Figure 6**

