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REVIEW PAPER

On the move: induced resistance in monocots

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Abstract

Although plants possess an arsenal of constitutive defences such as structural barriers and preformed antimicrobial defences, many attackers are able to overcome the pre-existing defence layers. In response, a range of inducible plant defences is set up to battle these pathogens. These mechanisms, commonly integrated as induced resistance (IR), control pathogens and pests by the activation of specific defence pathways. IR mechanisms have been extensively studied in the Dicotyledoneae, whereas knowledge of IR in monocotyledonous plants, including the globally important graminaceous crop plants, is elusive. Considering the potential of IR for sustainable agriculture and the recent advances in monocot genomics and biotechnology, IR in monocots is an emerging research field. In the following, current facts and trends concerning basal immunity, and systemic acquired/induced systemic resistance in the defence of monocots against pathogens and herbivores will be summarized.

Key words: Crops, inducible defence, plant immunity, systemic resistance.

Introduction

Plants are continuously confronted with an armada of different pathogens and pests. These potential attackers utilize diverse tactics to clash with the plant defensive system. Bacteria can invade plants through natural openings such as stomata or wounds, pathogenic fungi can violently break cell walls to enter the host cell (Fig. 1), and insect herbivores employ enzymes to attenuate plant toxins. Moreover, pathogens are able to manipulate plant immunity by delivering effector molecules that are hijacking the defence pathways. Nonetheless, only a few pathogens successfully infect a specific plant species, although plants, unlike animals, do not possess specialized and mobile defender cells. Thus, the self-protection plants have developed throughout the evolutionary arms race with their attackers has to be highly intricate and efficient to help in surviving the diverse biological stress situations.

In order to defend themselves, plants are armed with constitutive, pre-existing defences such as cell wall barriers or pre-formed and stored antimicrobial toxins. In such cases where attackers are able to overcome the constitutive defence layers, they face an arsenal of inducible defences (Fig. 1; Pieterse *et al.*, 2009; Spoel and Dong, 2012). During an initial phase, plant cells exert a so-called 'innate immunity'. In a first branch of this immunity, pathogen- or microbe-associated molecular patterns (PAMPs/MAMPs) such as chitin or flagellin are recognized by membrane-localized pattern-recognition receptors (PRRs) (Zipfel, 2009). The perception of MAMPs by PRRs leads to the activation of multiple downstream defence signalling events. The second branch of the plant innate immune system acts mostly in the cytoplasm; NB-LRR (nucleotide-binding leucine

Abbreviations: ABA, abscisic acid; AHL, *N*-acyl homoserine lactone; Avr, avirulence; BABA, β-aminobutyric acid; DAMP, damage-associated molecular pattern; DIMBOA, 2,4-dihydroxy-7-methoxy-2H-1,4-benzoxazin-3(4H)-one; ET, ethylene; ETI, effector-triggered immunity; GLV, green leaf volatiles; HAMP, herbivore-associated molecular pattern; HR, hypersensitive response; IR, induced resistance; ISR, induced systemic resistance; JA, jasmonic acid; LAR, local acquired resistance; LPS, bacterial lipopolysaccharides; MAMP, microbe-associated molecular pattern; MeJA, methyl jasmonate; MeSA, methyl salicylate; NB-LRR, nucleotide-binding leucine rich repeat; PAMP, pathogen-associated molecular pattern; PGPF, plant growth promoting endophytic fungi; PGPR, plant growth promoting rhizobacteria; PR, pathogenesis-related; PRR, pattern-recognition receptor; PTI, pattern-triggered immunity; R gene, resistance gene; ROS, reactive oxygen species; SA, salicylic acid; SAR, systemic acquired resistance; VOC, volatile organic compound.

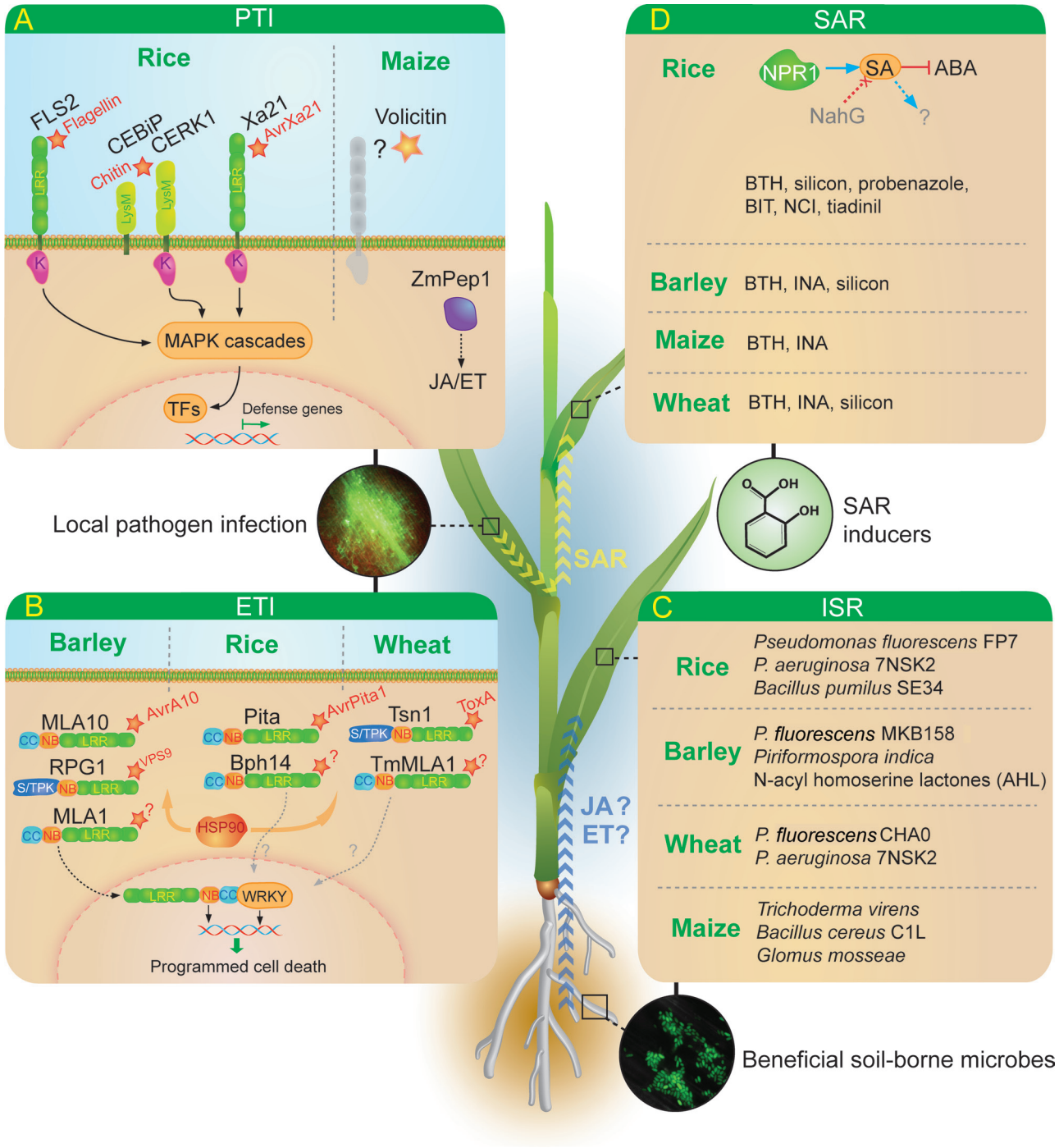

Fig. 1. Snapshot of IR mechanisms in monocots. (A) Molecular mechanisms of pattern-triggered immunity (PTI). The bacterial MAMP (microbe-associated molecular patterns) flagellin is recognized by FSL2, a PRR consisting of an extracellular LRR and cytoplasmic kinase (K) domain. The MAMP chitin is sensed by the LysM PRRs CEBiP and CERK1. MAMP-signalling activates MAPK cascades, which regulate transcription factors (TFs) driving the expression of defence genes. HAMPs (herbivore-associated molecular patterns) and damage-associated molecular patterns (DAMPs) are also triggering PTI. In maize, the HAMP volicitin is recognized by an unknown receptor, and the DAMP ZmPep1 functions as endogenous signal regulating jasmonic acid (JA)- and ethylene (ET)-dependent pathways during pathogen attack. (B) Effector-triggered immunity (ETI) mediated by NBS-LRR (nucleotide-binding leucine rich repeat) proteins. Pathogens employ effectors (represented by stars) to suppress PTI. Such effectors are contained by NBS-LRR proteins. Monocot NBS-LRR proteins usually have coiled-coil (CC) or serine/threonine protein kinase (S/TPK) domains and are localized in both the cytoplasm and nucleus. NBS-LRR proteins are folded into an active form by the heat shock protein 90 (HSP90). They interact directly with effectors and also regulate WRKY transcription factors. (C) Induced systemic resistance (ISR) following root infection by beneficial soil-borne microbes: examples of organisms triggering ISR in monocots. (D) Systemic acquired resistance (SAR). Mobile signals travel from attacked tissues to distant organs where systemic resistance responses are induced. In rice, the

rich repeat) proteins, which are encoded by plant resistance (*R*) genes, recognize pathogen-derived avirulence (Avr) proteins. These effector proteins help pathogens to overcome PAMP- or pattern-triggered immunity (PTI; Jones and Dangl, 2006). The recognition and attenuation of Avr proteins by plant R-proteins results in effector-triggered immunity (ETI), which is usually manifest in a hypersensitive response (HR; Greenberg and Yao, 2004).

PTI and ETI alleviate pathogen and pest attacks by inducing downstream responses that can result in a local and systemic induced resistance. Locally, these inducible defences consist of cell wall reinforcements through callose apposition and lignification, the production of secondary antimicrobial compounds, and the accumulation of pathogenesis-related (PR) proteins. Moreover, the attacked tissue is able to generate long-distance mobile alarm signals that are inducing systemic resistance in non-colonized organs (Shah, 2009). The systemic expression of defence in distal tissues can be observed upon infection with pathogens and is referred to as systemic acquired resistance (SAR). Resistance expressed following root colonization by non-pathogenic soil microbes is known as induced systemic resistance (ISR). SAR is predominantly effective against biotrophic pathogens (Vlot *et al*., 2008), whereas ISR is mainly counteracting necrotrophic pathogens and pests (Van Loon, 2007). Commonly, the inducible defence networks are regulated pivotally by phytohormones, which serve as specific chemical signals induced in response to particular attackers (Balmer and Mauch-Mani, 2012).

The vast majority of knowledge has been gathered from dicots such as cucumber, tobacco, and *Arabidopsis*. The knowledge about monocots remains elusive (Kogel and Langen, 2005). Monocots are a large group of about 59 300 species, amongst them the largest family is represented by orchids (Orchidaceae), followed by Poaceae, which include economically important plants such as rice, wheat, maize, sugarcane, and bamboo. Originating from a common angiosperm ancestor and going through an intimate co-evolution with plant pathogens, monocots and dicots are assumed to share most of the immune pathways. Here, we present the current knowledge of local and systemic IR mechanisms in monocots.

Pattern-triggered immunity (PTI): a stealth mission for pathogens?

Pathogens cannot sneak in: upon contact with invaders, plant cells use the first branch of their innate immune system by perceiving conserved microbial structures and peptides with the help of plasma membrane-localized PRRs (Fig. 1; Zipfel, 2009; Tsuda and Katagiri, 2010). In *Arabidopsis*, the best case study of this immune reaction is represented by the receptor-like kinase flagellin insensitive 2 (FLS2), which recognizes amino acids derived from bacterial flagellin. FLS2 interacts with BAK1, the brassinosteroid receptor BRI1-associated receptor kinase 1, to activate downstream defence responses (Chinchilla *et al*., 2007). Amongst monocots, various PRRs have been identified over the past few years (Table 1), notably in the model monocot rice (*Oryza sativa*; Chen and Ronald, 2011). FSL2 homologues are found in all higher plants, and the rice homologue OsFLS2 has been demonstrated to act as a functional flagellin receptor (Takai *et al*., 2008). Moreover, a variety of different MAMPs have been shown to be active in rice, including bacterial lipopolysaccharides (LPS; Desaki *et al*., 2006) and chitin (Kishimoto *et al*., 2010). In rice, chitin is perceived by the plasma membrane glycoprotein CEBiP, which forms a dimer with the chitin elicitor receptor kinase 1 (CERK1, also known as Lys-M-RLK1; Shimizu *et al*., 2010). As for *Arabidopsis*, chitin reception in rice then triggers the generation of reactive oxygen species (ROS) and the expression of *PR* genes. The best-studied example of PTI in monocots is the *Xa21*-mediated disease resistance in rice. *Xa21* encodes a receptor exhibiting an extracellular LRR domain, as well as an intracellular non-RD (non-arginine–aspartate) domain. XA21 perceives the 194-amino acid bacterial protein Ax21, which is conserved in all known *Xanthomonas* strains (Lee *et al*., 2009). As for OsFLS2, XA21 induces downstream defence mechanisms by activating MAPK cascades, thereby actuating transcription factors, triggering the expression of *PR* genes and the development of HR (Tena *et al*., 2011). *Xa21* homologues have been found in *Brachypodium*, sorghum, and maize (Tan *et al*., 2012). Several other non-RD receptor kinases have been identified in monocots. In rice, the B-lectin receptor kinase Pi-d2 confers resistance against *Magnaporthe grisea* (Chen *et al*., 2006).

Table 1. Selected monocot sensors recognizing conserved molecular patterns

Plant species	Protein name	Molecular pattern	Pathogen	Reference
Rice	CEBiP	Chitin	*Magnaporthe grisea*	Shimizu *et al*., 2010
	OsFLS2	Flagellin	*Pseudomonas avenae* *Acidovorax avenae*	Takai *et al*., 2008
	Pi-d2	Unknown	*M. grisea*	Chen *et al*., 2006
	XA21	Sulphated Ax21	*Xanthomonas* spp.	Lee *et al*., 2009
Barley	HvCEBiP	Chitin	*M. oryzae*	Tanaka *et al*., 2010
Wheat	WKS1 (Yr36)	Unknown	*Puccinia striiformis*	Fu *et al*., 2009
Maize	Unknown	ZmPep1	(Endogenous elicitor)	Huffaker *et al*., 2011

SAR key player NPR1 down-regulates genes. SA suppresses the abscisic acid (ABA) pathway. Expressing the SA-degrading enzyme NahG in rice reduces pathogen resistance. SAR can also be triggered in monocots by the application of SAR inducers such as BTH (*S*-methyl benzo-1,2,3-thiadiazole-7-carbothioate), INA (2,6-dichloroisonicotinic acid), BIT (1,2-benzisothiazole-1,1-dioxide) or NCI (*N*-cyanomethyl-2-chloroisonicotinamide). Image of the rhizobacteria *P. fluorescens* CHA0: courtesy of P Kupferschmied and C Keel, University of Lausanne.

In addition to PAMPs and MAMPs, so-called damage-associated molecular patterns (DAMPs) are also recognized during pathogen attack. Known DAMPs are polysaccharides released from plant cell walls, or endogenous peptides such as the 23-amino acid peptide AtPep1 in *Arabidopsis*. Recently, the maize ZmPep1 peptide has been identified as an orthologue of AtPep1 (Huffaker *et al*., 2011), suggesting a similar role of DAMPs in monocots and dicots. In conclusion, PTI mechanisms are highly conserved in both monocots and dicots, although some PRRs such as EFR, the *Arabidopsis* receptor of bacterial EF-TU (elongation factor unstable), are not found in monocots (Boller and He, 2009). Nevertheless, the fact that rice encodes a higher variety of non-RD domain receptor kinases than *Arabidopsis* (Dardick and Ronald, 2006) indicates that, although PTI signalling is conserved in all angiosperms, both monocots and dicots underwent particular evolutionary adaptations.

Effector-triggered immunity (ETI): Special Forces striking back

Once detected by plant cells and facing PTI-triggered defences, successful pathogens are able to perturb the first inducible defence lines (Jones and Dangl, 2006). Bacteria, fungi, and oomycetes are delivering effectors behind enemy lines to suppress PTI. There, these effectors manipulate host cellular mechanisms to favour subsequent invasion steps. Examples of such effectors are AvrPtoB and AvrPto, effectors from *Pseudomonas syringae* strains targeting the kinase domains of EFR, FLS2, and BAK1 (Boller and He, 2009). In contrast to bacterial effectors, eukaryotic pathogen effectors are less well studied. The oomycete *Hyaloperonospora arabidopsidis* produces ATR1 and ATR13 effectors (Sohn *et al*., 2007), and the fungus *Blumeria graminis* f.sp. *hordei* delivers AVRK and AVRA10 proteins into barley cells (Ellis *et al*., 2007). Pathogen effectors are able to render a plant susceptible, thus being a serious threat for plant survival. However, plants are promptly counterstriking by sending in recon troops that recognize effectors, thus triggering ETI (Fig. 1; Table 2). These recon troops are mostly NB-LRR proteins encoded by resistance (*R*) genes (Elmore *et al*., 2011). NB-LRR proteins usually exhibit an N-terminal TIR (Toll/Interleukin-1 Receptor) domain or coiled-coil (CC) motif. Activation of NB-LRRs induces local and systemic defence signalling involving hormonal networks, ROS-generation, and gene expression adaptations by WRKY and TGA transcription factors (Jones and Dangl, 2006). According to the guard hypothesis, some *R* genes can directly recognize pathogen molecules (effectors), while other *R* genes indirectly recognize metabolic perturbations due to the presence of the pathogen (Jones and Dangl 2006). In cereals, the prevailing situation seems to consist of direct surveillance as, in most cases, a direct interaction between the resistance gene and the corresponding effector is the rule (Table 2).

NB-LRR encoding genes represent one of the largest and widely conserved gene families in plants, with over one-hundred family members for the majority of sequenced plants (Jones and Dangl, 2006), including monocots and dicots. Despite the extensive knowledge of NB-LRRs in monocots, their elucidation has been mainly limited to rice and, more recently, to wheat and sorghum. Compared with dicots, monocot genomes encode higher numbers of CC-NB-LRRs (Martin *et al*., 2011). Intriguingly, genes coding for TIR-NB-LRRs homologues are rare in monocots (Kim *et al*., 2012). The majority of described rice NB-LRRs is promoting resistance *to M. grisea*, such as Pita, Pib, Piz-t, Pikm, and Pit (reviewed in Chen and Ronald, 2011). Bph14 confers resistance to the brown planthopper (Du *et al*., 2009), and XA1 mediates resistance against *Xanthomonas oryzae* (Yoshimura *et al*., 1998). Despite the large number of rice NB-LRRs, most of their target effectors are unknown. Only four *M. grisea* effectors are described, AvrPiz-t (Shang *et al*., 2009), AvrPita (Jia *et al*., 2000), AvrPia and AvrPik/km/kp (Qu *et al*., 2006). AvrPita is recognized by the rice NBS-LRR protein Pita; direct binding of Pita to AvrPita induces cell death that retards the spread of *M. grisea* on rice (Jia *et al*., 2000). Other *R*-genes conferring resistance to *Xanthomonas oryzae* pv. *oryzae* in rice do not exhibit NBS or LRR domains, such as xa13 and Os11N3 (Antony *et al*., 2010). Xa13, a recessive allele belonging to the NODULIN3 (N3) gene family, triggers immunity by recognizing the *Xanthomonas* effectors AvrXA7. In turn, the type III effector AvrXa7 drives the expression of the rice susceptibility gene OS-8N3, which defeats Xa13 and induces effector-triggered susceptibility (ETS; Antony *et al*., 2010). The extensive synteny between the genomes of several major cereal species and the high colinearity between large portions of these genomes facilitates synteny-based positional cloning. The availability of detailed rice (International Rice Genome Sequencing Project,

Table 2. Selected monocot proteins recognizing pathogen effectors

Plant Species	Protein name	Effector	Pathogen	Reference
Rice	Bph14	Unknown	Brown planthopper	Du *et al*., 2009
	Os11N3	AvrXA7	*Xanthomonas* spp.	Antony *et al*., 2010
	Pita	AvrPita1	*Magnaporthe grisea*	Jia *et al*., 2000
	Piz-t	AvrPiz-t	*M. grisea*	Li *et al*., 2009
	XA27	AvrXA27	*Xanthomonas* spp.	Gu *et al*. 2005
Barley	RDG2A	Unknown	*Pyrenophora graminea*	Bulgarelli *et al*., 2010
	RPG1	Urediniospore effectors (protein with a fibronectin type III susceptibility domain; vacuolar protein sorting associated protein 9)	*Puccinia graminis*	Brueggeman *et al*., 2002
Wheat	TmMla1	Unknown	*Blumeris graminis* f.sp. *hordei*	Jordan *et al*., 2011
	Tsn1	ToxA	*Stagonospora nodorum*	Faris et al. 2010
Sorghum	Cs1A & Cs2A	Unknown	*Colletotrichum sublineolum*	Biruma *et al*., 2012

2005) and, recently, barley genomic data as well (Mayer *et al*., 2011) will allow the identification of genes playing a crucial role in IR in major cereal species and, hopefully, lay the basis for genomics-based breeding strategies for defence in these plants.

In other monocot species, NB-LRRs are less explored. Nonetheless, in the genomes of *Brachypodium distachyon*, *Sorghum bicolor*, and *Zea mays*, conserved NB-LRR-encoding genes were identified (Kim *et al*., 2012). In sorghum, a CC-NB-LRR encoding gene cluster that confers resistance to *Setosphaeria turcica* has recently been discovered (Martin *et al*., 2011). The corresponding resistance gene has been found to be conserved in maize, rice, foxtail millet, and in *Brachypodium distachyon*. In addition, the NB-LRR encoding *R* genes *Cs1A* and *Cs2A* were shown to mediate the resistance of sorghum against *Colletotrichum sublineolum* (Biruma *et al*., 2012). In wheat, the recently identified CC-NB-LRR protein TmMla1 functions in resistance against *Blumeria graminis* f.sp. *hordei* (Jordan *et al*., 2011). Wild wheat (*Triticum turgidum* L. ssp. *dicoccoides*) possesses the *Yr36* gene, which encodes a kinase and putative START lipid-binding domain and confers resistance to *Puccinia striiformis* (Fu *et al*., 2009). In barley, the CC-NB-LRR-type gene *Rdg2a* has been discovered to confer resistance to *Pyrenophora graminea* (Bulgarelli *et al*., 2010). Another barley gene, *Rpg1,* regulates resistance against *Puccinia graminis* f.sp. *tritici* (Brueggeman *et al*., 2002). RPG1 interacts with two effector proteins from urediniospores, one of them is characterized as a vacuolar protein sorting-associated protein (VPS9). This leads to rapid phosphorylation followed by the degradation of RPG1. The resulting HR then confers resistance to the rust fungus (Nirmala *et al*., 2011). Thus far, ETI-mechanisms in monocots and dicots are highly conserved.

Systemic acquired resistance: a defence in depth in monocots?

Upon locally induced defence, plants employ an intricate defence mechanism that activates resistance responses in not-yet-attacked tissues. In the case of a local challenge by leaf pathogens, mobile alarm signals are sent to distal leaves to induce a systemic resistance against a broad range of subsequent attackers. This mechanism is known as SAR (Shah, 2009). SAR has been extensively studied in the two dicot models tobacco and *Arabidopsis*, leading to the identification of specific molecular components and of a set of mobile defence signals (Vlot *et al*., 2008). Salicylic acid (SA) has been found to be the main chemical regulator of SAR. SA exerts its canonical action on NPR1 (non-expressor of *PR* genes, also known as NIM1). Originally, *npr1* was discovered as recessive mutation conferring a SAR^- phenotype (Cao *et al*., 1997). Now it is known that NPR1 is a transcription factor activator that is present in the cytosol in an oligomeric form. SA accumulation leads to its constitutive monomerization. As a monomer, NPR1 enters the nucleus to interact with transcription factors (Mou *et al*., 2003), triggering extensive changes in the defence gene transcriptome (Maleck *et al*., 2001). Novel evidence shows that two paralogues of NPR1, NPR3 and NPR4, are SA receptors with different binding affinities to SA. They regulate NPR1 stability and activity depending on the SA level in the cell. In unchallenged plants, NPR4 mediates the degradation of most of the NPR1. When a pathogen triggers ETI, a gradient of SA builds up from the local to the systemic part and the elevated SA levels trigger an HR. Further expansion of cell death is then restricted through NPR3/NPR1 interactions in the cells adjacent to the HR (Fu *et al*., 2012). Prior activation of defence genes in distal tissues renders them more resistant against future attacks. A common marker of SAR in dicots is the up-regulation of PR genes such as *PR1* and *PR5*.

For an effective SAR reaction, mobile alarm signal(s) have to be sent from locally infested leaves to distant tissues. In *Arabidopsis*, several mobile SAR signals have been discovered, such as glycerol-3-phospate (G3P; Chanda *et al*., 2011), azelaic acid (Jung *et al*., 2009), and the volatile methyl salicylate (MeSA) (Park *et al*., 2007). Recent findings also propose dehydroabietinal (DA), a diterpenoid aldehyde, as the SAR-signal in *Arabidopsis* (Chaturvedi *et al*., 2012). The known SAR signals are generally controversial as they are highly conditional, depending on the experimental systems. This abundance of different signals could be considered as a safety mechanisms to prevent accidental activation of the cost-intensive immune response. Through cross-interaction between signals or even requirement of parallel activation, an appropriate induction of IR for a given specific situation might be achieved (Dempsey and Klessig, 2012). SAR can also be induced by the application of various synthetic chemical compounds such as INA (2,6-dichloroisonicotinic acid; Métraux *et al*., 1990), BTH (*S*-methyl benzo-1, 2,3-thiadiazole-7-carbothioate; Görlach *et al*., 1996), probenazole (3-allyloxy-1,2-benziso-thiazole-1,1-dioxide; Nakashita *et al*., 2002*a*), BIT (1,2-benzisothiazole-1,1-dioxide; Yoshioka *et al*., 2001) NCI (*N*-cyanomethyl-2-chloroisonicotinamide; Nakashita *et al*. 2002*b*) or tiadinil (3'-chloro-4,4'-dimethyl-1,2,3 -thiadiazole-5-carboxanilide; Yasuda *et al*., 2004).

Compared with dicots, the knowledge of SAR in monocots is scarce. NPR1, the master regulator of SAR in dicots, has been confirmed for all monocots where genomic data is available (Kogel and Langen, 2005). In rice, over-expression of both *AtNPR1* (Chern *et al*., 2001) and the endogenous homologue *OsNH1* (Chern *et al*., 2005) resulted in an enhanced resistance to *Xanthomonas oryzae* pv. *oryzae*. Transcriptomic analysis of *OsNPR1* knockdown and over-expressing rice lines showed that OsNPR1 is dominantly involved in the down-regulation of genes, and in the SA-mediated suppression of abscisic acid (ABA)-responsive genes (Sugano *et al*., 2010). Chemical SAR inducers were also found to be active in monocots, such as BTH and INA in maize (Morris *et al*., 1998), BTH in wheat (Görlach *et al*., 1996), and INA in barley (Kogel *et al*., 1994). Similarly to *Arabidopsis*, BTH-treatment of maize triggers the expression of PR proteins such as PR1 and PR5 (Morris *et al*., 1998). Monocot and dicot PR protein sequences were found to share extensive similarities. However, when performing an unrooted phylogenetic tree analysis using PR1 homologues from different species, dicot PR1 genes grouped together in a cluster distant from monocot sequences (Lu *et al*., 2011*a*). Thus, PR1 probably underwent the main diversifications after the monocot–dicot separation. Other resistance inducers in addition are described for monocots, such as the effect of probenazole in rice (Umemura *et al*., 2009). Probenazole strongly up-regulates *OsSGT1*, which encodes an UDP-glucose:SA glucosyltransferase. OsSGT1 is believed to

support rice defence mechanisms by converting free SA to conjugated SA-*O*-β-glucoside (SAG) which, in turn, can be converted back into SA when needed. SA-levels itself were not found to be altered upon probenazole-treatment, suggesting an exquisite role of SAG during SAR in rice (Umemura *et al*., 2009). In barley induced with INA, the situation presents itself differently: here, defence reactions against *Blumeria graminis* f.sp. *hordei* neither depend on, nor induce SA accumulation (Hückelhoven *et al*., 1999). In contrast to dicots, the role of SA during SAR in monocots has yet to be elucidated. Rice contains high endogenous levels of SA (Silverman *et al*., 1995), and pathogen infection does not up-regulate these levels. However, transgenic rice plants expressing the SA-degrading enzyme salicylate hydroxylase (NahG) exhibit a diminished resistance against *Magnaporthe grisea* (Yang *et al*., 2004), although PR gene expression profiles were found to be unaltered. The role of SA in other monocot models is less studied. Some reports on wheat and barley showed a 'local acquired resistance" (LAR) where a first fungal inoculation on a leaf makes a second attack on the same leaf less efficient (Thordal-Christensen and Smedegaard-Petersen, 1988; Jørgensen *et al*., 1998). In both studies, SA levels were found to be unaffected. Nevertheless, a recent study of *P. syringae* pv. *tomato*-induced LAR in barley demonstrated similarities between gene expression profiles during LAR in barley and SAR in *Arabidopsis* (Colebrook *et al*., 2012).

Although general chemical and molecular SAR players such as NPR1, *PR* genes and transcription factors are conserved in monocots and dicots, only a few reports describe biological SAR phenomena in monocots. Infection of rice by *P. syringae* pv. *syringae* leads to a systemic resistance *against M. grisea* (Smith and Métraux, 1991). In wheat, SAR against stem and leaf rust has been noted (Barna *et al*., 1998). Nevertheless, these SAR phenomena are highly conditional, corroborated by the lack of reproducibility by other laboratories (Kogel and Langen, 2005). However, the intricate signalling process during SAR is highly conditional, depending on multiple factors such as type of attackers, age of plant, and growth conditions. In *Arabidopsis*, MeSA is not required for SAR when plants are exposed for more than 3.5 h to light after a primary pathogen infection (Liu *et al*., 2011). Strong light conditions trigger SAR in *Arabidopsis* upon *P. syringae* pv. *maculicola* infection without the accumulation of either SA or PR1 in systemic leaves (Zeier *et al*., 2004). Hence, particular molecular or chemical SAR factors have to be specifically determined for a given pathosystem, which might, in turn, explain the discrepant mode of action of certain SAR regulators between dicots and monocots.

Induced systemic resistance: support from underground alliances

Colonization of plant roots by some soil microbes, such as plant growth-promoting rhizobacteria (PGPR) or endophytic fungi (PGPF), can directly stimulate plant growth by improving nutrient uptake or photosynthesis (Spaepen *et al*., 2009; Trillas and Segarra, 2009) or indirectly by suppressing soil-borne pathogens through the production of antibiotic compounds (De Vleesschauwer and Höfte, 2009). Moreover, these beneficial microorganisms can also indirectly reduce plant disease through an induction of a systemic resistance, named ISR. ISR confers a resistance against a wide spectrum of attackers, mostly necrotrophic pathogens and pests (Van Wees *et al*., 2008; Pineda *et al*., 2010). Similarly, mycorrhizae have been reported to induce plant resistance in a way resembling that of ISR (reviewed by Pozo and Azcón-Aguilar, 2007). Various beneficial microorganisms are known to induce ISR in monocots. In cereals, endophytic fungi, PGPR or mycorrhizae are reported to induce resistance against pathogens and insect herbivores (Table 3). The potential resistance induced by PGPR in monocots depends on the host-PGPR combination and on the type of attacker. *P. aeruginosa* 7NSK2 and *Serratia plymuthica* IC1270 induce resistance against *Magnaporthe oryzae* in rice, but they enhance disease severity caused by *Rhizoctonia solani* (De Vleesschauwer *et al*., 2006, 2009). However, some pseudomonads induce resistance of rice against *R. solani* (Table 3). Induction of resistance by a specific strain of PGPR is not restricted to only one plant species: for example, *P. aeruginosa* 7NSK2 triggers ISR in rice (De Vleesschauwer *et al*., 2006) and wheat (Muyanga *et al*., 2005). Application of a PGPR mixture enhances the efficacy of resistance induction compared with the use of individual strains in both dicots (De Boer *et al*., 2003) and monocots (Lucas *et al*., 2009).

Diverse microbial molecules have been identified as ISR elicitors in monocots. Exopolysaccharides produced by *Pantoea agglomerans* induce defence responses in wheat cells by triggering an increased accumulation of hydrogen peroxide and an augmented peroxidase activity (Ortmann and Moerschbacher, 2006). Siderophores and antibiotics produced by *Pseudomonas* strains, such as pseudobactins and pyocyanin, are important defence elicitors in rice against *M. oryzae* (De Vleesschauwer *et al*., 2008; De Vleesschauwer and Höfte, 2009). In contrast to tomato and bean, pyocyanin was shown to be the only component compulsory for triggering ISR in rice. Certain fungal endophytes have also been shown to trigger IR. A beneficial *Penicillium* primes *Arabidopsis* for defence against *P. syringae* (Hossain *et al*., 2008) and *Glomus mossae* protects tomatoes from infection by *Phytophthora* (Pozo *et al*., 2002). *Trichoderma virens*, an endophytic fungus that triggers ISR in maize, has been shown to facilitate resistance via the release of a proteinaceous elicitor (Djonovic *et al*., 2007). *Piriformospora indica* induces IR in both dicots and monocots but is probably best-known for this effect on barley. Here, it was shown to induce resistance without having to rely on the classical defence pathways involving SA, JA or ET (Waller *et al*., 2005). A barley leaf transcriptome and metabolite analysis revealed that *P. indica*-induced plants over-expressed a small set of defence-related genes including transcripts coding for PR and heat-shock proteins (Molitor *et al*., 2011). In creeping bentgrass (*Agrostis stolonifera)* which is closely related to cereals, treatment with (2R, 3R)-butanediol, a bacterial-derived volatile, induces resistance against *Microdochium nivale* (Cortes-Barco *et al*., 2010). Rhizobacteria can also produce hormones that manipulate phytohormone pathways. SA produced by *P. aeruginosa* strains triggers peroxidase accumulation in rice leading to an increase in resistance to *R. solani* (Saikia *et al*., 2006).

Some N-acyl homoserine lactones (AHL) controlling quorum sensing in bacteria (Miller and Bassler, 2001) also have the capacity to induce resistance. AHLs from *Serratia liquefaciens* and *P. putida* induce resistance against *Alternaria* in tomato (Schuhegger *et al*., 2006). Intriguingly, *P. indica* is

Table 3. Examples of established cereal ISR pathosystems

Plant species	Beneficial microorganisms	Plant attackers	References
Rice	*Pseudomonas fluorescens* PF1	*Cnaphalocrocis medinalis*	Radja Commarea *et al*., 2002
	P. fluorescens FP7		
	Pseudomonas fluorescens PF1	*Rhizoctonia solani*	Radjacommarea *et al*., 2004
	P. fluorescens Pf1, TDK1, PY15	*Cnaphalocrocis medinalis*	Saravanakumar *et al*., 2007
	P. fluorescens WCS374r	*Magnaporthe oryzae*	De Vleesschauwer *et al*., 2008
	P. fluorescens Aur6 *Chryseobacterium balustinum* Aur9	*Magnaporthe oryzae*	Lucas *et al*., 2009
	P. aeruginosa	*Rhizoctonia solani*	Saikia *et al*., 2006
	P. aeruginosa 7NSK2	*Magnaporthe oryzae*	De Vleesschauwer *et al*., 2006
		Rhizoctonia solani	
	Bacillus pumilus SE34 *Bacillus subtilis* GB03	*Xanthomonas oryzae* pv. *oryzae*	Chithrashree *et al*., 2011
	Serratia plymuthica IC1270	*Magnaporthe oryzae*	De Vleesschauwer *et al*., 2009
		Cochliobolus myiabeanus	
		Rhizoctonia solani	
Maize	*Trichoderma virens* T22	*Colletotrichum graminicola*	Djonovic *et al*., 2007
	Bacillus cereus C1L	*Cochliobolus heterostrophus*	Huang *et al*., 2010
	Glomus mosseae	*Rhizoctonia solani*	Song *et al*., 2011
Wheat	*P. fluorescens* CHA0	*Fusarium graminearum*	Henkes *et al*., 2011
	P. fluorescens CHA0	*Gaeumannomyces graminis* var. *tritici*	Sari *et al*., 2008
	P. fluorescens MKB158	*Fusarium graminearum*	Petti *et al*., 2008
	P. aeruginosa 7NSK2	*Blumeria graminis*	Muyanga *et al*., 2005
		Cochliobolus sativus	
	Chaetomium globosum	*Pyrenophora tritici-repentis*	Istifadah and McGee, 2006
	Fungal endophytes	*Puccinia recondite* f.sp. *tritici*	Dingle and McGee, 2003
Barley	*Piriformospora indica*	*Blumeria graminis* f.sp. *hordei*	Molitor *et al*., 2011
	P. fluorescens MKB158	*Fusarium graminearum*	Petti *et al*., 2010
	Fusarium oxysporum f.sp. *radicis-lycopersici*	*Blumeria graminis* f.sp. *hordei*	Nelson, 2005
Pearl millet	*B. pumilus* INR7	*Sclerospora graminicola*	Raj *et al*., 2003
	B. pumilus SE34		
	B. subtilis GB03		
	P. fluorescens UOM SAR 14	*Sclerospora graminicola*	Raj *et al*., 2004
Sorghum	*B. cereus* KBS2-6	*Pythium utlimum*	Itris *et al*., 2008
	B. cereus KFP9-A		
	Serratia marcescens KBS9-R		

closely associated with an endobacterium, *Rhizobium radiobacter* (Sharma *et al*., 2008), that produces a series of AHLs. Application of these AHLs to barley induces resistance against powdery mildew (Sharma *et al*., 2008). This raises the question as to whether the observed IR capacity of *P. indica* might not actually be due to the presence of the endophytic bacteria.

The efficacy of ISR in monocots against necrotrophic pathogens has been demonstrated repeatedly but only in a few cases, the involved defence signalling pathway has been investigated. ISR induced by *P. fluorescens* WCS374r against *M. oryzae* in rice depends on a jasmonic acid (JA)/ethylene (ET)-modulated signal but is independent from SA-signalling (De Vleesschauwer *et al*., 2008). Involvement of JA-signalling in ISR was also shown in maize (Djonovic *et al*., 2007; Song *et al*., 2011) and barley (Petti *et al*., 2010). Interestingly, ISR triggered by *T. virens* in maize also seems to be associated with the priming of genes involved in the production of volatile compounds called green leaf volatiles (GLV) (Djonovic *et al*., 2007). Several defence-related genes involved in SA- and JA-dependent pathways are strongly induced when mycorrhized maize plants are challenged with *R. solani* (Song *et al*., 2011). ISR in monocots is mostly linked to JA-dependent defences. However, some PGPR or PGPF induced an SA-dependent pathway effective against biotrophic pathogens (Muyanga *et al*., 2005; Molitor *et al*., 2011).

Overall, recent studies on ISR triggered by PGPR, PGPF or mycorrhiza in monocots and more specifically in cereals tend to point to common mechanisms with dicotyledonous plants.

Induced resistance against insect herbivores: protection against air-borne assaults

Plants are confronted with a wide variety of insect herbivore attacks. To counteract these attacks promptly and specifically by inducing defence mechanisms, plants recognize molecules originating either from wounding damage or from compounds derived from the herbivore itself, such as oral secretions (OS) and oviposition fluids. These elicitors, called herbivore associated molecular patterns (HAMPs), have been found in several monocot pathosystems. Volicitin, a hydroxyl fatty acid-amino acid conjugate found in *Spodoptera exigua* OS, induces volatile

emission in maize (Alborn *et al*., 1997) and caeliferins from *Schistocerca americana* OS trigger IR in maize (Alborn *et al*., 2007). Plant perception of HAMPs is widely elusive, but similarities to MAMP-recognition have been proposed (Bonaventure *et al*., 2011). In maize, volicitin is perceived by a plasma membrane protein (Truitt *et al*., 2004), which is so far the only known HAMP-receptor in monocots.

Upon perception of an herbivore, IR mechanisms are mediated by different defence-related hormones. Plant-induced defences against phloem-feeding herbivores seem to share a common plant reaction to biotrophic pathogens by activating SA-dependent pathways associated with the production of PR proteins (Alagar *et al*., 2010) and callose deposition at the feeding site (Hao *et al*., 2008). In rice, defence induced by an attack of the phloem-feeding brown planthopper is mediated by a SA-related signalling and is associated with an accumulation of PR proteins and an HR (Zhou *et al*., 2009). In resistant wheat cultivars, but not in susceptible ones, infestation by gall insects induces changes in SA levels (Tooker and De Moraes, 2011). By contrast, plants induce JA and ET-dependent pathways against chewing herbivores. In maize, JA and ET are important in plant defence against *S. frugiperda* (Shivaji *et al*., 2010; Harfouche *et al*., 2006). JA was also shown to have an important role in IR of wheat against pests (El-Wakeil *et al*., 2010). In rice, the JA-dependent pathway induces resistance against insect herbivores and suppression of JA activity results in an improved larval performance of the striped stem borer and leaf folder (Zhou *et al*., 2009). Ethylene is another key player in fending off herbivores. ET emission induced by elicitors of *S. frugiperda* OS influences the expression of direct defences such as defence proteins and secondary metabolites (Harfouche *et al*., 2006). In rice, the ethylene responsive factor ERF3 mediates between SA, JA, and ET pathways and thus orchestrates the response to chewing or phloem-feeding insects (Lu *et al*., 2011*b*).

After herbivore attack, plants can induce defences that will directly act against insect herbivore. The maize insect resistance 1-cysteine protease (Mir1-CP) content increases in roots and leaves in response to larvae feeding on leaves, conferring a systemic induction of plant defence against herbivores (Lopez *et al*., 2007). Trypsin proteinase inhibitors are important defence compounds against herbivores such as the striped stem borer and leaf folder in rice (Wang *et al*., 2011; Zhou *et al*., 2011). Secondary metabolites, such as the hydroxamic acids in cereals, can also have a direct negative effect on insect herbivores (Chen, 2008). Direct local defence can enhance direct plant defence systemically. Infestation of rice plants with *S. frugiperda*, for example, increases resistance against a subsequent attack by the rice water weevil (Hamm *et al*., 2010). Similarly, root infestation of maize by *Diabrotica virgifera virgifera* induces resistance in the leaves against *S. littoralis* and the necrotrophic pathogen *Setosphaeria turcica* (Erb *et al*., 2009). This illustrates that an induction of below-ground defences can induce above-ground resistance in maize.

Many plants respond to insect herbivory or wounding by emitting blends of volatile organic compounds (VOCs). VOCs release is an important cue for systemic defence signalling within an attacked plant as well as for plant–plant communication. Exposure of a maize plant to VOCs from infested plants primes the defence response against the generalist *S. littoralis* (Ton *et al*., 2007). Green leaf volatiles (GLVs), specific VOCs emitted by plants upon wounding damages, can also activate defence mechanisms in neighbouring intact plants (Ruther and Furstenau, 2005).

Induced resistance (IR) in non-cereal monocots: the last bastion

Because of their economic importance, most of the research on IR in monocots has been conducted on cereals. Nevertheless, IR such as SAR and ISR can also be found in non-cereal monocots. In *Lilium formonasum*, a previous infection with *Botrytis elliptica* suppresses a secondary infection with the same pathogen in systemic tissues (Lu *et al*., 2007). Classical synthetic chemical SAR inducers have been reported in diverse non-cereal monocot systems. *L. formosanum* can be protected against *B. elliptica* by probenazole. Here, resistance is associated with a stomatal closure and increased callose deposition (Lu *et al*., 2007). SA-treatment primes callose accumulation in onion, which confers enhanced resistance to downy mildew (Polyakovskiy and Dmitriev, 2011). BTH enhances plant defence in banana against *Colletotrichum musae* via a higher chitinase defence gene expression (Ma *et al*., 2009). Curcuma (Radhakrishnan *et al*., 2011) and sugarcane (Ramesh Sundar *et al*., 2006) were also protected by BTH treatment against *Pythium aphanidermatum* and *Colletotrichum falcatum*, respectively. Functional ISR has also been reported in non-cereal monocots, here mostly against necrotrophic fungal pathogens. For example, *Bacillus cereus* C1L was efficient in eliciting ISR in *Lilium formonasum* against *Botrytis elliptica* (Liu *et al*., 2008). In banana plants, a combination of the rhizobacteria *Pseudomonas fluorescens* CHA0 and chitin induces systemic resistance against banana bunch top virus (Kavino *et al*., 2008). A mixture of several PGPRs seems to have an increased positive effect compared with a single strain use on resistance in gladiolus (Shanmugam *et al*., 2011) and in banana (Sangeetha *et al*., 2010). ISR induced by a hypoagressive isolate of *Fusarium oxysporum* in date palm against *Fusarium oxysporum* f.sp. *albedinis* is characterized by a primed reaction of the plant with a faster induction of peroxidase activity and a higher amount of phenolics (El Hassni *et al*., 2004).

Conclusion

Historically, the majority of research on IR has been performed in dicot model plants. Recent advances in monocot genomics, however, are helping to identify the key components of IR signalling. Further improvements in monocot biotechnology such as plant transformation methods will provide a more profound insight into IR mechanisms. Moreover, a variety of cereal and non-cereal IR model systems are now well established, making IR in monocots a research field ready to move forward. Novel insights into the functioning of IR in monocots are expected to have a positive impact on sustainability in modern agriculture.

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