Phylotranscriptomics of the Pentapetalae Reveals Frequent Regulatory Variation in Plant Local Responses to the Fungal Pathogen *Sclerotinia sclerotiorum*

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One-sentence summary: Comparative transcriptome analyses reveal a major contribution of regulatory divergence in conserved genes during the response of Pentapetalae plants to the fungal pathogen *Sclerotinia sclerotiorum*.

Keywords: Quantitative disease resistance, *Sclerotinia sclerotiorum*, gene expression, fungal pathogen, evolution of plant immunity, regulatory divergence, exaptation, ABC transporter.

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ABSTRACT

Quantitative disease resistance (QDR) is a conserved form of plant immunity that limits infections caused by a broad range of pathogens. QDR has a complex genetic determinism. The extent to which molecular components of the QDR response vary across plant species remains elusive. The fungal pathogen *Sclerotinia sclerotiorum*, causal agent of white mold disease on hundreds of plant species, triggers QDR in host populations. To document the diversity of local responses to *S. sclerotiorum* at the molecular level, we analyzed the complete transcriptomes of six species spanning the Pentapetalae (*Phaseolus vulgaris*, *Ricinus communis*, *Arabidopsis thaliana*, *Helianthus annuus*, *Solanum lycopersicum*, and *Beta vulgaris*) inoculated with the same strain of *S. sclerotiorum*. About one third of plant transcriptomes respond locally to *S. sclerotiorum*, including a high proportion of broadly conserved genes showing frequent regulatory divergence at the interspecific level. Evolutionary inferences suggested a trend towards the acquisition of gene induction relatively recently in several lineages. Focusing on a group of ABCG transporters, we propose that exaptation by regulatory divergence contributed to the evolution of QDR. This evolutionary scenario has implications for understanding the QDR spectrum and durability. Our work provides resources for functional studies of gene regulation and QDR molecular mechanisms across the Pentapetalae.
INTRODUCTION

The plant immune system includes multiple molecular mechanisms for pathogen detection and defense. Gene-for-gene resistance involves single dominant resistance (‘R’) genes often belonging to the nucleotide-binding domain and leucine-rich repeat (NLR) family of proteins (Dodds and Rathjen, 2010). This form of resistance typically protects plants against a few genotypes of biotrophic pathogens, which keep host cells alive during infection. There are however no dominant R genes described to function against many necrotrophic pathogens (which actively kill host cells during infection), such as the fungus Sclerotinia sclerotiorum (Mbengue et al., 2016; Guyon et al., 2014). S. sclerotiorum is the causal agent of white and stem mold diseases on a broad range of dicot plants (Bolton et al., 2006). The host range of S. sclerotiorum covers all major groups of the Pentapetalae, a subclade of the core eudicots including Rosidae, Asteridae and Caryophyllaceae (Moore et al., 2010; Navaud et al., 2018). This fungus notably causes severe damage on oil crops such as soybean (Glycine max) and rapeseed (Brassica napus) when conditions are favorable (Derbyshire and Denton-Giles, 2016). Plants typically exhibit quantitative disease resistance (QDR) against S. sclerotiorum, a form of immunity resulting in a full continuum of disease symptom severity in plant populations (Roux et al., 2014). The distribution of disease symptoms on plants challenged by S. sclerotiorum follows an approximately Gaussian distribution in populations of sunflower (Helianthus annuus, Asterales) (Fusari et al., 2012), soybean (G. max, Fabales) (Bastien et al., 2014), rapeseed (B. napus, Brassicales) (Wu et al., 2013) and Arabidopsis thaliana (Brassicaceae) (Perchepied et al., 2010) for instance. QDR is a complex type of resistance involving genes of very diverse families (Corwin and Kliebenstein, 2017). Due to the functional diversity and complexity of QDR, the genetic bases and evolution of QDR remain largely unexplored.

How many genes contribute to the QDR response to a given pathogen is an unresolved question of fundamental and practical interest (Corwin and Kliebenstein, 2017). This information is indeed critical to understand the evolutionary dynamics of plant pathogens with a broad host range, and to optimize breeding for disease resistance in crops. Genome Wide Association (GWA) mapping in A. thaliana natural populations and the analysis of fungal small RNA putative targets identified five QDR genes against S. sclerotiorum (Badet et al., 2017, 2019; Barbacci et al., 2018; Derbyshire et al., 2019). The inactivation of these genes caused 16 to 36% variation in the QDR phenotype each, in line with previous quantitative trait loci analyses for QDR in crops (Micic et al., 2004; Poland et al., 2011; Bonhomme et al., 2014). Together with other approaches (Mbengue et al., 2016; Perchepied et al., 2010; Stotz et al., 2011), these studies bring to a few dozens the number of known A. thaliana genes contributing to QDR against S. sclerotiorum. Global transcriptomic analyses revealed that the number of genes differentially expressed upon S. sclerotiorum challenge adds up to 4,703 in A. thaliana and 3,513 in tomato (Solanum lycopersicum; Badet et al., 2017), suggesting that the number of QDR genes may exceed by far the current validated list. In agreement, GWA mapping for resistance against four distinct Botrytis cinerea strains in 110 A. thaliana accessions identified 3,504 genes as candidates for controlling QDR (Corwin et al., 2016). Extending the analysis to several parameters...
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describing the disease lesion (size, shape, color) increased to 7,940 the number of candidate QDR genes in the A. thaliana – B. cinerea interaction (Fordyce et al., 2018).

These findings provide molecular foundations for an infinitesimal model (or “polygenic model”) of plant QDR. The infinitesimal model of quantitative genetics postulates that continuous variation in a trait is often controlled by environmental factors and a large number of loci, each making a very small (infinitesimal) contribution to the phenotype (Fisher, 1919; Nelson et al., 2013). The existence of very large numbers of causal genetic variants is proposed to explain the small proportion of phenotypic variance resulting from variants identified in GWA studies, a problem known as the “missing heritability” (Manolio et al., 2009; Barton et al., 2017). Because the infinitesimal model notably assumes linkage equilibrium and additive phenotypic effect of loci, the genetic architecture of disease resistance probably involves an additional layer of complexity (Barton et al., 2017; Turelli, 2017). In a recent variant of the infinitesimal model coined the “omnigenic” model, Boyle et al. suggest that any gene differentially regulated in infected tissues has a non-null impact on the disease outcome (Boyle et al., 2017). Following this view, a modest number of core genes would directly and strongly affect QDR and could each be modulated by numerous small effect genes as a result of their interactions in global gene networks (Boyle et al., 2017; Liu et al., 2019). Although the most efficient way to move forward in understanding the genetics of complex traits is debatable, the omnigenic model emphasizes the relevance of large-scale transcriptomics for studying diseases (Boyle et al., 2017; Wray et al., 2018).

A global knowledge of gene regulation is also key to study complex traits from an evolutionary point of view according to the “cis-regulatory hypothesis”. This theory predicts that because cis-regulatory mutations are likely to have reduced pleiotropic effects, they should lead to phenotypic evolution more frequently than mutations in coding regions (Stern and Orgogozo, 2008, 2009). A literature survey covering multicellular plants and animals provided support for the cis-regulatory hypothesis over long time scales (beyond the species level) (Stern and Orgogozo, 2008). In the harlequin ladybird beetle (Harmonia axyridis), allelic variation in the cis-regulatory region of the transcription factor gene pannier is responsible for over 200 distinct patterns of red and black on the elytra (the hardened forewing), providing a striking example of phenotypic variation caused by cis-regulatory variants at the intraspecific level (Ando et al., 2018; Gautier et al., 2018). In our previous work, we showed that the ARPC4 gene encoding an actin-related protein complex member controls the dynamics of the actin filament network with an impact on QDR against S. sclerotiorum (Badet et al., 2019). Two major alleles of ARPC4 were detected in A. thaliana populations, associated with contrasted levels of QDR. Remarkably, the two ARPC4 alleles encoded identical proteins, but differed markedly in their induction upon S. sclerotiorum inoculation. The Arp2/3 complex involving ARPC4 is conserved in all eukaryotes (Deeks and Hussey, 2005), indicating that transcriptional regulation of conserved pleiotropic genes can underlie variation in plant QDR phenotypes. Similarly, the POQR gene showed conserved expression pattern and function in QDR upon S. sclerotiorum challenge in A. thaliana and tomato (Badet et al., 2017). These results suggest that some QDR determinants are conserved between distantly related plants and that gene expression polymorphisms could be drivers of QDR evolution.
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*S. sclerotiorum* has one of the broadest host ranges among plant pathogens (Derbyshire et al., 2017; Navaud et al., 2018), providing a rare opportunity to analyze the diversity and long-term evolution of transcriptional programs in the interaction of various plants with a single fungal pathogen species. To document how diverse the local plant responses to *S. sclerotiorum* are at the molecular level, we analyzed the complete transcriptomes of six plant species spanning the Pentapetalae group inoculated with the same strain of *S. sclerotiorum*. Differentially expressed genes (DEGs) were enriched with core ortholog genes (conserved in all six species) but depleted from lineage-specific genes. Among core orthologs, patterns of gene expression varied significantly across species and deviated significantly from a null model of expression heritability. Only 4.34% of core orthogroups contained up-regulated genes from all six species, including a group of ABCG transporters. Functional analysis of *AtABCG40* supports a scenario of exaptation in which ancestral stress response genes gained a function in local disease resistance to *S. sclerotiorum* recently in plant evolution.

**RESULTS**

Common features of *S. sclerotiorum*-responsive gene pools across distant species

To document the transcriptional changes induced locally by *S. sclerotiorum* infection in taxonomically diverse eudicot plant species, we performed RNA-sequencing of healthy and inoculated leaves from six plant species, chosen to cover a broad diversity in the Pentapetalae group of angiosperms (Zeng et al., 2017) (Chanderbali et al., 2017). *Phaseolus vulgaris* (common bean, Order Fabales) and *Ricinus communis* (castor bean, Order Malpighiales) were chosen as representatives of the fabid group of the Rosids Clade and *Arabidopsis thaliana* (thale cress, Order Brassicales) as a representative of the malvid group of the Rosids Clade. *Helianthus annuus* (sunflower, Order Asterales) and *Solanum lycopersicum* (tomato, Order Solanales) were chosen as representatives of the Asterids Clade and *Beta vulgaris* (beetroot, Order Caryophyllales) as a representative of basal Superasteridae lineages (Zeng et al., 2017) (Fig. 1A, Supplemental Data Set 1.1). A reference genome is available for all these species, to which we aligned RNA-sequencing reads to quantify plant gene expression. In non-inoculated plants, a minimum of 90% of the total sequence reads were mapped to the corresponding plant reference genome, except for *R. communis* in which only 74.26% of sequence reads mapped to annotated transcripts.

With an aim to study comparable stages of infection on all six plant species, we harvested the edge of disease lesions before they reached 25mm diameter (Badet et al., 2017; Peyraud et al., 2019) (Supplemental Methods). As expected due to the presence of sequence reads corresponding to *S. sclerotiorum* RNAs, an average 32.5% of sequence reads mapped to their corresponding plant genome in infected samples (Fig. 1B, Supplemental Data Set 1.2). To estimate the degree of fungal colonization in these samples, we mapped RNA-sequencing reads to *S. sclerotiorum* reference genome (Derbyshire et al., 2017). Our results suggest that *S. sclerotiorum* colonization was consistent across replicates and across species, with an average 58.25 ± 4.83% of reads aligned to the fungal genome (Fig. 1B).
The number of genes expressed both in non-inoculated and infected leaves ranged from 19,353 (R. communis) to 42,351 (H. annuus), reaching a total 156,497 genes across the six plant species (Fig. 1C, Table 1, Supplemental Data Set 1.3). The proportion of expressed genes was remarkably consistent across all six plant species corresponding to 78.48 ± 2.47% of complete transcriptomes (Fig 1C).

To determine the number of plant genes mis-regulated upon S. sclerotiorum infection in each genome, we calculated log fold change (LFC) of expression in healthy and inoculated plant samples. We used LFCs shrinkage by the empirical Bayes approach implemented in DESeq2 to avoid bias in genes with low read coverage (Love et al., 2014) (Supplemental Methods). Genomic median for LFC ranged from -0.19 in A. thaliana to 0.07 in B. vulgaris (Fig. 1D, Supplemental Data Set 1.4-1.9). The distribution of LFC values were very similar between genomes, with a stronger dispersion from the median in Rosid species. We considered as differentially expressed genes (DEGs) showing a log2 fold change higher than 1.5 and a P-value lower than 0.01. We detected 6,148 DEGs in B. vulgaris, 6,843 in P. vulgaris, 7,510 in R. communis, 8,069 in S. lycopersicum, 9,844 in H. annuus and 10,496 in A. thaliana (Table 1, Supplemental Data Set 1.3). Although there was a 2.7 fold variation in total number of genes per genome (from 22,336 in R. communis to 58,138 in H. annuus), the number of DEGs varied by 1.7 fold only. DEGs represented an average 32.63 ± 4.17% of expressed genes (Fig. 1E). The lowest proportion of DEGs corresponded to species from the Superasteridae clade (average 26.7% ± 1.65), whereas species from the Rosids clade showed 38.6% ± 3.75 of DEGs in average. In all species, the proportion of down-regulated genes exceeded that of up-regulated genes (Student’s t test p-value= 0.0013), with an up-/down-regulated ratio of 0.82 ± 0.03 (Fig. 1E, Table 1). In average, expressed genes included 17.9% ± 2.27 down-regulated DEGs, 14.7% ± 1.93 up-regulated DEGs and 67.4% ± 4.17 genes not differentially expressed. These results highlight a number of common global features of S. sclerotiorum-responsive plant transcriptomes across distant plant lineages.

S. sclerotiorum-responsive genes are enriched with core orthologs

To compare local transcriptional responses to S. sclerotiorum in the six selected plant species, we performed sequence-based clustering of all predicted proteins from the six species using OrthoMCL (Li et al., 2003). This identified 14,983 orthogroups shared between two or more species and containing from 2 to 110 expressed genes, for a total of 97,763 genes (Fig. 2A, Table 1, Supplemental Data Set 1.10). 62,838 genes (40.2%) classified into 7,918 orthogroups we designated as ‘Pentapetalae Core’ that contained genes from all six-plant species and could represent ancestral gene groups. There were 58,726 expressed genes (37.5%) unique to one species and not assigned to orthogroups, classified as ‘lineage specific’ (see methods). The 7,065 ‘other ortholog groups’, shared between two to five species, included a total of 34,925 genes (22.3%) (Fig. 2A, Supplemental Data Set 1.11). The proportion of Pentapetalae Core genes represented 41.5 ± 2.9% per species, other ortholog genes represented 23.1 ± 2.1% per species (Fig. 2B, Table 1, Supplemental Data Set 1.12). The proportion of lineage specific genes was more variable ranging from 23.8% in R. communis to 48.1% in H. annuus (average 35.6 ± 4.8%).
To test for the relative contribution of conserved genes in the plant transcriptional responses to *S. sclerotiorum*, we calculated for each species the proportion of DEGs that were core Pentapetalae, other ortholog, and lineage specific genes (Fig. 2C). On average, 46.7% ± 2.6 of DEGs were Core Pentapetalae genes, 24.7% ± 1.7 were other core ortholog genes, 28.6% ± 3.6 were lineage-specific genes. The percentage Core Pentapetalae in DEGs was significantly higher than that of other core ortholog genes (Student t test p-value= 4.2e−08) and that of lineage-specific genes (p-value= 2.3e−06). The proportion of up- and down-regulated genes was similar among the ‘other core ortholog’ and ‘lineage specific’ classes. It showed a slight difference among Core Pentapetalae genes in which 43.15% ± 1.7 were up-regulated and 49.63% ± 3.6 were down-regulated (p-value= 0.058). The percentage of DEGs being lineage specific showed the strongest variation between species, especially regarding down-regulated genes that included 17.6% of lineage-specific genes in *P. vulgaris* and up to 42.1% in *H. annuus*.

To determine whether the prevalence of Core Pentapetalae genes in DEGs was conserved at different phylogenetic levels, we analyzed separately the distribution of DEGs in the Rosids and Superasteridae clades (Fig 2D, Supplemental Data Set 1.13). In both clades, DEGs were significantly more abundant in Core Pentapetalae genes than in any other class (p-value<5.5e−10 for Rosids, p-value<0.005 for Superasteridae). In the Rosids, other core orthologs and lineage-specific genes contributed to a similarly low proportion of DEGs (in average 26.9% and 22.9% respectively, p-value= 0.14). By contrast, in the Superasteridae, DEGs were significantly less abundant among other orthologs than among lineage-specific genes (in average 22.7% and 34.7% respectively, p-value= 7.0e−05). As a complementary analysis, we used chi-squared tests to show that Core Pentapetalae genes were significantly enriched with DEGs in all six species, between 1.041 fold (p-val 1.74e−04) in *R. communis* and 1.196 fold (p-val 3.11e−46) in *B. vulgaris* (Table 2, Supplemental Data Set 1.13). Other ortholog genes were significantly enriched with DEGs in Superasterids species only, between 1.129 fold (p-val 6.91e−12) in *S. lycopersicum* and 1.157 fold (p-val 3.97e−17) in *H. annuus*. Conversely, lineage specific genes were depleted with DEGs in all six species, between 0.751 fold (p-val 5.46e−81) in *B. vulgaris* and 0.885 (p-val 9.79e−12) in *R. communis*.

We conclude that the plant transcriptome is enriched with core ortholog genes during local responses to *S. sclerotiorum*. Because of a relatively high proportion of lineage-specific genes differentially regulated in species from the Superasteridae clade, this trend is less pronounced in the Superasteridae than in Rosids. Comparing the transcriptome of resistant and susceptible genotypes at the species level, together with further functional assays, will be required to determine which of these responsive genes contribute to the QDR phenotype.

**Accumulation of *S. sclerotiorum*-induced genes during the evolution of the Pentapetalae**

To document global patterns of evolution in the repertoire of *S. sclerotiorum*-responsive genes across plants, we inferred gene gains and losses along the Pentapetalae phylogeny. Following a simple parsimony hypothesis, we considered a gain along a branch of the tree when genes were present in all derived clades (among ortholog groups or lineage-specific)
but not in three or more paraphyletic clades (Fig. 3A, Supplemental Data Set 1.14-1.15).

Reciprocally, we considered a loss along a branch when genes were present in all paraphyletic clades but absent from all derived clades. This identified a total of 5,131 loss events associated with 10,143 DEGs and 10,630 gain events associated with 16,046 DEGs.

Considering Pentapetalae Core genes as the ancestral state, we calculated the change in gene content along each branch of the tree relative to the ancestral state (Fig. 3B). There were 3,148 orthogroups (including 4,083 DEGs) that could not be associated unambiguously to one branch of the tree and were excluded from this analysis. We used estimated dates of divergence in the Pentapetalae phylogeny (Zeng et al., 2017) (Fig. 3B) to calculate the proportion of genes gained and lost per million year (Mya) along branches of the tree. Except the two most ancestral branches (Pentapetalae to Superasteridae and Pentapetalae to Rosids), all branches showed higher rates of gene gain than gene losses. Along intermediate and terminal branches of the tree, whole genome losses were 0.95 ± 0.53%/Mya and whole genome gains 0.35 ± 0.21%/Mya (paired t test p-val=0.012) (Fig. 3C, Supplemental Data Set 1.15). For DEGs, losses were 0.12 ± 0.06%/Mya and gains were 0.25 ± 0.12% (p-val=0.009), indicating that S. sclerotiorum-responsive gene pools were less variable than whole genomes and that all lineages accumulated DEGs since ~120 Mya.

Next, we calculated the ratio between the number of genes up-regulated and down-regulated upon S. sclerotiorum inoculation among orthogroups containing among genes gained and lost along intermediate and terminal branches of the phylogeny (Fig. 3A, D, Supplemental Data Set 1.15). The up/down ratio was ~1.08 ± 0.20 in gained DEGs but only ~0.80 ± 0.12 in lost DEGs (paired t test p-val=0.0098), suggesting a trend to favor the retention of up-regulated genes rather than down-regulated genes. In agreement, the up/down ratio was 0.72 in Core Pentapetalae genes and ~0.82 ± 0.02 in average in the six plant species. These analyses suggest the existence of mechanisms leading to the progressive accumulation of genes up-regulated upon S. sclerotiorum inoculation during the evolution of Pentapetalae plants.

Local transcriptomic responses to S. sclerotiorum involve core and lineage-specific functions

To document the functional diversity of genes differentially expressed upon S. sclerotiorum inoculation, we collected Gene Ontology (GO) and PFAM domains annotations for the six plant species. Between 47.1% (H. annuus) and 74.1% (A. thaliana) of expressed genes were annotated with GO, and between 76.1% (H. annuus) and 89.7% (A. thaliana) of expressed genes were annotated with at least one Pfam domain per species (Table 1, Supplemental Data Set 1.16-1.18, Supplemental Files 1 and 2). These annotations included between 2,381 (B. vulgaris) and 3,792 (A. thaliana) distinct GOs and between 4,128 (R. communis) and 5,913 (H. annuus) distinct PFAMs represented at least three times (Table 1). To highlight gene functions associated with the local response to S. sclerotiorum, we analyzed GO and PFAM annotation enrichment with upregulated and downregulated genes from each species. We mapped GOs to enriched PFAM domains to summarize annotations as GOs. We found 262 GOs significantly enriched with upregulated genes (chi-squared adjusted p-
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val<0.01) in at least one species, including 103 “Biological Process” ontologies, 28 “Cellular Component” ontologies and 131 “Molecular Function” ontologies (Fig 4A, Supplemental Data Set 1.19). Six GOs (4.65%) were enriched with upregulated genes in all species including protein phosphorylation and kinase activities (GO:0006468, GO:0004674, GO:0004672). Two hundred and four GOs were enriched with upregulated genes in a single species, revealing the prevalence of abscisic acid signaling (GO:0009738) in R. communis and jasmonic acid signaling (GO:0009753, GO:0016629) in A. thaliana and P. vulgaris. These GOs also point towards lineage-prevalent defense mechanisms, such as the biosynthesis of terpenes (GO:0010333) and inhibition of endopeptidases (GO:0004866) in S. lycopersicum, flavonoid glucuronidation (GO:0052696) in H. annuus, vitamin B6 metabolism (GO:0042816) and ceramide biosynthesis (GO:0046513) in P. vulgaris. Fifty-two GOs were enriched with upregulated genes in two to five species, including defense response (GO: 0006952), response to wounding (GO:0009611), exocytosis (GO:0006887), response to chitin (GO:0010200), toxin catabolic process (GO:0009407) and flavonoid biosynthesis (GO:0009813). We found 411 GOs significantly enriched with downregulated genes (chi-squared adjusted p-val<0.01) in at least one species, including 187 “Biological Process” ontologies, 57 “Cellular Component” ontologies and 167 “Molecular Function” ontologies (Fig 4B, Supplemental Data Set 1.19). Fifteen GOs were enriched with downregulated genes in all species, mostly related to the photosynthesis process (GO:0015979, GO:009654, GO:009522, GO:009507, GO:0016168). Three hundred forty GOs were enriched with downregulated genes in a single species, such as response to auxin (GO:0009733) in B. vulgaris, amine metabolism (GO:0009038) in H. annuus. GOs enriched with downregulated genes also included several GOs related to RNA processing (GO:0009451, GO:0001510), the regulation of protein translation (GO:0006400, GO:0006417) and DNA maintenance (GO:0004519, GO:0006298, GO:0006260, GO:0006508) in several species. Thirty-one GOs were enriched both with up- and down-regulated, leading to a total 642 GOs enriched with DEGs.

To visualize hierarchical relationships between GOs enriched with DEGs, we constructed a network of biological process ontologies in which nodes are color-coded according to enrichment with up- or down-regulated genes (Fig 4C). Biological Process ontologies harboring an excess of upregulated over down-regulated genes notably included “response to toxin” (up/down ratio 2.19), “response to chitin” (up/down ratio 5.80), “response to wounding” (up/down ratio 2.14) and “cell surface receptor signaling” (up/down ratio 1.19) (Fig. 4C). Ontologies with an excess of upregulated genes tended to cluster in defined sectors of the GO network related to secondary metabolism and detoxification on one side, signaling and response to stress on the other side (Fig. 4C). Biological Process ontologies harboring an excess of down-regulated over upregulated genes included “response to light stimulus” (up/down ratio 0.25), “starch biosynthetic process” (up/down ratio 0.13) and “RNA methylation” (up/down ratio 0.03). DEG-enriched ontologies spanned a broad range of the GO network, highlighting the diversity of processes involved in the response to S. sclerotiorum.
To study the relationship between conservation and regulation among major gene functions responsive to *S. sclerotiorum* infection, we classified gene ontologies according to their content in core genes and their content in upregulated genes, focusing on the 282 Biological Process GOs enriched in DEGs. To this end, we determined the ratio between the number of Pentapetalae core genes and the number of lineage-specific DEGs as well as the ratio between the number of upregulated and down-regulated DEGs for all GOs (Supplemental Data Set 1.19). For instance, ontology “toxin catabolic process” (GO:0009407) was identified in 71 upregulated genes, 12 down-regulated genes and 51 genes not differentially regulated (Fig 5A, up/down ratio 5.92), including 23 core Pentapetalae DEGs and 21 lineage-specific genes (core/LS ratio 1.09). Ontology “chlorophyll biosynthesis process” (GO:0015995) was identified in 79 down-regulated genes, 11 upregulated genes and 48 genes not differentially regulated across six species (Fig 5B, up/down ratio 0.14), including 76 core Pentapetalae DEGs and 9 lineage-specific DEGs (core/LS ratio 15.2). Fifty-one GOs did not contain upregulated or core genes and were excluded from the analysis. We observed a correlation between the content in Pentapetalae core genes and the content in down-regulated genes (Spearman’s ρ = 0.325, p=4.41e-0.7), suggesting a high degree of conservation or convergence in gene functions down-regulated upon *S. sclerotiorum* infection (Fig 5C). A number of ontologies were exceptions to this trend, harboring a high content in core genes and high content in upregulated genes, such as callose deposition (GO:00052542), chorismate biosynthesis (GO:0009423), ceramide biosynthesis (GO:0046513), hypersensitive response (GO:0009626) and systemic acquired resistance (GO:0009862). Gene families lacking annotation, not covered by this analysis, may also contribute to the repertoire of genes responsive to *S. sclerotiorum*.

The transcriptional response of core genes to *S. sclerotiorum* varies significantly between species

We found only a few GOs enriched with DEGs in all species (Fig. 4A-B). This could be due to (i) GOs enriched with DEGs being weakly conserved across species, or to (ii) conserved GOs harboring variable content in DEGs across species. To test for the second alternative, we analyzed the distribution of DEGs among genes, ontologies and PFAM domains conserved in all six species. Genes, GOs and PFAMs retrieved in a maximum of five species were excluded from the following analyses. The Core Pentapetalae class includes 62,838 genes conserved in all six species and distributed in 7,918 orthogroups of 6 to 132 genes. Among those, 9,520 genes (15.2%) were up-regulated upon *S. sclerotiorum* infection, distributed between 3,659 orthogroups (46.2%). This included 159 orthogroups (2.01%) containing up-regulated genes from all six plant species (Fig. 6A, Supplemental Data Set 1.11, 1.21). There was 13,201 Pentapetalae core genes down-regulated (21.0%), distributed in 4,962 orthogroups (62.7%), including 282 orthogroups (3.56%) containing down-regulated genes from all six plant species. Overall, 1,039 Pentapetalae core groups did not contain DEGs (13.1%), 438 contained DEGs in all six plant species (5.53%), and 6,441 contained DEGs in one to five plant species (81.3%) (Fig. 6A, Supplemental Figure 1-2), indicating that a majority of conserved gene groups showed divergent regulation upon *S. sclerotiorum* inoculation in distinct plant species.
To test whether this observation holds true for conserved gene functions, we conducted a comparative analysis of GOs and PFAM domains expressed in all six species (corresponding to 3,536 GOs and 5,919 Pfam domains, Fig. 6B-C, Supplemental Data Set 1.22-1.23). We found that 71.5% of GOs and 64.5% of PFAM domains were associated with DEGs from one to five plant species. In agreement with frequent species-specific regulatory patterns for conserved genes, although detected in all six plant species, only 23% of GOs and 33.6% of Pfam domains were associated with DEGs in all six plant species (Fig. 6B, C).

To characterize species-specific regulation across gene ontologies, we color-coded GO networks according to the number of plant species in which they are associated with upregulated genes (0 to 6) (Fig. 6D). Ontologies associated with upregulated genes in five or six species where highly connected at the center of the network. Exceptions included the ontologies “ATP transport”, “Negative regulation of apoptosis”, “vacuolar acidification”, “Lipid homeostasis”, “Cysteine biosynthesis from serine”, “removal of superoxide radicals” and “negative regulation of endopeptidase” that were terminal nodes of the network associated with upregulated genes in five or six species. This prompted us to test for a relationship between association with upregulated genes in multiple species and the level of GOs in the ontology hierarchy. We found that the number of species in which GOs associate with upregulated genes decreased with ranks in the GO hierarchy, from an average rank 12.96 for GOs associated with upregulated genes in all six plant species to an average rank 17.66 for GOs associated with upregulated genes in a single species (Student’s t-test p-value 3.7e-09) (Fig. 6E, Supplemental Data Set 1.22). This highlights significant variations in genes and biological functions regulated locally upon S. sclerotiorum inoculation across diverse plant species.

To evaluate further the divergence in biological functions regulated upon S. sclerotiorum inoculation across six plant species, we analyzed 893 GOs represented in all six species and including at least one DEG and one Pentapetalae core gene in each species. As a reference, we calculated pairwise correlations for GOs content in Pentapetalae core genes between plant species. We found an average Spearman ρ 0.601 ± 0.078 (Fig. 6F, Supplemental Data Set 1.24). We then calculated pairwise correlations for GOs content in DEGs between plant species and found an average Spearman ρ 0.370 ± 0.064 (Fig. 6G, Supplemental Data Set 1.24). Next, we analyzed the top 100 GOs enriched in DEGs in each plant species. In total, the selection included 410 GOs for the six species, indicated a limited overlap in individual top 100 lists. Indeed, only seven GOs related to the down-regulation of photosynthesis belonged to the top 100 enriched in DEGs in all six species (Fig. 6H, Supplemental Data Set 1.25). Forty-four (S. lycopersicum) to 64 (P. vulgaris) GOs were part of the top 100 enriched in DEGs in a single species, including for instance “structural constituent of cytoskeleton” in A. thaliana, “carotenoid biosynthesis” in B. vulgaris, “flavonoid biosynthesis” in H. annuus and “glucosylceramidase activity” in P. vulgaris. For comparison purposes, we analyzed top 100 GOs depleted in DEGs in each plant species (Fig. 6I, Supplemental Data Set 1.25-1.26). Eleven GOs were highly depleted in DEGs in all six species, between 14 (H. annuus) to 38 (A. thaliana and P. vulgaris) GOs were highly depleted in GOs in a single species. This supports...
the view that the repertoire of core gene ontologies enriched with DEGs is rather diverse at
the interspecific level.

**Patterns of core genes expression upon S. sclerotiorum inoculation deviate significantly
from a null model of heritability**

Out of 3,659 Pentapetalae core orthogroups including upregulated genes, only 159 (4.3%)
included upregulated genes from all six species analyzed (Supplemental Data Set 1.21),
suggesting a high degree of regulatory divergence at the interspecific level. Since genes from
a given core Pentapetalae orthogroup most likely derive from a common ancestor, we
considered the null hypothesis that their expression pattern is completely inherited, leading
to similar expression patterns within orthogroups. We compared observed distributions of
gene expression upon S. sclerotiorum inoculation to modelled distributions to test this
hypothesis and estimate the degree of regulatory variation within Core Pentapetalae genes.

First, we analyzed the distribution of the number of species harboring up-regulated genes in
each Pentapetalae core orthogroup. For this, we shuffled 100 times Core Pentapetalae genes
among orthogroups. Under the null hypothesis of complete inheritance, upregulation is
inherited in 6 species. We conducted our boot strap analysis by constraining identical
expression (up-regulated or not) in 6 species within an ortholog group (h=6) to test this
hypothesis. We next considered relaxed degrees of expression pattern inheritance (h=5 to
2), down to a complete random assignment of expression patterns (h=1) (Fig. 7A,
Supplemental Data Set 1.27, Supplemental File 3). We used the sum of squared residuals
(SSR) to estimate the deviation between observed and simulated distributions. The lowest
SSR value (38.0) is obtained when gene expression is constrained as identical in two species
only (h=2). A similar analysis on the distribution of down-regulated genes also identified h=2
as the best approximation to observed distribution. This suggests that constraints on core
genes upregulation are weak, allowing switches between up-regulated and not up-regulated
states in 4 out of 6 species in average.

Second, we clustered genes based on their LFC using the Silhouette method (Charrad et al.,
2014) within each orthogroup, and we determined the size of the largest expression cluster
relative to the size of each orthogroup (Supplemental Data Set 1, Supplemental File 4).
Under the null hypothesis of complete inheritance, gene expression patterns should be
highly consistent within each Pentapetalae core orthogroup, and clusters should tend to
cover complete orthogroups. By contrast, under the hypothesis that genes forming an
orthogroup are fully independent (expression patterns are randomly distributed), expression
clusters would correspond to one single gene. To determine the extent to which genes
expression departs from independence in each Pentapetalae core orthogroup, we
computed a “consistency of gene expression index” (CEI) corresponding to the difference
between the relative size of the largest expression cluster observed, and the relative size of
the largest theoretical expression cluster under gene independence (Supplemental Data Set
1.28, Fig 7B). For instance, orthogroup #56 containing 50 genes (theoretical expression
cluster size under gene independence was 1/50) included seventeen expression clusters, the
largest of which contained six genes (12% of the orthogroup, CEI = 0.12 – 0.02 = 0.1) (Fig.
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In our dataset, CEI ranged from 0.0602 (92.8% of genes showed independent expression pattern) to 0.8824 (94.1% of genes showed consistent expression pattern). The CEI average was 0.46, meaning that in average about half of the genes composing a core orthogroup had consistent patterns of expression (Fig 7B). There were only 831 orthogroups (10.5%) with CEI ≥ 0.67 (largest expression cluster including at least 67.42% of genes in the orthogroup). One of them is orthogroup #1067 including thirteen genes distributed into two expression clusters, the largest containing ten genes (CEI = 0.692) (Fig. 7D). There were 2,500 orthogroups (31.6%) with CEI ≤ 0.33 (largest expression cluster including less than 34.1% of genes in the orthogroup). We conclude that core orthogroups with weakly consistent expression patterns are abundant, suggesting that expression consistency in weakly constrained in many orthogroups. Our bootstrap and clustering approaches both lead to the conclusion that the expression of core genes upon *S. sclerotiorum* inoculation is only weakly heritable at the interspecific level.

**Evidence for exaptation into QDR in a group of ABCG/PDR transporters**

To illustrate regulatory divergence in response to *S. sclerotiorum* at the interspecific level, we focused on the orthogroup #4. This group is remarkable for including up-regulated genes in each of the six plant species, a property restricted to 2.01% of the Pentapetalae core class. Gene expression log fold change (LFC) ranges from -6.2 (XM_015728348.1) to 12.7 (PHASIBEAM10F011181T1) in orthogroup #4, ranking it 14/15,685 for intragroup LFC variation. Orthogroup #4 contains 96 expressed genes encoding “class G” ABC transporters (“pleotropic drug resistance” or PDR class), among which 28 are upregulated and 14 are down-regulated upon *S. sclerotiorum* inoculation (Fig. 8A, Supplemental Data Set 1.29, Supplemental File 5). A phylogenetic analysis of orthogroup #4 reveals six monophyletic clades each containing genes from all six plant species, suggesting the divergence of these clades early in land plants, in agreement with previous reports (Hwang et al., 2016a). Upregulated genes were restricted to one species in clade 1 and 3, three species in clade 2 and four species in clade 4. Clade 5 did not include upregulated genes while clade 6 contained upregulated genes from each of the six plant species, including *A. thaliana* ABCG40/PDR12 (AT1G15520, LFC 12.48) (Fig. 8A).

Because the six plant lineages analyzed here diverged before the emergence of the *Sclerotinia* genus (Navaud et al., 2018), we hypothesized that the ABCG clade induced in all species were exapted into QDR (Gould and Vrba, 1982). In this evolutionary scenario, an ancestor of ABCG clade 6 would have been responsive to signals of the ancestral environment, in which the *Sclerotinia* genus had not emerged yet, and underwent regulatory and functional changes associated with enhanced QDR against *S. sclerotiorum*. Because of the extensive resources available for this plant, we focused on *A. thaliana* Clade 6 gene ABCG40 to test this exaptation scenario. For this, we first analyzed the expression of *A. thaliana* ABCG genes from orthogroup #4 in twelve stress conditions reported in the literature, including challenge by the fungal pathogens *S. sclerotiorum*, *B. cinerea*, *Alternaria brassicicola*, *Verticillium dahlia* and *Colletotrichum incanum*. The other treatment surveyed covered inoculation by the fungal endophyte *C. tofeldiae*, inoculation by avirulent (carrying
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the *AvrRPS4* avirulence gene) and virulent (DC3000 cor-) strains of the bacterial pathogen *Pseudomonas syringae* pv. tomato (*Pst*), inoculation by the nematode *Heterodera schachtii*, inoculation by the cabbage leaf curl virus (CalCuV) and upon heat and salt stress ([Fig. 8B, Supplemental Data Set 1.30]). Six *ABCG* genes were significantly induced by two or more stresses. Salt treatment, inoculation by *C. tofeldiae* endophytic fungus, the virulent *Pst* DC3000 cor- strain, *H. schachtii* nematodes and CalCuV did not significantly induce any of the *ABCG* genes tested. The *ABCG40/PDR12* gene was significantly induced by the broadest range of treatments (6/12), including fungal pathogens *S. sclerotiorum*, *B. cinerea* (LFC 8.08), *A. brassicicola* (LFC 4.15), *V. dahliae* (LFC 1.58), avirulent *Pst* (LFC 9.04) and heat (LFC 8.16).

Second, we analyzed the disease resistance phenotype of four T-DNA mutant lines altered in the *AtABCG40* gene. To characterize mutant lines at the transcript level, we first assessed *AtABCG40* expression in healthy plants ([Fig. 8C, Supplemental Data Set 1.31]). The *abcg40-2* line (SALK_005635) (Campbell et al., 2003) was strongly impaired in *ABCG40* expression (<4% of wild type expression, p-val=1.4e⁻⁴), while *ABCG40* was only weakly reduced in *abcg40-3* (SAIL_885_009) and *abcg40-4* lines (SALK_148565) (~62% and ~54% of wild type respectively), and *abcg40-5* (SALK_013945) showed an increased accumulation of *ABCG40* transcripts (~165% of wild type). We inoculated leaves of these plants with a GFP-expressing strain of *S. sclerotiorum* to measure the area colonized by the fungus 24 hours after inoculation, using the *rlp30-1* mutant (Zhang et al., 2013) as a susceptible control ([Fig. 8D]). A strong genotype effect with no significant experiment replicate effect were detected on lesion size (ANOVA P-value 9.81e⁻⁶ and 0.05 respectively). Average area colonized by *S. sclerotiorum* was 85.0 ± 9.7 mm² in Col-0 and 123.7 ± 16.9 mm² in *rlp30-1* (Student’s t test adjusted p-val = 9.9e⁻⁵, [Fig. 8E, Supplemental Data Set 1.32]). Decrease in *ABCG40* expression was associated with an increase in susceptibility in *abcg40-3* (108.1 ± 13.0 mm² colonized, adj. p-val 0.02), and *abcg40-2* (130.5 ± 17.4 mm² colonized, adj. p-val 2.5e⁻⁵). The *abcg40-4* and *abcg40-5* mutants were weakly but not significantly impaired in resistance to *S. sclerotiorum* (106.5 ± 10.1 and 98.4 ± 9.7 mm² colonized respectively, adj. p-val > 0.1), suggesting that *ABCG40* transcripts may be functional in these lines. Overall, these results demonstrate a role for *AtABCG40* in disease resistance to *S. sclerotiorum* and suggest that *AtABCG40* was exapted into QDR from an ancestral stress response function through reinforcement of its transcription upon fungal pathogen challenge.

**DISCUSSION**

Our phylotranscriptomics analyses of local responses to *S. sclerotiorum* in plants from six Pentapetalae orders provide an unprecedented insight into the broad diversity of transcriptional reprogramming upon challenge with a single fungal pathogen strain. Comparative transcriptomics studies of plant interactions with eukaryotic pathogens typically focused on gene reprogramming in closely related plant cultivars (Zhao et al., 2009) and species (Powell et al., 2017), and on the transcriptome of several parasite strains (Cooke et al., 2012; Palma-Guerrero et al., 2016; Zhang et al., 2017) or species (Hacquard et al., 2016) infecting a given plant genotype. The remarkably wide range of plants that *S.
sclerotiorum can infect allowed us to document molecular responses to this fungus in host species that diverged over 140 million years ago.

**An estimate of the pan-genomic diversity of S. sclerotiorum responsive genes**

Recent sequencing efforts defined the complement of NLR genes across *A. thaliana* accessions (Van de Weyer et al., 2019). Here, we provide an estimate of the QDR complement responding to *S. sclerotiorum* locally in the Pentapetalae. Together, we identified 48,910 genes differentially expressed upon *S. sclerotiorum* across six species, enriched in 642 gene ontologies. Despite their evolutionary distance and strong variation in genome size, all species analyzed in this work upregulated ~15% of their genome and downregulated ~18% of their genome upon infection, suggesting the existence of a conserved optimal pool of *S. sclerotiorum*-responsive transcripts. In average, we found that ~47% of genes differentially expressed in a species upon *S. sclerotiorum* inoculation were conserved in the five others (core QDR orthogroups) while ~38% were specific to this species. Similarly, the core NLR orthogroups, conserved in >50 out of 65 accessions, represent 53% of NLR genes (Van de Weyer et al., 2019). This suggests that a significant fraction of *S. sclerotiorum*-responsive pan-transcriptome is likely to be functional in multiple plant species, as shown for instance for the POQR putative peptidase in *A. thaliana* and tomato (Badet et al., 2017). In this context, functional analyses of QDR genes in model species may offer relatively straightforward perspectives for improving QDR in crops.

Experimental evidence for a role in QDR is only available for a limited number of DEGs. Nevertheless, a diverse set of *A. thaliana* DEGs were shown to function in resistance against *S. sclerotiorum* (Table 3). According to the omnigenic model (Boyle et al., 2017), any DEG in our pan-transcriptome is expected to impact the quantitative disease resistance phenotype. Our work therefore suggests that about 32.4% of plant genes could have a non-null contribution to QDR against *S. sclerotiorum*. Because network connectivity is an important determinant of gene essentiality and conservation, it is tempting to speculate that core genes in the omnigenic model, that directly and strongly affect disease, would likely be among the 43% DEGs broadly conserved across species. Species-specific DEGs would modulate core genes activity according to genetic backgrounds, pathogen genotypes and other environmental variables and may underlie the frequent variation in core genes regulation that we observed. Our enrichment analyses are consistent with a set of genes that could directly control QDR through their protective or antifungal activity (e.g. terpene and flavonoid biosynthesis genes, endopeptidase inhibitors, toxin catabolism genes), the activity of which could be modulated by numerous transcriptional and post-transcriptional regulators (e.g. WRKY DNA-binding, ERF/AP2-type transcription factor and kinase domain proteins). We identified several DEGs encoding proteins with domains of unknown function (DUF2870, DUF969, DUF3403) representing promising candidates for novel defense proteins. Studies with *B. cinerea* in *A. thaliana* and tomato indicated that genes associated with QDR varied according to the fungal strain being inoculated (Corwin et al., 2016; Soltis et al., 2019), suggesting that the pan-genomic repertoire of QDR-associated transcripts may depend on pathogen genotypes and species, and may exceed current estimates.
Molecular bases of regulatory variation and QDR phenotype evolution

Although 47% of DEGs belonged to core orthogroups conserved in six plant species, only 2.01% of core orthogroups contained genes upregulated in six species (and 3.56% genes down-regulated in six species), highlighting frequent regulatory variation within orthogroups. In our previous work, we identified allelic variants of the ARPC4 gene differing in their promoter region, induction upon pathogen challenge, and associated with resistance to *S. sclerotiorum* (Badet et al., 2019). These findings are in line with a prominent role of regulatory variation in the evolution of QDR. It is also consistent with the alteration of host gene transcription being a major function of small RNA secreted by several fungal pathogens (Weiberg et al., 2013; Derbyshire et al., 2019). Species-specific gene expression patterns may result from directional selection on gene regulation or specific environmental influences (Romero et al., 2012). Recent progress in genomics allowed disentangling genetic and environmental factors in a number of cases, pointing towards several molecular mechanisms that may be responsible for expression polymorphisms in QDR genes. Molecular mechanisms underlying the evolution of gene expression include variations in epigenetic marks, gene copy number variation, and cis- and trans-regulatory variation.

Epigenetic marks such as DNA methylation control *A. thaliana* stress-responsive gene expression upon challenge by bacterial and fungal pathogens (Yu et al., 2013; Le et al., 2014). Histone acetylation, methylation and ubiquitination and the activity of chromatin remodeling complexes are other epigenetic mechanisms involved in the control of plant defense genes transcription (Ramirez-prado et al., 2018). Transcription is also controlled by the effect of cis-regulatory elements and trans-acting factors which may drive phenotypic evolution. For instance, the homeobox transcription factor RCO underwent regulatory changes, gene duplication and loss leading to leaf shape diversity among *Brassicaceae* species (Vlad et al., 2014). Cis-regulatory variation results from the emergence of novel enhancer sequences (Long et al., 2016) or from genome shuffling recruiting new genes to transcriptionally active regions. In both cases, whole genome duplications and transposable elements (TEs) can play prominent roles. Insertion of TE acting as an enhancer of the teosinte branched (*tb1*) transcription factor gene was a key molecular event in the acquisition of apical dominance during maize domestication (Studer et al., 2011). In some *A. thaliana* accessions, insertion of a TE-derived enhancer mediated WRKY33 binding to the promoter of the *CYP82C2* gene, making it inducible upon bacterial pathogen inoculation, activating biosynthesis of the antimicrobial 4-hydroxyindole-3-carbonylnitrile metabolite (Barco et al., 2019). A similar mechanism could have driven the emergence of a *S. sclerotiorum*-inducible allele of ARPC4 associated with enhanced QDR in *A. thaliana* (Badet et al., 2019). The pangenomic repertoire of QDR transcripts conserved across species but differentially regulated upon *S. sclerotiorum* inoculation provide an original resource for the identification of new transcription enhancer motifs. Since transcription factors often regulate multiple target genes, while cis-regulatory motifs act rather locally in the genome, cis-regulatory regions are considered less pleiotropic and therefore more likely to contribute to phenotypic evolution (Wray, 2007). Detailed promoter sequence analyses and cross-species functional validations will be required to estimate the relative contribution of cis- and trans-regulatory divergence in the evolution of QDR. The central role of gene expression
in QDR phenotypes suggests that engineering the expression or copy number of endogenous QDR genes, rather than introgressing new genes or alleles, could be an efficient strategy to control diseases in the field.

An exaptation scenario for the recruitment of genes into QDR

Under the Red Queen hypothesis, plant immunity genes represent true adaptations, evolved under selection pressure associated with specific pathogen populations (Van Valen, 1973). In agreement, signatures of diversifying selection were detected in Arabidopsis and Solanum NLR genes (Mondragón-Palomino et al., 2017; Stam et al., 2019). In natural populations of A. thaliana, polymorphism at the GS-ELONG locus controlling the structure of aliphatic glucosinolate defense compounds were associated with the presence of specific aphid species in the environment (Züst et al., 2012). In a selection experiments, aphids species drove selection for contrasted glucosinolate profiles in five plant generations, demonstrating that herbivores drive the rapid evolution of some plant defense genes. Alternatively, plant genes could have emerged prior to exposure to pathogens to serve non-immune functions, before being exapted into defense with the rise of pathogen populations.

The glucosinolate 3-hydroxypropylglucosinolate (3OHPGSL) produced by Brassicaceae plants has a role in defense and inhibits root growth in diverse dicot species (Malinovsky et al., 2017). Responses to 3OHPGSL involve the TOR (Target of Rapamycin) pathway conserved across eukaryotes, suggesting that the TOR pathway was exapted into glucosinolate response early in plant evolution. Similarly, A. thaliana CYP82C2 gene originated from the duplication the iron-stress response gene CYP82C4 and was exapted into defense through cis-regulatory variation (Barco et al., 2019).

We identified 159 orthogroups featuring genes upregulated by S. sclerotiorum in all six plant lineages, which diverged long before the emergence of the Sclerotinia genus (Navaud et al., 2018). For these genes, responsiveness to S. sclerotiorum likely evolved convergently in multiple plant lineages, through exaptation of ancient stress responses, or a combination of both processes. Here, we provide evidence for exaptation of the ABCG40/PDR12 transporter into QDR against S. sclerotiorum. The indirect pathway for Abscisic acid (ABA) synthesis probably evolved with the colonization of land (Hauser et al., 2011), similar to the ABCG/PDR class of transporters (Hwang et al., 2016b). It is therefore probable that the ABA importer function of ABCG40/PDR12 (Kang et al., 2010) evolved with the colonization of land, about 450 Mya, to control drought tolerance, seed germination and lateral root formation (Kang et al., 2010; Campbell et al., 2003). Consistent with an ancestral role in acclimation to abiotic stress, AtABCG40 was induced upon heat stress and down-regulated by salt stress (Suzuki et al., 2016). AtABCG40 is also induced upon inoculation by the fungal pathogens A. brassicicola, Fusarium oxysporum, and more strongly and more rapidly by S. sclerotiorum (Campbell et al., 2003). In line with suppression of ABA synthesis leading to susceptibility to S. sclerotiorum (Perche pied et al., 2010; Zhou et al., 2015; Mbengue et al., 2016), mutants defective in AtABCG40 were more susceptible to S. sclerotiorum than wild type. Exaptation of AtABCG40 into QDR against S. sclerotiorum associates with strong transcriptional activation of the gene upon challenge with S. sclerotiorum, through a mechanism that remains to be elucidated.
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**Broad spectrum and durability in the context of exaptation into resistance**

The evolution of QDR genes through exaptation of ancestral stress responses and developmental pathways could be related to QDR often being relatively broad spectrum and durable (Roux et al., 2014; Poland et al., 2009; Corwin and Kliebenstein, 2017). Signals created by pathogen attack on plants are either molecular signals, that can be specific of the pathogen genotype such as pathogen effectors (Guo et al., 2018; Guyon et al., 2014), or abiotic signals. Abiotic signals generated by pathogens during host colonization include altered pH (Xu et al., 2015; Fernandes et al., 2017), water status (Xin et al., 2016; Aung et al., 2018), physical strain (Wilson and Talbot, 2009). Since similar abiotic signals may be generated by diverse pathogens, as for alkalination by *Pseudomonas* phytotoxins (Bender et al., 1999) and by *Fusarium* RALF-like peptides (Masachis et al., 2016), plant responses mediating acclimation to these signals are likely to be exapted into QDR and to function against a broad spectrum of pathogens. A rapid transcriptional reprogramming was proposed to be critical for broad-spectrum efficiency of plant immune responses, supporting a central role of cis-regulatory divergence in adaptation to pathogens (Mine et al., 2018). In pathogens were single effector molecules are major determinants of the disease outcome, single amino acid substitution, gene presence absence and expression polymorphism are frequent mechanisms associated with evasion of recognition by the plant immune system (Raffaele and Kamoun, 2012). Abiotic signals are not directly encoded by pathogen genomes, therefore evasion of the corresponding plant responses may require very complex genetic alterations to pathogen genomes.

Upon escape from adaptive conflict, genes with exapted function may retain their ancestral functions (Conant and Wolfe, 2008). In some animal lineages, structural proteins forming the ocular lens retained their ancestral argininosuccinate lyase enzymatic activity (Platigorsky et al., 1988). A class of fungal dicarboxylic acid transporters were shown to have retained their ancestral activity and gained the ability to transport tricarboxylic acids after horizontal transfer to oomycetes (Savory et al., 2018). Plant enzymes in the dihydroflavonol-4-reductase and the SABATH methyltransferase families evolved novel substrate specificities during the diversification of dicots while retaining weak ancestral activity (Des Marais and Rausher, 2008; Huang et al., 2012). Due to such pleiotropic activity of some QDR genes, the suppression of their activity by pathogens may have adverse effects on availability of plant-derived nutrients or lead to pathogen detection, and therefore increase the durability of disease resistance. The reconstruction of immune gene networks may facilitate the systematic investigation of pleiotropy in the QDR gene pool and shed light on how genomic backgrounds affect the activity of disease resistance genes (Smakowska-Luzan et al., 2018; Wu et al., 2018; Peyraud et al., 2017; Mine et al., 2014).

**METHODS**

**Plant materials and Sclerotinia sclerotiorum infection**
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Arabidopsis thaliana Col-0 genotype obtained from the Nottingham Arabidopsis Stock Centre (accession number N1093), Solanum lycopersicum L. cv Ailsa Craig, Helianthus annuus cv XRQ, Ricinus communis cv Hale (accession number PI 642000) and Beta vulgaris L. subsp. vulgaris (accession number PI 355961) obtained from the USDA ARS Germplasm Resources Information Network, Phaseolus vulgaris (accession number G19833) obtained from the International Center for Tropical Agriculture (CIAT) were used for inoculations. Plants were grown in Jiffy pots under controlled conditions at 23°C, with a 9 hour light period at intensity of 120 μmol/m²/s under Osram OSLON SSL 80 LEDs. Five-week-old plants were inoculated on fully developed leaves by 0.5 cm-wide plugs of PDA (potato dextrose agar, Fluka) colonized by S. sclerotiorum strain 1980 (ATCC18683) (Supplemental Methods). Plugs of agar containing the fungal pathogen were placed on the adaxial surface of leaves and plants were incubated in trays closed with plastic wrap to maintain 80% humidity and placed in a Percival AR-41L3 chamber under the same day/light condition as for plant growth.

Tissue sampling, RNA extraction and sequencing

RNA-sequencing reads from A. thaliana and S. lycopersicum were obtained from NCBI Gene Expression Omnibus (GEO) database accession GSE106811 (Badet et al., 2017). For all infected plant species, the edge of 25mm-wide developed necrotic lesions (Peyraud et al., 2019) was isolated with a scalpel blade from leaves placed on a glass slide cooled down by liquid nitrogen, and immediately frozen in liquid nitrogen. Samples were harvested before lesions reached 25mm width, at various time after inoculation owing to the different susceptibility level of each species. All inoculations were performed five hours after lights were turned on, samples were harvested at 24 hours post inoculation for H. annuus, between 47 and 50 hours post inoculation for A. thaliana, P. vulgaris, R. communis and S. lycopersicum, 72 hours post inoculation for B. vulgaris. Samples from non-inoculated plants were harvested simultaneously to serve as controls and exclude biases due to synchrony of plants circadian clock. To determine the extent to which circadian synchrony varies across species in our dataset, we analyzed normalized read counts corresponding to ten genes of the circadian clock (LHY, TOC1, LUX, RVE8, PRR9, PRR3, PRR5, ZTL, GI, ELF3) in the six species, in inoculated and healthy plants (Supplemental Methods). In a principal component analysis, the first two dimensions summarized 86.8% of the total inertia. All species variables appeared very well correlated, excepted for A. thaliana which differed slightly in a higher TOC1 expression. Correlations between inoculated and healthy samples were significant for all species (ρ≥0.66, p-val≤0.04). Material obtained from leaves of three plants were pooled together for each sample, all samples were collected in triplicates. Samples were ground with glass beads (2.5 mm) in a Retschmill apparatus (24 htz for 2x1 min) before RNA extraction using NucleoSpin RNA extraction kits (Macherey-Nagel) following the manufacturer’s instructions. A 30 minutes DNase treatment (TURBO DNase; Ambion) was applied following manufacturer’s instructions to eliminate genomic DNA. The quality and concentration of RNA preparations were assessed with an Agilent 2100 Bioanalyzer and the RNA 6000 Nano kit. For H. annuus, P. vulgaris, R. communis and B. vulgaris samples, messenger RNA sequencing was outsourced to Fasteris SA (Switzerland) to produce Illumina paired reads (2 x 125bp) using a HiSeq 2500 instrument. Three independent biological samples were sequenced per condition. The corresponding raw RNAseq reads data was deposited in NCBI’s GEO under accession GSE138039.

Mapping of sequence reads and analysis of differentially expressed genes (DEGs)
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Reads were mapped to reference genomes listed in Supplemental Data Set 1.1 using the RNA-Seq analysis tool of the CLC Genomics Workbench 11.0.1 (Qiagen) software. The following mapping parameters were used: mismatch cost 2, insertion cost 3, deletion cost 3, length fraction 0.8, similarity fraction 0.8, both strands mapping, 10 hits maximum per read, with expression value given as total counts. All reads were mapped independently to S. sclerotiorum genome version 2 (Derbyshire et al., 2017) to estimate the fraction of fungal reads per sample. Number of uniquely mapped reads per transcript were exported in tab delimited text format. Differential gene expression analysis was conducted with the DESeq2 Bioconductor package version 1.8.2 (Love et al., 2014) in R 3.4.0 with total uniquely mapped read count per gene as input. Differential expression was calculated using expression in uninfected plants as a reference with ~replicates + treatment as the design formula. For interspecific comparisons, log fold changes were quantile normalized using the preprocessCore package in R (Bolstad 2017). Genes with log fold change ≥1.5 and ≤-1.5 and a Benjamini-Hochberg adjusted p-value < 0.01 in DESeq2 Wald test were considered significant for differential expression. For the analysis of AtPDR genes expression in multiple stress conditions, total read count per gene were retrieved from the NCBI GEO repository (accessions GSE66290, GSE83478, GSE104590, GSE70094, GSE116269, GSE90075, GSE72548, GSE56922 and GSE72806) and differential gene expression analysis performed in DESeq2 as described above.

Orthologous gene groups analyses

To define groups of orthologous genes, the complete proteomes of the six species were used in a Markov Clustering analysis through OrthoMCL (version 1.4) was used via the Family Companion server (Cottret et al., 2018). Parameters were set to pi_cutoff = 0, pv_cutoff = 0.00001, pmatch_cutoff = 80 and inflation= 1.5. Genes not expressed in our RNA-seq data (read count =0) were then discarded from orthogroups. Orthogroups including at least one expressed gene from each of the six plant species were designated as Pentapetalae core groups/genes. Orthogroups including one or more expressed gene from two to five plant species were designated as other orthologous groups/genes. Orthogroups including expressed genes from one single species together with expressed genes not included in orthogroups were designated as lineage-specific. The largest 100 orthogroups were manually inspected for consistency in protein sequence, Gene Ontology and PFAM annotation. R scripts used for the generation of figures in this manuscript are provided in Supplemental File 6.

Gene annotation and enrichment analyses

Gene ontology (GO) annotations for A. thaliana and S. lycopersicum were obtained from the Ensembl Plant database release 37 (Kersey et al., 2018). GO annotations were mapped onto complete plant proteomes of H. annuus, P. vulgaris, B. vulgaris and R. communis using the Blast2GO 5.2.5 (Conesa et al., 2005). Pfam domain annotations were obtained using the hmmscan function of the HMMER v3.1b2 software against the Pfam31.0 database (Schaeffer et al., 2017). For enrichment analyses, the number of differentially expressed genes and genes not differentially expressed was summed across the six species for each GO and
PFAM. Several occurrences of a same PFAM domain within a gene were counted once. GOS were mapped onto PFAM domains using the PFAM2GO file version 2019/12/14 provided by Interpro. Counts were used in a chi-squared test performed with R 3.5.0, and p-values adjusted with Bonferroni correction for multiple testing. Enrichment fold was defined as the ratio between proportion of DEGs in genes harboring a given GO or PFAM over the overall proportion of DEGs among expressed genes in each species. Annotations harboring enrichment fold > 1 and adjusted p-value < 0.01 were considered significantly enriched in DEGs. Annotations harboring enrichment fold < 1 and adjusted p-value < 0.01 were considered significantly depleted in DEGs. Gene ontology network rendering was performed using the BiNGO plugin in Cytoscape 3.6.1. To visualize enrichment across all species for the complete GO network, the following parameters were used: statistical test −−, significance level: 1.00, categories to be visualized: All categories, reference set: Use whole annotation as reference set, select ontology file: GO_Full, select organism/annotation: custom. As a custom annotation file, a list of 143,807 GO annotations from DEGs of the six species, including 6,962 distinct GO terms, was provided (Supplemental File 7). Using the ‘Import table from file’ option, an attribute table containing DEG enrichment score (-log of adjusted p-value from chi-squared test), up/down ratio and number of species with up-regulated genes for each of 6,962 GO (Supplemental File 8) was mapped onto the full GO network. Rank in the GO hierarchy were determined using the GOMFANCESTOR, GOCCANCESTOR and GOBPANCESTOR functions from the GO.db package version 3.8 from BiocManager in R 3.5.0. Violin plot rendering was performed using the easyGgplot2 package in R 3.4.0.

Bootstrapping and expression consistency analyses

Bootstrapping and consistency analysis was performed using homemade scripts in R 3.5.0, provided in Supplemental Files 3 and 4. To simulate distributions of upregulated genes among core orthogroups a total of 9,520 ‘upregulated’ gene states and 53,318 ‘not upregulated’ gene states were shuffled among 7,918 core orthogroups containing at least one DEG. Orthogroups in this simulation had the same number of genes from each species as experimentally determined core orthogroups. For each orthogroup, a minimal gene count (MGC) is determined, corresponding to the number of genes for the species with the least genes in the group. Gene states are drawn first for MGCs in each orthogroup, with ‘h’ species assigned the same draw. The remaining gene states are drawn randomly for each species in each orthogroup. The percentage of orthogroups featuring 0 to 6 species with at least one upregulated gene state was calculated of 100 bootstrap replicates. The procedure was repeated using 13,201 ‘downregulated’ gene states and 49,637 ‘not downregulated’ gene states. Residual sum of squares was calculated using the ssq function of the hydroGOF package in R 3.4.0. The index of gene expression consistency was computed on the basis of the number of clusters of core ortholog genes with similar expression. The number of clusters was computed by Silhouette analysis (Rousseeuw, 1987) performed by the factoextra R library (Charrad et al., 2014). The index of conservation expression was then computed as the difference between the proportion of genes in the largest cluster and the proportion associated to one gene of the cluster. By construction, an index of conservation
of expression equal to 0 indicates a group composed by genes with independent level of expression. An index tending towards 1 indicates an unique pattern of genes expression.

**Molecular and phenotypic characterization of abcg40 mutants**

*A. thaliana* T-DNA mutant lines SALK_005635 (*abcg40*-2), SAIL_885_E09 (*abcg40*-3), SALK_148565 (*abcg40*-4), and SALK_013945 (*abcg40*-5) were obtained from the European Arabidopsis Stock Centre (NASC). Plants were grown as described above. Homozygous T-DNA insertions were verified by PCR performed with primers given in **Supplemental Data Set 1.33**. The accumulation of *ABCG40* transcripts in *abcg40* mutants was assessed by reverse transcription quantitative PCR (RT-qPCR) performed on a LightCycler480 device (Roche) following manufacturer’s recommendation. RNA extraction, cDNA synthesis and quantitative PCR reactions were performed as described in (Badet et al., 2015) using primers given in **Supplemental Data Set 1.33** and the At2g28390 gene as a housekeeping reference. A strain of *S. sclerotiorum* 1980 expressing GFP fused to the endogenous OAH1 gene (Badet et al., 2017) was inoculated on four-week old plants as described above. Lesions were imaged 24 hours later with a Zeiss Axio Zoom V16 microscope under bright light and fluorescent illumination. Lesion areas were determined using the ImageJ 1.51k software. For this, fully developed lesions that have not reached the leaves borders were manually set as regions of interest. The area of the agar plug used for inoculation served as reference to normalize lesion measurements across images. Statistical analyses of disease phenotypes were performed in R via type III two-way ANOVA for unbalanced designs, significance of the difference between genotype was assessed using Student’s t test with Bonferroni correction for multiple testing since no experiment replicate effect was detected.

**Accession Numbers**

Sequencing data generated in this work has been deposited in the National Center for Biotechnology Information’s Gene Expression Omnibus and are accessible through Gene Expression Omnibus Series accession number GSE138039.
SUPPLEMENTAL DATA

Supplemental Data

Supplemental Figure 1. Species distribution of genes upregulated upon inoculation by S. sclerotiorum according to their Pfam domain annotation.

Supplemental Figure 2. Species distribution of genes downregulated upon inoculation by S. sclerotiorum according to their Pfam domain annotation.

Supplemental Methods Supplemental materials and methods providing information on the general aspect of plants at the time of inoculation and sampling, the analysis of circadian clock genes expression in healthy and inoculated plants, the analysis of leaf senescence genes expression in healthy and inoculated plants, the identification of differentially expressed genes in relation to read coverage and the analysis of AtABCG genes expression in twelve stress condition.

Supplemental Data Set 1. Includes supplemental datasets 1.1 to 1.33.

Supplemental Data Set 1.1 (related to Fig 1A) List of reference genomes and annotations used for mapping RNA-seq reads and calling gene expression.

Supplemental Data Set 1.2 (related to Fig 1B) Summary of read mappings for the 36 sequenced libraries.

Supplemental Data Set 1.3 (related to Fig 1C) Summary of gene expression analysis per species.

Supplemental Data Set 1.4 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Arabidopsis thaliana genes.

Supplemental Data Set 1.5 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Phaseolus vulgaris genes.

Supplemental Data Set 1.6 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Beta vulgaris genes.

Supplemental Data Set 1.7 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Ricinus communis genes.

Supplemental Data Set 1.8 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Helianthus annuus genes.

Supplemental Data Set 1.9 (related to Fig 1D) Log Fold change, differential expression analysis and orthogroup assignment for Solanum lycopersicum genes.

Supplemental Data Set 1.10 (related to Fig 2A) List of genes assigned to orthogroups.

Supplemental Data Set 1.11 (related to Fig 2A) Number of species, of expressed genes and DEGs per orthogoup.
Supplemental Data Set 1.12 (related to Fig 2B) Distribution of orthogroups per species.

Supplemental Data Set 1.13 (related to Fig 2D, Table 1) Complete statistical analysis for enrichment in DEGs among core, ortho and lineage-specific genes.

Supplemental Data Set 1.14 (related to Fig 3A) Parsimonious inference of orthogroups evolution.

Supplemental Data Set 1.15 (related to Fig 3B,C,D) Analysis of rates of gene loss and gain over evolution.

Supplemental Data Set 1.16 (related to Fig 4A) Summary of Gene Ontology and PFAM mappings per species.

Supplemental Data Set 1.17 (related to Fig 4A) Gene Ontology mapping with chi-squared test for DEG enrichment.

Supplemental Data Set 1.18 (related to Fig 4A) PFAM mapping with chi-squared test for DEG enrichment.

Supplemental Data Set 1.19 (related to Fig 4 & 5) List of gene ontologies (Gos) and PFAMs enriched with upregulated and downregulated genes identified using a chi-squared test on counts for each species (as presented in main text).

Supplemental Data Set 1.20 (related to Fig 5C) Summary table for GOs enriched in DEGs.

Supplemental Data Set 1.21 (related to Fig 6A) Species distribution for orthogroups expressed in all 6 species.

Supplemental Data Set 1.22 (related to Fig 6B, E) Species distribution for Gene Ontologies expressed in all 6 species.

Supplemental Data Set 1.23 (related to Fig 6C) Species distribution for PFAMs expressed in all 6 species.

Supplemental Data Set 1.24 (related to Fig 6F,G) Enrichment coefficients in core genes and DEGs for 893 GO and species correlation analysis.

Supplemental Data Set 1.25 (related to Fig 6H, I) Top100 GO enriched in DEGs in each species.

Supplemental Data Set 1.26 (related to Fig 6I) Top100 GO depleted in DEGs in each species.

Supplemental Data Set 1.27 (related to Fig 7A) Bootstrap analysis of the % of orthogroups including up/downreg. Genes per number of species.

Supplemental Data Set 1.28 (related to Fig 7B) Analysis of gene expression consistency within orthogroups.

Supplemental Data Set 1.29 (related to Fig 8A) Expression of members of the \textit{ABCG/PDR} orthogroup #4 upon inoculation by \textit{S. sclerotiorum}.
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**Supplemental Data Set 1.30** (related to Fig 8B) Expression of *A. thaliana PDR* genes in 12 stress conditions.

**Supplemental Data Set 1.31** (related to Fig 8C) Assessment of *AtPDR12* gene expression in *pdr12* mutants by RT-qPCR.

**Supplemental Data Set 1.32** (related to Fig 8E) Disease lesion area measured on *A. thaliana pdr12* mutants after *S. sclerotiorum* inoculation.

**Supplemental Data Set 1.33** (related to Methods) List of oligonucleotide primers used in this work.

**Supplemental File 1** (related to Fig 4) Complete list of Gene ontology annotations used in this work

**Supplemental File 2** (related to Fig 4) Complete list of PFAM annotations used in this work

**Supplemental File 3** (related to Fig 7A) Input data and homemade R script used for the bootstrap analysis of upregulated/downregulated genes distribution

**Supplemental File 4** (related to Fig 7B) Homemade R script used for the analysis of LFC consistency within orthogroups

**Supplemental File 5** (related to Fig 8A) Maximum likelihood tree of phylogenetic relationship between 96 *ABCG/PDR* genes in Newick format

**Supplemental File 6** (related to Fig1-8) R code used for the generation of figures and statistical analyses

**Supplemental File 7** (related to methods) List of GO annotations used as a reference for the construction of the full GO network

**Supplemental File 8** (related to methods) Attribute table used to visualize properties of GO annotation on the full GO network
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AUTHORS’ CONTRIBUTIONS

JS, MM and SR conceived and designed the experiments. JS, MM, MB and AD performed the experiments. JS, MD, AB and SR analyzed the data. JS and SR wrote the article.

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FIGURE LEGENDS

Figure 1. Overview of genes responsive locally to *S. sclerotiorum* inoculation in six plant species of the Pentapetalae clade. (A) Proportion of RNA-sequencing reads from non-infected samples mapped to a unique position in plant genomes (% of all reads). Results from samples collected in three independent biological replicates are shown, grey dotted line shows average for all samples. (B) Proportion of RNA-sequencing reads from infected samples mapped to a unique position in plant genomes (colors) and *S. sclerotiorum* genome (grey, % of all reads). Results from samples collected in three independent biological replicates are shown, grey dotted lines show the average for all samples. (C) Proportion of plant genes expressed in all samples, both in healthy and infected plants. Labels indicate number of expressed genes (% of complete annotated transcriptomes). (D) Distribution of gene expression Log2 fold change (LFC) in infected compared to healthy plants (% of transcripts). Dotted lines and labels indicate median LFC. (E) Proportion of differentially expressed (DEGs) in each species (colors) and overall (grey, % of transcripts). Plain bars correspond to the % of up-regulated genes, empty bars to the % of down-regulated genes, labels indicate the total number of DEGs per species. Error bars show standard deviation. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).

Figure 2. Distribution of genes differentially expressed upon *S. sclerotiorum* inoculation (DEGs) according to gene conservation. (A) Distribution of orthologous gene groups across species. Groups including genes from all six species are designated as ‘Pentapetalae Core’, groups including genes from 2 to 5 species are designated as ‘other orthologs’, genes not included in multi-species ortholog groups are designated as ‘lineage specific’. The number of gene group is indicated in bold, number of genes in italics and the proportion (% of all expressed genes) is indicated between square brackets. (B) Proportion of Pentapetalae (Penta.) Core genes, lineage specific (spe.) genes and other ortholog (ortho.) genes in each plant genome (% of expressed genes). (C) Relative proportion of DEGs belonging to Pentapetalae core groups (dark blue), other ortholog groups (green) and lineage-specific genes (yellow). Branches of the phylogenetic tree in inset are color-coded according to corresponding gene groups. Results are shown for down-regulated genes only (Down), upregulated genes only (Up) or both together (All). Plain dots show proportions for Superasteridae species, empty dots for Rosid species. Letters define groups of significance as determined by pairwise Student t tests (p-val < 0.01). (D) Relative proportion of DEGs, either down-regulated (empty circles) or upregulated (plain circles), in Rosids and Superasteridae species. Genes belonging to Pentapetalae core groups (dark blue), other ortholog groups (green) and lineage-specific genes (yellow) are shown separately. Letters define groups of significance as determined by pairwise Student t tests (p-val < 0.01). Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers). *At*, *Arabidopsis thaliana*; *Bv*, *Beta vulgaris*; *Ha*, *Helianthus annuus*; *Pv*, *Phaseolus vulgaris*; *Rc*, *Ricinus communis*; *Sl*, *Solanum lycopersicum*.
Figure 3. Relative increase in up-regulated genes inferred from the evolutionary history of *S. sclerotiorum*-responsive transcriptomes. (A) Principles of a parsimony approach to infer gene gains and losses in the evolutionary history of the Pentapetalae. In these hypothetical examples, a gene of interest is present in lineages with a green tick, absent in lineages with a red cross. We inferred presence along branches shown in green and absence along branches shown as dotted grey lines. (B) Inferred transcriptome evolution along the phylogeny of the Pentapetalae. The size of grey circles indicates the relative total number of genes gained (plain circle, left) and lost (dotted circles, right) along each branch. The size of colored circles indicates the relative differentially express genes (DEGs) gained (plain circle, left) and lost (dotted circles, right) along each branch. The color scale represents the ratio of up/down regulated genes for gained and lost DEGs. (C) Estimated relative number of genes gained and lost per million years (Mya) along intermediate and terminal branches of the Pentapetalae phylogeny (% of Pentapetalae Core set). Estimates are given for whole genomes and DEG pools. Significance of the difference between gains and losses was assess by a paired t test. (D) Ratio between the number of genes up- and down-regulated among DEGs gained and lost along intermediate and terminal branches of the Pentapetalae phylogeny. Significance of the difference between gains and losses was assess by a paired t test. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).

Figure 4. Distribution of differentially expressed genes according to gene ontologies (GOs). Distribution of GOs enriched with upregulated genes (A) and downregulated genes (B) across species. Selected ontologies are labelled on the figure, colored according to the species in which they are enriched with DEGs. Labels are italicized for ontologies enriched both with upregulated genes and with downregulated genes. (C) Network of ‘Biological Process’ (BP) GOs covering all GOBPs including expressed genes in at least on species. GOs are represented by circles sized according to the number of expressed genes per GO. GOs significantly enriched in DEGs in a chi-squared test (adjusted p-val < 0.01 after Bonferroni correction for multiple testing) are colored according to their enrichment score. Ontologies discussed in the main text are labelled. At, *Arabidopsis thaliana*; Bv, *Beta vulgaris*; Ha, *Helianthus annuus*; Pv, *Phaseolus vulgaris*; Rc, *Ricinus communis*; Sl, *Solanum lycopersicum*.

Figure 5. Distribution of gene ontologies according to their content in genes upregulated upon *S. sclerotiorum* inoculation and their content in core genes. Number of genes annotated with GO:0009407 ‘toxin catabolism’ (A) and GO:0015995 ‘chlorophyll biosynthesis’ (B) that are up-regulated (yellow), down-regulated (blue), or not differentially expressed (white) upon *S. sclerotiorum* inoculation in each of the six plant species. (C) Distribution of GOBPs significantly enriched with DEGs according to the ratio of the number of Pentapetalae core genes over the number of lineage-specific genes per annotation (X-axis) and the ratio of the number of upregulated genes over the number of down-regulated genes per annotation (Y-axis). Dotted lines indicate median values for all annotations, grey areas represent the interval containing the top 10%-top 90% annotations. Circles are sized according to the number of expressed genes per annotation and colored according their
enrichment with DEGs. At, Arabidopsis thaliana; Bv, Beta vulgaris; Ha, Helianthus annuus; Pv, Phaseolus vulgaris; Rc, Ricinus communis; Sl, Solanum lycopersicum.

**Figure 6. Diversity of gene functions differentially regulated upon S. sclerotiorum inoculation at the inter-specific level.** Distribution of Pentapetalae core groups (A), conserved gene ontologies (B) and conserved Pfam domains (C) according to the number of plant species in which genes are upregulated (red), downregulated (blue) or both (grey). For instance, 2.00% of the Pentapetalae core groups contain genes upregulated in all six species. The proportion of groups containing DEGs from six species are labeled. The sum of groups containing DEGs from 1 to 5 species is labeled to illustrate how frequent interspecific divergence in gene expression is. (D) Network of ‘Biological Process’ GOs represented by circles sized according to the number of DEGs per GO and colored according to the number of species in which genes are upregulated for each GO. Ontologies discussed in the main text are labelled. (E) Distribution of GOs’ level in the gene ontology hierarchy according to the number of species in which genes are up-regulated. Median values are shown by a black diamond and labelled. Significance of the difference to the 6-species distribution was assessed by a Student t test with p-values shown on top the plot. (F, G) Pairwise correlations for the content of GOs in Pentapetalae Core genes (F) or in DEGs (G) between the different plant species. Circles are sized and colored according to Spearman ρ. The average ρ and standard deviation for all pairwise comparisons is indicated. (H) Overlap between the list of top 100 GOs enriched in DEGs in each of the six species. Values shown in grey are common to 2 to 5 species. (I) Number of GOs in the top 100 enriched in DEGs (red) or depleted in DEGs (grey) that are shared between all six species, between 2-5 species or unique to one species. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers). At, Arabidopsis thaliana; Bv, Beta vulgaris; Ha, Helianthus annuus; Pv, Phaseolus vulgaris; Rc, Ricinus communis; Sl, Solanum lycopersicum.

**Figure 7. Analysis of interspecific regulatory variation within Core Pentapetalae gene groups.** (A) Simulated (grey) and observed (red) distributions of the number of up-regulated genes in each Core Pentapetalae group. Similar expression (upregulated or not) has been constrained in h=6 to 1 species (no constraint). The deviation between observed and simulated distribution was assessed using the sum of squared residuals (SSR). The lowest SSR value corresponded to h=2 species suggesting that constraints to maintain similar gene expression across species are weak. (B) Distribution of the consistency in gene expression index (CEI) across 7918 core ortholog gene groups. The CEI is determined using gene expression clustering within each orthogroups, and increases with the relative size of the largest expression cluster. Counts in the bottom quarter (CEI≤0.33) and top quarter (CEI≥0.67) of the range are labelled, red dotted line shows dataset average. (C, D) Detailed analysis of orthogroup #56 with low CEI (C) and orthogroup #1067 with high (D) CEI value. The dotplots show log2 fold change (LFC) of expression upon S. sclerotiorum inoculation for genes in each orthogroup. Arrows points towards the position of orthogroups #56 and #1067 in the CEI distribution. Pie charts show the relative size of expression clusters (as % of orthogroup size), with the largest expression cluster labelled.
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Figure 8. Extreme regulatory diversity and evidence for exaptation of an ancestral function into quantitative disease resistance in a group of ABCG transporters. (A) Phylogenetic relationship of 96 ABCG transporters from six Pentapetalae species covering orthogroup #4. The tree obtained by a maximum likelihood analysis is shown, with the number of substitution per site used as branch length, and branch support determined by an approximate likelihood-ratio test (red labels). Terminal nodes are color-coded according to plant species. The circular bar plot shows gene expression log2 fold change (LFC) upon inoculation by S. sclerotiorum, in blue when significantly down-regulated, in red when significantly up-regulated and in grey otherwise. Six major clades are delineated by shaded sectors, with the list of species including upregulated indicated by icons for each clade. A. thaliana genes are labelled in green with their ABCG and PDR identifiers. Inset: reverse cumulative distribution of orthogroups according to the delta LFC between most induced and most down-regulated genes per group. Red line indicates position of orthogroup #4. (B) Expression LFC for 14 A. thaliana ABCG genes upon 12 stress treatments. The size and color of bubbles relates to LFC, bubbles are outlined when genes are significantly up- or down-regulated. Adjusted p-values obtained by DESeq-2 were Bonferroni corrected for multiple testing. (C) Altered ABCG40 gene expression in A. thaliana abcg40 insertion mutant lines. Top diagram represents ABCG40 gene structure with exons as blue boxes, red arrows indicate the position of T-DNA insertions in each line. Boxplots show ABCG40 gene expression in healthy wild type (Col-0) and mutant plants obtained from three or four plants per genotype measured twice. Expression is given relative to AT2G28390 housekeeping gene expression. (D) Representative pictures of leaves infected by S. sclerotiorum carrying GFP shown under U.V. illumination at 24 hours post inoculation (hpi). Bar = 2 mm. (E) Measurements of leaf areas colonized by S. sclerotiorum 24 hpi. The rlp30-1 mutant was used as a susceptibility control (Zhang et al., 2013). Significance of the difference to wild type were assessed by Student’s t tests with Bonferroni correction for multiple testing, with p-values shown in green. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).
Table 1. General transcriptome statistics for the six species analyzed in this work. DEG, Differentially expressed gene; GO, gene ontology; No., number.

|                      | P. vulgaris | R. communis | A. thaliana | H. annuus | S. lycopersicum | B. vulgaris | ALL       |
|----------------------|-------------|-------------|-------------|-----------|----------------|-------------|-----------|
| Expressed genes      | 22,068      | 19,353      | 22,822      | 42,351    | 27,045         | 22,858      | 156,497   |
| DEG total            | 6,843       | 7,510       | 10,496      | 9,844     | 8,069          | 6,148       | 48,910    |
| (31.01%)             | (38.81%)    | (45.99%)    | (23.24%)    | (29.84%)  | (26.90%)       |             |
| Upregulated genes    | 3,012       | 3,545       | 4,692       | 4,402     | 3,513          | 2,867       | 22,031    |
| Downregulated genes  | 3,831       | 3,965       | 5,804       | 5,442     | 4,556          | 3,281       | 26,879    |
| Up/down ratio        | 0.79        | 0.89        | 0.81        | 0.81      | 0.77           | 0.87        | 0.82      |
| Mean reads per DEG   | 2,834.65    | 3,210.32    | 3,314.90    | 2,485.06  | 3,445.84       | 3,771.52    | 3,183.08  |
| (healthy)            |             |             |             |           |                |             |
| Mean reads per DEG   | 1,535.95    | 2,372.43    | 1,640.26    | 924.75    | 1,675.03       | 1,438.26    | 1,584.93  |
| (inoculated)         |             |             |             |           |                |             |
| Core orthologs Total | 10,307      | 8,919       | 10,551      | 13,906    | 10,206         | 8,949       | 62,838    |
| [DEGs]               | [3,611]     | [3,604]     | [5,358]     | [3,825]   | [3,444]        | [2,879]     | [22,721]  |
| Other orthologs Total| 5,693       | 5,837       | 5,224       | 8,057     | 5,668          | 4,446       | 34,925    |
| [DEGs]               | [1,795]     | [2,327]     | [2,427]     | [2,168]   | [1,910]        | [1,357]     | [11,984]  |
| Lineage-specific     | 6,068       | 4,597       | 7,047       | 20,388    | 11,171         | 9,463       | 58,734    |
| Total ; [DEGs]       | [1,437]     | [1,579]     | [2,711]     | [3,851]   | [2,715]        | [1,912]     | [14,205]  |
| % of expressed genes | 59.36%      | 69.21%      | 74.13%      | 47.08%    | 48.47%         | 59.69%      | 73.96%    |
| with GO ; PFAM       | 85.75%      | 89.45%      | 89.66%      | 76.12%    | 83.86%         | 80.49%      | 83.08%    |
| No. with ≥3 occurrences | 3,791      | 2,410       | 3,792       | 3,059     | 2,600          | 2,381       | 6,171    |
| GO ; PFAM            | 4,284       | 4,128       | 4,862       | 5,913     | 4,969          | 4,353       | 13,100    |
| % of core genes with GO; PFAM | 88.36% | 87.79% | 97.71% | 86.51% | 80.73% | 86.90% | 87.99% |
| No. GOs enriched in up/down-reg. genes | 156 | 31 | 54 | 57 | 55 | 25 | 262 |
Table 2. Distribution of differentially expressed genes (DEGs) among core orthologs, other orthologs and lineage-specific genes. Significance of enrichment (fold >1) or depletion (fold <1) was assessed using a chi-squared test followed by a Bonferroni correction for multiple testing.

| Species                      | % of DEGs in core genes | % of DEGs in other ortho genes | % of DEGs in lineage-spe. Genes | DEG enrichment fold in core genes | chi-squared p-val | DEG enrichment fold in other ortho genes | chi-squared p-val | DEG enrichment fold in lineage-spe. Genes | chi-squared p-val |
|------------------------------|--------------------------|-------------------------------|--------------------------------|----------------------------------|-------------------|------------------------------------------|-------------------|------------------------------------------|-------------------|
| *Phaseolus vulgaris*         | 52.77                    | 26.23                         | 21.00                          | 1.13                             | 8.40E-33          | 1.02                                     | 2.32E+00          | 0.76                                     | 1.20E-46          |
| *Ricinus communis*           | 47.99                    | 30.99                         | 21.03                          | 1.04                             | 1.74E-04          | 1.03                                     | 3.38E-01          | 0.89                                     | 9.79E-12          |
| *Arabidopsis thaliana*       | 51.05                    | 23.12                         | 25.83                          | 1.10                             | 2.06E-40          | 1.01                                     | 3.14E+00          | 0.84                                     | 1.78E-51          |
| *Helianthus annuus*          | 38.86                    | 22.02                         | 39.12                          | 1.18                             | 7.60E-47          | 1.16                                     | 3.97E-17          | 0.81                                     | 5.95E-92          |
| *Solanum lycopersicum*       | 42.68                    | 23.67                         | 33.65                          | 1.13                             | 6.07E-27          | 1.13                                     | 6.91E-12          | 0.81                                     | 1.64E-61          |
| *Beta vulgaris*              | 46.83                    | 22.07                         | 31.10                          | 1.20                             | 3.11E-46          | 1.13                                     | 9.81E-09          | 0.75                                     | 5.46E-81          |
| **ALL**                     |                          |                               |                                | 1.16                             | 9.53E-257         | 1.10                                     | 1.20E-43          | 0.77                                     | 0.00E+00          |
Table 3. A selection of *A. thaliana* genes involved in disease resistance to *S. sclerotiorum* and differentially expressed in this study. LFC, gene expression log2 fold change.

| TAIR gene id | Gene name | LFC (this work) | Reference |
|--------------|-----------|----------------|-----------|
| At1g15520    | ABCG40    | 12.48          | This work |
| At3g26830    | PAD3      | 9.97           | (Stotz et al., 2011) |
| At1g20380    | POQR      | 7.15           | (Badet et al., 2017) |
| At3g05360    | RLP30     | 4.46           | (Zhang et al., 2013) |
| At4g33430    | BAK1      | 2.88           | (Zhang et al., 2013) |
| At5g45900    | ATG7      | 2.54           | (Kabbage et al., 2013) |
| At1g64280    | NPR1      | 1.25           | (Guo and Stotz, 2007) |
| At3g48990    | AAE3      | 1.12           | (Foster et al., 2012) |
| At4g26080    | ABI1      | 1.03           | (Perchepied et al., 2010) |
| At4g14147    | ARPC4     | 1.02           | (Badet et al., 2019) |
| At2g39940    | COI1      | -1.23          | (Guo and Stotz, 2007) |
| At1g34210    | SERK2     | -2.25          | (Derbyshire et al., 2019) |
| At5g03280    | EIN2      | -2.89          | (Guo and Stotz, 2007) |
| At3g47450    | NOA1      | -3.22          | (Perchepied et al., 2010) |
| At1g0855     | NPQ1      | -3.95          | (Zhou et al., 2015) |
| At2g32680    | RLP23     | -5.41          | (Albert et al., 2015) |
| At5g61420    | MYB28     | -6.84          | (Stotz et al., 2011) |
Figure 1. Overview of genes responsive to *S. sclerotiorum* inoculation in six plant species of the Pentapetalae clade. (A) Proportion of RNA-sequencing reads from non-infected samples mapped to a unique position in plant genomes (% of all reads). Results from samples collected in three independent biological replicates are shown, grey dotted line shows average for all samples. (B) Proportion of RNA-sequencing reads from infected samples mapped to a unique position in plant genomes (colors) and *S. sclerotiorum* genome (grey, % of all reads). Results from samples collected in three independent biological replicates are shown, grey dotted lines shows average for all samples. (C) Proportion of plant genes expressed in all samples, both in healthy and infected plants. Labels indicate number of expressed genes (% of complete annotated transcriptomes). (D) Distribution of gene expression Log2 fold change (LFC) in infected compared to healthy plants (% of transcripts). Dotted lines and labels indicate median LFC. (E) Proportion of genes differentially expressed (DEGs) in each species (colors) and overall (grey, % of transcripts). Plain bars correspond to the % of up-regulated genes, empty bars to the % of down-regulated genes, labels indicate the total number of DEGs per species. Error bars show standard deviation. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).
Figure 2. Distribution of genes differentially expressed upon S. sclerotiorum inoculation (DEGs) according to gene conservation. (A) Distribution of orthologous gene groups across species. Groups including genes from all six species are designated as ‘Pentapetalae Core’, groups including genes from 2 to 5 species are designated as ‘other orthologs’, genes not included in multi-species ortholog groups are designated as ‘lineage specific’. The number of gene group is indicated in bold, number of genes in italics and the proportion (% of all expressed genes) is indicated between square brackets. (B) Proportion of Pentapetalae (Penta.) Core genes, lineage specific (spec.) genes and other ortholog (ortho.) genes in each plant genome (% of expressed genes). (C) Relative proportion of DEGs belonging to Pentapetalae core groups (dark blue), other ortholog groups (green) and lineage-specific genes (yellow). Branches of the phylogenetic tree in inset are color-coded according to corresponding gene groups. Results are shown for down-regulated genes only (Down), upregulated genes only (Up) or both together (All). Plain dots show proportions for Superasteridae species, empty dots for Rosid species. Letters define groups of significance as determined by pairwise Student t tests (p-val < 0.01). (D) Relative proportion of DEGs, either down-regulated (empty circles) or upregulated (plain circles), in Rosids and Superasteridae species. Genes belonging to Pentapetalae core groups (dark blue), other ortholog groups (green) and lineage-specific genes (yellow) are shown separately. Letters define groups of significance as determined by pairwise Student t tests (p-val < 0.01). Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers). At, Arabidopsis thaliana; Bv, Beta vulgaris; Ha, Helianthus annuus; Pv, Phaseolus vulgaris; Rc, Ricinus communis; Sl, Solanum lycopersicum.
Figure 3. Relative increase in up-regulated genes inferred from the evolutionary history of S. sclerotiorum-responsive transcriptomes. (A) Principles of a parsimony approach to infer gene gains and losses in the evolutionary history of the Pentapetalae. In these hypothetical examples, a gene of interest is present in lineages with a green tick, absent in lineages with a red cross. We inferred presence along branches shown in green and absence along branches shown as dotted grey lines. (B) Inferred transcriptome evolution along the phylogeny of the Pentapetalae. The size of grey circles indicates the relative total number of genes gained (plain circle, left) and lost (dotted circles, right) along each branch. The size of colored circles indicates the relative differentially express genes (DEGs) gained (plain circle, left) and lost (dotted circles, right) along each branch. The color scale represents the ratio of up/down regulated genes for gained and lost DEGs. (C) Estimated relative number of genes gained and lost per million years (Mya) along intermediate and terminal branches of the Pentapetalae phylogeny (% of Pentapetalae Core set). Estimates are given for whole genomes and DEG pools. Significance of the difference between gains and losses was assess by a paired t test. (D) Ratio between the number of genes up- and down-regulated among DEGs gained and lost along intermediate and terminal branches of the Pentapetalae phylogeny. Significance of the difference between gains and losses was assess by a paired t test. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).
Figure 4. Distribution of differentially expressed genes according to gene ontologies (GOs). Distribution of GOs enriched with upregulated genes (A) and downregulated genes (B) across species. Selected ontologies are labelled on the figure, colored according to the species in which they are enriched with DEGs. Labels are italicized for ontologies enriched both with upregulated genes and with downregulated genes. (C) Network of ‘Biological Process’ (BP) GOs covering all GOBPs including expressed genes in at least one species. GOs are represented by circles sized according to the number of expressed genes per GO. GOs significantly enriched in DEGs in a chi-squared test (adjusted p-val < 0.01 after Bonferroni correction for multiple testing) are colored according to their enrichment score. Ontologies discussed in the main text are labelled. At, Arabidopsis thaliana; Bv, Beta vulgaris; Ha, Helianthus annuus; Pv, Phaseolus vulgaris; Rc, Ricinus communis; Sl, Solanum lycopersicum.
Figure 5. Distribution of gene ontologies according to their content in genes upregulated upon *S. sclerotiorum* inoculation and their content in core genes. Number of genes annotated with GO:0009407 ‘toxin catabolism’ (A) and GO:0015995 ‘chlorophyll biosynthesis’ (B) that are up-regulated (yellow), down-regulated (blue), or not differentially expressed (white) upon *S. sclerotiorum* inoculation in each of the six plant species. (C) Distribution of GOBP significantly enriched with DEGs according to the ratio of the number of Pentapetalae core genes over the number of lineage-specific genes per annotation (X-axis) and the ratio of the number of upregulated genes over the number of down-regulated genes per annotation (Y-axis). Dotted lines indicate median values for all annotations, grey areas represent the interval containing the top 10%-top 90% annotations. Circles are sized according to the number of expressed genes per annotation and colored according their enrichment with DEGs. At, Arabidopsis thaliana; Bv, Beta vulgaris; Ha, Helianthus annuus; Pv, Phaseolus vulgaris; Rc, Ricinus communis; Sl, Solanum lycopersicum.
Figure 6. Diversity of gene functions differentially regulated upon *S. sclerotiorum* inoculation at the inter-specific level. Distribution of Pentapetalae core groups (A), conserved gene ontologies (B) and conserved Pfam domains (C) according to the number of plant species in which genes are upregulated (red), downregulated (blue) or both (grey). For instance, 2.00% of the Pentapetalae core groups contain genes upregulated in all six species. The proportion of groups containing DEGs from six species are labeled. The sum of groups containing DEGs from 1 to 5 species is labeled to illustrate how frequent interspecific divergence in gene expression is. (D) Network of Biological Process GOs represented by circles sized according to the number of DEGs per GO and colored according to Spearman ρ. The network is labeled. The sum of groups containing DEGs from 1 to 5 species is labeled to illustrate how frequent interspecific divergence in gene expression is. (E) Distribution of GOs’ level in the gene ontology hierarchy according to the number of species in which genes are upregulated for each GO. Ontologies discussed in the main text are highlighted. Median values are shown by a black diamond and labelled. Significance of the difference to the 6-species distribution was assessed by a Student t test with p-values shown on top the plot. (F, G) Pairwise correlations for the content of GOs in Pentapetalae Core genes (F) or in DEGs (G) between the different plant species. Circles are sized and colored according to Spearman ρ. The proportion of groups containing DEGs from 1 to 5 species is labeled to illustrate how frequent interspecific divergence in gene expression is. (H) Overlap between the list of top 100 GOs enriched in DEGs per species. Circles are sized and colored according to Spearman ρ. The proportion of groups containing DEGs from 1 to 5 species is labeled to illustrate how frequent interspecific divergence in gene expression is. (I) Number of GOs in the top 100 enriched or depleted in DEGs (grey) that are shared between all six species, between 2-5 species or unique to one species. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers). *At*, Arabidopsis thaliana; *Bv*, Beta vulgaris; *Ha*, Helianthus annuus; *Pv*, Phaseolus vulgaris; *Rc*, Ricinus communis; *Sl*, Solanum lycopersicum.
Figure 7. Analysis of interspecific regulatory variation within Core Pentapetalae gene groups. (A) Simulated (grey) and observed (red) distributions of the number of up-regulated genes in each Core Pentapetalae group. Similar expression (upregulated or not) has been constrained in h=6 to 1 species (no constraint). The deviation between observed and simulated distribution was assessed using the sum of squared residuals (SSR). The lowest SSR value corresponded to h=2 species suggesting that constraints to maintain similar gene expression across species are weak. (B) Distribution of the consistency in gene expression index (CEI) across 7918 core ortholog gene groups. The CEI is determined using gene expression clustering within each orthogroups, and increases with the relative size of the largest expression cluster. Counts in the bottom quarter (CEI≤0.33) and top quarter (CEI≥0.67) of the range are labelled, red dotted line shows dataset average. (C, D) Detailed analysis of orthogroup #56 with low CEI (C) and orthogroup #1067 with high (D) CEI value. The dotplots show log2 fold change (LFC) of expression upon S. sclerotiorum inoculation for genes in each orthogroup. Arrows points towards the position of orthogroups #56 and #1067 in the CEI distribution. Pie charts show the relative size of expression clusters (as % of orthogroup size), with the largest expression cluster labelled.
ABCG36 (PDR8)
ABCG29 (PDR1)
ABCG31 (PDR3)
ABCG38 (PDR10)
ABCG32 (PDR4)
ABCG39 (PDR11)
ABCG34 (PDR6)
ABCG37 (PDR9)
ABCG30 (PDR2)
ABCG41 (PDR13)

CLADE 1
induced in:
ABCG42
ABCG43

CLADE 2
induced in:
ABCG33

CLADE 3
induced in:
ABCG40

CLADE 4
induced in:
ABCG40

CLADE 5
not induced

CLADE 6
induced in:

Gene expression LFC

Sclerotinia sclerotiorum
Botrytis cinerea
Alternaria brassicicola
Verticillium dahliae
Colletotrichum incanum
Colletotrichum tofeldiae
P. syringae AvrRPS4
P. syringae DC3000 cor-
Heterodera schachtii
Cabbage leaf curl virus
Heat
Salt

Col-0
rlp30-1
abcg40-2
abcg40-3
abcg40-4
abcg40-5

AT/ABC40 relative expression (x 10^4)

Colonized area (mm²)

Col-0
rlp30-1
abcg40-2
abcg40-3
abcg40-4
abcg40-5
Figure 8. Extreme regulatory diversity and evidence for exaptation of an ancestral function into quantitative disease resistance in a group of ABCG transporters. (A) Phylogenetic relationship of 96 ABCG transporters from six Pentapetalae species covering orthogroup #4. The tree obtained by a maximum likelihood analysis is shown, with the number of substitution per site used as branch length, and branch support determined by an approximate likelihood-ratio test (red labels). Terminal nodes are color-coded according to plant species. The circular bar plot shows gene expression log2 fold change (LFC) upon inoculation by *S. sclerotiorum*, in blue when significantly down-regulated, in red when significantly up-regulated and in grey otherwise. Six major clades are delineated by shaded sectors, with the list of species including upregulated indicated by icons for each clade. *A. thaliana* genes are labelled in green with their ABCG and PDR identifiers. Inset: reverse cumulative distribution of orthogroups according to the delta LFC between most induced and most down-regulated genes per group. Red line indicates position of orthogroup #4. (B) Expression LFC for 14 *A. thaliana* ABCG genes upon 12 stress treatments. The size and color of bubbles relates to LFC, bubbles are outlined when genes are significantly up- or down-regulated. Adjusted p-values obtained by DESeq-2 were Bonferroni corrected for multiple testing. (C) Altered ABCG40 gene expression in *A. thaliana abcg40* insertion mutant lines. Top diagram represents ABCG40 gene structure with exons as blue boxes, red arrows indicate the position of T-DNA insertions in each line. Boxplots show ABCG40 gene expression in healthy wild type (Col-0) and mutant plants obtained from three or four plants per genotype measured twice. Expression is given relative to *AT2G28390* housekeeping gene expression. (D) Representative pictures of leaves infected by *S. sclerotiorum* carrying GFP shown under U.V. illumination at 24 hours post inoculation (hpi). Bar = 2 mm. (E) Measurements of leaf areas colonized by *S. sclerotiorum* 24 hpi. The *rlp30-1* mutant was used as a susceptibility control (Zhang et al., 2013). Significance of the difference to wild type were assessed by Student’s t tests with Bonferroni correction for multiple testing, with p-values shown in green. Boxplots show 1st and 3rd quartiles (box), median (thick line) and the most dispersed values within 1.5 times the interquartile range (whiskers).
**IN A NUTSHELL**

**Background:** Necrotrophic plant pathogens actively kill host cells to cause disease. This is notably the case for the fungus *Sclerotinia sclerotiorum*, causal agent of white and stem mold diseases on a broad range of dicot plants. Plants have developed specific resistance mechanisms to mitigate infections by necrotrophs, such as quantitative disease resistance (QDR). Studies on QDR so far have shown that this form of immunity is frequent in nature, often efficient against multiple pathogen genotypes (broad spectrum), and relatively stable over time (durable). QDR probably involves numerous genes with very diverse molecular functions, but the repertoire of genes contributing to QDR remains largely unknown.

**Question:** *S. sclerotiorum* causes disease on plant species from very diverse botanical families in the Pentapetalae group of the dicots, such as common bean, castor bean, thalecress, tomato, sunflower and beetroot. How diverse are transcriptional responses to *S. sclerotiorum* in these plants? Are conserved genes and lineage-specific genes strongly regulated during infection?

**Findings:** By studying the global transcriptome response of six plant species from diverse botanical families to the same strain of *S. sclerotiorum*, we showed that a large proportion of differentially express genes were conserved across species. However, despite being present in all species, “core” genes exhibited contrasting expression patterns during infection in each plant species.

These results suggested that some genes involved in QDR are broadly conserved stress-responsive genes that underwent changes in their transcriptional response to infection during evolution. We highlight one family of ABC transporter genes, conserved and induced by *Sclerotinia* infection in all plant species. Using *Arabidopsis thaliana* mutants, we show that *ABCG40*, the most induced member of this family, plays a role in QDR.

**Next steps:** We identified the repertoire of genes conserved and responding similarly to fungal infection across botanical families, as a resource to mine for quantitative resistance genes active in multiple plant species. We wish now to investigate the molecular and evolutionary mechanisms underlying interspecific expression variation in QDR genes.
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Phylotranscriptomics of the Pentapetalae Reveals Frequent Regulatory Variation in Plant Local Responses to the Fungal Pathogen Sclerotinia sclerotiorum

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