RESEARCH PAPER



Transcriptional regulation of receptor-like protein genes by environmental stresses and hormones and their overexpression activities in *Arabidopsis thaliana*

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Abstract

Receptor-like proteins (RLPs) have been implicated in multiple biological processes, including plant development and immunity to microbial infection. Fifty-seven *AtRLP* genes have been identified in Arabidopsis, whereas only a few have been functionally characterized. This is due to the lack of suitable physiological screening conditions and the high degree of functional redundancy among *AtRLP* genes. To overcome the functional redundancy and further understand the role of *AtRLP* genes, we studied the evolution of *AtRLP* genes and compiled a comprehensive profile of the transcriptional regulation of *AtRLP* genes upon exposure to a range of environmental stresses and different hormones. These results indicate that the majority of *AtRLP* genes are differentially expressed under various conditions that were tested, an observation that will help to select certain *AtRLP* genes involved in a specific biological process for further experimental studies to eventually dissect their function. A large number of *AtRLP* genes were found to respond to more than one treatment, suggesting that one single *AtRLP* genes, and generated and characterized transgenic Arabidopsis plants overexpressing the individual *AtRLP* genes, presenting new insight into the roles of *AtRLP* genes, as exemplified by *AtRLP3*, *AtRLP11* and *AtRLP28*. Our study provides an overview of biological processes in which *AtRLP* genes may be involved, and presents valuable resources for future investigations into the function of these genes.

Key words: Arabidopsis, hormone, overexpression, receptor-like protein, stress, transcriptional regulation.

Introduction

All living organisms exploit cell-surface receptors to perceive extracellular signals that are from self (e.g. endogenous signaling molecules), non-self (e.g. pathogen-derived molecules) or modified-self (e.g. self molecules that are modified by pathogens) (Cook *et al.*, 2015). In plants, most of these receptors contain extracellular leucine-rich repeats (eLRRs) that are thought to mediate protein–protein interactions (Kobe and Kajava, 2001; Matsubayashi, 2003). Receptor-like

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proteins (RLPs) represent an important class of such cellsurface receptors. Structurally RLPs consist of two eLRR domains interrupted by an island domain, a single-pass transmembrane domain and a short cytoplasmic tail that lacks obvious motifs for intracellular signaling, except for a putative endocytosis motif found in some members (Tör *et al.*, 2009; Wang *et al.*, 2010a).

RLPs have been shown to play important roles in development and disease resistance in several plant species (Kruijt et al., 2005; Tör et al., 2009; Wang et al., 2010a). Two Arabidopsis RLPs. CLAVATA2 (CLV2)/AtRLP10 and TOO MANY MOUTHS (TMM)/AtRLP17, are known to play a role in plant development. While CLV2 is involved in meristem and organ development, TMM regulates stomatal distribution (Jeong et al., 1999; Nadeau and Sack, 2002; Wang et al., 2008; Wang et al., 2010b; Wang et al., 2011). Apart from CLV2 and TMM, most RLPs characterized to date have been found to be involved in disease resistance. These include the Cf proteins, mediating resistance to the fungal pathogen Cladosporium fulvum (Rivas and Thomas, 2005; Thomma et al., 2005; Stergiopoulos and de Wit, 2009); LeEIX, mediating recognition of the ethylene-inducing xylanase (EIX) of the biocontrol fungus Trichoderma viride (Ron and Avni, 2004); HcrVf-2, conferring resistance to the apple scab fungus Venturia inaequalis (Belfanti et al., 2004); LepR3, providing race-specific resistance to the fungal pathogen Leptosphaeria maculans (Larkan et al., 2013); and Ve1, mediating resistance towards Verticillium vascular fungi expressing the avirulence gene Avel (Fradin et al., 2009; Fradin et al., 2011).

Over the years, an increasing number of Arabidopsis RLPs (AtRLPs) have been assigned functions in pathogen resistance. We reported previously the assembly of a genomewide collection of T-DNA insertion lines for the 57 AtRLP genes in the Arabidopsis genome (Wang et al., 2008). After an extensive screening only a few novel phenotypes were discovered, including the reported phenotypes for CLV2 and TMM. While AtRLP41 was found to mediate abscisic acid (ABA) sensitivity, AtRLP30 and AtRLP18 were found to influence non-host resistance towards Pseudomonas syringae pv. phaseolicola (Wang et al., 2008). In addition, AtRLP52 is required for basal defense against the powdery mildew pathogen Erysiphe cichoracearum (Ramonell et al., 2005). SNC2/AtRLP51 and AtRLP55 were suggested to be implicated in basal defense against the bacterial pathogen Pseudomonas syringae pv. tomato DC3000 (Zhang et al., 2010). ReMAX/AtRLP1 was found to provide recognition of eMAX from Xanthomonads (Jehle *et al.*, 2013), while the fungal pattern sensor RBPG1/AtRLP42 confers resistance to fungal endo-polygalacturonases (Zhang et al., 2014). RFO/ AtRLP3 has been implicated in resistance to the vascular wilt fungus Fusarium oxysporum forma specialis matthioli (Shen and Diener, 2013). As a final example, AtRLP23 was recently found to perceive a conserved 20-amino-acid fragment present in most necrosis and ethylene-inducing peptide (NEP) 1-like proteins, thereby mediating immune activation that, similar to what was observed for the Cf proteins, is dependent on SOBIR1 and SERK3/BAK1 (Liebrand et al., 2013; Albert et al., 2015; Postma et al., 2016). However, the biological functions of the majority of the *AtRLP* genes still remain unclear.

The major challenge currently is to understand the biological function of AtRLP genes that lack an obvious phenotype in a single mutant background (Wang *et al.*, 2008). One reason is the lack of suitable screening conditions in which the phenotype might only be visible in a conditionspecific manner. Interestingly, studies on several AtRLP genes have revealed gene expression changes, as well as the emergence of phenotypic alterations, with specific elicitors (Wang et al., 2008; Wang et al., 2010a). Therefore, it may be necessary to test a broad range of physiological conditions, in combination with high-resolution screening for phenotypes. To this end, a comprehensive profile of the transcriptional response of AtRLP genes under various conditions, including exposure to biotic and abiotic stress and hormones, will be very helpful. The lack of assignment of biological functions to *AtRLP* genes may also be explained by a strong functional redundancy among the various AtRLP genes (Wang et al., 2008). In particular, most of the closely related AtRLP genes are located at one locus on the chromosomes (Fig. 1), making it impossible to generate highorder mutant combinations. RNA interference studies that silence multiple AtRLP genes simultaneously also failed to uncover new biological functions for several sets of closely related AtRLP genes (Ellendorff et al., 2008). As an alternative approach, analysis of the gain-of-function phenotypes has yielded valuable information on the function of AtRLP genes, including TMM, Vel and AtRLP23 (Fradin et al., 2009; Fradin et al., 2011; Yan et al., 2014; Albert et al., 2015).

To overcome the functional redundancy and further understand the role of AtRLP genes, in this work we studied the evolution of AtRLP genes and compiled a comprehensive profile of the transcriptional regulation of AtRLP genes upon exposure to environmental stresses and hormones. This will help to select AtRLP genes that might be involved in a specific biological process for further experimental studies aimed at dissecting AtRLP function. In addition, we performed a genome-wide cloning of AtRLP genes, and generated and characterized transgenic Arabidopsis overexpressing individual AtRLP genes. The data presented in this study provide valuable resources for future investigations into the biological role of AtRLP genes.

Materials and methods

Plant materials and growth conditions

The Arabidopsis ecotypes Columbia (Col-0) and Landsberg *erecta* (Ler) were used as wild-types (WT) for all phenotypic analyses. The *clv2-1* and *rlp10-1* mutants were described previously (Kays and Clark, 1998; Wang *et al.*, 2008). Plants were grown in soil in the greenhouse or on 1/2 MS medium supplemented with 1% sucrose under a 16h light–8h dark regime at 22 °C. For the *in vitro* growth of Arabidopsis plants, seeds were surface-sterilized for 3h by mixing 10mL water, 10mL 99% NaClO and 5mL 99% HCl, and subsequently sown on 1/2 MS solidified with 1% agar. The plates were incubated at 4 °C in the dark for 3 days and transferred to growth chambers.



Fig. 1. AtRLP genes scatter over the different chromosomes of the Arabidopsis genome. The numbers at the top indicate the chromosome number.

Chromosomal locations and analysis of duplication of genes in the AtRLP gene family

The chromosomal location of each member of the *AtRLP* family and its location-related *receptor-like kinase* (*RLK*) gene was determined with the Chromosome Map Tool at TAIR (http://www.arabidopsis.org/jsp/ChromosomeMap/tool.jsp). The location of each gene in relation to major chromosomal duplication events in the Arabidopsis genome was determined with tools provided at http:// wolfe.gen.tcd.ie/athal/dup and/or defined by Blanc *et al.* (2003). Tandem duplicated genes were identified based on criteria described by Shiu and Bleecker (2003). Briefly, tandem repeats of *AtRLP* genes were defined as genes that are located within 30 kb or are separated by five or fewer non-homologous spacer genes.

Gene expression data analysis

The gene expression data of each experiment presented in this study were obtained from AtGenExpress (http://www.weigel-world.org/resources/microarray/AtGenExpress) (Kilian *et al.*, 2007; Goda *et al.*, 2008), and were normalized using the GC-RMA method (Wu *et al.*, 2004). Fifty-two of the 57 *AtRLP* genes were available in the dataset. The fold-change of expression of *AtRLP* genes under each condition was determined by the expression change relative to the respective controls. Fold-change ratios were subsequently transformed to log₂ values to indicate the transcript change. We set a two-fold change threshold as a cut-off to identify differentially expressed genes, in which the false discovery rate was found to be around 0.2% (Zhu and Wang, 2000). Overlapping of *AtRLP* gene sets is defined as *AtRLP* genes that display similar responses to the selected conditions.

AtRLP cloning and generation of transgenic plants

The primers to amplify AtRLP genes were designed according to the predicted ORF sequences that were retrieved from the TAIR database (https://www.arabidopsis.org). Total RNA was extracted from Arabidopsis seedlings using the EZNATM Plant RNA Kit (Omega, USA) and reverse-transcribed into cDNA using the RevertAidTM First Strand cDNA Synthesis Kit (MBI, Fermentas, USA). The PCR reaction was conducted using PhusionTM High-Fidelity DNA Polymerase (Finnzymes, Finland). The PCR products were subsequently purified using the Quick DNA Purification Kit (Cwbio, China). The purified PCR products were cloned into pDONR207 and sequenced. After sequence verification, all entry clones were subsequently recombined into the destination vector pGD625 that contained the CaMV 35S promoter through LR recombination reactions. In the case of AtRLP32 and AtRLP46, CaMV 35S promoters containing destination vectors pFAST-R02 and pB2GW7, respectively, were used.

The resulting constructs were introduced into *Agrobacterium tumefaciens* and transformed into WT plants or the *clv2-1* or *rlp10-1* mutants using the floral dip method (Clough and Bent, 1998). Seeds from transformed plants were selected using corresponding antibiotics until at least three homozygous transgenic lines were obtained for each *AtRLP* gene.

Stress induction and gene expression analysis by quantitative real-time RT-PCR (qPCR)

The sterilized seeds were sown in 1/2 MS liquid medium containing 1% (w/v) sucrose. After sowing, the medium was incubated at 4 °C

in the dark for 3 d and subsequently grown on a roller shaker for 7 d with 16h light–8h dark at 21 °C. For NaCl and mannitol induction, the seedlings were treated with 150 mM NaCl and 400 mM mannitol, respectively, and sampled at 0, 3, and 6h. The qPCRs were performed in triplicate with SYBR Green PCR Master Mix (Thermo Scientific) using a Bio-Rad IQ5 (Bio-Rad). The *Actin2* gene was used as control to normalize expression levels. For each independent biological replicate, the relative transcript amount was calculated as the mean of three technical replicates. The relative expression levels were normalized to a value of 1 in the respective control samples. All primers used for qPCRs are listed in Supplementary Table S1 at *JXB* online.

Phenotypic analyses

The carpel number was counted using mature siliques under a dissection microscope. For biological statistics, there was a minimum of 30 plants of which 20 siliques per plant were counted to determine the mean carpel numbers for individual genotypes. For the salt and mannitol tests, seeds were sown on 1/2 MS medium supplemented with different concentrations of salt and mannitol as indicated. The germination rate was analysed in triplicate for each line (around 60 seeds each).

Results and discussion

Duplication of AtRLP genes in the Arabidopsis genome

We found that AtRLP genes are distributed over all five chromosomes with many clusters containing two or more AtRLP genes (Fig. 1), suggesting a major role of tandem duplications in the enlargement of the AtRLP gene family. We therefore investigated the evolutionary relationship and duplication events of AtRLP family members. To this end, we determined the chromosomal locations and the duplicated chromosome segments in which AtRLP genes are found (Fig. 1 and Table 1). A total of 35 AtRLP genes were found to be present in tandem repeats, representing about 60% of all AtRLP genes (Table 1). Out of 57 AtRLP genes, 27 were found in the hypothesized duplicated regions, whereas 30 were located outside these regions (Table 1). Within the duplicated regions, 11 AtRLP genes were found to have duplicated pair(s), whereas the remaining 16 AtRLP genes were found to have no corresponding duplicated pair although their locations were surrounded by duplicated segments (Table 1). Specifically, four pairs of AtRLP genes (AtRLP23 and AtRLP42; AtRLP33 and AtRLP53; AtRLP44 and AtRLP57; and AtRLP51 and AtRLP55) constitute pairs of duplicated genes in segmental duplicated blocks of chromosomes. In addition, three AtRLP genes, AtRLP1, AtRLP4 and AtRLP17/TMM, were found to be duplicated counterparts of the eLRR-containing

Table 1. Duplication of AtRLP genes in the Arabidopsis genome

	Outside the duplication region	Within the duplication region			
		With duplicated genes	Without duplicated genes		
Singular	3	8	11		
Tandem repeats	27	3	5		
Total	30	11	16		

genes At1g74360 (*eLRR-receptor-like kinase* (*RLK*) gene), At2g14440 (*eLRR-RLK* gene) and At3g126102 (containing an eLRR domain), respectively. No traceable duplication event was found for three genes, namely *AtRLP7*, *AtRLP36*, and *AtRLP52*. Altogether, these results suggest that a major role of the tandem duplications, in conjunction with segmental duplications, has been to contribute to enlargement of the *AtRLP* gene family in Arabidopsis.

In a previous study, Shiu and Bleecker (2003) found that some of the AtRLP genes locate close to an RLK. We identified nine AtRLP genes that were located in relatively close proximity (10 predicted genes) to an *RLK* gene (see Supplementary Table S2 at JXB online), thus resulting in RLP-RLK combinations. To determine a possible functional significance of such associations, the expression patterns of these genes were compared using ATTED-II (Obayashi et al., 2014). Unfortunately, co-expression could not be confirmed for any of the AtRLP-RLK combinations, suggesting that these RLP-RLK combinations do not have a biological relevance. Indeed, for instance, sequence comparison revealed that AtRLP52 and At5g25930, encoding an RLK, show a high degree of similarity in their extracellular domains (see Supplementary Fig. S1 at JXB online), while they do not exhibit an overlap in expression patterns. It is also possible that several of these AtRLP genes that are located in *RLK* gene clusters may have arisen through unequal crossovers (Shiu and Bleecker, 2003), as might be the case for AtRLP52 and At5g25930.

AtRLP genes display comprehensive and distinct transcriptional regulation upon exposure to external stimuli and hormones

It was suggested that the majority of AtRLP genes are involved in plant defense, as has been shown by phylogenomic analysis (Fritz-Laylin et al., 2005). The data suggest potential transcriptional regulation of AtRLP genes upon exposure to environmental stimuli. Therefore, we started to investigate the transcriptional regulation of the entire AtRLP gene family by external stimuli and hormones. The availability of microarray datasets allowed us to identify specifically regulated AtRLP genes. To deepen our understanding of the transcriptional regulation of the AtRLP genes, we explored and visualized the expression of the genes under various growth conditions by using AtGenExpress (Kilian et al., 2007). AtRLP27, AtRLP38, AtRLP50, AtRLP51 and AtRLP53 are not present in the AtGenExpress, which resulted in a total of 52 AtRLP genes that were analysed in our study. AtRLP8 was found not to exhibit a response to any treatment tested (Supplementary Table S3). An overview of the differentially expressed AtRLP genes for all conditions tested can be found in Supplementary Tables S4-S7 at JXB online. As the environmental stimuli tested were by no means comprehensive, it is possible that AtRLP8 expression is responsive to other environmental factors. Only AtRLP29 was differentially expressed upon various light treatments (Supplementary Table S7), suggesting that the expression of most AtRLPgenes is not perturbed by light. To confirm the validity of the microarray data, we selected AtRLP23, AtRLP28, AtRLP30, *AtRLP33* and *AtRLP37*, based on our study interests, to examine their expression in response to NaCl and mannitol at different time points. By qPCR, we confirmed that out of 20 samples tested, 14 showed similar expression patterns to the microarray data (Supplementary Fig. S2 at *JXB* online), which represents most of the genes and the two treatments.

A total of 51 *AtRLP* genes exhibited differential expression, with a two-fold change or more, under at least one of the tested conditions (Fig. 2, Table 2 and Supplementary Tables S4–S7). A list of *AtRLP* genes showing the highest differential expression per treatment is presented in Table 2. We found that a number of up-regulated *AtRLP* genes were significantly over-represented for multiple stress conditions and hormones tested (Fig. 2, Table 2 and Supplementary Tables S4–S7). In particular, the up-regulated *AtRLP* genes were significantly enriched among seven of the nine abiotic stress conditions and nine of the 11 biotic stress conditions analysed (Fig. 2, Table 2 and Supplementary Tables S4–S7). Amongst all conditions tested, biotic stresses perturbed the expression of the largest proportion of *AtRLP* genes (87%), compared with the abiotic stresses (80%) and hormone

treatments (40%) (Fig. 2). In more detail, the largest proportion of differentially expressed *AtRLP* genes was found for the treatments of UV-B, cold, heat, osmotic stress, salt stress, ABA, bacterial pathogens and bacterial pattern HrpZ (Fig. 2 and Supplementary Tables S4–S7).

Interestingly, the down-regulated AtRLP genes were also enriched in four abiotic conditions and two biotic conditions (Fig. 2, Table 2 and Supplementary Tables S4-S7). Surprisingly, no AtRLP gene was differentially expressed upon treatment with the pathogen elicitor lipopolysaccharide (Fig. 2 and Supplementary Table S5). Nevertheless, a number of AtRLP genes were differentially expressed under different hormone treatments (Supplementary Table S6). More than half (53%) of the AtRLP genes were not perturbed by hormones, whereas only seven and nine AtRLP genes were not responsive to biotic and abiotic stresses, respectively (Fig. 2 and Supplementary Table S3). This is consistent with the hypothesis that the majority of AtRLP genes are involved in plant stress signaling, as was suggested by phylogenomic analyses (Fritz-Laylin et al., 2005). Indeed, most of the functionally characterized AtRLP genes are involved in pathogen



Fig. 2. Overview of the amounts of differentially expressed *AtRLP* genes per treatment. The number and proportion of *AtRLP* genes that are differentially expressed per treatment are indicated. An *AtRLP* with a two-fold change or greater is considered as a differentially expressed *AtRLP* gene. (A) Number and proportion of differentially expressed *AtRLP* genes which are regulated by abiotic treatments. (B) Number and proportion of differentially expressed *AtRLP* genes that are regulated by biotic treatments. (D) Number and proportion of differentially expressed *AtRLP* genes that are regulated by biotic treatments. (D) Number and proportion of differentially expressed *AtRLP* genes that are regulated by biotic streatments. (D) Number and proportion of differentially expressed *AtRLP* genes in three major classes of treatments, abiotic stress, hormones, and biotic stress. The total number represents the sum of number of up-regulated and down-regulated genes. As a result of dynamic responses, the total number in some cases was smaller than the sum of up-regulated and down-regulated genes.

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Table 2. The most transcriptionally responsive AtRLP genes per treatment

Cold APPL PST APPL PST <th< th=""><th>Treatment</th><th>Gene</th><th>Accession no.</th><th>Log₂</th><th>Treatment</th><th>Gene</th><th>Accession no.</th><th>Log₂</th></th<>	Treatment	Gene	Accession no.	Log ₂	Treatment	Gene	Accession no.	Log ₂
AFR.129 AB301550 -2.8 ARLPS ATQ5150 -2.2 AFR.129 AFG.129 ARLPS ATQ5150 -2.1 AFR.129 AFG.129 ARLPS	Cold	AtRLP33	At3g05660	7.3	Heat	AtRLP37	At3g23110	-3.0
AARLP6 AARLP6<		AtRLP37	At3g23110	-3.8		AtRLP9	At1g58190	-2.2
AFR/198 AM9/1900 2.6 APR/145 APS(200) -2.0 Ouncois APR/172 APS(200) 5.1 P syntyss APR/122 APS(200) 5.1 APR/172 APS(200) 5.1 APR/122 APS(200) 6.1 APR/1748 APS(2000) 3.5 APR/126 APS(200) 6.1 APR/1748 APS(2000) 3.5 APR/126 APS(2000) 2.6 APR/1749 APS(2000) 3.2 APR/126 APS(2000) 2.6 APR/1749 APS(2010) 3.0 APR/176 APS(2010) 2.6 APR/1749 APS(2010) 3.0 APR/176 APS(2010) 2.6 APR/174 APS(2010) 3.0 APR/176 APS(2010) 2.1 APR/174 APS(2010) 3.0 APS(2010) 3.1 APS(2010) 3.1 APR/174 APS(2010) 3.0 APS(2010) 3.1 APS(2010) 3.1 APR/174 APS(2010) 3.1 APS(2010) 3.1		AtRLP32	At3g05650	2.8		AtRLP6	At1g45616	2.1
AHLP178 AH2 AHLP36 AH2P172 AH2p120 C-0.0 AHLP27 AH2p2200 5.1 AHRLP21 AH2p22070 4.4 AHRLP20 AH2p2200 5.1 AHRLP21 AH2p22070 4.4 AHRLP20 AH2p2200 3.5 AHRLP20 AH2p220 4.5 AHRLP20 AH2p200 3.5 AHRLP20 AH2p200 4.5 AHRLP20 AH2p200 3.5 AHRLP20 AH2p200 4.5 AHRLP20 AH2p200 3.2 AHRLP20 AH2p200 2.5 AHRLP30 AH3p19100 3.2 AHRLP30 AH3p19100 2.6 AHRLP30 AH3p2010 3.6 Cinorua AHRLP30 AH3p19100 2.2 AHRLP30 AH3p20500 2.1 AHRLP30 AH3p19100 2.1 AHRLP30 AH3p20500 2.1 AHRLP30 AH3p19100 2.1 AHRLP30 AH3p20500 2.1 AHRLP30 AH3p1910 3.1 3.1 3.1 3.1 <		AtRLP49	At4g13900	2.6		AtRLP45	At3g53240	-2.1
Omnubi AFR/E33 Adag.02600 5.1 AFR/E23 Adag.22640 5.1 AFR/E43 Adag.22630 5.1 AFR/E43 Adag.22640 4.3 AFR/E43 Adag.25630 3.7 AFR/E46 4.33 AFR/E43 Adag.256308 3.5 AFR/E46 Ad3.25640 4.3 AFR/E43 Ad3.056608 4.3 Primetarus AFR/E46 Ad3.05000 2.6 AFR/E47 Ad3.056070 3.2 AFR/E46 Ad3.05000 2.6 AFR/E47 Ad3.0520710 3.0 AFR/E46 Ad3.05000 2.3 AFR/E47 Ad3.0520710 3.0 AFR/E46 Ad3.05000 2.1 AFR/E47 Ad3.0520710 3.0 AFR/E46 Ad3.05000 2.1 AFR/E47 Ad3.050750 2.1 AFR/E46 Ad3.05000 2.1 AFR/E47 Ad3.050750 1.1 AFR/E47 Ad3.05000 2.1 AFR/E47 Ad3.050750 1.6 AFR/E47 Ad3.05000 2.1 <td< td=""><td></td><td>AtRLP18</td><td>At2g15040</td><td>-2.4</td><td></td><td>AtRLP54</td><td>At5g40170</td><td>-2.0</td></td<>		AtRLP18	At2g15040	-2.4		AtRLP54	At5g40170	-2.0
ARP. R9 Ang. Pag. Ang. Pag. Ang. Pag. ArP. Pag. Ang. Pag. Ang. Pag. Pag. Pag. Pag. Pag. Pag. Pag. Pa	Osmotic	AtRLP33	At3g05660	5.4	P. syringae	AtRLP22	At2g32660	5.1
ARIL Mag Alag 13800 3.7 ARIL Prop ARR Prop Alag 2460 4.3 ARL Prop Alag 26800 3.5 ARR Prop Alag 26800 -4.1 ARR Prop Alag 26800 3.5 ARR Prop Alag 16800 2.6 ARR Prop Alag 26800 3.5 ARR Prop Alag 1600 2.6 ARR Prop Alag 2600 3.0 ARR Prop Alag 1600 2.2 ARR Prop Alag 2600 3.0 ARR Prop Alag 1604 2.2 ARR Prop Alag 2600 3.0 ARR Prop Alag 1604 2.6 Drought ARR Prop Alag 2600 3.0 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600 3.0 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600 3.0 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600 3.1 ARR Prop Alag 2600		AtRLP23	At2g32680	5.1		AtRLP21	At2g25470	4.4
AHL/H09 AH302000 3.5 AHR.190 A4320500 4.43 AHR.193 AH3025000 4.5 Printestans AHR.191 AA3015000 2.8 AHR.193 AH3075000 3.2 AHR.191 AA3015000 2.8 AHR.194 AH301900 3.2 AHR.194 AH301900 2.5 AHR.197 AH323010 3.2 AHR.194 AH301900 2.6 AHR.197 AH323100 3.5 B. cineres AHR.196 AA31100 2.6 AHR.197 AH323100 3.5 B. cineres AHR.196 AA31100 2.1 AHR.197 AH323100 3.8 E. cronti AHR.197 AH321000 2.1 AHR.197 AH32100 3.1 AHR.198 AH321000 3.1 AHR.197 AH32100 3.1 AHR.293 AH321000 3.2 AHR.193 AH32100 1.6 AHR.194 AH322000 3.1 AHR.293 AH321000 3.2 AHR.193 AH321000		AtRLP49	At4g13900	3.7		AtRLP20	At2g25440	4.3
ARILP28 Adag23080 3.5 ARILP28 Arag23050 4.4 Sait ARILP27 Alsg0F80 3.2 ARILP30 A2 ARILP37 Alsg0F80 3.2 ARILP30 A33 ARILP30 A33 ARILP38 Adsg1710 3.0 ARILP38 Arg17130 2.3 ARILP38 Adsg1710 3.3 ARILP38 Arg17130 2.3 ARILP38 Adsg1710 3.3 B. chorca ARILP38 Arg17130 2.4 ARILP37 Alsg05800 2.1 ARILP38 Arg17130 2.1 Arg1713 2.4 ARILP37 Alsg05800 2.1 ARILP38 Arg17100 2.4 Arg17130 2.1 Arg17130 2.1 Arg17130 2.1 Arg17130 2.2 Arg171300 2.2		AtRLP46	At4g04220	3.5		AtRLP40	At3g24954	4.3
Sait ARILP3 Atag.1000 4.3 P. infestions ARILP30 Atag.1000 2.8 ARILP40 Atag.1000 3.2 ARILP30 Atag.1000 2.4 ARILP37 Atag.2010 2.5 ARILP38 Atag.1000 2.4 ARILP37 Atag.2010 2.5 ARILP38 Atag.1040 4.4 Drought ARILP37 Atag.2010 2.5 ARILP38 Atag.1040 4.8 ARILP37 Atag.2010 2.5 ARILP38 Atag.1040 4.8 ARILP37 Atag.2010 3.3 B. cheres ARILP38 Atag.10500 2.1 ARILP38 Atag.1000 1.8 C. corrii ARILP38 Atag.1000 3.7 ARILP3 Atag.2010 1.3 ARILP38 Atag.2010 3.7 ARILP3 Atag.2010 1.3 ARILP38 Atag.2010 3.7 ARILP3 Atag.2010 1.3 ARILP38 Atag.2010 3.7 ARILP3 Atag.2010 1.4 ARILP39		AtRLP28	At2g33080	3.5		AtRLP26	At2g33050	-4.1
ARLP7 Arlg Ar80 3.2 ARLP40 Arlg Ar80 2.4 ARLP37 Al3g23110 3.0 ARRP18 Ar12g1500 2.3 ARLP36 Al3g26010 2.6 ARRP18 Ar12g1500 2.3 ARRP38 Al3g2600 3.9 ARRP18 Ar12g1500 2.3 ARRP3 Al3g2600 3.9 ARRP18 Ar12g1500 2.4 ARRP3 Al3g26600 2.1 AR1P48 Ar12g3500 2.1 ARRP3 Al3g2660 2.1 AR1P48 Ar12g3500 2.1 ARRP23 Al3g2680 2.3 AR1P48 Ar12g3500 3.2 ARRP23 Al3g2680 2.3 AR1P48 Ar12g3500 3.1 ARRP27 Al3g2510 1.7 AR1P478 Ar12g3500 3.1 ARRP3 Ar12g3500 1.9 AR1P28 Ar12g3500 3.1 ARRP3 Ar12g3500 1.9 AR1P28 Ar12g3200 3.1 ARRP3 Ar12g3200 1.8 AR1P28<	Salt	AtRLP33	At3g05660	4.3	P. infestans	AtRLP19	At2g15080	2.8
ARLP40 Ard[1500 3.2 ARLP40 Ard[1500 2.4 ARR.P36 Al3g2010 2.6 ARRP46 Ard[1500 2.2 ARR.P36 Al3g2010 2.6 ARRP46 Ard[1500 2.2 ARR.P37 Al3g21500 3.5 ARRP46 Ard[1500 2.4 ARR.P37 Al3g2500 -2.2 ARRP47 Al3g0560 2.1 ARRP28 Al3g0560 2.1 ARRP28 Al3g0560 2.1 ARRP28 Al3g0560 3.7 Al4g1300 2.4 Al4g1300 2.4 Al4g1300 2.1 ARRP28 Al4g1300 3.7 Al4g1280 2.2 Al4g1300 3.7 Al4g1280 3.7 Al4g1280 3.7 Al4g1280 3.7 Al4g1280 3.7 Al4g1280 3.7 Al4g1280 3.8 Al4g1280 3.8 Al4g1290 Al4g1280 3.8 Al4g1290 3.7 Al4g1280 3.8 Al4g1290 3.8 Al4g1290 3.8 Al4g1290 3.8 Al4g1290 3.8 Al4g1290 3.		AtRLP7	At1g47890	3.2		AtRLP30	At3g05360	2.6
ARE.PS7 A3922110 3.0 ARE.PS6 A		AtRLP49	At4g13900	3.2		AtRLP49	At4g13900	2.4
APRLP36 A322010 2.6 APRLP36 Ar32200 2.6 Drought APRLP38 A322010 3.9 B. cnwraa APRLP30 A231500 -4.6 APRLP37 A4323110 3.9 B. cnwraa APRLP30 AA3035380 2.1 APRLP37 A4302500 1.9 APRLP20 APRLP30 2.2 APRLP37 A4302500 1.9 APRLP37 AP302500 3.7 APRLP37 A4302310 3.1 APRLP31 AP302500 3.7 APRLP37 A4302310 3.1 APRLP33 AP302500 2.9 APRLP37 A4302310 2.9 APRLP33 AP302580 2.9 APRLP37 A13023310 2.9 APRLP30 APRLP30 APRLP30 3.0 APRLP37 AP30238080 1.8 APRLP30 APR0 3.0 3.0 APRLP30 AP30238080 1.8 APRLP30 AP3025800 2.3 APRLP30 AP30238080 5.5 APRLP30 AP302580		AtRLP37	At3g23110	3.0		AtRLP18	At2q15040	2.3
AHR_178 AH3_02400 3.9 AHR_178 AH3_02400 3.9 Dought AHR_178 AH3_0560 3.1 AHR_178 AH3_0560 2.1 AHR_178 AH3_0500 3.1 AHR_178 AH3_0500 3.8 E cronth AHR_178 AH3_0500 3.7 AHR_178 AH3_0500 3.8 E cronth AHR_178 AH3_0500 3.7 AHR_178 AH3_022880 2.3 AHR_178 AH3_0580 3.2 AHR_178 AH3_022880 2.3 AHR_178 AH3_0580 2.8 AHR_178 AH3_022880 3.1 AHR_178 AH3_0580 2.8 AHR_178 AH3_02380 1.9 AHR_178 AH3_0580 2.8 AHR_178 AH3_02580 1.3 AHR_178 AH3_0580 2.8 AHR_178 AH3_02580 1.4 AHR_172 AH3_02580 2.8 AHR_178 AH3_02580 5.5 AHR_172 AH3_02580 2.8 AHR_178 AH3_02580 <		AtRLP36	At3q23010	2.6		AtRLP46	At4q04220	2.2
Drought ARPLP37 ANS_023110 3.9 B. cineres APRLP39 ANS_00580 3.1 ARPLP18 ALSg15040 -2.2 ARPLP39 ALSg15040 2.1 ARPLP37 ALSg15040 -2.2 ARPLP28 ALSg25010 2.1 ARPLP37 ALSg05650 1.9 ARPLP28 ALSg25010 3.7 ARPLP37 ALSg25010 3.1 ARPLP28 ALSg25010 3.2 ARPLP3 ALSg25010 1.7 ARPLP28 ALSg2500 2.8 ARPLP3 ALSg2500 1.9 ARPLP30 ALSg2500 3.1 ARPLP3 ALSg2500 1.8 ARPLP30 ALSg2500 3.1 ARPLP3 ALSg2500 1.8 ARPLP30 ALSg2500 3.1 ARPLP3 ALSg2500 1.8 ARPLP30 ALSg2500 3.1 ARPLP3 ALSg25000 1.8 ARPLP30 ALSg2500 3.1 ARPLP3 ALSg25000 1.8 ARPLP31 ALSg25000 3.1 ARPL		AtRLP39	At3g24900	3.9		AtRLP18	At2q15040	-4.6
ARRUP16 AND 15040 2.2 ARRUP30 AAG 13800 2.4 ARRUP32 AA306660 2.1 ARRUP32 AA205300 2.1 ARRUP33 AA306660 1.9 ARRUP30 ARRUP30 3.7 ARRUP3 AA3030660 1.9 ARRUP30 ARRUP30 3.7 ARRUP3 AA30300660 2.3 ARRUP30 ARRUP30 3.1 ARRUP3 AA303110 2.1 ARRUP30 AA300300 3.1 ARRUP3 AA303110 2.9 HrpZ ARRUP30 AA300300 3.1 ARRUP3 AA302310 2.9 HrpZ ARRUP30 AA300300 3.1 ARRUP3 AA302300 1.8 ARRUP30 AA300300 3.1 ARRUP3 AA302300 1.9 ARRUP30 A3305300 3.0 ARRUP3 AA3023800 1.4 ARRUP31 A2232640 3.0 ARRUP3 AA3023800 6.5 GST-NPP1 ARRUP32 A23030600 2.3 <td< td=""><td>Drought</td><td>AtRLP37</td><td>At3q23110</td><td>3.9</td><td>B. cinerea</td><td>AtRLP30</td><td>At3q05360</td><td>3.1</td></td<>	Drought	AtRLP37	At3q23110	3.9	B. cinerea	AtRLP30	At3q05360	3.1
ARR_P32 AdSq05650 2.1 ARR_P23 AdSq05660 2.1 ARR_P33 AdSq05660 1.9 ARR_P21 AdSq25401 1.8 Genctoxic ARR_P37 AdSq0310 3.1 ARR_P32 AdSq2501 3.7 ARR_P37 AdSq0310 3.1 ARR_P32 AdSq03680 3.1 ARR_P37 AdSq0310 1.9 ARR_P33 AdSq0580 3.1 ARR_P37 AdSq0310 1.9 HIPZ ARR_P30 AdSq0580 3.1 ARR_P3 AdSq0580 1.8 ARR_P30 AdSq0580 3.1 ARR_P3 AdSq0580 1.4 ARR_P30 AdSq0580 3.1 ARR_P3 AdSq0580 1.4 ARR_P30 AdSq0580 3.1 ARR_P3 AdSq0580 5.5 ARR_P12 AdSq0580 2.1 UV-B ARR_P3 AdSq0580 5.5 ARR_P3 AdSq0580 2.3 ARR_P3 AdSq0580 5.5 ARR_P3 AdSq0580 2.3 <td< td=""><td></td><td>AtRI P18</td><td>At2q15040</td><td>-2.2</td><td></td><td>AtRI P49</td><td>At4a13900</td><td>2.4</td></td<>		AtRI P18	At2q15040	-2.2		AtRI P49	At4a13900	2.4
ARRI P33 At3g05680 1.9 ARRIP20 A2225440 1.9 Genotoxic ARRI P49 Av4g13900 3.8 E. oronli ARRI-P1 Al222500 3.7 ARRI-P23 At2g22580 2.3 ARRI-P23 Az2g1560 3.1 ARRI-P24 At3g1010 1.9 ARRI-P23 Az2g32680 3.1 ARRI-P3 At3g1010 2.9 HrpZ ARRI-P23 At3g05860 2.6 Oxidative ARRI-P3 At3g05360 1.8 ARRI-P32 At3g05860 3.1 ARRI-P3 At3g05860 1.8 ARRI-P32 At3g05860 3.0 ARRI-P3 At3g05860 1.8 ARRI-P32 At3g05860 2.8 ARRI-P3 At3g05860 5.5 ARRI-P32 At3g05860 2.8 ARRI-P3 At3g05860 4.8 ARRI-P32 At2g32680 1.8 ARRI-P3 At3g05860 4.8 ARRI-P32 At2g32680 3.8 ARRI-P3 At3g05860 5.5 ARRI-P32 <t< td=""><td></td><td>AtRI P32</td><td>At3q05650</td><td>2.1</td><td></td><td>AtRI P25</td><td>At2q33030</td><td>2.1</td></t<>		AtRI P32	At3q05650	2.1		AtRI P25	At2q33030	2.1
Genotoxic AIRLP49 Alag13900 3.8 E. oronli AIRLP47 Al3g25010 3.7 ARRLP37 Al3g250110 3.1 ARRLP3 Al2g15040 3.2 ARRLP37 Al3g25010 3.1 ARRLP35 Al2g15040 3.2 ARRLP3 Al3g25302 2.3 ARRLP3 Al2g15080 2.9 ARRLP3 Al3g25310 2.9 HrpZ AlRLP30 Al3g05360 3.1 ARRLP3 Al3g25300 1.8 ARRLP21 Al3g25470 3.0 ARRLP30 Al3g25800 -1.4 ARRLP21 Al3g25600 2.1 ARRLP3 Al2g32680 -1.4 ARRLP21 Al3g25600 2.8 ARRLP31 Al3g05800 5.6 ARRLP22 Al2g32680 2.8 ARRLP34 Al4g13900 4.8 ARRLP30 Al3g05800 2.8 ARRLP32 Al3g05800 4.8 ARRLP32 Al3g05800 3.8 ARRLP33 Al3g05860 4.6 Fig22 ARRLP32 Al3g2580		AtRI P33	At3q05660	1.9		AtRI P20	At2a25440	1.8
Dokumon Alfigue27 Algg23110 3.1 Alfiller Alfigue23 Algg23680 3.1 ARRLP3 Algg23680 2.3 ARRLP3 Algg23680 3.1 ARRLP3 Algg23680 2.3 ARRLP3 Algg23680 3.1 ARRLP3 Algg23680 1.9 ARRLP3 Algg23680 3.1 ARRLP3 Algg23680 1.9 HrpZ ARRLP30 Algg25670 3.0 ARRLP3 Algg25800 1.8 ARRLP1 Algg26470 3.0 ARRLP3 Algg25800 -1.4 ARRLP1 Algg26460 2.6 ARRLP3 Algg25800 -1.4 ARRLP22 Algg26460 2.4 ARRLP3 Algg25800 -1.4 ARRLP22 Algg26800 2.4 ARRLP4 Algg25800 -1.4 ARRLP21 Algg2680 2.4 ARRLP3 Algg25800 5.5 ARRLP32 Algg2680 2.3 ARRLP4 Algg25800 4.6 ARRLP32 Algg23800 2.8	Genotoxic	AtRI P49	At4a13900	3.8	E orontii	AtRI P41	At3a25010	3.7
ARRLP3 Alsg22800 2.3 ARRLP3 Alsg22800 3.1 ARRLP7 Alfg47800 1.9 ARRLP3 Alsg22800 2.6 ARRLP3 Alsg211010 2.9 HrpZ ARRLP3 Alsg25800 3.1 ARRLP37 Alsg25800 1.9 ARRLP32 Alsg25800 3.1 ARRLP37 Alsg25800 1.8 ARRLP21 Alsg2580 3.0 ARRLP30 Alsg25800 -1.4 ARRLP21 Alsg2580 2.6 ARRLP30 Alsg25800 -1.4 ARRLP21 Alsg25800 2.8 ARRLP30 Alsg25800 5.5 ARRLP12 Alsg25800 2.3 ARRLP30 Alsg25800 5.5 ARRLP10 Alsg25800 2.3 ARRLP30 Alsg23310 4.8 AIRLP10 Alsg2580 1.8 Wounding ARRLP30 Alsg2580 2.3 ARRLP30 Alsg2580 3.5 ARRLP37 Alsg2580 2.3 ARRLP30 Alsg2580 2.3 ARRLP	Conotoxio	AtRI P37	At3a23110	3.1	E. oronta	AtRI P18	At2q15040	3.0
Angle So Algo So 2.3 Angle So 3.1 ARR.P34 Algo F800 1.9 ARR.P30 Algo Sos 2.9 ARR.P34 Algo F800 1.9 ARR.P30 Algo Sos 2.9 ARR.P34 Algo Sos 1.9 ARR.P30 Algo Sos 3.1 ARR.P34 Algo Sos 1.9 ARR.P30 Algo Sos 3.1 ARR.P32 Algo Sos 1.8 ARR.P21 Algo Sos 3.1 ARR.P32 Algo Sos 5.5 ARR.P40 Algo Sos 2.8 ARR.P30 Algo Sos 5.5 ARR.P31 Algo Sos 2.8 ARR.P46 Algo Sos 5.5 ARR.P30 Algo Sos 2.8 ARR.P47 Algo Sos 5.5 ARR.P30 Algo Sos 2.8 ARR.P47 Algo Sos 4.8 ARR.P30 Algo Sos 2.8 ARR.P47 Algo Sos 4.8 ARR.P30 Algo Sos 2.8 ARR.P47 Algo Sos 4.8 ARR.P30		Atric 07	At2a32680	2.3		Atric 10	At2g73040	3.1
Angle Market Angle Mass 1.3 Angle Mass 2.6 Oxidative ARR/P37 Al3g23110 2.9 HrpZ ARR/P30 Al3g05360 3.1 ARR/P37 Al3g25380 1.8 ARR/P12 Al2g2680 3.1 ARR/P30 Al3g05380 1.8 ARR/P12 Al2g2680 3.0 ARR/P32 Al2g2680 -1.4 ARR/P12 Al2g2680 2.8 ARR/P32 Al2g2680 5.5 ARR/P12 Al2g2680 2.8 ARR/P37 Al3g05380 5.5 ARR/P10 Al3g05380 2.3 ARR/P37 Al3g05310 4.8 ARR/P7 Al1g47890 1.9 ARR/P37 Al3g05660 4.6 Fig22 ARR/P7 Al1g47890 1.8 Wounding ARR/P32 Al3g05650 2.3 ARR/P32 Al3g05650 2.3 ARR/P32 Al3g05650 2.3 ARR/P32 Al2g32680 3.8 ARR/P32 Al3g05650 2.3 ARR/P32 Al2g32680 2.6		AUNLI 23 Atel e7	At1a/7800	2.0		AUNLI 23 Atel p35	Al2932000	20
And LP37 Al3g1010 L7 And LP30 Al3g05360 2.0 Oxidative ARILP37 Al3g05380 1.1 ARILP30 Al3g05380 3.1 ARILP33 Al2g32880 1.8 ARILP21 Al2g32860 3.0 ARILP33 Al2g32880 -1.4 ARILP32 Al3g05380 2.8 ARILP30 Al3g05380 5.5 ARILP32 Al3g23805 2.8 ARILP30 Al3g05380 5.5 ARILP32 Al3g05380 2.3 ARILP30 Al3g05380 5.5 ARILP32 Al3g05380 2.3 ARILP33 Al3g05380 5.5 ARILP32 Al3g05380 2.3 ARILP33 Al3g05860 4.8 ARILP32 Al3g05380 2.3 ARILP49 At4g13900 4.7 ARILP32 Al3g05380 2.3 ARILP44 Al4g13900 4.7 ARILP32 Al2g32800 3.8 ARILP42 Al4g13900 4.7 ARILP32 Al2g32860 2.3 ARILP42		AUNLI 7 Atol D24	At2a11010	1.5		AUNLI 33	At2405260	2.9
OAAdame AndLP30 AbgLo110 2.3 InfL AndLP2 AbgLo300 3.1 ARLP30 Ad305380 1.8 ARLP21 Al2g32660 3.1 ARLP30 Ad305380 1.8 ARLP21 Al2g32680 3.0 ARLP41 Al3g25010 -1.4 ARLP21 Al2g32680 2.6 ARLP40 Al3g25010 -1.4 ARLP22 Al2g32680 2.8 ARLP46 Al4g04220 4.8 ARLP23 Al3g05360 2.3 ARLP46 Al4g04220 4.8 ARRP23 Al3g05360 2.3 ARRP46 Al4g04220 4.8 ARRP23 Al3g05360 2.3 ARRP47 Al3g23110 4.8 ARRP23 Al3g05860 3.5 ARRP23 Al3g05860 2.3 ARRP23 Al3g05860 2.3 ARRP42 Al3g05860 2.3 ARRP30 Al3g05860 2.4 ARRP24 Al3g05860 2.3 ARRP30 Al3g05860 2.4 ARRP3 <t< td=""><td>Ovidativa</td><td>ALNLF 34</td><td>Alog11010</td><td>1.7</td><td>Um7</td><td>ALALF30</td><td>A13905300</td><td>2.0</td></t<>	Ovidativa	ALNLF 34	Alog11010	1.7	Um7	ALALF30	A13905300	2.0
ARILPY9 ARIQ (38.0) 1.9 ARILP22 AR0252470 3.0 ARILP23 AR2932880 -1.4 ARILP21 AR025470 2.6 ARILP41 AR395300 -1.4 ARILP22 AR0252680 2.6 ARILP43 AR2932880 6.0 GST-NPP1 ARILP22 AR0252680 2.8 ARILP30 AR3905880 5.5 ARILP32 AR0305800 2.3 ARILP44 Ardq13900 4.7 ARILP32 AR0305360 2.3 ARILP44 Ardq13900 4.7 ARILP32 AR0252470 3.1 Wounding ARILP32 AR0305680 4.6 Fg22 ARILP32 AR0252470 3.2 ARILP40 AR0305680 2.3 ARILP32 AR030580 2.4 ARILP52 AR0305680 2.3 ARILP32 AR030580 2.4 ARILP52 AR0305680 2.3 ARILP32 AR030580 2.4 ARILP52 AR0305680 2.7 ARILP21 AR030580 2.3	Oxidative		Al3923110	2.9	hipz	ALALF30	At0g00000	0.1
ARRLP30 ARRLP30 I.63 ARRLP12 ARRLP30 Composition Composition <td></td> <td>AIRLP49</td> <td>Al4g13900</td> <td>1.9</td> <td></td> <td>AIRLP22</td> <td>AL2932000</td> <td>3.1</td>		AIRLP49	Al4g13900	1.9		AIRLP22	AL2932000	3.1
ARRLP23 AL2g32800 -1.4 ARRLP40 AIR1P10 2.0 ARRLP41 Al3g26010 -1.4 ARRLP40 Al3g24864 2.1 UV-B ARRLP30 Al3g25300 5.5 ARRLP22 Al2g32860 2.8 ARRLP30 Al3g05300 5.5 ARRLP20 Al3g05360 2.3 ARRLP37 Al3g05300 4.8 ARRLP23 Al3g05360 2.3 ARRLP37 Al3g05600 4.6 FIg22 ARRLP23 Al2g32800 1.8 ARRLP33 Al3g05600 4.6 FIg22 ARRLP23 Al2g32800 3.8 ARRLP33 Al3g05650 2.3 ARRLP30 Al3g05860 2.4 ARRLP32 Al3g05650 2.3 ARRLP30 Al3g05860 2.4 ARA ARRLP32 Al3g05650 2.3 ARRLP30 Al3g05860 2.4 ABA ARRLP32 Al3g05650 2.3 ARRLP30 Al3g0560 2.0 ARA ARRLP33 Al3g05650 2.3 ARRLP		AIRLP30	Al3g05360	1.8		AIRLP21	Al2g25470	3.0
AHRLP41 Ab3g23010 -1.4 AHRLP40 AH3g24854 2.1 UV-B AHRLP23 At3g23280 6.0 GST-NPP1 AHRLP20 A2522660 2.8 AHRLP46 AH3g03300 5.5 AHRLP30 A13g05360 2.3 AHRLP47 A13g23110 4.8 AHRLP20 A1477990 1.9 AHRLP40 A13g24954 4.1 AHRLP23 A12g32660 3.8 AHRLP40 A13g24954 4.1 AHRLP22 A12g32660 3.5 AHRLP5 A13g24954 4.1 A14g233020 3.8 AHRLP5 A13g25650 2.3 AHRLP21 A12g32680 2.4 ABA AHRLP37 A13g2580 2.3 AHRLP30 A13g05800 2.4 ABA AHRLP33 A13g05660 2.3 AHRLP40 A13g2580 2.4 ABA AHRLP33 A13g05660 2.7 AHRLP40 A13g0560 2.4 AFR AHRLP10 A11g71400 -3.0 AHRLP17 A119600		ATRLP23	At2g32680	-1.4		AtRLP12	At1g71400	2.6
OV-B AREP23 AL2932680 6.0 GST-NPP1 AREP22 AL2932680 2.8 ARR ARR ARR ARR ARR 2.8 ARR 2.8 ARR ARR ARR ARR ARR 2.8 3.3 ARR ARR ARR ARR ARR 3.3 3.3 ARR ARR ARR ARR ARR 3.333 3.333 3.333 <		ATRLP41	At3g25010	-1.4		AtRLP40	At3g24954	2.1
AHRLP30 AK390380 5.5 AHRLP20 AIT AIT <td>UV-B</td> <td>AtRLP23</td> <td>At2g32680</td> <td>6.0</td> <td>GSI-NPP1</td> <td>AtRLP22</td> <td>At2g32660</td> <td>2.8</td>	UV-B	AtRLP23	At2g32680	6.0	GSI-NPP1	AtRLP22	At2g32660	2.8
AHHLP36 At4304220 4.8 AHHLP30 At305380 2.3 AHRLP37 At3025110 4.8 AHRLP7 At1947890 1.9 AHRLP30 At3025660 4.6 FIg22 At2932680 3.8 AHRLP30 At3026560 4.6 FIg22 At293260 3.8 AHRLP30 At3905650 2.3 AHRLP21 At2925470 3.2 AHRLP32 At3905650 2.3 AHRLP30 At3924954 2.6 AHRLP32 At3905650 2.3 AHRLP30 At392580 2.3 ABA AHRLP32 At3905650 4.0 IAA AHRLP30 At392580 2.3 ABA AHRLP32 At3905650 4.0 IAA AHRLP30 At390580 2.4 ABA AHRLP32 At3905660 2.7 AHRLP30 AHRLP30 1.7 AFRLP33 At3905660 2.7 AHRLP1 At1907390 1.7 AFRLP33 At3905660 -2.2 MJ AHRLP3 At3905660 -2.9 AHRLP33 At3905660 -2.4 AHRLP1 <td></td> <td>AtRLP30</td> <td>At3g05360</td> <td>5.5</td> <td></td> <td>AtRLP12</td> <td>At1g/1400</td> <td>2.4</td>		AtRLP30	At3g05360	5.5		AtRLP12	At1g/1400	2.4
AHLP3/ At5g23110 4.8 AHLP/ At5g4890 1.9 AHRLP49 At4g13900 4.7 AHRLP23 At5g23260 1.8 Wounding AHRLP33 At5g05660 4.6 FIg22 AHRLP24 At2g32260 3.8 AHRLP40 At3g24954 4.1 AHRLP21 At2g2260 3.5 AHRLP32 At3g05650 2.3 AHRLP20 At3g24954 2.6 AHRLP32 At3g05650 2.3 AHRLP30 At3g24954 2.6 AHRLP32 At3g05650 4.0 IAA AHRLP30 At3g24954 2.6 ABA AHRLP32 At3g05650 -3.0 AHRLP30 At3g0560 2.4 AHRLP12 At1g17100 -3.0 AHRLP17 At1g03050 2.0 AHRLP33 At3g05660 2.7 AHRLP1 At1g07300 1.7 ACC AHRLP3 At3g05660 -2.2 MJ AHRLP3 At3g05660 -2.9 AHRLP1 At1g07390 1.4 AHRLP1 At1g07390 -2.5 AHRLP3 At2g32680 -1.4 <t< td=""><td></td><td>AtRLP46</td><td>At4g04220</td><td>4.8</td><td></td><td>AtRLP30</td><td>At3g05360</td><td>2.3</td></t<>		AtRLP46	At4g04220	4.8		AtRLP30	At3g05360	2.3
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ARRLP6 ARI245616 3.5 ARRLP21 At2g25470 3.2 AHRLP32 At3g05650 2.3 AtRLP40 At3g05560 2.4 ABA AHRLP32 At3g05650 4.0 IAA AHRLP23 At2g32680 2.3 ABA AHRLP10 At1g65380 -3.0 AtRLP17 At1g0080 2.0 AHRLP32 At3g05660 2.7 AtRLP14 At1g07390 1.7 AHRLP3 At3g05660 -2.2 AHRLP1 At1g07390 1.7 ACC AHRLP3 At3g05660 -2.2 MJ AtRLP1 At1g07390 -2.5 AHRLP1 At1g07390 1.4 AtRLP1 At1g07390 -2.5 AHRLP3 At3g05660 -2.2 MJ AtRLP1 At1g07390 -2.5 AHRLP1 At1g07390 1.4 AtRLP1 At1g07390 -2.5 AHRLP1 At1g07390 -1.4 AtRLP12 At1g07390 -2.5 AHRLP1 At1g07390 -1.4 AtRLP12 At1g07400 -1.7 BL AHRLP3 At2g32680 2.6<		AtRLP40	At3g24954	4.1		AtRLP22	At2g32660	3.5
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AtRLP37 At3g23110 2.1 AtRLP30 At3g05360 2.4 ABA AtRLP32 At3g05560 4.0 IAA AtRLP23 At2g32680 2.3 AtRLP10 At1g65380 -3.0 AtRLP17 At1g08080 2.0 AtRLP33 At3g05660 2.7 AtRLP46 At4g04220 1.7 AtRLP46 At4g04220 -2.2 AtRLP21 At2g25470 1.7 AtRLP53 At3g05660 -2.2 MJ AtRLP3 At3g05660 -2.9 AtRLP51 At1g07390 1.7 AtRLP3 At3g05660 -2.9 AtRLP7 At1g07390 1.7 AtRLP1 At1g07390 -2.5 AtRLP7 At1g07390 1.4 AtRLP22 At2g32660 2.1 AtRLP7 At1g07390 -1.3 AtRLP23 At3g05660 1.9 AtRLP7 At1g00380 -1.4 AtRLP33 At3g05660 1.9 AtRLP7 At1g07390 -1.3 AtRLP33 At3g05660 1.9 AtRLP7 At1g08080 2.4 AtRLP33 At3g05660 1.2		AtRLP32	At3g05650	2.3		AtRLP40	At3g24954	2.6
ABA AtRLP32 At3g05650 4.0 IAA AtRLP23 At2g32680 2.3 ARLP10 At1g65380 -3.0 AtRLP17 At1g80080 2.0 AtRLP33 At3g05660 2.7 AtRLP1 At1g07390 1.7 ACC AtRLP33 At3g05660 -2.2 MJ AtRLP33 At3g05660 -2.9 AtRLP1 At1g07390 1.7 At1g07390 -2.5 AtRLP1 At1g07390 -2.5 AtRLP1 At1g07390 1.4 AtRLP21 At3g05660 -2.9 AtRLP1 At1g07390 1.4 AtRLP33 At3g05660 -2.9 AtRLP1 At1g07390 1.4 AtRLP11 At1g07390 -2.5 AtRLP17 At1g08080 -1.4 AtRLP22 At2g32660 2.1 AtRLP17 At1g05080 2.6 Zeatin AtRLP33 At3g05660 1.7 AtRLP17 At1g05080 2.4 AtRLP33 At3g05660 1.7 AtRLP17 At1g05080 2.4 AtRLP34 At3g05660 1.7 AtRLP17 At1g05080 <td></td> <td>AtRLP37</td> <td>At3g23110</td> <td>2.1</td> <td></td> <td>AtRLP30</td> <td>At3g05360</td> <td>2.4</td>		AtRLP37	At3g23110	2.1		AtRLP30	At3g05360	2.4
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AtRLP37 At3g23110 2.1 AtRLP54 At5g40170 -1.2 AtRLP41 At3g25010 2.0 AtRLP46 At4g04220 -1.1 GA AtRLP33 At3g05660 -2.7 SA AtRLP17 At1g80080 -4.8 AtRLP10 At1g65380 -1.7 At8g11010 3.9 AtRLP7 At1g47890 -1.3 At8LP37 At3g23110 3.6 AtRLP37 At3g23110 1.3 At8LP33 At3g05660 2.7 AtRLP30 At3g23100 1.3 At8LP33 At3g05660 2.7		AtRLP17	At1g80080	2.4		AtRLP17	At1g80080	1.2
AtRLP41 At3g25010 2.0 AtRLP46 At4g04220 -1.1 GA AtRLP33 At3g05660 -2.7 SA AtRLP17 At1g80080 -4.8 AtRLP10 At1g65380 -1.7 At8LP34 At3g11010 3.9 AtRLP7 At1g47890 -1.3 At8LP37 At3g05660 2.7 AtRLP37 At3g23110 1.3 At8LP33 At3g05660 2.7 AtRLP37 At3g23110 1.3 At8LP33 At3g05660 2.7 AtRLP30 At3g05360 1.9 At3g05360 1.9		AtRLP37	At3q23110	2.1		AtRLP54	At5q40170	-1.2
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AtRLP10 At1g65380 -1.7 AtRLP34 At3g11010 3.9 AtRLP7 At1g47890 -1.3 AtRLP37 At3g23110 3.6 AtRLP37 At3g23110 1.3 AtRLP33 At3g05660 2.7 AtRLP30 At3g05360 1.9	GA	AtRLP33	At3g05660	-2.7	SA	AtRLP17	At1g80080	-4.8
AtRLP7 At1g47890 -1.3 AtRLP37 At3g23110 3.6 AtRLP37 At3g23110 1.3 AtRLP33 At3g05660 2.7 AtRLP30 At3q05360 1.9 At3q05360 1.9		AtRLP10	At1q65380	-1.7		AtRLP34	At3q11010	3.9
AtRLP37 At3g23110 1.3 AtRLP33 At3g05660 2.7 AtRLP30 At3q05360 1.9		AtRLP7	At1q47890	-1.3		AtRLP37	At3q23110	3.6
Atrace At		AtRLP37	At3a23110	1.3		AtRLP33	At3a05660	2.7
			5	-		AtRLP30	At3g05360	1.9

defense, whereas only a few of them, e.g. *AtRLP41*, are involved in the response to hormone treatments (Wang *et al.*, 2008).

It was found that with respect to abiotic stresses the most notable perturbation was observed for many AtRLP genes in the aerial parts of Arabidopsis seedlings. However, this strong induction was not seen in roots (see Supplementary Table S4 at JXB online). These data indicate that the response of AtRLP genes to abiotic stresses possibly is tissue or stage specific. In addition, we found that AtRLP genes, as a whole, tend to be upregulated by avirulent *P. syringae*, especially in the case of treatments with the avirulent strains P. syringae pv. tomato DC3000 HrcC, P. syringae pv. tomato DC3000 avrRpm1 and P. syringae pv. phaseolicola (Fig. 2 and Supplementary Table S5). However, many AtRLP genes were significantly down-regulated by P. syringae pv. tomato DC3000, a virulent bacterial strain capable of infecting Arabidopsis (Fig. 2 and Supplementary Table S5). These observations suggest that AtRLP genes are involved in basal defense networks that are suppressed by DC3000. Notably, several AtRLP genes, including AtRLP30, were significantly induced by flg22, a peptide corresponding to the most conserved domain of bacterial flagellin (Supplementary Table S5). It would be of great interest to examine whether those AtRLPs are also involved in mediating flagellin signaling.

Generally, our results indicate that a large number of AtRLP genes are responsive to abiotic and biotic stresses, as well as to hormones. In addition, our study provides an overview of the biological processes in which AtRLP genes may be involved. Given the fact that phenotypic changes often are observed under suitable physiological conditions, the specific treatments and corresponding AtRLP gene expression profiles identified here serve as a resource for targeted screening of individual AtRLP genetic mutants.

Individual AtRLP genes are transcriptionally regulated by multiple external stimuli and hormones

It has been suggested previously that some AtRLP genes might be responsive to several factors and thereby participate in different signaling pathways (Wang et al., 2008; Fradin et al., 2011). To elucidate this phenomenon, we firstly counted the number of treatments in which individual AtRLP genes were differentially expressed across all the treatments tested (see Supplementary Fig. S3 at JXB online). Of the 52 AtRLP genes, only AtRLP8 displayed no response to any of the treatments tested (Supplementary Table S3), and six AtRLP genes displayed an increase or decrease in expression under only one condition (see Supplementary Fig. S3 and Supplementary Tables S4–S7). The remaining AtRLP genes exhibited an increased or decreased expression upon more than one treatment (Supplementary Fig. S3 and Supplementary Tables S4–S7), suggesting that a single *AtRLP* gene may be involved in several physiological processes or in a common process initiated by more than one condition. Our observation that a large number (45 out of 52) of AtRLP genes respond to more than one treatment also supports the existence of extensive cross-talk and signal integration among different signaling pathways.

Next, we compared sets of three treatments that are known to induce physiological responses to identify overlapping AtRLP genes that display multiple responses (Fig. 3). In general, we found a large number of the same AtRLP genes had increased expression, while only a few overlapping AtRLP genes showed decreased expression upon any of the three treatments we examined (Fig. 3). Thus, no clear stimulusspecific AtRLP gene expression patterns could be deduced. Treatment with UV-B, fungi and bacteria showed the largest number of overlapping AtRLP genes with increased expression, namely 18, which was followed by 14 shared AtRLP genes upon exposure to hormones, biotic and abiotic stresses (Fig. 3). Osmotic stress, salt stress, and cold are intricately linked in various physiological processes (Xiong and Zhu, 2002; Zhu, 2002). With these three treatments there were five AtRLP genes with increased response and five AtRLP genes with decreased expression (Fig. 3). Eight AtRLP genes showed increased expression, while no AtRLP showed decreased expression upon exposure to SA, fungi and bacteria (Fig. 3). Among the abiotic stress-related treatments, wounding, UV-B and heat treatment shared five respondents with increased expression and two with decreased expression (Fig. 3). ABA, SA, and methyl jasmonate (MJ) are well known to have cooperative effects in plant development and plant defense (Cutler et al., 2010). Surprisingly, we found that exposure to ABA, SA, and MJ revealed no overlapping genes with either increased or decreased expression (Fig. 3). Similarly, there was very little overlap with either increased or decreased expression found for MJ, fungi, and bacteria (Fig. 3). This may indicate that some of the AtRLP genes are conditionally responsive or play a role outside of the crosstalk networks. Nevertheless, the various conditions that we analysed are not exhaustive and more overlap might be found upon additional treatments.

Around ten of the 57 AtRLP genes have already been implicated in various physiological programs, as has been discussed (Tör et al., 2009; Wang et al., 2010a). Among them, we found that the expression of ReMAX/AtRLP1, RFO/AtRLP3, AtRLP18, AtRLP23, AtRLP30 and *RBPG1/AtRLP42*, but not *AtRLP55*, is perturbed by a broad set of external stimuli and hormones (Fig. 2 and Supplementary Tables S4–S7). Surprisingly, two developmental AtRLP genes, CLV2 and TMM, are differentially expressed upon several external stimuli (Supplementary Tables S4–S7), suggesting dual functions of the encoded proteins in plant development and in the response to stress. For example, *CLV2* is repressed by virulent *P. syringae* pv. tomato DC3000 and Phytophthora infestans, while TMM is repressed by osmotic stress (Supplementary Tables S4-S5). However, AtPDO2/AtRLP4, another putative developmentally related *AtRLP* gene identified through phylogenomic analysis (Fritz-Laylin et al., 2005), exhibited no alteration in expression under most external conditions tested (Supplementary Tables S4–S7).

In conclusion, our observations reveal that a large number of AtRLP genes display differential expression upon more than one treatment, indicating that a single AtRLPgene may be involved in several physiological processes. We 3346 | Wu et al.



Fig. 3. The number of overlapping *AtRLP* genes showing differential regulation in response to different treatments. (A) The number of up-regulated overlapping *AtRLP* genes in three selective treatment sets. (B) The number of down-regulated overlapping *AtRLP* genes in three selective treatment sets as shown in (A). (C) The number of differentially expressed *AtRLP* genes in three selective treatment sets as shown in (A). (C) The number of differentially expressed *AtRLP* genes in three selective treatment sets as shown in (A) and (B). Differentially expressed *AtRLP* genes represent the sum of up-regulated and down-regulated genes. As a result of dynamic responses, the number of differentially expressed *AtRLP* genes in some cases was smaller than the sum of up-regulated and down-regulated genes.

found that 14 AtRLP genes with increased expression and eight AtRLP genes with decreased expression, a sum of 22 AtRLP genes showing differential expression, are shared

among the three major classes of treatments, abiotic stress, biotic stress, and hormones (Fig. 3). The results thus reveal a large number of overlapping AtRLP genes responding

to the examined treatments. Notably, our analysis also highlights that several known AtRLP genes exhibit distinct responses to specific treatments, which has not been described previously.

Cloning of AtRLP genes and sequence analysis of the isolated AtRLP genes

The cDNA fragments containing the respective coding sequences of individual *AtRLP* genes were obtained by RT-PCR (Supplementary Tables S8 and S9 at *JXB* online). *AtRLP18* and *AtRLP49* were not amplified, as they were annotated as pseudogenes in the TAIR10 release (see Supplementary Table S9). PCR products were obtained for 51 of the remaining 54 predicted *AtRLP21* failed to amplify by RT-PCR and were excluded from our study (Supplementary Table S9). Purified PCR products were introduced into pDONR207 to produce the entry clones. Plasmid DNA from entry clones was subsequently recombined into the CaMV 35S promoter containing binary vector pGD625, pFAST-R02 or pB2GW7 to generate over-expression constructs.

A total of 51 cDNA sequences were successfully cloned into pDONR207 and sequenced (Supplementary Table S9). Among them, 44 out of the 51 isolated clones carrying cDNA sequences were identical to those predicted in TAIR, whereas the other seven isolated sequences differed from their corresponding predictions in TAIR (Fig. 4; Supplementary Table S9). The isolated

sequence of AtRLP4 showed single base substitution, which caused a non-synonymous mutation (Fig. 4 and Supplementary Fig. S4 at JXB online). The isolated sequences of AtRLP13, AtRLP20, and AtRLP40 showed different intron-exon boundaries as compared with the predicted sequences, resulting in different gene products (Fig. 4 and Supplementary Fig. S4). The predicted introns of AtRLP24 and AtRLP52 were eliminated in their isolated sequences and have integrated as a part of the exon, which results in the presence of one single exon instead of two exons (Fig. 4 and Supplementary Fig. S4). An unpredicted exon was found in the isolated sequence of AtRLP56 (Fig. 4 and Supplementary Fig. S4). These observations indicate that the annotations are probably not correct. It is also possible that the isolated and the predicted sequences are both present in planta, which may suggest that some AtRLP genes have undergone alternative splicing, probably in different tissues and organs, or upon applying different stimuli.

In this study, a total of 51 *AtRLP* cDNAs containing complete coding sequences were generated, and these will provide useful tools for further functional analyses of this important gene family. For instance, the entry clones can be recombined into any Gateway-compatible destination vector and introduced into Arabidopsis to dissect the resulting phenotypes, which will indicate the possible functions of target genes. It was revealed previously that some conserved residues and motifs of RLPs are of functional significance (Fritz-Laylin *et al.*, 2005; Wang *et al.*, 2008; Wang *et al.*, 2010b). Therefore, the cloned genes could also be mutated in these conserved residues and/or motifs



Fig. 4. Schematic comparisons of cloned *AtRLP* sequences and predicted mRNA sequences derived from TAIR. Black boxes indicate exons, and lines between exons represent introns. Red boxes represent new exon sequences in the cloned *AtRLP* gene, and open boxes show the missed exon sequences in the cloned *AtRLP* gene. The vertical blue line indicates a single base substitution.

by site-directed mutagenesis to create mutant variants. In line with this hypothesis, it has been shown that transgenic plants expressing a mutation in the conserved GxxxG motif, which is known to aid in protein–protein interactions, that is located on the transmembrane domain of *SNC2/AtRLP51* exhibit constitutively activated defense responses (Zhang *et al.*, 2010). In summary, the resources generated in this study will provide useful tools for future functional examination of *AtRLP* genes.

Generation of transgenic Arabidopsis plants overexpressing AtRLP genes

To facilitate functional analysis, we generated transgenic plants overexpressing the individual AtRLP genes. To this end, the resulting CaMV 35S-driven expression constructs were transformed into wild-type (Col-0 and/or Ler) plants and/or the *clv2* mutant (see Supplementary Table S10 at *JXB* online). For each construct, at least 20 independent transgenic lines were initially analysed in the T2 generation, and then at least three independent homozygous lines (T3 or T4 plants) were obtained for each AtRLP gene (Supplementary Table S10). Altogether, we generated a collection of 167 homozygous overexpression (AtRLP-OX) lines for 51 AtRLP genes. This collection of transgenic plants could be used for the analysis of developmental aspects and studies on RLP function in pathogen defense and stress conditions, thus providing a valuable resource for future investigations into the biological role of AtRLPs.

Overexpression of AtRLP3 and AtRLP11 rescues the clv2-1 mutant

A phenotypical analysis of 4- to 6-week-old homozygous *AtRLP-OX* lines with respect to their growth and development under normal growth conditions did not reveal any abnormalities. Therefore, additional tests need to be performed on these *AtRLP-OX* lines to study the phenotype of various organs at multiple growth and developmental stages.

We reported previously that two AtRLPs, AtRLP2 and AtRLP12, which share high sequence similarity to CLV2, are able to rescue the *clv2* mutant phenotype when expressed under the control of the CLV2 promoter, suggesting that the specialization among CLV2, AtRLP2 and AtRLP12 is largely ascribed to differences in their expression patterns (Wang et al., 2010b). Intriguingly, AtRLP3 and AtRLP11 are duplicated genes of AtRLP2 and AtRLP12, respectively, which may suggest a similar function for these paralogues. To test this hypothesis and to investigate the biological role of AtRLP3 and AtRLP11, we analysed the transgenic plants overexpressing AtRLP3 and AtRLP11 in either the wild-type plants or the *clv2* mutant (Supplementary Table S10). Wildtype plants developed an invariant two carpels per flower, while *clv2-1* mutants produced multiple carpels per flower (Kayes and Clark, 1998; Wang et al., 2008). Interestingly, AtRLP3-OX and AtRLP11-OX transformed into the clv2-*1* mutant completely complemented its phenotype, showing a mean carpel number that is comparable to the wild-type plants (Fig. 5), which is similar to what has been shown for AtRLP2 and AtRLP12 (Wang et al., 2010b). The overall growth and appearance of AtRLP3-OX and AtRLP11-OX in the wild-type were indistinguishable from wild-type plants grown under normal growth conditions. Additionally, the atrlp3-1 and atrlp11-1 mutants displayed no meristem defects (Wang et al., 2008), despite our observation that AtRLP3-OX and AtRLP11-OX are able to rescue the phenotype of the clv2-1 mutant. These results suggest that the functional diversity among these closely related genes is primarily due to divergence of gene expression, rather than of their proteincoding regions. CLV2 exhibited overlapping expression with AtRLP2, AtRLP3, AtRLP11 and AtRLP12 in some organs, suggesting that CLV2 may have overlapping functions with these members in those organs.

It has been shown that *RFO2/AtRLP3* confers resistance to the vascular wilt fungus *Fusarium oxysporum*, whereas *AtRLP2* does not (Shen and Diener, 2013). The eLRRs of *RFO2/AtRLP3* and AtRLP2 are interchangeable for resistance, while the less conserved membrane-proximal domains of *RFO2/AtRLP3* specify resistance (Shen and Diener, 2013). It was thus suggested that *AtRLP2* was a non-functional pseudogene, similar to the case where a loss-of-function polymorphism accounts for the susceptible allele of *Ve1* (Fradin *et al.*, 2009). Conversely, ectopic expression of *AtRLP2* could suppress the *clv2* mutant, suggesting that *AtRLP2* is functional. Combined with our results, this suggests that, unlike AtRLP2, AtRLP3 has a dual function in plant development and immunity (Fig. 5; Shen and Diener, 2013).

AtRLP28-OX lines show enhanced salt stress tolerance in Arabidopsis

In addition to developmental phenotyping, we initiated an assay of the AtRLP-OX lines to test the involvement of individual AtRLP in the response to salt stress. Our transcriptional analyses have shown that several AtRLP genes are responsive to salt (see Supplementary Table S4). However, no evidence is available on their physiological roles in coping with salt stress.

To determine whether any AtRLP gene plays a role in tolerance to salt stress, we have tested the AtRLP-OX lines for their ability to germinate, compared with wild-type seeds, on medium in the presence NaCl. Three independent AtRLP28-OX lines exhibited significantly higher germination rates as compared with wild-type seeds (Fig. 6), implying that AtRLP28 is involved in the tolerance to salt stress. However, the germination rate of AtRLP28-OX lines is comparable to that of WT in the presence of mannitol (see Supplementary Fig. S5 at JXB online). The elevated expression of AtRLP28was confirmed by quantitative RT-PCR for these independent lines (Fig. 6).

To further determine a possible link between *AtRLP28* expression and salt tolerance, the expression of *AtRLP28* was evaluated on exposure to NaCl and mannitol by qPCR. *AtRLP28* transcripts were up-regulated significantly in response to NaCl and mannitol (see Supplementary Fig. S2), which is partially inconsistent with the microarray data. The discrepancy may be due to the difference in the samples



Fig. 5. *AtRLP3-OX* and *AtRLP11-OX* rescue the *clv2-1* mutant. (A) Representative siliques of Ler, *clv2-1*, *AtRLP3-OX* in *clv2-1* and *AtRLP11-OX* in *clv2-1* plants. (B) The mean number of carpels for multiple independent transgenic lines of *AtRLP3-OX* and *AtRLP11-OX* that were transformed into *clv2-1* relative to the wild-type Ler and the *clv2-1* mutant. For each genotype, a minimum of 30 transgenic plants with 20 siliques per plant were analysed. An asterisk indicates a significant difference (*P*<0.01) compared with the wild-types.



Fig. 6. Germination phenotype of the wild-type (WT) and *AtRLP28-OX* lines in response to NaCl treatment. (A) Expression levels of *AtRLP28* in WT and three independent transgenic lines overexpressing *AtRLP28* were determined by qPCR. (B) Germination percentages of WT and three independent *AtRLP28-OX* seeds grown for 2 d on the 1/2 MS medium supplemented with different concentrations of NaCl. Asterisks indicate statistically significant differences compared with WT (* indicates *P*<0.05). (C) Germination percentages of WT and three independent *AtRLP28-OX* seeds grown on 1/2 MS medium containing 150 mM NaCl at the indicated times.

collected. Indeed, in a previous study, AtRLP28 expression was shown to be significantly up-regulated under NaCl treatment (Jung *et al.*, 2008). The data thus confirm our qPCR analyses. In conclusion, these results indicate that high levels of AtRLP28 expression enhance plant salt tolerance. However, how AtRLP28 mediates salt stress tolerance requires further biological investigation.

Conclusions

The Arabidopsis genome contains 57 AtRLP genes, the majority of which have yet to be assigned biological roles. In this study, we compiled a detailed expression profile of the transcriptional regulation of AtRLP genes upon exposure to a broad range of environmental stresses and hormones. Our results indicate that a large number of AtRLP genes are differentially regulated upon various conditions tested, thus providing an overview of the processes in which AtRLP genes may be involved. Furthermore, our data revealed that a large number of AtRLP genes display differential expression upon more than one treatment, indicating that a single AtRLP gene may be involved in multiple physiological processes. The specific processes and the alteration of the expression of the corresponding AtRLP genes identified here serve as a tool for targeted screenings of individual AtRLP mutants and AtRLP-OX lines. In addition, we performed a genome-wide cloning of AtRLP genes, and generated and characterized transgenic plants overexpressing individual AtRLP genes. As an initial attempt to elucidate the biological role of AtRLP genes using these AtRLP-OX lines, we found that AtRLP3-OX and AtRLP11-OX are able to rescue the phenotype of the *clv2-1* mutant, which indicates that, similar to their duplicated genes AtRLP2 and AtRLP12, the functional specificity of these genes is determined at the level of their transcriptional regulation. Furthermore, AtRLP28 was found to mediate salt stress tolerance. Taken as a whole,

the comprehensive profile and the generated AtRLP-OX lines provide valuable resources for future investigations into the biological role of AtRLP genes.

Supplementary data

Supplementary data are available at JXB online.

Figure S1. The sequence comparison of the extracellular domains of AtRLP52 and At5g25930.

Figure S2. The expression of *AtRLP23*, *AtRLP28*, *AtRLP30*, *AtRLP33* and *AtRLP37* in response to NaCl and mannitol at indicated times.

Figure S3. Number of treatments in which a given *AtRLP* gene is up-regulated, down-regulated and differentially expressed.

Figure S4. Sequence comparisons of cloned *AtRLP* sequences, genomic DNA sequences and predicted mRNA sequences derived from TAIR.

Figure S5. Osmotic effects on the seeds germination of WT and *AtRLP28-OX* lines using mannitol.

Table S1. A list of quantitative real-time PCR primers used in this study.

Table S2. AtRLP genes which locate close to an RLK gene.Table S3. AtRLP genes displaying no transcriptionalresponses to the experimental conditions.

Table S4. Gene expression of AtRLPs under abiotic stress.

Table S5. Gene expression of AtRLPs under biotic stress.

Table S6. Gene expression of *AtRLPs* upon treatment with hormones.

 Table S7. Gene expression of *AtRLPs* under different light conditions.

Table S8. A list of primers used in the cloning of *AtRLP* genes.

Table S9. Overview of the cloning results of *AtRLP* genes. Table S10. Summary of the *AtRLP-OX* transgenic plants.

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References

Albert I, Böhm H, Albert M, et al. 2015. An RLP23–SOBIR1–BAK1 complex mediates NLP-triggered immunity. Nature Plants 1, 15140.

Blanc G, Hokamp K, Wolfe KH. 2003. A recent polyploidy superimposed on older large-scale duplications in the *Arabidopsis* genome. Genome Research **13**, 137–144.

Belfanti E, Silfverberg-Dilworth E, Tartarini S, Patocchi A, Barbieri M, Zhu J, Vinatzer BA, Gianfranceschi L, Gessler G, Sansavini S. 2004. The *HcrVf2* gene from a wild apple confers scab resistance to a

transgenic cultivated variety. Plant Biology **101**, 886–890. **Clough SJ, Bent AF.** 1998. Floral dip: a simplified method for *Agrobacterium*-mediated transformation of *Arabidopsis thaliana*. The Plant Journal **16**, 735–743.

Cook DE, Mesarich CH, Thomma BPHJ. 2015. Understanding plant immunity as a surveillance system to detect invasion. Annual Review of Phytopathology **53**, 541–563.

Cutler SR, Rodriguez PL, Finkelstein RR, Abrams SR. 2010. Abscisic acid: Emergence of a core signaling network. Annual Review of Plant Biology **61**, 651–679.

Ellendorff U, Zhang Z, Thomma BPHJ. 2008. Gene silencing to investigate the roles of receptor-like proteins in *Arabidopsis*. Plant Signaling and Behavior **3**, 893–896.

Fradin EF, Abd-EI-Haliem A, Masini L, van den Berg GC, Joosten MH, Thomma BPHJ. 2011. Interfamily transfer of tomato Ve1 mediates *Verticillium* resistance in Arabidopsis. Plant Physiology **156**, 2255–2265.

Fradin EF, Zhang Z, Ayala JC, Castroverde CC, Nazar RN, Robb J, Liu CM, Thomma BPHJ. 2009. Genetic dissection of *Verticillium* wilt resistance mediated by tomato *Ve1*. Plant Physiology **150**, 320–332.

Fritz-Laylin LK, Krishnamurthy N, Tör MT, Sjölander KV, Jones JDG. 2005. Phylogenomic analysis of the receptor-like proteins of rice and *Arabidopsis*. Plant Physiology **138**, 611–623.

Goda H, Sasaki E, Akiyama K, et al. 2008. The AtGenExpress hormone and chemical treatment data set: experimental design, data evaluation, model data analysis and data access. The Plant Journal **55**, 526–542.

Jehle AK, Lipschis M, Albert M, Fallahzadeh-Mamaghani V, Fürst U, Mueller K, Felix G. 2013. The receptor-like protein ReMAX of *Arabidopsis* detects the microbe-associated molecular pattern eMax from Xanthomonas. The Plant Cell **25**, 2330–2340.

Jeong S, Trotochaud AE, Clark SE. 1999. The *Arabidopsis CLAVATA2* gene encodes a receptor-like protein required for the stability of the CLAVATA1 receptor-like kinase. The Plant Cell **11**, 1925–1933.

Jung C, Seo JS, Han SW, Koo YJ, Kim CH, Song SI, Nahm BH, Choi YD, Cheong JJ. 2008. Overexpression of *AtMYB44* enhances stomatal closure to confer abiotic stress tolerance in transgenic Arabidopsis. Plant Physiology **146**, 623–635.

Kayes JM, Clark SE. 1998. *CLAVATA2*, a regulator of meristem and organ development in *Arabidopsis*. Development **125**, 3843–3851.

Kilian J, Whitehead D, Horak J, Wanke D, Weinl S, Batistic O, D'Angelo C, Bornberg-Bauer E, Kudla J, Harter K. 2007. The AtGenExpress global stress expression data set: protocols, evaluation and model data analysis of UV-B light, drought and cold stress responses. The Plant Journal **50**, 347–363.

Kobe B, Kajava AV. 2001. The leucine-rich repeat as a protein recognition motif. Current Opinion in Structural Biology **11**, 725–732.

Kruijt M, de Kock MJD, de Wit PJGM. 2005. Receptor-like proteins involved in plant disease resistance. Molecular Plant Pathology **6**, 85–97.

Larkan NJ, Lydiate DJ, Parkin IA, Nelson MN, Epp DJ, Cowling WA, Rimmer SR, Borhan MH. 2013. The *Brassica napus* blackleg resistance gene *LepR3* encodes a receptor-like protein triggered by the *Leptosphaeria maculans* effector AVRLM1. New Phytologist **197**, 595–605.

Liebrand TW, van den Berg GC, Zhang Z, et al. 2013. Receptor-like kinase SOBIR1/EVR interacts with receptor-like proteins in plant immunity against fungal infection. Proceedings of the National Academy of Sciences of the United States of America **110**, 10010–10015.

Matsubayashi Y. 2003. Ligand-receptor pairs in plant peptide signaling. Journal of Cell Science **116**, 3863–3870.

Nadeau JA, Sack FD. 2002. Control of stomatal distribution on the *Arabidopsis* leaf surface. Science **296**, 1697–1700.

Obayashi T, Okamura Y, Ito S, Tadaka S, Aoki Y, Shirota M, Kinoshita K. 2014. ATTED-II in 2014: evaluation of gene coexpression in agriculturally important plants. Plant and Cell Physiology **55**, e6.

Postma J, Liebrand TW, Bi G, Evrard A, Bye RR, Mbengue M, Kuhn H, Joosten MH, Robatzek S. 2016. Avr4 promotes Cf-4 receptor-like protein association with the BAK1/SERK3 receptor-like kinase to initiate receptor endocytosis and plant immunity. New Phytologist **210**, 627–642. Ramonell K, Berrocal-Lobo M, Koh S, Wan J, Edwards H, Stacey G, Somerville S. 2005. Loss-of-function mutations in chitin responsive genes show increased susceptibility to the powdery mildew pathogen *Erysiphe cichoracearum*. Plant Physiology **138**, 1027–1036.

Rivas S, Thomas CM. 2005. Molecular interactions between tomato and the leaf mold pathogen *Cladosporium fulvum*. Annual Review of Phytopathology **43**, 395–436.

Ron M, Avni A. 2004. The receptor for the fungal elicitor ethylene inducing xylanase is a member of a resistance-like gene family in tomato. The Plant Cell **16**, 1604–1615.

Shen Y, Diener AC. 2013. *Arabidopsis thaliana RESISTANCE TO FUSARIUM OXYSPORUM 2* implicates tyrosine-sulfated peptide signaling in susceptibility and resistance to root infection. PLoS Genetics **9**, e1003525.

Shiu SH, Bleecker AB. 2003. Expansion of the receptor-like kinase/Pelle gene family and receptor-like proteins in *Arabidopsis*. Plant Physiology **132**, 530–543.

Stergiopoulos I, de Wit PJGM. 2009. Fungal effector proteins. Annual Review of Phytopathology **47**, 233–263.

Thomma BPHJ, van Esse HP, Crous PW, de Wit PJGM. 2005. *Cladosporium fulvum* (syn. *Passalora fulva*), a highly specialized plant pathogen as a model for functional studies on plant pathogenic Mycosphaerellaceae. Molecular Plant Pathology **6**, 379–393.

Tör M, Lotze MT, Holton N. 2009. Receptor-mediated signaling in plants: molecular patterns and programmes. Journal of Experimental Botany **60**, 3645–3654.

Wang G, Ellendorff U, Kemp B, et al. 2008. A genome-wide functional investigation into the roles of receptor-like proteins in *Arabidopsis*. Plant Physiology **147**, 503–517.

Wang G, Fiers M, Ellendorff U, Wang Z, de Wit PJGM, Angenent G, Thomma BPHJ. 2010a. The diverse roles of extracellular leucine-rich

repeat-containing receptor-like proteins in plants. Critical Reviews in Plant Sciences **29**, 285–299.

Wang G, Long Y, Thomma BPHJ, de Wit PJGM, Angenent G, Fiers M. 2010b. Functional analyses of the CLAVATA2-like proteins and their domains that contribute to CLAVATA2 specificity. Plant Physiology **152**, 320–331.

Wang G, Zhang Z, Angenent G, Fiers M. 2011. New aspects of CLV2, a versatile gene in the regulation of Arabidopsis development. Journal of Plant Physiology **168**, 403–407.

Wu Z, Irizarry RA, Gentleman R, Martinez-Murillo F, Spencer F. 2004. A model-based background adjustment for oligonucleotide expression arrays. Journal of the American Statistical Association 99, 909–917.

Xiong L, Zhu JK. 2002. Salt tolerance. Arabidopsis Book 1, e0048.

Yan L, Cheng X, Jia R, Qin Q, Guan L, Du H, Hou S. 2014. New phenotypic characteristics of three *tmm* alleles in *Arabidopsis thaliana*. Plant Cell Reports **33**, 719–731.

Zhang L, Kars I, Essenstam B, Liebrand TWH, Wagemakers L, Elberse J, Tagkalaki P, Tjoitang D, van den Ackerveken G, van Kan JA. 2014. Fungal endopolygalacturonases are recognized as microbeassociated molecular patterns by the Arabidopsis receptor-like protein RESPONSIVENESS TO BOTRYTIS POLYGALACTURONASES1. Plant Physiology **164**, 352–364.

Zhang Y, Yang Y, Fang B, Gannon P, Ding P, Li X, Zhang Y. 2010. *Arabidopsis* snc2-1D activates receptor-like protein-mediated immunity transduced through WRKY70. The Plant Cell **22**, 3153–3163.

Zhu JK. 2002. Salt and drought stress signal transduction in plants. Annual Review of Plant Biology **53**, 247–273.

Zhu T, Wang X. 2000. Large-scale profiling of the *Arabidopsis* transcriptome. Plant Physiology **124**, 1472–1476.