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Induced resistance: an enhancement of basal resistance?

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Abstract: Upon primary pathogen attack, plants activate resistance mechanisms at the site of infection. Besides this so-called basal resistance, plants have also the ability to enhance their defensive capacity against future pathogen attack. There are at least two types of biologically induced resistance. Classic systemic acquired resistance (SAR) results from infection by a necrotizing pathogen and is dependent on endogenous accumulation of salicylic acid (SA). Root colonization by non-pathogenic rhizobacteria can trigger an induced systemic resistance (ISR) response as well, which functions independently of SA and requires intact responsiveness to the plant hormones jasmonic acid (JA) and ethylene (ET). A screen for genotypes impaired in either ISR or SAR revealed that ecotypes RLD1 and Ws-0, as well as the enhanced disease susceptibility mutants *eds4-1*, *eds8-1* and *eds10-1*, are impaired in WCS417r-mediated ISR, whereas mutants *eds5-1* and *eds12-1* are impaired in pathogen-induced SAR. Analysis of JA-, ET-, and SA-responsiveness revealed that the ISR-impaired genotypes are affected in signaling compounds contributing to JA/ET-dependent basal resistance against *P. syringae*, while the SAR-impaired genotypes are affected in compounds contributing to SA-dependent basal resistance against *P. syringae*. To further examine the relationship between basal resistance and induced resistance, the effectiveness of SAR and ISR was assessed against different Arabidopsis pathogens that are resisted through JA/ET-dependent basal resistance, SA-dependent basal resistance, or a combination of JA/ET- and SA-dependent basal resistance. This analysis revealed that ISR is effective predominantly against pathogens that are resisted through JA/ET-dependent basal resistance, whereas SAR is effective against pathogens that are resisted through SA-dependent basal resistance. Collectively, our results suggest that ISR constitutes an enhancement of JA/ET-dependent basal resistance, whereas SAR is achieved through an enhanced expression of SA-dependent basal resistance.

Key words: *Arabidopsis*, resistance, jasmonic acid, ethylene, salicylic acid

Introduction

Once a plant is exposed to pathogen attack, it activates a diverse array of defense mechanisms at the site of infection. If this interaction is compatible, the plant reacts inefficiently, or too late to halt the pathogen. Resistance against so-called virulent pathogens is considerably less than that against avirulent pathogens that evoke a hypersensitive response. However, in a compatible interaction, the plant still has strategies to restrain the ongoing virulent pathogen. This type of resistance is not well defined, but is sometimes referred to as polygenic, horizontal, or basal resistance, and acts in slowing down the rate of disease development.

Besides basal resistance against primary pathogen attack, plants can also enhance their defensive capacity against future pathogen attack. This phenomenon is referred to as induced resistance. There are at least two types of biologically induced resistance. Classic induced resistance results from localized infection by a necrotizing pathogen, leading to a systemic acquired resistance (SAR) in plant parts distant from the site of infection (Sticher et al., 1997). Alternatively, root colonization by non-pathogenic rhizobacteria can trigger an induced systemic resistance (ISR) response as well (Van Loon et al., 1998). Most ISR-eliciting

rhizobacteria belong to the group of the fluorescent *Pseudomonas* spp.. Both pathogen-induced SAR and rhizobacteria-mediated ISR are effective against different types of pathogens, and are typically characterized by a restriction of pathogen growth and a suppression of disease development compared to primary infected, non-induced plants. However, the signaling pathways controlling pathogen-induced SAR and rhizobacteria-mediated ISR differ. Whereas SAR requires endogenous accumulation of salicylic acid (SA; Gaffney et al., 1993; Lawton et al., 1995), the signaling pathway controlling ISR functions independently of SA, and requires intact responsiveness to the plant hormones jasmonic acid (JA) and ethylene (Pieterse et al., 1998). Apart from these differences, SAR and ISR are both dependent on the defense-regulatory protein NPR1. Downstream NPR1 both signaling pathways diverge, because SAR is accompanied by a transcriptional activation of genes encoding pathogenesis-related proteins, whereas ISR is not (Pieterse et al., 1998).

Interestingly, plant genotypes that are impaired in the expression of SAR and/or ISR are often characterized by a reduced level of basal resistance against primary infection against certain pathogens (Delaney et al., 1994; Thomma et al., 1998; Pieterse et al., 1998; Norman-setterblad et al., 2000), suggesting that components controlling induced resistance also contribute to basal resistance. In this review, we provide several lines of evidence that ISR and SAR in Arabidopsis are achieved through an enhanced expression of specific basal resistance responses.

Results and discussion

Identification of the ISR1 locus controlling ISR and basal resistance against P. syringae pv. tomato

To examine naturally occurring variation in ISR- and SAR-inducibility, ten Arabidopsis ecotypes were tested for their ability to express WCS417r-mediated ISR and pathogen-induced SAR against the virulent leaf pathogen *Pseudomonas syringae* pv. *tomato* DC3000 (*Pst*). This screen revealed that all ecotypes were unaffected in pathogen-induced SAR, whereas two ecotypes, RLD1 and Ws-0, failed to develop ISR upon treatment of the roots with WCS417r bacteria. Interestingly, ecotypes RLD1 and Ws-0 also displayed a remarkably low level of basal resistance against *Pst* (Ton et al., 1999; Table 1). Based on this association between ISR-inducibility and basal resistance against *Pst*, a genetic approach was initiated to identify (a) genetic determinant(s) involved in the regulation of ISR and basal resistance against *Pst*. Analysis of the progeny from crosses between ISR-inducible and ISR-noninducible Arabidopsis ecotypes revealed that both the potential to express ISR and the relatively high basal resistance against *Pst* of the ISR-inducible ecotypes are controlled by a single locus on chromosome III, designated *ISR1* (Ton et al., 1999). Apparently, the *ISR1* locus is not only involved in WCS417r-mediated ISR, but it also contributes to basal resistance against *Pst*.

To assess the physiological role of the *ISR1* locus, RLD1 and Ws-0 were tested for their responsiveness to JA and ET. This analysis revealed that both ecotypes react normally to JA, but exhibit reduced responsiveness to ethylene. Compared to the ISR-inducible ecotype Columbia (Col-0), RLD1 and Ws-0 exhibited a reduced triple response, a decrease in the expression of ethylene-inducible genes, and no induced resistance against *Pst* after treatment with various concentrations of the ethylene precursor 1-aminocyclopropane-1-carboxylate (ACC) (Table 1; Ton et al., 2001). Moreover, in the F_3 progeny of a cross between the ISR-inducible ecotype Col-0 and the ISR-noninducible ecotype RLD1, the reduced ethylene sensitivity of the RLD1 parent co-segregated with the recessive alleles of the *ISR1* locus, whereas the unaffected ethylene sensitivity of the Col-0 parent co-segregated with the

dominant alleles of the *ISR1* locus (Ton et al., 2001). These results suggest that the *ISR1* locus controls ethylene-dependent basal resistance, which is during ISR. Therefore, genotypes affected in the *ISR1* locus exhibit a reduced level of basal resistance, and concomitantly lack the ability to express ISR.

Table 1: Naturally occurring variation in ISR-inducibility and basal resistance against *Pst* among *Arabidopsis* ecotypes

Ecotype	ISR [a]		SAR [a]	Basal resistance [b]
	WCS417r	ACC	*avrPst*	
Col-0	43.3 *	32.9 *	61.2 *	2.4 ± 0.2
RLD1	-3.8	3.6	62.4 *	3.6 ± 0.1
Ws-0	-5.0	3.0	43.9 *	3.7 ± 0.2

[a] ISR was induced by transferring 2-week-old seedlings to soil containing *P. fluorescens* WCS417r bacteria at $5x10^7$ cfu.g^{-1}, or by dipping the leaves in a 0.25 M ACC solution containing 0.01 % Silwet three days before challenge inoculation. Five-week-old plants were challenge inoculated by dipping the leaves in a bacterial suspension of *Pst* at $2.5x10^5$ cfu.mL^{-1}. SAR was induced by pressure infiltrating a suspension of avirulent *Pst* (*avrPst*) at 10^7 cfu/mL^{-1} 3 days before challenge.

[b] Values presented are means (±SD) of the log of the proliferation of Pst over a 3-day time interval. Five-week-old plants were infected by pressure infiltrating a suspension of virulent *Pst* at $5x10^5$ cfu.mL^{-1} into the leaves. Immediately after infiltration, and 3 days later, the number of *Pst* bacteria per gram of leaf fresh weight was determined.

ISR and SAR in enhanced disease susceptibility mutants

Based on previous observations that *Arabidopsis* genotypes impaired in ISR and/or SAR show a reduced level of basal resistance against *Pst*, a collection of Arabidopsis mutants with enhanced disease susceptibility to pathogenic *P. syringae* pathovars was screened for induced resistance. Out of 11 *eds* mutants tested, three mutants (*eds4*, *eds8* and *eds10*) were affected in the expression of WCS417r-mediated ISR, whereas two (*eds5* and *eds12*) were blocked in the expression of pathogen-induced SAR. Further analysis of the ISR-impaired mutants revealed that *eds8* is disturbed in JA signaling and *eds4-1* in ethylene signaling. Although blocked in rhizobacteria-, MeJA-, and ACC-induced resistance against *Pst*, mutant *eds10* showed normal responsiveness to both methyl jasmonate (MeJA) and ACC, indicating that it harbors a mutation downstream the perception of ET in the defense pathway. Whereas *eds5* is known to be blocked in pathogen-induced accumulation of SA (Nawrath and Métraux, 1999), further analysis of *eds12* revealed that the SAR-impaired phenotype of this mutant is caused by a reduced sensitivity to SA, as evidenced by a reduced *PR1* transcription and an impaired SAR response upon treatment with SA. These results demonstrate that components contributing to SA-dependent basal resistance against *P. syringae* (EDS5 and EDS12) are required for the expression of SAR, whereas components contributing to JA/ethylene-dependent basal resistance (EDS4, EDS8 and EDS10) are required for WCS417r-mediated ISR.

Differential effectiveness of ISR and SAR

To further elucidate the relationship between basal resistance and induced resistance, we compared the effectiveness of SAR and ISR against different Arabidopsis pathogens that are primarily resisted through either SA-dependent, JA/ethylene-dependent, or a combination of SA- and JA/ethylene-dependent basal resistance. Activation of ISR resulted in a significant

level of protection against the fungal pathogen *Alternaria brassicicola*, which is resisted through JA/ethylene-dependent basal defenses (Thomma et al., 1998). Conversely, SAR was ineffective against this pathogen. Disease caused by the oomycete *Peronospora parasitica* or by turnip crinkle virus (TCV), which are both predominantly resisted through SA-dependent basal resistance (Delaney et al., 1994; Kachroo et al., 2000), were considerably reduced in plants expressing SAR, whereas activation of ISR yielded only weak, and no protection against these pathogens, respectively. Induction of SAR or ISR was equally effective against *X. campestris* pv. *armoraciae* that, like *Pst*, is resisted through a combination of SA- and JA/ethylene-dependent basal resistance. Apparently, SAR is effective against pathogens that are resisted through SA-dependent basal resistance, whereas ISR is effective predominantly against pathogens that are resisted through JA/ethylene-dependent basal resistance. Pathogens resisted through both SA- and JA/ET-dependent basal resistance are sensitive to both types of induced resistance (Table 2).

Table 2: Differential effectiveness of ISR and SAR against five *Arabidopsis* pathogens, as related to JA/ET-dependent and SA-dependent basal resistance.

Pathogen	Induced resistance [a]		Basal resistance [b]	
	ISR	SAR	JA/ET-dependent	SA-dependent
A. brassicicola	++		+	
X. campestris	++	++	+	+
P. syringae	++	+++	+	+
P. parasicita	+	+++		+
TCV	–	++		+

[a] +: weak resistance; ++: moderate resistance; +++:strong resistance

[b] based on the enhanced disease susceptibility of transgenics/mutants of Arabidopsis, impaired in either JA/ET-dependent, or SA-dependent defense signaling.

Concluding remarks

Collectively, our results suggest that induced resistance constitutes an enhancement of SA-dependent basal defenses in the case of pathogen-induced SAR, and of JA/ethylene-dependent basal resistance in the case of WCS417r-mediated ISR. Thus, defense responses that are active locally upon primary pathogen attack are enhanced by induction of ISR or SAR. Such association between induced resistance and basal resistance fits perfectly with the phenomenon variously referred to as "priming", "potentiation" or "sensitization". Potentiation is manifested upon challenge inoculation of plants expressing induced resistance as a stronger and faster activation of specific defenses compared to non-induced plants. Therefore, we conclude that ISR is achieved by a potentiated expression of JA- and ethylene-dependent basal defenses, whereas SAR is achieved by a potentiated expression of SA-dependent defenses.

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