EMBO reports

scientific report

Chromatin modification acts as a memory for systemic acquired resistance in the plant stress response

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Priming of defence genes for amplified response to secondary stress can be induced by application of the plant hormone salicylic acid or its synthetic analogue acibenzolar *S*-methyl. In this study, we show that treatment with acibenzolar *S*-methyl or pathogen infection of distal leaves induce chromatin modifications on defence gene promoters that are normally found on active genes, although the genes remain inactive. This is associated with an amplified gene response on challenge exposure to stress. Mutant analyses reveal a tight correlation between histone modification patterns and gene priming. The data suggest a histone memory for information storage in the plant stress response.

Keywords: chromatin; systemic acquired resistance; plant promoter control; systemic signalling

EMBO *reports* (2011) **12**, 50–55. doi:10.1038/embor.2010.186

INTRODUCTION

After localized infection by a pathogen, plants often acquire systemic immunity to further infections (Durrant & Dong, 2004). This requires the accumulation of the plant hormone salicylic acid in tissue distal from the infection site and is called systemic acquired resistance (SAR). Exogenous application of salicylic acid and some salicylic acid analogues, such as acibenzolar *S*-methyl (BTH) is sufficient to trigger resistance to biotic and abiotic stress (Ryals *et al*, 1996; Senaratna *et al*, 2000). In the SAR response, defence genes in the infected and remote tissue show the 'priming' phenomenon; they are able to respond faster and/or to a greater extent to subsequent challenge (Kohler *et al*, 2002; Conrath, 2009). The promoters of many of these genes contain at least one 'W-box' that provides binding sites for WRKY transcription factors (Maleck *et al*, 2000; Rushton *et al*, 2010). Genes encoding WRKY factors are themselves transcriptionally induced by either pathogen infection or treatment with microbe-associated molecular patterns, such as flagellin (Asai *et al*, 2002; Dong *et al*, 2003).

Mutants that are attenuated in pathogen defence are often also compromised in gene priming. For example, the *npr1* mutant of *Arabidopsis thaliana* is deficient in SAR (Durrant & Dong, 2004) and cannot be primed for enhanced gene expression (Kohler *et al*, 2002; Beckers *et al*, 2009). By contrast, defence genes are often constitutively primed for enhanced activation in mutants with permanently enhanced immunity to pathogens such as *sni1*, *cpr1* and *edr1* (Frye & Innes, 1998; Frye *et al*, 2001; Kohler *et al*, 2002; Mosher *et al*, 2006).

Chromatin structure is important for the regulation of gene expression. The basal repeat unit of chromatin is the nucleosome containing 147 base pairs of DNA wrapped around a protein core particle comprising two copies each of histones H2A, H2B, H3 and H4 (Luger *et al*, 1997). Histones are subject to many covalent modifications. Acetylation of lysines in the amino-terminal tails of histones H3 and H4 has been associated with active genes (Eberharter & Becker, 2002). This modification reduces the ionic interaction between positively charged lysine side chains and the negatively charged DNA backbone (Garcia-Ramirez *et al*, 1995). Moreover, lysine acetylation provides docking sites for transcriptional coactivator proteins containing bromodomains (Kanno *et al*, 2004). For histone methylation the situation is more complex because lysine and arginine residues can be methylated and up to three methyl groups can be added to each residue. Furthermore, specific methylation patterns are associated with both gene activation and repression. The strongest correlation between histone methylation and gene activity is found for trimethylation of Lys 4 on histone H3 (H3K4me3) on promoters and coding sequences of active genes (Ruthenburg *et al*, 2007). By contrast, the roles of dimethylation and monomethylation of the same residue in gene regulation are less defined.

Although gene priming is a widespread phenomenon and has also been described for the defence response in animals (Hayes *et al*, 1995), little is known about the mechanisms for it at the molecular level. On the basis of mutant analyses, it has been suggested recently that defence genes are poised for enhanced activation during SAR by replacement on gene promoters of

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Received 19 March 2010; revised 23 October 2010; accepted 26 October 2010; published online 3 December 2010

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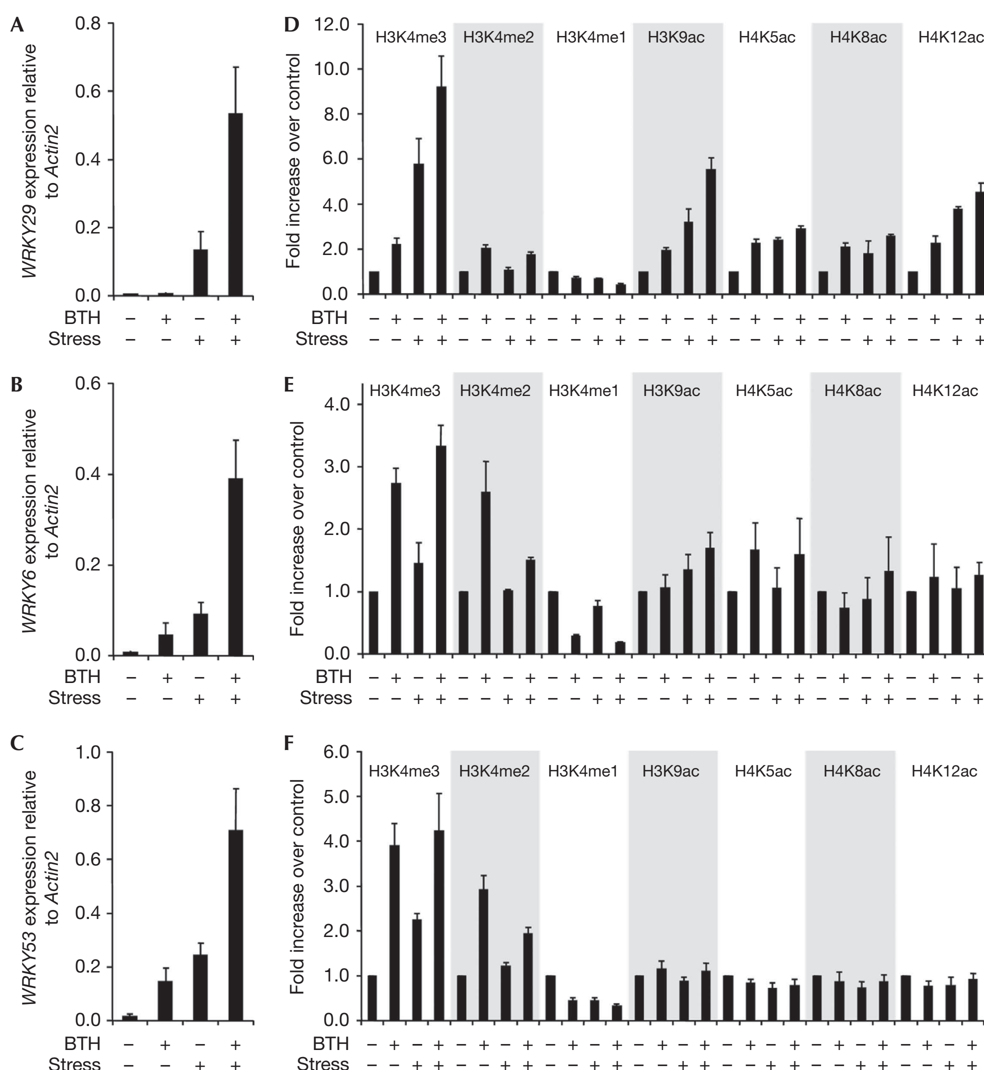

Fig 1 | Transcript abundance and histone modifications after priming and potentiated activation of three *WRKY* transcription factor genes. Plants were treated with 100 μM BTH or wettable powder (control). After 72 h, half of the plants were stressed by infiltrating water into their leaves. After 3 h, leaves were collected and RNA and chromatin were isolated. **(A–C)** Transcript abundance as determined by RT–qPCR. Data are standardized for abundance of the *Actin2* transcript. **(D–F)** H3K4 methylation (me) and histone acetylation (ac) on the gene promoters. Data are standardized for histone modification levels in the absence of inducer and stress treatment. Each data point is based on four independent replicates. Error bars indicate s.e.m. values. BTH, acibenzolar *S*-methyl; RT–qPCR, reverse transcriptase–quantitative PCR.

histone H2A with its variant H2A.Z (March-Díaz *et al*, 2008; van den Burg & Takken, 2009). In this study, we show that histone modifications—such as H3 and H4 acetylation—and H3K4 methylation are systemically set during a priming event. These modifications might create a memory of the primary infection that is associated with an amplified reaction to a second stress stimulus.

RESULTS AND DISCUSSION

Chromatin states control cellular memory and differentiation in animals and plants (Roh *et al*, 2006; Zhang, 2008). Thus, we hypothesized that primed genes could be poised for enhanced activation of gene expression by histone modifications. To identify potential target genes of priming, we tested 11 *Arabidopsis* genes encoding WRKY transcription factors (*WRKY6*, *WRKY11*, *WRKY18*, *WRKY22*, *WRKY23*, *WRKY26*, *WRKY29*, *WRKY31*, *WRKY48*, *WRKY53* and *WRKY66*) for gene priming after BTH application (data not shown). BTH was chosen as the elicitor of priming because it induces gene priming at moderate concentrations (100–300 μM; Kohler *et al*, 2002; Beckers *et al*, 2009). *WRKY29*, *WRKY6* and *WRKY53* showed a typical priming response in expression (Fig 1A–C); application of the priming agent BTH alone did not activate *WRKY29*, and only activated *WRKY6* and *WRKY53* to a limited extent. Similar levels of gene expression were observed when plants were stressed by infiltration of water into their leaves. This has previously been used as a challenging stress (Kohler *et al*, 2002; Beckers *et al*, 2009) because it elicits a cell collapse or wound stress response in the entire leaf

that is more uniform than, for example, bacterial infection. Water infiltration after BTH treatment resulted in strongly enhanced gene activation, compared with plants that were stressed without previous BTH treatment (Fig 1A–C).

From the same samples, by using chromatin immunoprecipitation we analysed methylation of histone H3 Lys 4 (H3K4me) and acetylation of several lysine residues on histones H3 and H4 (H3ac, H4ac) on the promoters of the selected *WRKY* genes. The specificity of the chromatin immunoprecipitation reaction was evaluated in advance by measuring histone modifications on genes that were known to be transcriptionally activated or suppressed by BTH treatment (supplementary Fig S1A,B online). On the *WRKY29* promoter (Fig 1D), trimethylation (H3K4me3) and dimethylation (H3K4me2) of H3K4 and all acetylations tested increased after BTH application although this did not induce *WRKY29* transcription (Fig 1A). Thus, chromatin marks normally associated with active genes (Pokholok *et al*, 2005) are set by the priming stimulus before gene activation. Particularly after previous priming, a stress stimulus enhanced some of the modifications—H3K4me3, H3K9ac and H4K12ac—on *WRKY29* (Fig 1D). For *WRKY6* and *WRKY53*, only minor changes in histone acetylation were observed after both priming and/or stress treatment (Fig 1E,F). However, for these genes, H3K4me3 was induced by BTH treatment alone, to levels that are otherwise only found on the fully active gene (BTH treatment plus subsequent stress exposure). Induction of H3K4me2 was stronger with BTH alone than with stress treatment, whereas H3K4me1 showed a reciprocal reduction (Fig 1E,F). Importantly, the enhancement of H3K4 trimethylation and dimethylation after BTH treatment was not caused by the concomitant gene induction (Fig 1B,C), as transcripts accumulated to higher levels after direct stress exposure. However, changes in histone trimethylation and dimethylation were weaker after stress application than they were after BTH treatment (Fig 1E,F). As an additional control, we measured transcript levels and histone modifications on the *Ubiquitin5* (*UBQ5*) gene (supplementary Fig S1C,D online). Transcript abundance was slightly reduced by stress treatment in the absence of BTH, concomitant with a decrease in H3K4me3 levels. All other modifications remained unchanged under these conditions. Moreover, nucleosome occupancy on the tested *WRKY* gene promoters was only slightly affected by the treatments (supplementary Fig S1E online). Together, these data imply that pre-stress application of BTH induces chromatin modifications on *WRKY* gene promoters that facilitate the activation of gene expression by subsequent stress. This might be due to increased accessibility of DNA in acetylated compared with non-acetylated chromatin (Eberharter & Becker, 2002; Kanno *et al*, 2004) or the provision of docking sites for gene activators by histone modifications (de la Cruz *et al*, 2005; Vermeulen *et al*, 2007).

We investigated whether histone modifications on *WRKY* gene promoters can be detected in leaves distal to localized foliar infection by the pathogen *Pseudomonas syringae* pv. *maculicola*. Localized *P. s. maculicola* infection primed the *WRKY* promoters in remote leaves for an augmented response to secondary stress (Fig 2A) and, furthermore, the transcriptional responses in distal leaves were similar to those observed with BTH (Fig 1). Our analysis of histone modifications focused on comparison between the primed and non-primed state and on modifications that were induced by BTH in the previous assay (Fig 2B). On the three

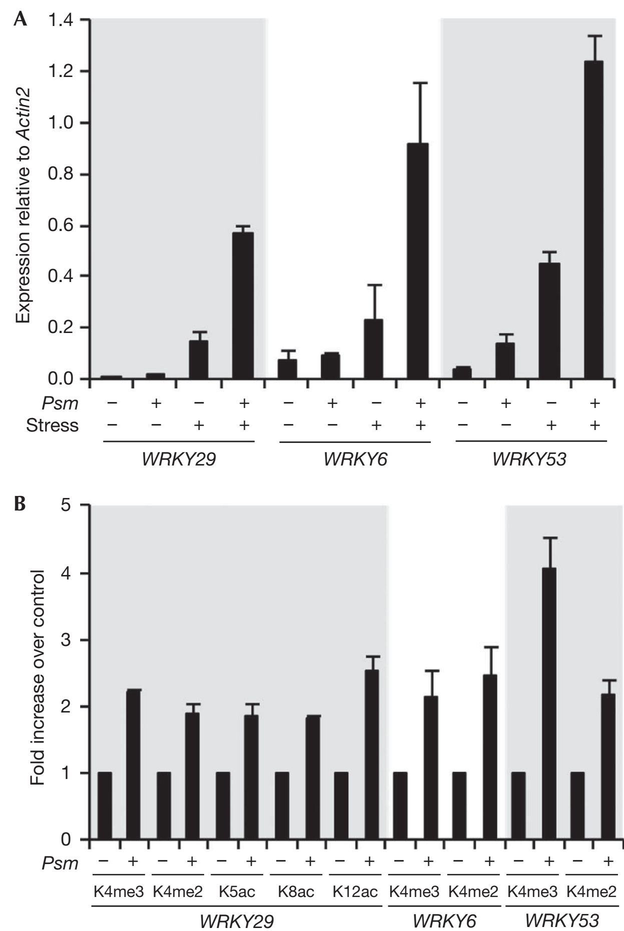

Fig 2 | Pathogen-induced priming for augmented gene activation. **(A)** Lower leaves were infected with *Psm*. After 72 h, upper leaves were left untreated or stressed by the infiltration of water. After 3 h, upper leaves were collected and analysed for transcript abundance. Data are standardized for abundance of the *Actin2* transcript. **(B)** Histone modifications in upper leaves 72 h after infection of lower leaves with *Psm*. Data are standardized for histone modification levels in the absence of pathogen infection. Each data point is based on at least three independent replicates. Error bars indicate s.e.m. values. ac, acetylation; me, methylation; *Psm*, *Pseudomonas syringae* pv. *maculicola*.

WRKY gene promoters, clear increases in histone modifications were observed after pathogen infection (Fig 2B). The response amplitude after perception of the systemic signals for SAR was similar to that observed after BTH treatment (Fig 1). Thus, pathogen exposure induces one or more systemic signals that are stored on gene promoters in remote leaves in the form of histone modifications.

Enhanced trimethylation of H3K4 concomitant with gene priming is a common feature of the assayed *WRKY* promoters. Next, we measured this histone modification in mutants that are attenuated in gene priming (*npr1*) or show permanent priming (*cpr1*, *edr1*) and constitutive pathogen resistance (*sni1*; see Introduction).

The transcriptional response of *WRKY29* to BTH and stress treatment is shown in Fig 3A. In the *npr1* mutant, *WRKY29* was responsive to stress treatment, but this response was not

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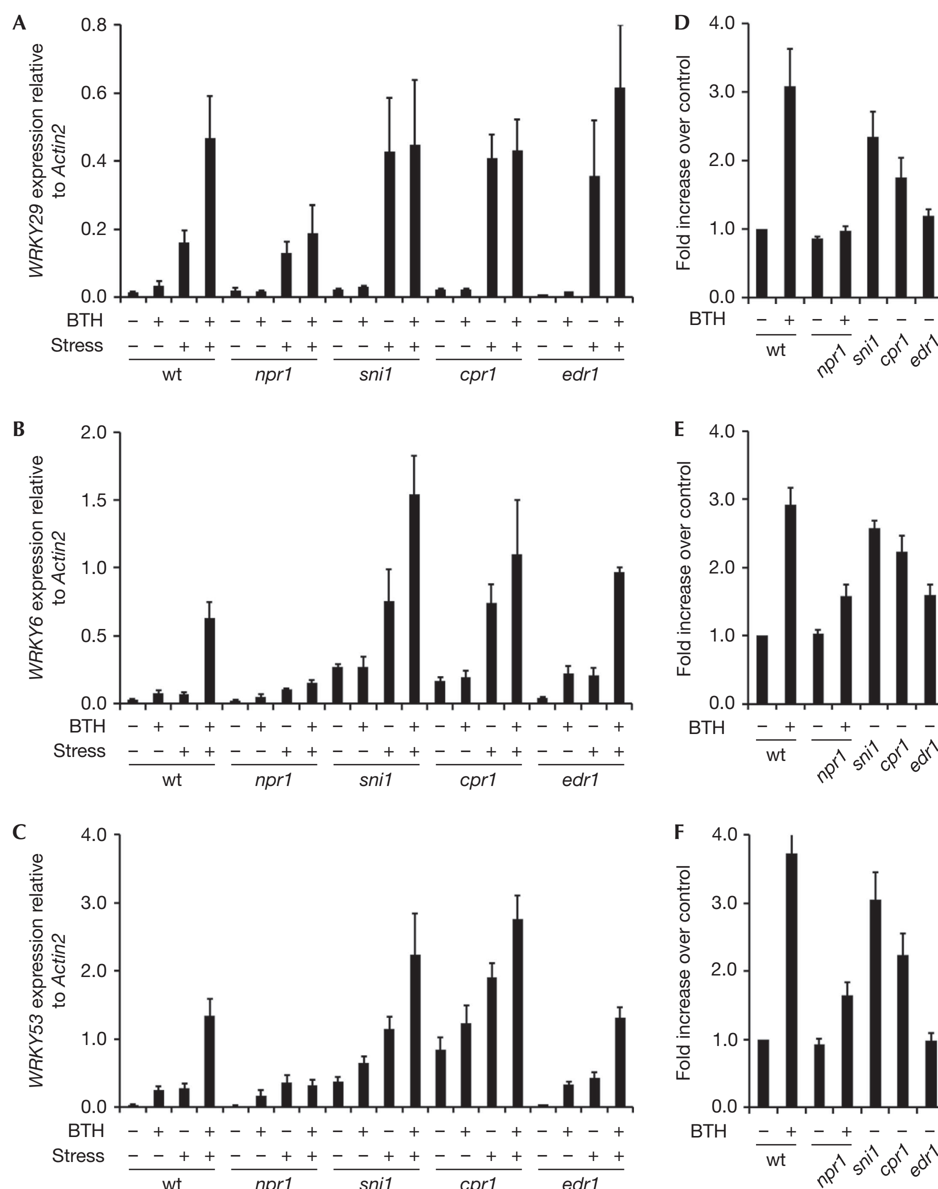

Fig 3 | Potentiated gene activation and H3K4 trimethylation in *npr1*, *sni1*, *cpr1* and *edr1* mutants. Wild-type and mutant plants were treated with 100 μM BTH or wettable powder (control). After 72 h, some of the plants were additionally stressed by infiltrating water into their leaves. Three hours later, leaves were collected and RNA and chromatin were extracted. (**A–C**) Transcript abundance in wild-type and mutant plants as determined by RT–qPCR. Data are standardized for abundance of the *Actin2* transcript. (**D–F**) Histone H3 Lys 4 trimethylation on gene promoters in wild-type and mutant plants. Data are standardized for wild-type histone modification levels in the absence of BTH. Data represent at least three independent replicates. Error bars indicate s.e.m. values. BTH, acibenzolar *S*-methyl; RT–qPCR, reverse transcriptase–quantitative PCR; wt, wild type.

augmented by earlier BTH application. By contrast, in the *sni1*, *cpr1* and *edr1* mutants, BTH treatment was not required for the strongest *WRKY29* activation in response to stress exposure. Transcription levels detected in these mutants in the absence of BTH were similar to those observed in the stress-exposed wild type after priming with BTH. This indicates that *WRKY29* was already primed in these mutants, in the absence of the inducer. Consistent with the transcriptional response, BTH induced trimethylation of H3K4 on the *WRKY29* promoter in the wild type, but not on the priming-deficient *npr1* mutant (Fig 3D). In the constitutively primed *sni1* and *cpr1* mutants (Fig 3A), H3K4me3 levels were already enhanced in the absence of BTH pretreatment. However, this was not found for the *edr1* mutant in which H3K4me3 levels were low.

In the assayed mutants, the results were similar for *WRKY6* and *WRKY53* expression and histone modifications. Neither gene showed augmented expression after BTH pretreatment and stress stimulus in the *npr1* mutant (Fig 3B,C). This correlated with the impaired ability of *npr1* to induce high H3K4me3 levels on the *WRKY6* and *WRKY53* promoters in response to BTH (Fig 3E,F).

In the *sni1* and *cpr1* mutants, the basal response to stress was augmented to levels normally observed in wild-type plants only after priming by BTH, although some additional induction of transcription was observed when the mutants were pretreated with BTH. For the *WRKY6* and *WRKY53* promoters, constitutively high H3K4me3 levels were detected in *sni1* and *cpr1* (Fig 3E,F). In the *edr1* mutant, the transcriptional response of *WRKY6* and *WRKY53* to BTH application and stress treatment was similar to the pattern found in the wild-type, indicating that the genes were not strongly primed in this mutant. Consequently, compared with the wild type, enhancement of basal H3K4me3 levels was almost undetectable (*WRKY6*) or absent (*WRKY53*). Together, our mutant analyses link H3K4 trimethylation as a molecular footprint to gene priming as the functional outcome. Whereas the association between H3K4me3 modification and gene priming is given in *npr1*, *sni1* and *cpr1*, constitutive priming of *WRKY29* in *edr1* does not seem to require high H3K4me3 levels. This might indicate the presence of a second independent process controlling priming in this mutant. Alternatively, weak or transient changes in histone modification might not have been detected in our experiments.

Not many examples exist that correlate histone modifications with a transcriptionally poised state. In maize, the tissue specificity of photosynthetic genes is controlled by H3K4me3 and is established independently of transcriptional activation (Offermann *et al*, 2006; Danker *et al*, 2008; Horst *et al*, 2009). Similar stimulus-dependent control of histone modifications was described for the vernalization response in *Arabidopsis* (He & Amasino, 2005) and the hormonal regulation of the *beta-phaseolin* promoter in beans (Ng *et al*, 2006). A genome-wide study in human cells revealed that about half of the inactive genes have nucleosomes that carry H3K4me3 or histone acetylations (Guenther *et al*, 2007). In our study, the abundance of H3K4me2 on primed genes before stress treatment (Figs 1D–F, 2B and 3B,C) is intriguing. H3K4me2 often colocalizes with H3K4me3 in vertebrates (Ruthenburg *et al*, 2007), but H3K4me2 has also been associated with poised states of genes in yeast and vertebrates (Pokholok *et al*, 2005; Bernstein *et al*, 2006). As the WD repeat-containing protein 5 component of the human methyltransferase complex preferentially binds to histone H3 when dimethylated at Lys 4 (Wysocka *et al*, 2005), high levels of H3K4me2 might speed-up or enhance subsequent trimethylation, facilitating the recruitment of chromatin remodelling factors and other effector proteins (Wysocka *et al*, 2006; Ruthenburg *et al*, 2007). As gene priming is part of the induced immune response in plants (Conrath, 2009) and animals (Chen *et al*, 1992; Pham *et al*, 2007), it will be interesting to see whether pre-stress modification of chromatin on defence gene promoters also has a function in animal defence.

METHODS

A. thaliana (accession Columbia-0) wild-type plants and *npr1*, *sni1*, *cpr1* and *edr1* mutants were grown in short day conditions (8 h light, $100\,\mu mol\,m^{-2}\,s^{-1}$) at 20 °C in a growth chamber. Treatments with wettable powder or 100 μM BTH and water infiltration were as described previously (Beckers *et al*, 2009). For pathogen-induced priming, three lower leaves were infiltrated with a suspension of *P. s. maculicola* (5×10^5 colony-forming units per millilitre).

RNA was isolated from leaves by using the TRIZOL method (Chomczynski, 1993). Transcript abundance was measured by reverse transcriptase–quantitative PCR on an ABI Prism 7300 sequence detector system (Applied Biosystems) using gene-specific primers (supplementary Table S1 online) and SYBR Green fluorescence (Platinum SYBR Green qPCR Mix, Invitrogen) for detection. Data were standardized for *Actin2* transcript abundance.

Chromatin isolation and immunoprecipitation were performed as described previously (Haring *et al*, 2007). The antibodies used for precipitation of modified histones from 2 g of leaf material are listed in supplementary Table S2 online. The abundance of DNA sequences in the precipitate was measured by quantitative PCR using the primers listed in supplementary Table S1 online. Background signals with serum derived from rabbits that were immunized with an unrelated potato protein never exceeded 10% of positive signals.

Supplementary information is available at EMBO *reports* online (http://www.emboreports.org).

ACKNOWLEDGEMENTS
We thank G.J.M. Beckers, I. Horst and J.-P. Métraux for providing valuable comments on the manuscript. S. Offermann introduced M.J. to molecular techniques. X. Dong kindly provided us with *sni1* mutant seeds. We also thank M. Okzakzin for excellent technical assistance. This study was supported by funds from the German Science Foundation (Deutsche Forschungsgemeinschaft).

CONFLICT OF INTEREST
The authors declare that they have no conflict of interest.

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