Plant Pathology (2013) **62** (Suppl. 1), 43–55 Doi: 10.1111/ppa.12105

Costs and benefits of hormone-regulated plant defences

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Plants activate defence responses to protect themselves against microbial pathogens and herbivorous insects. However, induction of defences comes at a price, as the associated allocation costs, autotoxicity costs and ecological costs form fitness penalties. Upon pathogen or insect attack, resources are allocated to defences instead of to plant growth and reproduction, while above- and below-ground interactions with beneficial organisms may also be disturbed. The phytohormones salicylic acid and jasmonic acid are major players in the regulation of induced defences and their associated fitness costs. Hormone-controlled signalling pathways cross-communicate, providing the plant with a finely tuned defence regulatory system that can contribute to a reduction of fitness costs by repressing ineffective defences. However, this sophisticated regulatory system causes ecological costs, because activated resistance to one organism can suppress resistance to another. Moreover, the system can be hijacked by invading organisms that manipulate it for their own benefit. Priming for enhanced defence emerged as a defence mechanism with limited fitness costs. Because priming results in a faster and stronger activation of defence only after pathogen or insect attack, the limited costs of the primed state are often outweighed by the benefits in environments with pathogen or herbivore pressure. The balance between protection and fitness is crucial for a plant's success and is therefore of great interest for plant breeders and farmers. By combining molecular knowledge and ecological relevance of defence mechanisms, one can gain fundamental insight into how and why plants integrate different immune signals to cope with their natural multitrophic environment in a cost-effective manner.

Keywords: costs, fitness, hormone crosstalk, jasmonic acid, priming, salicylic acid

Introduction

During their lifetime, plants encounter innumerable attackers, including microbial pathogens and herbivorous insects that try to retrieve nutrients from the plant. Plants can ward off the majority of these attackers for which they rely on preformed defences and activation of their innate immune system. Preformed plant defences include physical barriers such as thick cuticles, rigid cell walls, thorns, needles and trichomes, and chemical weapons such as toxic or repellent compounds (Osbourn, 1996). In a second line of defence, inducible defences can be activated when pattern recognition receptors of plants recognize general features of microbial pathogens, such as flagellin, lipopolysaccharides, peptidoglycan, β-glucans and chitin, referred to as pathogen- or microbe-associated molecular patterns (PAMPs or MAMPs; Jones & Dangl, 2006; Pel & Pieterse, 2013). Similarly, recognition of so-called damage-associated molecular patterns (DAMPs), such as galacturonides, systemins and AtPep1, which are endogenous elicitors that accumulate as a result of enzymatic degradation of plant cell walls or proteins upon attack by pathogens or insects, leads to activation of defences (Fig.1; Lotze *et al.*, 2007; Boller & Felix, 2009; Heil, 2009; Ferrari *et al.*, 2013). Other defence-inducing compounds are the herbivore-associated molecular patterns (HAMPs), such as fatty acid–amino acid conjugates (FACs) from oral secretions (Felton & Tumlinson, 2008; Mithöfer & Boland, 2008) and effectors of pathogens that are produced to suppress immune responses but that the plants, under evolutionary pressure, have learned to recognize (Jones & Dangl, 2006).

The immune response that is activated upon pathogen or insect attack is modulated by the induced production of a phytohormonal blend in the plant. The phytohormones salicylic acid (SA), jasmonic acid (JA), ethylene (ET) and abscisic acid (ABA) are important regulators of induced defence mechanisms (Robert-Seilaniantz *et al.*, 2011; Pieterse *et al.*, 2012). Whereas SA and JA are the main players, ET and ABA have more modulating roles (van Loon *et al.*, 2006a; Ton *et al.*, 2009). The SA pathway is primarily induced by and effective against biotrophic pathogens, whereas the JA pathway is primarily induced by and effective against necrotrophic pathogens and herbivorous insects (Fig. 1; Glazebrook, 2005; Howe & Jander, 2008). The quantity, composition and timing of the hormonal signal signature tailors the defence response specifically to the attacker at hand, thereby prioritizing effective over ineffective defences, which minimizes fitness costs (De Vos *et al.*, 2005; Pieterse & Dicke, 2007).

The benefits of plant defences are obvious; they help the plant to survive in the presence of harmful organisms

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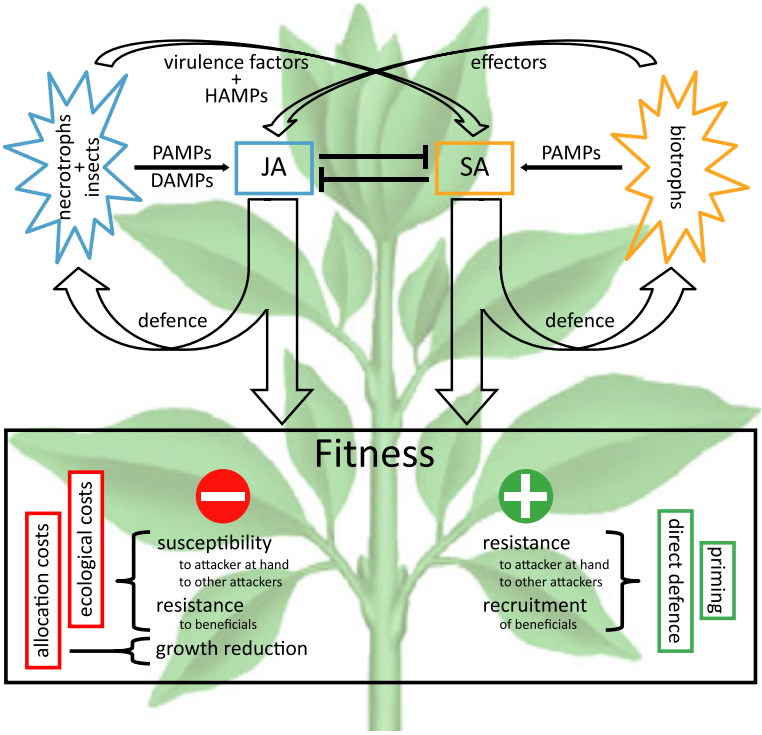

Figure 1 Schematic overview of hormone-regulated inducible defence responses and their effects on plant fitness. Upon attack by a necrotrophic pathogen or herbivorous insect, pathogen- or herbivore-derived pathogen-associated molecular patterns (PAMPs) and damage-associated molecular patterns (DAMPs) are recognized, leading to activation of jasmonic acid (JA)-dependent defence responses. Upon attack by a (hemi-) biotrophic pathogen, its PAMPs are recognized and salicylic acid (SA)-dependent defence responses are activated. These SA- and JA-dependent defence signalling pathways antagonize each other. The induced defence mechanisms have positive effects on the plant's fitness by enhancing resistance through direct activation and priming of defence and through recruitment of beneficial microorganisms. Negative effects of induced plant defences on plant fitness occur as well. Ecological costs are incurred via pathway crosstalk, through which an increase in resistance to one attacker leads to an increase in susceptibility to another attacker. In addition, pathway crosstalk can be hijacked by an attacker to antagonize effective defences, resulting in increased susceptibility. Necrotrophic pathogens and herbivorous insects can produce virulence factors and herbivore-associated molecular patterns (HAMPs), respectively, that activate the SA pathway leading to suppression of effective JA-dependent defence responses. (Hemi-)biotrophic pathogens can produce effectors that activate JA or other hormone signalling that acts antagonistically on SA-dependent defences. Furthermore, as ecological costs, beneficial microorganisms can be warded off by the plant's own defence mechanism. Allocation costs are incurred during activation of the plant's defence mechanisms, because valuable resources are used for defence rather than for growth and reproduction. Allocation costs during direct activation of defences are considerably larger than during priming of defences.

(Fig. 1). However, the inducible character of plant defences leaves a time slot between attack and the expression of defences in which the plant is vulnerable to the invading organism. Constitutive expression of defence traits does not have this drawback, making it probable that inducible defences have other selective advantages over constitutive defences (Heil & Baldwin, 2002). Fitness costs that are associated with defences have been postulated to be a driving force behind the evolution of inducible defences (Simms & Fritz, 1990). There are also costs associated with the genetic maintenance of inducibility, such as receptors and defence signal transduction routes, which all constitutively require energy and resources (Purrington, 2000; Cipollini *et al.*, 2003). These maintenance costs may be minimal, because many inducible pathway components have been co-opted from other processes, such as growth and development (Pieterse *et al.*, 2009).

The actual induced resistance status entails direct and indirect fitness costs. Direct resistance costs include allocation and autotoxicity costs (Heil & Baldwin, 2002; Strauss *et al.*, 2002). The latter is inflicted on plants by induced secondary chemicals that are toxic to the plant itself as well (Baldwin & Callahan, 1993; Heil & Baldwin, 2002; Strauss *et al.*, 2002). Allocation costs occur when valuable resources are allocated to resistance instead of to growth and reproduction (Herms & Mattson, 1992; Heil & Baldwin, 2002; Strauss *et al.*, 2002; Walters & Heil, 2007). Re-allocation of plant resources has also been postulated as a means by which the plant starves the pathogen in order to halt the infection (Canet *et al.*, 2010). Once induced, the enhanced resistance status needs to be maintained, but this is less costly (Van Hulten *et al.*, 2006).

Indirect resistance costs, also known as ecological costs (Heil & Baldwin, 2002; Strauss *et al.*, 2002), occur

as a result of the changed physiology of the plant that in turn affects interactions with other biotic and abiotic environmental factors, such as beneficial or harmful organisms, competing plants and resource availability (Heil, 2002; Cipollini *et al.*, 2003; Kessler & Halitschke, 2007; Poelman *et al.*, 2008; Traw & Bergelson, 2010). In this review, an overview of current knowledge on benefits and costs associated with inducible defences that are controlled by phytohormones is provided and how this knowledge can be applied for improved crop protection is discussed.

SA-inducible defences

SA is a phenolic compound that plays a key role in disease resistance signalling in plants (Mishina & Zeier, 2007; Vlot *et al.*, 2009). Besides its role in plant defence, SA also influences seed germination, vegetative growth, photosynthesis, respiration, thermogenesis, flower formation, seed production, senescence and responses to abiotic stress (reviewed in Rivas-San Vicente & Plasencia, 2011). Plants rapidly synthesize SA upon pathogen infection (Malamy *et al.*, 1990; Métraux *et al.*, 1990). SA can be synthesized via two distinct enzymatic pathways that both require chorismate. The bulk of pathogen-induced SA is produced from isochorismate via ISOCHORISMATE SYNTHASE 1 (ICS1), but chorismate can also be converted into SA via a series of enzymatic reactions initially catalysed by PHENYLALANINE AMMONIA LYASE (PAL; Vlot *et al.*, 2009). Defence signalling downstream of SA is largely regulated by the regulatory protein NONEXPRESSOR OF *PR* GENES1 (NPR1; Dong, 2004). Activation of the SA signalling pathway leads to a change in the cellular redox state, which reduces NPR1 from its inactive oligomeric form to its active monomeric form. Monomeric NPR1 is then translocated to the nucleus where it interacts with TGA transcription factors (Mou *et al.*, 2003; Dong, 2004; Moore *et al.*, 2011), resulting in the activation of a large set of defence-related genes, amongst which are genes coding for PATHOGENESIS-RELATED (PR) proteins and WRKY transcription factors (van Loon *et al.*, 2006b; Rushton *et al.*, 2010; van Verk *et al.*, 2011).

Benefits: SA triggers disease resistance

The first indication for a role of SA in disease resistance signalling came from White (1979), who showed that exogenous application of SA to tobacco plants enhanced resistance against *Tobacco mosaic virus* (TMV). Nowadays, numerous examples exist that demonstrate the resistance-inducing capacity of SA in a wide variety of plants against (hemi-)biotrophic pathogens and some phloem-feeding insects (Klessig & Malamy, 1994; Walling, 2008; Vlot *et al.*, 2009). The significance of SA was further shown by the use of mutant or transgenic plants (mostly in *Arabidopsis thaliana*, tobacco and tomato) that are affected in the production or the perception of SA. For example, transgenic *NahG* plants, which are incapable of accumulating SA, and mutant *npr1* plants, which are impaired in SA signalling, are more susceptible to oomycete, fungal, bacterial and viral pathogens (reviewed in Glazebrook, 2005).

Trade-offs: allocation costs of SA-inducible defences

Several studies investigated the costs of SA-inducible defences. In general, exogenous application of SA or its chemical analogue benzothiadiazole (BTH) has been shown to reduce plant growth and seed production of different plant species (Heil *et al.*, 2000; Cipollini, 2002; Canet *et al.*, 2010). However, environmental conditions such as growing period, competition with neighbouring plants, and nitrogen supply, can influence these fitness effects and sometimes avert the growth costs associated with SA-inducible defences (Heidel *et al.*, 2004; Dietrich *et al.*, 2005). *Arabidopsis* mutants constitutively expressing SA-inducible defences, such as *constitutive expressor of PR genes 1*, *5* and *6* (*cpr1*, *cpr5* and *cpr6*), were shown to be dwarfed and severely affected in seed production (Bowling *et al.*, 1994; Heil & Baldwin, 2002; Heidel *et al.*, 2004; Van Hulten *et al.*, 2006). Conversely, SA-deficient *NahG* and *SA induction deficient2* (*sid2*, mutated in the SA biosynthesis gene *ICS1*) *Arabidopsis* plants had higher growth rates and seed production compared to wildtype plants under pathogen-free conditions (Cipollini, 2002; Abreu & Munné-Bosch, 2009), confirming the negative effects of SA on growth and reproduction. The decrease in growth that was observed after treatment with BTH was reduced in the SA signalling mutant *npr1*, implying a pivotal role of NPR1 in inhibiting plant growth when SA-dependent resistance mechanisms are activated (Van Hulten *et al.*, 2006; Canet *et al.*, 2010). However, after infection with the SA-inducing downy mildew pathogen *Hyaloperonospora arabidopsidis*, *npr1* mutant plants displayed a lower fitness than wildtype plants (Heidel & Dong, 2006). This demonstrates that, although costly, SA-inducible defences are beneficial when plants grow under pathogen pressure. The beneficial effects of SA-regulated defences was particularly apparent under low-nutrient conditions (Heidel & Dong, 2006), which supports the theory on allocation costs as a driver of the evolution of inducible defences. Mutants *cpr1* and *cpr5* that constitutively express SA-regulated defences failed to show a fitness benefit under pathogen pressure, supporting the hypothesis that the inducible character of SA-dependent resistance prevents excessive fitness costs (Heidel & Dong, 2006; Van Hulten *et al.*, 2006).

Although negative effects of SA on fitness have mostly been ascribed to allocation costs (Heil *et al.*, 2000; Walters & Heil, 2007), toxic effects of SA may also contribute to reduced fitness (Bi *et al.*, 2010; Asaduzzaman & Asao, 2012). However, most studies focusing on autotoxicity costs of SA have not included plant genotypes that rule out effects of allocation costs, e.g. SA signalling mutants such as *npr1*, which makes claims on a role for

SA in autotoxicity costs obscure. Moreover, most studies on allocation costs of SA signalling that have tested *npr1* made use of BTH as inducer of the SA pathway, which induces SA signalling and resistance without the toxic side effects of SA (Lawton *et al.*, 1996), thereby omitting autotoxicity effects of SA in their studies. One of the few studies that applied SA to *npr1* (Cipollini, 2002) found no decrease in seed set in comparison to non-treated *npr1* plants, whereas SA treatment did decrease seed set in wildtype plants, indicating that the costs incurred by SA are (mostly) allocation costs and not autotoxicity costs. Besides direct allocation costs, SA-inducible defences also inflict ecological costs, which include crosstalk effects between the SA and JA signalling pathways, which are described in the section on crosstalk.

JA-inducible defences

JA is a key regulator in the defence response against herbivorous insects and necrotrophic pathogens (Glazebrook, 2005; Howe & Jander, 2008). Besides its essential role in regulating disease and pest resistance, JA has also been implicated in senescence, root growth, fruit ripening, tendril coiling, pollen development, tuberization and responsiveness to abiotic stress (Wasternack & Hause, 2013).

JA is an oxylipin that accumulates rapidly in plants in response to wounding, herbivory and infection by necrotrophs (Creelman *et al.*, 1992; Penninckx *et al.*, 1996). The initial phase of JA formation takes place in the chloroplasts, where fatty acids of membrane lipids (e.g. linoleic acid) are metabolized by lipoxygenases to generate oxylipins including the JA precursor 12-oxo-phytodienoic acid (OPDA). Subsequently, OPDA is transported to the peroxisomes where it undergoes three steps of β-oxidation to generate JA (reviewed in Wasternack & Hause, 2013). JA can be conjugated to amino acids, such as L-isoleucine, resulting in JA-Ile, the most biologically active member of the JAs (Staswick & Tiryaki, 2004; Fonseca *et al.*, 2009). The F-box protein CORONATINE INSENSITIVE1 (COI1) is a key regulator of the JA signalling pathway (Xie *et al.*, 1998), as it is part of the JA receptor complex (Yan *et al.*, 2009; Sheard *et al.*, 2010). Binding of JA to COI1 targets JASMONATE ZIM-DOMAIN (JAZ) proteins for degradation via the 26S proteasome pathway (Chini *et al.*, 2007; Thines *et al.*, 2007). In the uninduced state, JAZ proteins repress JA-responsive gene expression by binding to transcriptional activators, such as MYC2, 3 and 4 (Fernández-Calvo *et al.*, 2011). Accumulation of JA triggers the degradation of JAZ proteins, resulting in derepression of JA-regulated genes.

In *Arabidopsis*, there are two distinct branches of the JA signalling pathway that antagonize each other. The ERF-branch is activated upon infection with necrotrophic pathogens and is regulated by the APETALA2/ETHYLENE RESPONSE FACTOR (AP2/ERF)-domain containing transcription factors ERF1 and ORA59 (Anderson *et al.*, 2004; Pré *et al.*, 2008). The ERF-branch of the JA response pathway also requires ET and results in the activation of the marker gene *PLANT DEFENSIN1.2* (*PDF1.2*; Penninckx *et al.*, 1998; Lorenzo *et al.*, 2003). The MYC-branch is activated upon wounding and feeding by herbivorous insects and is regulated by the MYC transcription factors MYC2, 3 and 4 in a synergistic action with ABA (Anderson *et al.*, 2004; Fernández-Calvo *et al.*, 2011; Niu *et al.*, 2011). Activation of the MYC-branch leads to induced expression of the marker gene *VEGETATIVE STORAGE PROTEIN2* (*VSP2*; Lorenzo *et al.*, 2004).

Benefits: JA triggers disease resistance

Many JA-inducible defence responses and their effectiveness in plant resistance against diseases and pests were identified by exogenous application of JA and by the analysis of mutants with defects in JA signalling compounds such as COI1, MYCs and ERFs. This demonstrated that JA signalling is indispensable for resistance to a wide range of necrotrophic pathogens and herbivorous insects, whereby in general the ERF-branch is associated with resistance against necrotrophic pathogens (Berrocal-Lobo *et al.*, 2002; Lorenzo *et al.*, 2003) and the MYC-branch with resistance against herbivorous insects (Lorenzo *et al.*, 2004; Howe & Jander, 2008; Kazan & Manners, 2012).

Trade-offs: allocation costs of JA-inducible defences

Studies on the costs of JA-inducible defences have mostly been executed with plants that were infested with insects. In contrast, cost studies on JA-inducible defences that are associated with infection by necrotrophic pathogens are scarce. Infestation by insects and exogenous application of JA comes with costs, which is apparent from a decreased seed set and delayed flowering and fruit ripening (Agrawal *et al.*, 1999; Redman *et al.*, 2001; van Dam & Baldwin, 2001). In addition, the *Arabidopsis* mutant *cev1* and the transgenic line overexpressing JA carboxyl methyltransferase (JMT), both constitutively expressing JA-dependent defences, showed reduced growth phenotypes (Ellis & Turner, 2001; Cipollini, 2010). The effect on delayed flowering of JMT-overexpressing lines was especially apparent under low nutrient conditions (Cipollini, 2010). Furthermore, competition with neighbouring plants increased JA-induced fitness costs in tobacco (van Dam & Baldwin, 2001). The JA-associated trade-offs in reduced plant performance have therefore been mostly explained by allocation costs. However, JA is also known to directly regulate several plant developmental processes such as growth and seed production (Creelman & Mullet, 1997; Yang *et al.*, 2012), which complicates the assignment of the origin of the fitness decrease detected in plants expressing JA responses. Despite the fact that JA-induced responses are costly, they benefit plants when under attack, even in field situations (Baldwin, 1998). To our knowledge no studies on autotoxicity costs of JA have been described

so far. Ecological costs of JA-inducible defences include crosstalk effects between the ERF- and MYC-branches of the JA signalling pathway, which are described in the section below on crosstalk.

Hormonal crosstalk in defence signalling

Plant hormones are integral to plant immune responses and are differentially effective against different types of attackers. During plant–attacker interactions, multiple hormones are induced that together steer the immune response of the plant (De Vos *et al.*, 2005). Hormone homeostasis is vital for a successful immune response upon attack, as extensive cross-communication between defence signalling pathways allows the plant to fine-tune the defence response to the attacker at hand (Reymond & Farmer, 1998). Hormonal crosstalk has often been interpreted as a cost-saving strategy and may have evolved as a means of the plant to reduce allocation costs by repression of unnecessary defences that are ineffective against the attacker that is encountered (Pieterse & Dicke, 2007; Thaler *et al.*, 2012). However, proof for this hypothesis has not been demonstrated yet, as to the authors' knowledge there has been no study that measured the fitness levels of plants exhibiting hormonal crosstalk in comparison to that of crosstalk mutant plants. In this review crosstalk between SA and JA signalling, and between the ERF- and MYC-branches of the JA signalling pathway is covered, but other hormones have also been reported to modulate hormone-controlled immune signalling (Robert-Seilaniantz *et al.*, 2011; Pieterse *et al.*, 2012).

Crosstalk between the SA and JA pathways

The first indication of crosstalk between SA and JA signalling came from Doherty *et al.* (1988), who showed that SA and its acetylated derivative aspirin are strong antagonists of the JA pathway. Many cases of crosstalk between the SA and JA pathways have been reported since (Bostock, 2005; Stout *et al.*, 2006; Pieterse *et al.*, 2012). Pharmacological experiments with *Arabidopsis* revealed that the JA-responsive genes *PDF1.2* and *VSP2* are highly sensitive to suppression by SA. The antagonistic effect of SA on JA signalling was observed in a large number of *Arabidopsis* accessions (Koornneef *et al.*, 2008) and was even reported to remain active in the next generation (Luna *et al.*, 2012), highlighting the potential significance of this phenomenon in the regulation of induced plant defences in nature.

Several important regulatory proteins of SA–JA pathway crosstalk have been identified, including NPR1 (Spoel *et al.*, 2003; Luna *et al.*, 2012; Pieterse *et al.*, 2012). Timing and concentration of ET was shown to modulate the strength of SA–JA crosstalk and its NPR1-dependency. Recent data showed that, in *Arabidopsis*, SA does not affect JA biosynthesis but it affects JA signalling downstream of COI1, at the level of transcriptional regulation of JA-responsive genes (Leon-Reyes *et al.*, 2010b; van der Does *et al.*, 2013). The ERF transcriptional activator ORA59, which regulates many JA-responsive genes, was identified as an important target for SA (van der Does *et al.*, 2013).

Many studies found an antagonistic effect of SA signalling on the JA pathway. However, in several cases JA signalling could suppress the SA pathway as well. A few molecular players have been reported to play a role in this JA–SA crosstalk, such as COI1 and MYC2 (Zheng *et al.*, 2012). Furthermore, neutral and synergistic interactions between SA and JA have also been reported (Schenk *et al.*, 2000; van Wees *et al.*, 2000; Mur *et al.*, 2006). Timing, sequence of initiation and the relative concentration of each hormone are important for the outcome of the SA–JA crosstalk (Mur *et al.*, 2006; Koornneef *et al.*, 2008; Leon-Reyes *et al.*, 2010a). Besides the aforementioned ET, other hormones can also modulate SA–JA crosstalk (Pieterse *et al.*, 2012). Under different plant–attacker conditions, simultaneous inductions of SA and JA signalling do not always lead to predicted outcomes, highlighting the complexity of the hormonal interactions.

Crosstalk between the ERF- and MYC-branches of the JA pathway

As described above, defence responses to necrotrophic pathogens and herbivorous insects are regulated by distinct branches of the JA signalling pathway: the ERF- and the MYC-branch, respectively (Lorenzo & Solano, 2005; Kazan & Manners, 2008; Pieterse *et al.*, 2012). Transcriptional changes in response to these diverse types of attackers show limited overlap, suggesting that the context in which the induced JA signal is perceived is crucial in tuning the JA response (De Vos *et al.*, 2005). The hormones ET and ABA determine the direction in which the JA pathway is steered, towards the ERF- or the MYC-branch, respectively (Verhage *et al.*, 2011; Pieterse *et al.*, 2012). Activation of the ERF-branch resulted in reduced expression of the MYC-branch marker gene *VSP2*, whilst silencing of *ORA59* or mutating the ET pathway caused enhanced levels of *VSP2* expression (Lorenzo *et al.*, 2004; Verhage *et al.*, 2011). Reciprocally, activation of the MYC-branch suppressed transcription of the ERF-branch marker gene *PDF1.2*, whereas mutation of MYC transcription factors genes or ABA signalling components led to enhanced expression of *PDF1.2* and the ERF transcription factor gene *ORA59* (Verhage *et al.*, 2011). These data clearly indicate a mutually antagonistic interaction between the different branches of the JA pathway.

Ecological costs of defence signalling

Fitness costs associated with induced defence arise from allocation and ecological costs (Fig. 1). Allocation costs are incurred when resources are allocated to resistance instead of to growth and reproduction (Heil & Baldwin,

2002) and have been described in the previous sections on SA- and JA-inducible defences. Ecological costs arise when defence-induced plants have altered abilities to interact with their biotic and abiotic environment (adjusted from Heil, 2002). For example, induction of the JA defence pathway resulted in reduced numbers of visitations by beneficial pollinators (Strauss *et al.*, 2002) and JA-regulated herbivore-induced plant volatiles (HIPVs) alter the interaction of a plant with herbivores, carnivores and competing plants (Dicke & van Loon, 2000). Also above- and below-ground interactions with beneficial microbes can be affected by the activation of defence in foliar tissue. Exogenous application of SA to the soil inhibited the growth and formation of root nodules in the *Rhizobium*–legume symbiosis, whereas growth of *Rhizobium* cells itself was not affected by SA (Sato *et al.*, 2002; Mabood & Smith, 2007). Furthermore, de Román *et al.* (2011) found that foliar application of acibenzolar-S-methyl (ASM), a functional analogue of SA, to soybean led to a transient reduction in arbuscular mycorrhizal colonization of roots. Negative effects of foliar herbivory on colonization of the roots by mycorrhizal fungi have also been reported (Barber *et al.*, 2012). In contrast, recruitment of soilborne beneficial microbes upon stress induction in the leaves has also been shown (Fig. 1). Beneficial *Bacillus subtilis* bacteria were recruited to the rhizosphere upon foliar infection of *Arabidopsis* with the bacterial pathogen *Pseudomonas syringae* (Rudrappa *et al.*, 2008). Moreover, foliar application of JA and wounding of the leaves of *Medicago truncatula* resulted in enhanced JA signalling and enhanced mycorrhization by *Rhizophagus irregularis* (formerly known as *Glomus intraradices*; Landgraf *et al.*, 2012). These results show the importance of testing effects of altered defences in plants under realistic environmental conditions, because otherwise relevant ecological costs might be missed. The net effect of induced defence signalling on plant fitness strongly depends on the community context.

Ecological costs as a result of hormonal crosstalk are becoming increasingly recognized. Whereas hormonal crosstalk may be advantageous for the plant to keep allocation costs in check, evidence is accumulating that crosstalk at the level of gene expression is translated into crosstalk at the level of resistance. When plants encounter multiple attackers simultaneously or successively, the induction of a hormone signalling route might elevate the resistance to one attacker, but at the same time hormonal crosstalk can decrease the resistance to another attacker (Pieterse *et al.*, 2012). Furthermore, as a common virulence strategy, successful pathogens and insects can hijack crosstalk mechanisms by targeting plant hormone biosynthesis and perception to rewire immune signalling, rendering the plants more susceptible (Fig. 1; Grant & Jones, 2009; Verhage *et al.*, 2010; Pieterse *et al.*, 2012). Ecological costs of crosstalk between different hormones are described in the next sections, focusing on crosstalk between SA and JA signalling, and between the ERF- and MYC-branches of JA signalling.

Ecological costs of SA–JA crosstalk

Many examples of ecological costs of SA–JA crosstalk have been described. For instance, in *Arabidopsis* infection by the hemibiotrophic pathogen *P. syringae* leads to induction of the SA pathway, resulting in an effective resistance response against this pathogen. However, through SA–JA crosstalk mechanisms JA signalling is suppressed, which renders the infected leaves more susceptible to the necrotrophic fungus *Alternaria brassicicola* (Spoel *et al.*, 2007). Similarly, induction of SA signalling in *Arabidopsis* by exogenous application of SA inhibited JA-induced resistance to the generalist herbivores *Spodoptera exigua* and *Trichoplusia ni* (Cipollini *et al.*, 2004; Cui *et al.*, 2005). In tobacco, *Manduca sexta* larvae consumed up to 2·5-times more leaf tissue from plants exhibiting increased SA signalling after inoculation with TMV than from mock-treated plants (Preston *et al.*, 1999). Furthermore, reduced SA signalling in *Arabidopsis* genotypes *NahG* and *npr1* was correlated with reduced feeding by *T. ni* in comparison to wildtype plants (Cui *et al.*, 2002). Crosstalk the other way around, namely of JA on SA, was also effective on the level of disease resistance: the JA-insensitive mutant *coi1* showed enhanced expression of SA-dependent defences and enhanced resistance to *P. syringae* (Kloek *et al.*, 2001).

Several pathogens have evolved ways to hijack host crosstalk mechanisms as a virulence strategy. One of the best-studied examples is the production of coronatine by *P. syringae*. Coronatine is a pathogen-derived functional and structural mimic of JA-Ile that suppresses SA signalling, thereby promoting susceptibility to this pathogen (Kloek *et al.*, 2001; Brooks *et al.*, 2005; Cui *et al.*, 2005; Zheng *et al.*, 2012). Furthermore, the necrotrophic pathogen *Botrytis cinerea* was shown to produce an exopolysaccharide that acts as an elicitor of the SA pathway and causes suppression of the JA pathway and consequently promotes pathogen growth (El Oirdi *et al.*, 2011). Likewise, nymphs of the phloem-feeding silverleaf whitefly *Bemisia tabaci* activated SA-responsive gene expression in *Arabidopsis*, thereby suppressing the JA signalling pathway. This was shown to be associated with accelerated nymphal development, suggesting that the nymphs of *B. tabaci* can rewire the plant's immune signalling network to their own benefit (Zarate *et al.*, 2007). Additionally, eggs of *Pieris brassicae* butterflies have been reported to induce SA signalling upon oviposition, which suppresses JA signalling and provides an advantage for the freshly hatched larvae (Bruessow *et al.*, 2010).

Ecological costs of crosstalk between the ERF- and MYC-branches

Indications of effects of crosstalk between the ERF- and MYC-branches on the level of resistance to insects and necrotrophic pathogens have come mainly from studies with *Arabidopsis* mutants affected in one of the branches. In a two-choice setup, larvae of the specialist

insect herbivore *Pieris rapae* preferred to feed from *Arabidopsis* genotypes that highly expressed the ERF-branch of the JA pathway, such as MYC2 mutant *jin1* plants and ORA59-overexpressing plants, over wildtype plants that highly expressed the MYC-branch upon feeding by the caterpillars (Verhage *et al.*, 2011). Furthermore, the *jin1* mutant and the ABA biosynthesis mutant *aba2* were more resistant to the necrotrophic pathogens *B. cinerea*, *Plectosphaerella cucumerina* and *Fusarium oxysporum* due to a potentiated expression of the ERF-branch in these mutants (Anderson *et al.*, 2004; Lorenzo *et al.*, 2004; Nickstadt *et al.*, 2004; Adie *et al.*, 2007). A comparable mechanism may underlie findings from the early 1990s with black bean aphids, *Aphis fabae*, that displayed a higher growth rate and fecundity on bean leaves infected with the necrotrophic pathogen *Botrytis fabae*, compared to uninfected leaves (Zebitz & Kehlenbeck, 1991).

Indications of hijacking of hormone crosstalk mechanisms by attackers have been found for ERF–MYC crosstalk as well. For example, application of the oral secretion of *P. rapae* caterpillars into wounded leaf tissue stimulated expression of the ERF-branch, suggesting that insecticidal compounds have the potential to manipulate the plant response towards the caterpillar-preferred ERF-branch (Verhage *et al.*, 2011; Fig. 1). Together, these data support the existence of high ecological costs of crosstalk on the level of resistance to pathogens and pests.

Priming for enhanced defence

Besides the fact that inducible defences involve fitness costs, the inducibility of defences comes with an unsafe time slot between attack and the expression of defences. This might have been a driving force for the development of a sophisticated, cost-effective way to activate inducible defence responses, namely by priming (Fig. 1). Plants that are primed for enhanced defence do not express defences in the absence of an attacker, but show a faster and stronger activation of cellular defence responses upon attack compared to non-primed control plants (Conrath *et al.*, 2002, 2006; Frost *et al.*, 2008). Prior activation of defences is not a prerequisite for the primed state, which makes priming a cost-efficient form of induced immunity. Another benefit of priming is that it offers enhanced resistance against a broad spectrum of attackers. Multiple inducers of priming for defence have been identified, including beneficial microbes, pathogens and herbivorous insects, but also chemical elicitors and wounding (Conrath *et al.*, 2002, 2006). The diverse forms of priming are described in the sections below.

Various mechanisms underlying priming have been reported. Inactive cellular proteins that play a role in cellular signal amplification have been shown to accumulate in primed plants where they remain dormant until activation by stressors, resulting in an accelerated response. Examples of such dormant signal transducers implicated in priming are transcription factors and mitogen-activated protein kinases (MPKs; Pozo *et al.*, 2008; Beckers *et al.*, 2009; Van der Ent *et al.*, 2009). Chromatin modifications at the promoters of priming-associated genes have also been implicated in the regulation of the primed state (Jaskiewicz *et al.*, 2011; Luna *et al.*, 2012; Rasmann *et al.*, 2012). Priming has in several cases been demonstrated to be transferred to the plant's offspring, which in some cases was associated with epigenetic changes, allowing plants to retain memory of a threatening situation into one or more successive plant generations (Luna *et al.*, 2012; Pieterse, 2012; Rasmann *et al.*, 2012; Slaughter *et al.*, 2012).

SA-dependent systemic acquired resistance

Systemic acquired resistance (SAR) is a well-studied form of induced resistance in which priming is thought to play an important role (Conrath *et al.*, 2002, 2006; Durrant & Dong, 2004; Vlot *et al.*, 2009). SAR is activated locally and systemically upon infection by a (hemi-)biotrophic pathogen and enhances resistance of uninfected plant parts to subsequent infection by the same or a broad range of other pathogens. SAR was first described by Ross (1961), who demonstrated that uninfected leaves of TMV-infected tobacco plants became more resistant to subsequent infection by TMV. SAR is associated with endogenous accumulation of SA, both at the site of infection and in healthy systemic tissues. Mutant plants that are impaired in SA signalling, including the *npr1* mutant, are incapable of developing SAR, indicating that SAR requires SA signalling (Durrant & Dong, 2004). Recently, several long-distance signals involved in the communication between SAR-induced tissue and systemic SAR-expressing tissue have been identified (Vlot *et al.*, 2009; Dempsey & Klessig, 2012; Shah & Zeier, 2013). SAR is accompanied by priming of SA-dependent defences, resulting in potentiated expression of SA-responsive genes, such as *PR1* (Mur *et al.*, 1996; van Wees *et al.*, 1999). Additionally, SA-independent callose deposition is primed during SAR, resulting in accelerated strengthening of the cell wall at the site of pathogen penetration (Ton & Mauch-Mani, 2004). Exogenous application of low concentrations of SA or BTH does not directly activate defences, but primes plants for enhanced expression of cellular defences after pathogen attack (Conrath *et al.*, 2002, 2006). This indicates that SA-mediated priming is an intrinsic part of pathogen-activated SAR.

JA/ET-dependent induced systemic resistance

Induced systemic resistance (ISR) triggered by nonpathogenic microbes is another well-studied form of induced resistance in which priming plays an important role. Plant roots contain a large number of rhizosphere-associated microbes, called the root microbiome, that aid in plant growth and reproduction (Berendsen *et al.*, 2012). Beneficial ISR-inducing microbes include soilborne plant

growth-promoting rhizobacteria (PGPR) and fungi (PGPF) as well as mycorrhizal fungi (Van Hulten *et al.*, 2010; Zamioudis & Pieterse, 2012). Like SAR, ISR offers a broad-spectrum resistance to foliar and root pathogens, but on top of that it is also effective against certain herbivores (van Wees *et al.*, 2008; Pineda *et al.*, 2010). In contrast to SAR, rhizobacteria-mediated ISR was shown to be independent of SA and instead requires JA, ET and ABA signalling (van Wees *et al.*, 2008; Van der Ent *et al.*, 2009; Pieterse *et al.*, 2012). Most SA-signalling components in *Arabidopsis* proved to be dispensable for ISR, except the NPR1 protein that acts downstream of JA and ET in ISR (Pieterse *et al.*, 1998; van Wees *et al.*, 2000).

Large-scale gene expression analysis revealed that induction of ISR can occur with only minor changes in gene expression in the leaves (Verhagen *et al.*, 2004; van Wees *et al.*, 2008; Van der Ent *et al.*, 2009). However, upon encounter with an invader, ISR-expressing plants show enhanced and accelerated expression of JA- and ET-regulated genes and accumulation of callose-rich papillae at the site of infection (Verhagen *et al.*, 2004; Pozo & Azcón-Aguilar, 2007; Pozo *et al.*, 2008). Concordantly, ISR is predominantly effective against necrotrophic pathogens and herbivorous insects, but also offers protection against biotrophs that are sensitive to cell wall defences (Ton *et al.*, 2002; van Oosten *et al.*, 2008). In roots, the induction of ISR results in altered expression of several genes, including the transcription factor gene *MYB72* (Verhagen *et al.*, 2004) that emerged as an important component of ISR, as *myb72* mutants were abolished in their ability to express ISR (Van der Ent *et al.*, 2008; Segarra *et al.*, 2009).

Wound-induced resistance

Priming has most often been studied in the context of plant–pathogen interactions, but plants can also be primed by signals associated with herbivore feeding. Tissue damage can lead to wound-induced resistance (WIR), which can be induced in neighbouring plants via HIPVs or in systemic leaves of the same plant via internal signals or externally via HIPVs (Heil & Silva Bueno, 2007; Frost *et al.*, 2008). These HIPVs prime the plant for JA-inducible defence mechanisms, or act as signals to attract parasitic and predatory insects to combat attacking herbivores (Baldwin *et al.*, 2006; Ton *et al.*, 2007). In addition to the effect on insect performance, WIR can also prime the plant for enhanced resistance against microbial pathogens (De Vos *et al.*, 2006).

Crosstalk during priming

Despite the shared dependency on the NPR1 protein, distinct signalling cascades are important for SAR and ISR, requiring SA or JA and ET, respectively (Pieterse *et al.*, 1998; van Wees *et al.*, 2000). Crosstalk between the SA and JA pathways could entail high ecological costs, as described in a previous section, but is this also true for SA- and JA-dependent priming? Simultaneous induction of SAR and ISR was shown to result in an additive effect on the level of resistance against *P. syringae* (van Wees *et al.*, 2000). In plants mutated in either the SA or JA signalling pathway, this additive effect was not found. Furthermore, induction of ISR did not affect expression of SAR-induced *PR1*. Thus, there is no evidence for SA–JA crosstalk during simultaneous activation of SAR and ISR. The *Arabidopsis* accession Bur-0 is constitutively primed for both *PR1* and *PDF1.2* expression upon exogenous application of SA and JA, respectively. Consequently, Bur-0 is more resistant to the hemibiotrophic pathogen *P. syringae* and also to the necrotrophic pathogen *P. cucumerina* (Ahmad *et al.*, 2011). Together, these results suggest that there is no SA–JA crosstalk during priming. However, when SAR was inherited in the next generation, the progeny showed a weaker induction of the JA-inducible gene *PDF1.2*, which was accompanied by increased susceptibility to the necrotrophic pathogen *B. cinerea*. This was associated with a chromatin modification at the *PDF1.2* promoter that is associated with transcriptional silencing (Luna *et al.*, 2012). Mycorrhizal fungi and PGPR have been reported to induce resistance to leaf chewing insects but also to increase susceptibility to phloem feeders (Koricheva *et al.*, 2009; Pineda *et al.*, 2010). These findings were recently expanded with studies on the effects of the ISR-inducing *Pseudomonas fluorescens* on the attraction of parasitoids by volatiles of aphid-infested plants. Pineda *et al.* (2013) reported that ISR-expressing plants attracted less parasitoids. It is unknown whether there is a role for SA–JA crosstalk in this ecological cost of ISR.

Benefits: limited allocation costs of priming

Priming of *Arabidopsis* with low concentrations of β-amino-butyric acid (BABA) was shown to have only marginal effects on plant growth and seed production in the absence of pathogens, suggesting that there are no or only limited allocation costs associated with priming (Van Hulten *et al.*, 2006). In the presence of pathogens, a clear fitness advantage was observed for primed plants over non-primed plants and plants expressing constitutive defences. Walters *et al.* (2008) also found that priming in barley by saccharin did not incur fitness costs, both in greenhouse and field conditions. Furthermore, there are several studies that show that PGPR not only prime for defence but also increase plant growth and seed production, although these traits are not causally related (Raupach & Kloepper, 1998; Zehnder *et al.*, 2001; Zamioudis *et al.*, 2013). These results indicate that there are fitness benefits for plants that interact with PGPR whilst no allocation costs are associated with this. Ahmad *et al.* (2011) found that the *Arabidopsis* accession Bur-0 is constitutively primed for enhanced defence against pathogens and insects, without growth restraints. Together these results show that the benefits of priming outweigh the marginal costs of it in environments in which disease occurs. Therefore, priming for enhanced

defence seems to be a very useful tool for application in crop protection.

Inducible defences and trade-offs with crop protection

In complex natural environments, plants encounter a multitude of pathogens and pests. In agriculture this leads to tremendous annual crop losses, representing a total value of over €450 billion worldwide. Allocation and ecological costs of (induced) plant defences are a major problem for the implementation of induced resistance in agriculture (Walters & Heil, 2007). To successfully use inducible defences in crop protection, a functional understanding of the physiological and ecological consequences of the induced state is indispensable and demands more research (Bostock, 2005; Koornneef & Pieterse, 2008). Hormonal pathway crosstalk presents a challenge for translating fundamental knowledge into crop disease resistance traits. Plants often have to deal with simultaneous or subsequent attack by very different attackers. Genetic traits that are associated with contrasting resistance mechanisms to different attackers, for example SA signalling that causes elevated resistance to biotrophs, but reduced resistance to necrotrophs, can greatly impact plant fitness and thus crop yield. The extensive interactions between different hormone signalling routes that are activated upon encounter of a plant with an attacker, and the concentration-, space- and time-dependent context in which this occurs, need to be dissected. However, to fully comprehend the plant's immune system, so that this knowledge can be applied to sustainable agriculture, plants need to be studied in agricultural and natural environments as well, because predictions on hormonal interactions and fitness effects during the encounter of plants with their biotic and abiotic environment do not always lead to the predicted outcomes (Clarke *et al.*, 2009; Ritsema *et al.*, 2010; Ballaré, 2011; Cerrudo *et al.*, 2012).

Breeders usually select for plant traits, such as yield and quality, while disease resistance is rarely in the top three of selected traits (Brown, 2002). Elevated resistance is usually correlated with detrimental effects on yield, but genetically and physiologically it is possible to heighten disease resistance while conserving plant fitness (Bechtold *et al.*, 2010). Research into the mechanisms of how plants successfully combine high disease resistance and high yield could open up new possibilities for the development of valuable crop species. Furthermore, priming for enhanced defence provides also an opportunity to protect plant species while minimizing the costs of resistance (Van Hulten *et al.*, 2006). Simultaneous activation of ISR and SAR provides an attractive tool for the improvement of crop species (van Wees *et al.*, 2000). Overall, understanding of the functioning of the complex defence signalling network and the fitness costs involved is necessary for successful application of defence traits in crops. Therefore, molecular biologists and ecologists should join forces to place molecular mechanisms of inducible plant defences in an ecological perspective.

Acknowledgements

The authors of this review are supported by VIDI grant no. 11281 of the Dutch Technology Foundation STW, which is part of the Netherlands Organization of Scientific Research, and ERC Advanced Investigator Grant no. 269072 of the European Research Council.

Conflicts of interest

The authors have no conflicts of interest to declare.

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