T1N6_22 positively regulates *Botrytis cinerea* resistance but negatively regulates *Pseudomonas syringae* pv. tomato DC3000 resistance in *Arabidopsis thaliana*

Qiao-yun Weng, Jin-hui Song, Ya-ting Zhao, Xu Zheng, Cong-cong Huang, Guan-yu Wang, Jing Zhang, Ji-hong Xing and Jin-gao Dong

*Department of Plant Protection, College of Agriculture and Forestry, Hebei North University, Zhangjiakou, P.R. China; Mycotoxin and Molecular Plant Pathology Laboratory, College of Life Science, Hebei Agricultural University, Baoding, P.R. China*

**ABSTRACT**

T1N6_22, a short-chain dehydrogenase/reductase family protein, was identified as a positive regulator in *Arabidopsis thaliana* resistance against *Botrytis cinerea* and *Alternaria brassicicola* in our preliminary study. In this study, we found that the expression levels of the T1N6_22 gene were induced and up-regulated in *A. thaliana* ecotype Columbia (Col-0) after *B. cinerea* and *Pseudomonas syringae* pv. *tomato* DC3000 inoculation. Compared with the Col-0 and t1n6_22/T1N6_22 plants, the expression of PAL, PR4, PPO, SOD and CAT genes were down-regulated in the T1N6_22 plants. In Col-0 plants treated with salicylic acid (SA) and the SA analogue benzo(1,2,3)thiadiazole-7-carbothioic acid S-methyl ester (BTH), the expression levels of T1N6_22 were significantly enhanced, whereas the expression levels of T1N6_22 were reduced by jasmonic acid treatment. Meanwhile, the t1n6_22 mutant exhibited enhanced resistance, whereas the wild-type Col-0 and complemented plants (t1n6_22/T1N6_22) showed susceptibility to *Pst* DC3000. After inoculation with *B. cinerea* and *Pst* DC3000, the expression levels of defence-related genes *PR1*, *PR3*, *PR5*, *NPR1* and *PDF1.2* in t1n6_22 were significantly different from those in Col-0 and t1n6_22/T1N6_22 plants. Taken together, the T1N6_22 gene played a negative role in *Arabidopsis* resistance to *Pst* DC3000. The T1N6_22 gene may be involved in the regulation of salicylic acid and jasmonic-acid-signalling pathways to affect the resistance of *Arabidopsis* to *B. cinerea* and *Pst* DC3000.

**Introduction**

Grey mould, caused by the fungus *Botrytis cinerea*, occurs worldwide and causes significant economic losses every year. Isolation and functional analyses of resistance genes related to *B. cinerea* infection will provide a theoretical basis for elucidation and control of the molecular mechanisms of plant–pathogen interaction. Many reports indicate that plant defence responses against pathogens are mediated by either salicylic acid (SA), or jasmonate/ethylene (JA/ET) signalling pathway [1–3]. Tomato plants pretreated with ethylene or methyl jasmonate (MeJA) show decreased susceptibility to *B. cinerea*. SA accumulation has been reported to be required for local resistance to *B. cinerea* in *Arabidopsis thaliana* [4]. Treatment of *A. thaliana* with exogenous SA could enhance the resistance to subsequent challenge with *B. cinerea* [5,6]. *A. thaliana* plants expressing the NahG gene, which inhibits the accumulation of SA, show more susceptibility to *B. cinerea* than *A. thaliana* ecotype Columbia (Col-0) [7]. *A. thaliana* mutants with increased SA levels, showing spontaneous cell death phenotypes, show increased susceptibility to *B. cinerea*. Concurrent with the hypersensitive response, the SA levels in the inoculated leaves increase [8]. Different defence mechanisms are involved in the response against different pathogens: the SA-dependent response is deployed against the biotrophic pathogens *Pseudomonas syringae* or *Hyaloperonospora parasitica*, whereas the JA/ET response is activated by the necrotrophic pathogens *B. cinerea* or *Alternaria brassicicola* [9–11]. These two molecules play important roles in the regulation of signalling networks in induced defence responses. *NPR1* (NON-EXPRESSOR OF PATHOGENESIS-RELATED PROTEINS 1) plays a role downstream in the SA-signalling pathway and also induces the expression of *PR1*, *PR2* and *PR5* [12,13]. *PR1* (PATHOGENESIS RELATED 1) and *NPR1* are commonly used as marker genes that have been found to have a profound impact on the SA-signalling pathway [14,15]. The plant hormones jasmonate and ethylene have been shown to be involved in induced systemic resistance. Plant defence responses have been achieved through activation of genes encoding antimicrobial...
pathogenesis-related proteins or enzymes, which is regulated by jasmonate and ethylene. PDF1.2 (PLANT DEFENCE SIN1.2) and THI2.1 (THIONIN2.1) are marker genes of the JA-signalling pathway [16–18].

In the preliminary studies, the T1N6_22 gene, encoding a short chain dehydrogenase/reductase (SDR) family protein, was isolated based on a T-DNA insertion allele. The loss-of-function mutant of T1N6_22 showed enhanced susceptibility to infection by B. cinerea and Alternaria brassicae, suggesting that T1N6_22 was a positive regulator of the basal defence response [19]. In this study, we investigated the function of T1N6_22 in A. thaliana resistance to Pst DC3000 and further analysed the mechanism of T1N6_22 gene regulation in Arabidopsis resistance to B. cinerea and Pst DC3000.

**Materials and methods**

**Plant materials and growth conditions**

A. thaliana ecotype Columbia (Col-0), T-DNA insertion mutant t1n6_22 and the complemented transgenic plant t1n6_22/T1N6_22 were obtained from the Mycotoxin and Molecular Plant Pathology Laboratory, Hebei Agricultural University. A. thaliana seeds were placed at 4 °C for 3 days prior to germination. Plants were grown on soil under fluorescent lights (150 μE·m⁻²·s⁻¹) at 22 °C with 60% relative humidity and 12h light/12h dark cycle. For axenic growth, seeds were sterilized and sown on a medium solidified with 0.8% agar that contained the salts of Murashige Skoog and 1% (w/v) sucrose. The conditions for axenic growth were 12h light of 60 μE·m⁻²·s⁻¹.

**B. cinerea inoculation**

B. cinerea was grown on potato dextrose agar medium and incubated at 20–25°C. Spore inoculums were prepared by harvesting spores in water, filtration through glass wool to remove hyphae and suspension in half-strength sterile grape juice to a concentration of 4–8 × 10⁶ spores mL⁻¹. Inoculation of A. thaliana plants with B. cinerea was performed on four-week-old plants. Inoculated plants were collected 0, 24 and 48h after inoculation for expression analysis of T1N6_22 and pathogen-inducible defence response genes.

**Pst DC3000 inoculation**

Pst DC3000 was grown in solid King's medium B (KB) medium (containing 25 μg mL⁻¹ of rifampicin) at 28°C. A single colony was transferred to 3mL liquid KB medium (containing 25 μg mL⁻¹ of rifampicin). The resulting Pst DC3000 liquid culture was agitated at 28°C overnight until mid-log growth phase. The bacteria were harvested by centrifugation at 4000 × g for 7min, and resuspended in 5mL of 10 mmol L⁻¹ MgCl₂. Four-week-old plants were syringe-infiltrated with Pst DC3000 suspension. To maintain high humidity, the inoculated plants were covered with a transparent plastic film. Inoculated plants were collected 0, 0.5, 1.5, 4 and 8 h after inoculation for expression analysis of T1N6_22 and pathogen-inducible defence response genes.

** Colony-forming units assay**

A. thaliana leaves inoculated with Pst DC3000 were washed three times with sterile water and were cut into leaf discs of 0.5 cm diameter. Leaf discs were grinded to homogenate with 1mL of sterile water, and then serial dilutions were prepared. One hundred microliters of diluted liquid of leaf homogenate were spread on solid KB plates with 25 μg mL⁻¹ of rifampicin and were incubated for 2 days at 28 °C to score the colony-forming units (CFUs). The experiments were repeated three times and experimental data were analysed by using DPS software.

**Hormone treatments**

For treatments with signalling hormones, 1 mmol L⁻¹ SA (North Tianyi chemical reagent company of Tianjin), 300 μmol L⁻¹ benzo(1,2,3)thiadiazole-7-carboxthioic acid S-methyl ester (BTH; SA analogue, purchased from TransGen Biotech, Beijing, China), 100 μmol L⁻¹ JA (purchased from Sigma–Aldrich, United States) and ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC; purchased from Shanghai Dibo Biotechnology Co. Ltd. Shanghai, China) were sprayed on the leaves of four-week-old plants. The controls were sprayed with distilled H₂O. Using the method of semi-quantitative reverse transcription polymerase chain reaction (RT-PCR), expression analysis of T1N6_22 was done after treatment with hormones at 0 and 12h. The primers are listed in **Table 1**. Each treatment was performed at least in triplicate.

**RNA extraction, semi-quantitative RT-PCR and quantitative real-time PCR**

Total RNA was extracted with TransZol™ Plant reagent (TransGen Biotech, Beijing, China), according to the manufacturer's instructions. The samples were treated with RNase-free DNase to remove the genomic DNA contamination. Two micrograms of total RNA were used as a template for first-strand cDNA synthesis with SuperScript II (Invitrogen, United States) and an oligo (dT) primer.
The semi-quantitative RT-PCR program was the following: 94°C for 4min, 28–33 cycles at 94°C for 30s, 55–60°C for 30s, 72°C for 45 s, and a final elongation step of 5min at 72°C. The PCR products were resolved by electrophoresis in a 2% agarose gel and images were captured by the AlphaImager 3400 system (Alpha Innotech, United States). The *A. thaliana* housekeeping gene *ACTIN* was employed for normalization of samples. The *ACTIN* and pathogen-inducible defence response gene primers are listed in Table 1.

**Table 1. PCR primer sequences.**

| Primer name Primer sequence (5’-3’) |
|-----------------------------------|
| Primers for semi-quantitative RT-PCR |
| 18S rRNA-F GTTGCAGTTAAAAAGCTCGT |
| 18S rRNA-R TTGATTTCTCATAAGGTGCC |
| ACTIN-F ATGGCTGATGGTGAAGACATTC |
| ACTIN-R TCCATGCTCAATAGGGTACTTG |
| T1N6_22-F CGGCAAAACTCGATATTCTGGT |
| T1N6_22-R CAAAAGTTGGAGACATTGGCG |
| PAL-F TGTGAAACTGACGAGATGTAAGC |
| PAL-R TGGCTTCTAACTATCTCTGGTG |
| SOD-F TTGTGGATGACTATGGGAGG |
| SOD-R TTGGTGCTTGATGACTTG |
| CAT-F GCCAATGACGCTTCCTCC |
| CAT-R TTCTGGTTGTATCTCGGTGTA |
| PP1-F TGGAGGAAGTTGAGCCG |
| PP1-R CAGATCCAGAAAGAGGGG |
| PR1-F ATGAAATTCTACTGCTCTCG |
| PR1-R TTAGTGGCCTTCGTTCA |
| Primers for quantitative real-time PCR |
| PR3-F CGGGCGACCTCTCTCCCTCC |
| PR3-R GAAATGACGCGGTCTGCG |
| PR5-F GGCGCAACAAAGATTCAGATG |
| PR5-R GAGACACAGCTGCGTTATTCG |
| PDF1.2-F TGGCTTCTGACACAACCTC |
| PDF1.2-R TGGCTTCTGACACAACCTC |
| NPR1-F CACCTTATCTTCTCGTCTT |
| NPR1-R CACCTTATCTTCTCGTCTT |
| T1N6_22-F TCTCCACCCCAAGACACATC |
| T1N6_22-R AACATGCGCCCTCACAAC |

The expression of *T1N6_22* gene was induced by *B. cinerea* and *Pst* DC3000

Using the method of quantitative real-time PCR, the expression level of the *T1N6_22* gene in Col-0 after *B. cinerea* and *Pst* DC3000 inoculation was detected. The results showed that the expression level of the *T1N6_22* gene was induced by *B. cinerea* and *Pst* DC3000 (Figure 2). In Col-0, *B. cinerea* infection promoted a nine-fold increase ($p < 0.05$) in the expression of *T1N6_22* at

**Results and discussion**

*T1N6_22* gene played a negative role in *A. thaliana* resistance to *Pst* DC3000

To study the function of the *T1N6_22* gene in *A. thaliana* resistance to *Pst* DC3000, the symptoms and bacterial population in the Col-0, *t1n6_22* and *t1n6_22/T1N6_22* plants inoculated with *Pst* DC3000 were investigated. It was found that the *t1n6_22* plants exhibited enhanced resistance to *Pst* DC3000, whereas the Col-0 and *t1n6_22/T1N6_22* plants showed obvious susceptibility to *Pst* DC3000 (Figure 1(A)). Consistent with this phenotype, the CFUs of *Pst* DC3000 in the *t1n6_22* plants were significantly lower than those in the Col-0 and *t1n6_22/T1N6_22* plants (Figure 1(B)). The results showed that the *t1n6_22* mutant had strong resistance to *Pst* DC3000, and indicated that the *T1N6_22* gene played a negative role in *A. thaliana* resistance to *Pst* DC3000.

The expression of *T1N6_22* gene was induced by *B. cinerea* and *Pst* DC3000

Using the method of quantitative real-time PCR, the expression level of the *T1N6_22* gene in Col-0 after *B. cinerea* and *Pst* DC3000 inoculation was detected. The results showed that the expression level of the *T1N6_22* gene was induced by *B. cinerea* and *Pst* DC3000 (Figure 2). In Col-0, *B. cinerea* infection promoted a nine-fold increase ($p < 0.05$) in the expression of *T1N6_22* at

Figure 1. Response of Col-0, *t1n6_22* and *t1n6_22/T1N6_22* plants to *Pst* DC3000. Symptoms (A) and bacterial concentration in the leaves (B) of plants infected by *Pst* DC3000.
48 h post-inoculation compared with the non-inoculated control. Upon inoculation with Pst DC3000, the expression level of the T1N6_22 gene was 10 times higher ($p < 0.05$) than that in the non-inoculated control at 8 h post-inoculation.

**T1N6_22 gene deficiency altered the expression of defence-related genes**

To investigate the molecular mechanism of T1N6_22 in plant defence responses, semi-quantitative RT-PCR was used to examine the expression levels of important defence-related genes. Significant ($p < 0.05$) up-regulation of PAL (L-phenylalanin ammonia-lyase), PR4 (pathogenesis related protein), PPO (polyphenol oxidase), SOD (superoxide dismutase) and CAT (catalase) expression was observed in the t1n6_22 mutant (Figure 3). In Col-0 and t1n6_22/T1N6_22, the changes in the expression levels of these defence-related genes were basically consistent. These results indicated that the T1N6_22 gene played a negative role in regulating the expression of PAL, PR4, PPO, SOD and CAT.

**Expression of the T1N6_22 gene was induced by SA but inhibited by JA**

To test the possible relationship of T1N6_22 with the SA-dependent and JA/ET-dependent signalling pathways, the effect of exogenous application of a number of signal molecules in Col-0 plants was investigated. In plants treated with SA and the SA analogue BTH, the expression levels of the T1N6_22 gene were significantly ($p < 0.05$) enhanced. Conversely, the expression level of the T1N6_22 gene was reduced by JA treatment ($p < 0.05$). The T1N6_22 gene expression level did not change significantly after treatment with the ethylene precursor ACC (Figure 4). These results indicated that the
expression of the T1N6_22 gene was induced by SA and inhibited by JA.

**T1N6_22 gene deficiency alters the expression of key SA-, JA/ET-signalling pathway genes in response to B. cinerea**

Induction of defence-related genes expression in plants is one of the effective mechanisms to recognize and respond to infections caused by pathogens. To investigate whether the mutation of the T1N6_22 gene affects the pathogen-inducible defence responses, we determined the expression of PR1 (encoding pathogenesis-related protein 1), PR3 (encoding a basic chitinase), PR5 (encoding a thaumatin-like protein) and PDF1.2 (a plant defensin gene) after inoculation with B. cinerea by quantitative real-time PCR (Figure 5). In wild-type Col-0 plants, the expression of PR1 and PR5 genes could be induced by B. cinerea and the expression level gradually increased with the progression of infection. The expression levels of PR1 and PR5, however, were more strongly reduced at 48h post-inoculation in the t1n6_22 mutant plants compared to those in the wild-type Col-0 plants. The expression patterns of PR3 and PDF1.2 in Col-0 plants and t1n6_22 plants were also significantly (p < 0.05) different. In the t1n6_22 mutant plants, the expression of PR3 and PDF1.2 was increased at 24h post-inoculation, and the expression of PR3 and PDF1.2 was significantly (p < 0.05) lower at 48h post-inoculation. The expression levels of PR3 and PDF1.2 showed no significant difference in Col-0 and t1n6_22/T1N6_22 plants. These results suggested that the T1N6_22 gene affected the expression of these defence genes in response to B. cinerea.

**T1N6_22 gene deficiency alters the expression of key SA-, JA/ET-signalling pathway genes in response to Pst DC3000**

To test whether the expression of defence genes in response to Pst DC3000 was affected by T1N6_22 gene mutation, quantitative real-time PCR was used to detect the expression of PR1, PR3, PR5 and NPR1 genes after inoculation with Pst DC3000 (Figure 6). The expression patterns of PR1, PR3, PR5 and NPR1 genes in Col-0 plants were basically consistent. In the early stage of pathogen infection, the expression of PR1, PR3, PR5 and NPR1 genes was induced and the highest levels were observed at 1.5h or 4h post-inoculation; then the expression levels decreased gradually. In t1n6_22 plants, the expression patterns of PR1, PR3, PR5 and NPR1 genes were significantly different (p < 0.05) from those in Col-0 plants: the expression of PR1, PR5 and NPR1 genes was significantly reduced, whereas the expression of the PR3 gene was strongly induced at 4h post-inoculation in t1n6_22 plants. These results suggested that the T1N6_22 gene regulates the expression of PR1, PR3, PR5 and NPR1 genes in the resistance response of A. thaliana against Pst DC3000.

It is known that infection of plants by various pathogens triggers a complex battery of defence responses dependent on activation of distinct signalling pathways [20,21]. The SA pathway and the JA/ET pathway are the major molecular mechanisms of plant defence responses. For example, the study of Hyung et al. on the expression of PR1 and PDF1.2 and the content of SA and JA in A. thaliana mutants with enhanced resistance or enhanced susceptibility to B. cinerea implicated RST1 (RESURRECTION 1) as a negative regulator for JA.
Figure 5. Expression levels of defence-related genes in A. thaliana plants after B. cinerea inoculation.

Figure 6. Expression levels of defence-related genes in A. thaliana plants after Pst DC3000 inoculation.
synthesis or signalling [22]. Two A. thaliana genes, VQ12 and VQ29, which are highly responsive to B. cinerea infection, have been suggested to be partially involved in the JA-signalling pathway and to negatively regulate the plant basal resistance to B. cinerea [23]. GhAATAF1, a cotton NAC transcription factor gene, could be highly induced by MeJA, SA and V. dahliae; overexpressing GhAATAF1 increased cotton plant susceptibility to the fungal pathogens V. dahliae and B. cinerea, coupled with the suppression of JA-mediated signalling and the activation of SA-mediated signalling [24]. Gonorazky et al. [25] reported that tomato SIPLC2 transcript levels were increased after inoculation with B. cinerea. Meanwhile, the transcript levels of the SA-defence pathway marker gene SIP1RNA were diminished, whereas the transcripts of SIPI-I and SIPI-II (JA-defence pathway marker genes) were increased in SIPLC2 silenced plants. Rice GF14b is highly expressed during blast infection: GF14b positively regulates panicle blast resistance, and GF14b-mediated disease resistance is associated with the JA- and SA-dependent pathway [26]. The tomato SIHUB1 and SIHUB2 genes could be induced by B. cinerea and Pst DC3000 inoculation and by treatment with SA and ACC; both SIHUB1 and SIHUB2 contribute to the resistance against B. cinerea, most likely through modulating the balance between the SA- and JA/ET-mediated signalling pathways [27].

So far, resistance genes against B. cinerea including BOS1, BOS2, BOS3, BOS4, BIK1 and MYB30 have been cloned from A. thaliana and most of them are regulated by the SA- or the JA/ET-signalling pathway [28–32]. T1N6_22 was identified as a positive regulator of the basal defence response in our preliminary study. In this study, we found that the expression levels of T1N6_22 gene could be induced and up-regulated in wild-type Col-0 plants after B. cinerea and Pst DC3000 inoculation. The expression level of the SA- defence pathway marker gene SlPR1a was diminished, whereas the transcripts of SlHUB1 and SlHUB2 genes were induced and up-regulated in wild-type A. thaliana plants after B. cinerea and Pst DC3000 inoculation. In the t1n6_22 plants, the expression of PAL, PR4, PPO, SOD and CAT genes was down-regulated. T1N6_22 was a negative regulator in the response to Pst DC3000 infection. T1N6_22 gene deficiency altered the expression of key genes of the SA-, JA/ET-signalling pathway in response to B. cinerea and Pst DC3000 infection. We could speculate that the T1N6_22 gene may be involved in the regulation of the SA- and JA-signalling pathways to impact the A. thaliana resistance to B. cinerea and Pst DC3000.

**Conclusions**

The A. thaliana T1N6_22 gene, which encodes an SDR family protein, was shown to be a positive regulator of the basal defence response against B. cinerea infection. In this study, we found that the expression levels of the T1N6_22 gene could be induced and up-regulated in wild-type Col-0 A. thaliana plants after B. cinerea and Pst DC3000 inoculation. In the t1n6_22 plants, the expression of PAL, PR4, PPO, SOD and CAT genes was down-regulated. T1N6_22 was a negative regulator in the response to Pst DC3000 infection. T1N6_22 gene deficiency altered the expression of key genes of the SA-, JA/ET-signalling pathway in response to B. cinerea and Pst DC3000 infection. We could speculate that the T1N6_22 gene may be involved in the regulation of the SA- and JA-signalling pathways to impact the A. thaliana resistance to B. cinerea and Pst DC3000.

**Disclosure statement**

The authors declare no conflict of interest.

**Funding**

This research was funded by the Project of Natural Science Foundation of Hebei Province [grant number C2014405010].

**ORCID**

Qiao-yun Weng http://orcid.org/0000-0001-6241-6683

**References**

[1] John MM, Jeffery LD. Signal transduction in the plant immune response. Trends Biochem Sci. 2000;25(2):79–82.
[2] Durner J, Shah J, Klessig DF. Salicylic acid and disease resistance in plants. Trends Plant Sci. 1997;2(7):266–274.
[3] Gundlach H, Müller MJ, Kutchan TM, et al. Jasmonic acid is a signal transducer in elicitor-induced plant cell cultures. Proc Natl Acad Sci USA. 1992;89(6):2389–2393.
[4] Diaz J, ten Have A, van Kan JA. The role of ethylene and wound signaling in resistance of tomato to Botrytis cinerea. Plant Physiol. 2002;129(3):1341–1351.
[5] Thomma BP, Eggermont K, Tieren KF, et al. Requirement of functional ethylene-insensitive 2 gene for efficient resistance of Arabidopsis to infection by Botrytis cinerea. Plant Physiol. 1999;121(4):1093–1102.
[6] Si-Ammour A, Mauch-Mani B, Mauch F. Quantification of induced resistance against phytophthora species expressing GFP as a vital marker: beta-aminobutyric acid but not BTH protects potato and Arabidopsis from infection. Mol Plant Pathol. 2003;4(4):237–248.
[7] Lisa S, Barbara C, Jane H, et al. Rhamnolipids elicit defense responses and induce disease resistance against biotrophic, hemibiotrophic, and necrotrophic pathogens that require different signaling pathways in Arabidopsis and highlight a central role for salicylic acid. Plant Physiol. 2012;160(3):1630–1641.
[8] Kachhoo P, Shanklin J, Shah J, et al. A fatty acid desaturase modulates the activation of defense signaling pathways in plants. Proc Natl Acad Sci USA. 2001;98(16):9448–9453.
[9] de Torres-Zabala M, Truman W, Bennett MH, et al. Pseudomonas syringae pv. tomato hijacks the Arabidopsis abscisic acid signalling pathway to cause disease. EMBO J. 2007;26:1434–1443.
[10] Laurent Z, Jean-Pierre M, Brigitte MM. β-Aminobutyric acid-induced protection of Arabidopsis against the necrotrophic fungus Botrytis cinerea. Plant Physiol. 2001;126(2):517–523.

[11] Thomma BPHJ, Nelissen I, Eggermont K, et al. Deficiency in phytoalexin production causes enhanced susceptibility of Arabidopsis thaliana to the fungusAlternaria brassicicola. Plant J. 1999;19(2):163–171.

[12] Pape S, Thurov C, Gatz C. The Arabidopsis PR-1 promoter contains multiple integration sites for the coactivator NPR1 and the repressor SNI1. Plant Physiol. 2010;154(4):1805–1818.

[13] Canet JV, Dobon A, Roig A, et al. Structure-function analysis of npr1 alleles in Arabidopsis reveals a role for its paralogs in the perception of salicylic acid. Plant Cell Environ. 2010;33(11):1911–1922.

[14] Brosche M, Kangasjarvi J. Low antioxidant concentrations impact on multiple signalling pathways in Arabidopsis thaliana partly through NPR1. J Exp Bot. 2012;63(5):1849–1861.

[15] Attaran E, Zeier T E, Griebel T, et al. Methyl salicylate production and jasmonate signaling are not essential for systemic acquired resistance in Arabidopsis. Plant Cell. 2009;21(3):954–971.

[16] Manners JM, Penninckx IA, Vermaere K, et al. The promoter of the plant defensin gene PDF1.2 from Arabidopsis is systemically activated by fungal pathogens and responds to methyl jasmonate but not to salicylic acid. Plant Mol Biol. 1998;38(6):1071–1080.

[17] Ochoa-Zarzosa A, Loesa-Angelo H, Sagreiro-Cisneros E, et al. Antibacterial activity of thionin Thi2.1 from Arabidopsis thaliana expressed by bovine endothelial cells against Staphylococcus aureus isolates from bovine mastitis. Vet Microbiol. 2008;127(3–4):425–430.

[18] Chan YL, Prasad V, Sanjaya, et al. Transgenic tomato plants expressing an Arabidopsis thionin (Thi2.1) driven by fruit-inactive promoter battle against phytopathogenic attack. Planta. 2005;221(3):386–393.

[19] Xing JH, Weng QY, Hao CC, et al. T1N6, 22 gene is required for biotic and abiotic stress responses in Arabidopsis. Russian J Genet. 2012;48(12):1191–1198.

[20] Uknes S, Mauch-Mani B, Moyer M, et al. Acquired resistance in Arabidopsis. Plant Cell. 1992;4(6):645–656.

[21] Ryals JA, Neuenschwander UH, Willits MG, et al. Systemic acquired resistance. Plant Cell. 1996;8(10):1809–1819.

[22] Hyung GM, Kristin AL, Eugene PP, et al. The Arabidopsis RESURRECTION1 gene regulates a novel antagonistic interaction in plant defense to biotrophs and necrotrophs. Plant Physiol. 2009;115(1):290–305.

[23] Wang H, Hu Y, Pan J, et al. Arabidopsis VQ motif-containing proteins VQ12 and VQ29 negatively modulate basal defense against Botrytis cinerea. Sci Rep. 2015 [cited 2016 Oct 25];5:14185. DOI:10.1038/srep14185

[24] He X, Zhu L, Xu L, et al. GhATAF1, a NAC transcription factor, confers abiotic and biotic stress responses by regulating phytohormonal signaling networks. Plant Cell Rep. 2016;35:2167–2179.

[25] Gororazyk G, Guzzo MC, Laxalt AM. Silencing of the tomato phosphatidylinositol-phospholipase C2 (SIPLC2) reduces plant susceptibility to Botrytis cinerea. Mol Plant Pathol. 2016;17(9):1354–1363.

[26] Liu Q, Yang J, Zhang S, et al. OsGF14b positively regulates panicle blast resistance but negatively regulates leaf blast resistance in rice. Mol Plant Microbe Interact. 2016;29(1):46–56.

[27] Zhang Y, Li D, Zhang H, et al. Tomato histone H2B monoubiquitination enzymes SIHUB1 and SIHUB2 contribute to disease resistance against Botrytis cinerea through modulating the balance between SA- and JA/ET-mediated signaling pathways. BMC Plant Biol. 2015 [cited 2016 Oct 25];15:252. DOI:10.1186/s12870-015-0614-2

[28] Govrin EM, Levine A. Infection of Arabidopsis with a necrotrophic pathogen, Botrytis cinerea, elicits various defense responses but does not induce systemic acquired resistance (SAR). Plant Mol Biol. 2002;48:267–276.

[29] Veronese P, Nakagami H, Bluhm B, et al. The membrane-anchored BOTRYTIS-INDUCED KINASE1 plays distinct roles in Arabidopsis resistance to necrotrophic and biotrophic pathogens. Plant Cell. 2006;18(1):257–273.

[30] Denby K J, Kumar P, Klebenstein DJ. Identification of Botrytis cinerea susceptibility loci in Arabidopsis thaliana. Plant J. 2004;38(3):473–486.

[31] Sylvain R, Susana R. Regulate and be regulated: integration of defense and other signals by the AtMYB30 transcription factor. Front Plant Sci. 2013 [cited 2017 Apr 05];4:98. DOI:10.3389/fpls.2013.00098

[32] Raffaee S, Rivas S, Roby D. An essential role for salicylic acid in AtMYB30-mediated control of the hypersensitive cell death program in Arabidopsis. FEBS Lett. 2006;580(14):3498–3504.