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Review

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Induced resistance to pests and pathogens in trees

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Summary

Tree resistance can be enhanced by a variety of biotic and abiotic inducers, including nonpathogenic and pathogenic microbes, and herbivores, resulting in enhanced protection against further biotic injury. Induced resistance (IR) could be a valuable tool in sustainable pest management. IR has been actively studied in herbaceous plant species, and, in recent years, in woody plant species, and is fast emerging as an intriguing, eco-friendly concept for enhancing tree resistance. However, before application of IR becomes possible, there is a need to increase our knowledge of the mechanisms of defence in forest trees. A richer understanding of these phenomena will play a critical role in developing sustainable integrated pest management strategies. This review summarizes our current knowledge of IR in forest trees, focusing on inducible defence mechanisms, systemic induction of resistance and phytohormone signalling networks. We conclude by discussing the potential advantages and limitations of applying IR-based management tools in forest systems.

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I. Introduction

In any plant–insect and plant–pathogen interaction there is a continuum of possible outcomes, ranging from extreme susceptibility to complete resistance. Plant resistance can be described on several mechanistic levels. These include basal resistance, parasite- and race-specific resistance (Jones & Dangl, 2006; Kiraly *et al.*, 2007), age-related (ontogenetic) resistance (Develey-Rivière & Galiana, 2007), organ-specific resistance (Blodgett *et al.*, 2007) and acquired or induced resistance (IR) (Agrawal *et al.*, 1999). In its broadest sense, IR is a form of resistance caused by activation of the host plant's own genetically programmed defence pathways, resulting in changes that diminish the effects of subsequent biotic attack (Agrawal *et al.*, 1999; Hammerschmidt, 2007). IR elicited by microorganisms in plants to other pathogenic microorganisms has been recognized for over 100 yr (Chester, 1933). By contrast, knowledge of plant resistance induced by insect herbivores has had a much shorter history of < 40 yr (Green & Ryan, 1972).

Most of the current knowledge on plant defence mechanisms, particularly as they relate to IR, has been obtained through studies on herbaceous annuals or short-lived perennials. These include the model plant species, *Arabidopsis thaliana*, *Cucumis sativus* (cucumber), *Lycopersicon* spp. (tomato), *Medicago truncatula*, *Nicotiana tabacum* (tobacco), *Oryza* spp. (rice), *Solanum* spp. (potato) and *Zea* spp. (maize). Many comprehensive overviews have been published on IR in herbaceous plants (Karban & Baldwin, 1997; Agrawal *et al.*, 1999; Gatehouse, 2002), but much less is known for trees, both angiosperms and gymnosperms. Tree and herbaceous species share common plant features but trees have certain unique features when compared with herbaceous plants. They are usually much larger, have much longer life spans (sometimes of millennia), characterized by life histories that have no equals among herbaceous model plants, and exhibit different architectural forms linked to secondary growth. Trees may be subject to different patterns of herbivore and pathogen pressure and require different modes of protection. In view of this, while we draw from the knowledge gained in the more studied herbaceous model species, findings from these models may not always apply to forest trees (Hammerschmidt, 2006).

Induced resistance has been well studied in many horticultural and agricultural systems and its application has proved effective (Vallad & Goodman, 2004; Walters, 2009). For example, the synthetic chemical primer/activator, acibenzolar-*S*-methyl (trade name Actigard or Bion, Syngenta Crop Protection, Basel, Switzerland), has been successfully used as a broad-spectrum crop protectant in the past decade (Leadbeater & Staub, 2007). IR does not involve the manipulation of genes, therefore the societal issues that are associated with the use of genetically modified organisms are not relevant to IR tools. IR functions as a multilayered, highly integrated defence system, and therefore IR can be durable and effective against a wide spectrum of pests and pathogens (Vallad & Goodman, 2004). These features are of particular importance in trees that are long-lived and confined to a particular pest environment, often for decades and even centuries.

Current methods of pest and disease management in trees vary greatly (Eyles *et al.*, 2008). They can range from the deployment of resistant material and pesticide application, through to the use of biological control and silvicultural management. In many cases, because of economic and environmental constraints, very little is done. While the development of tree protection methods based on IR mechanisms is still very much in its infancy, IR could provide alternative forms of protection in the future, either applied with other management tools or used alone. The use of IR has the potential to offer more eco-friendly options than current pesticides available for forest trees. In this review, we summarize findings from recent ecological and molecular studies on IR to both pests and pathogens in trees. In Section II, we describe the inducible defence mechanisms underlying IR, followed by a discussion on the systemic aspects of IR (Section III) and the phytohormone signalling networks that regulate IR (Section IV). In Section V, we highlight the large knowledge gaps that will need to be addressed in order to realize the potential offered by the application of IR in forest systems.

II. Induced defence mechanisms in trees

Insect herbivores and pathogens must overcome the diverse defence strategies that trees have evolved. This includes multiple constitutive and inducible defences that impede access to, deter or kill insects and inhibit or exclude pathogens physically and/or chemically (Pearce, 1996; Franceschi *et al.*, 2005). Constitutive defences, both below and above ground, are present at all times and represent the first lines of defence. When these barriers are breached, induced defences are triggered (Fig. 1). Trees presumably evolved induced defences because they incur lower resource allocation costs than constitutive resistance traits (Bonello *et al.*, 2006; Bolton *et al.*, 2009). For the purpose of this review, induced defence mechanisms, both direct and indirect, are divided up into five categories. In reality, defence mechanisms, regardless of the category into which they fall, are likely to function as a highly integrated and coordinated response that is modulated by biotic and abiotic factors (Bonello *et al.*, 2006); that is, a tree response may include all or several of the mechanisms in the following categories:

(1) Inducible chemical defences – toxic, antimicrobial, antinutritive and antidigestive activity via low-molecular-weight (LMW) compounds such as phenolic compounds, terpenoids and alkaloids

(2) Inducible protein-based defences – toxic, antimicrobial, antinutritive and antidigestive activity via proteins and

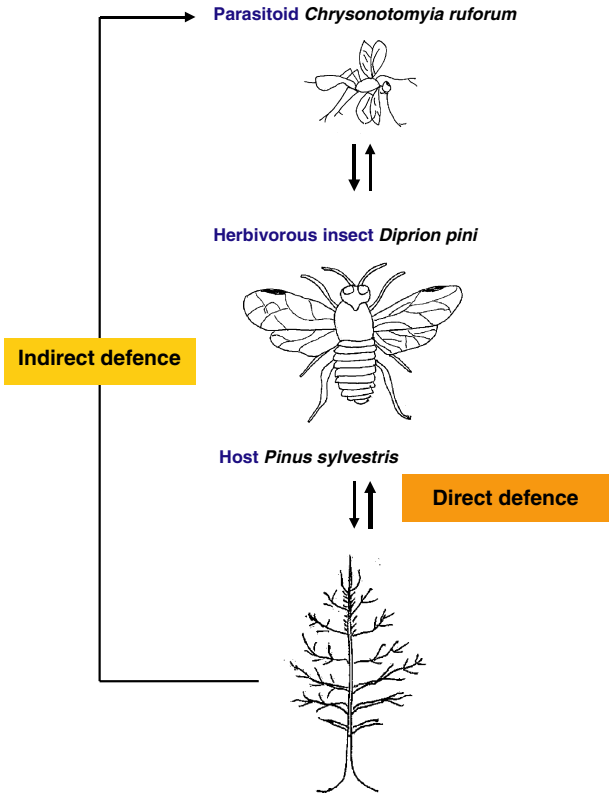

Fig. 1 Direct defence of a tree (in this case, *Pinus sylvestris*) has a direct adverse effect on the invading organism (*Diprion pini*). Indirect defences rely on the attraction of natural enemies (e.g. *Chrysonotomyia ruforum*) of *D. pini*. (Diagram based on findings reported by Hilker *et al.*, 2002.)

peptides, for example, oxidative and hydrolytic enzymes, and proteinase inhibitors

(3) Inducible anatomical defences – mechanical and structural barriers

(4) Inducible ecological or indirect defences – attraction of the natural enemies of the insect pest that is attacking

(5) Inducible civilian defences – reallocation of resources for regrowth (i.e. tolerance).

1. Inducible chemical defences

Low-molecular-weight compounds involved in defence are secondary metabolites that are classified according to their biosynthetic pathways and include terpenoids (> 40 000 known structures from the isoprenoid pathway; Keeling & Bohlmann, 2006), phenolic compounds (> 8000 known structures from the phenylpropanoid pathway; Bernards & Bastrup-Spohr, 2008), and alkaloids (> 12 000 known strucutures from the alkaloid pathway; Facchini, 2001). In plant–pathogen interactions, LMW antimicrobial compounds that are synthesized *de novo* upon infection are described as phytoalexins (Hammerschmidt, 1999), while pre-existing LMW antimicrobial compounds are called phytoanticipins (van Etten *et al.*, 1994).

Oleoresin is a complex mixture chiefly composed of monoterpenes and diterpenes, with smaller amounts of sesquiterpenes and other compounds, for example, phenolics (Keeling & Bohlmann, 2006). In many conifer species, the enhanced biosynthesis and accumulation of oleoresin are integral components of the induced chemical defence system against pathogens (Zeneli *et al.*, 2006) and insects (Franceschi *et al.*, 2005; Keeling & Bohlmann, 2006). This viscous liquid is produced in the resin ducts and related secretory structures of foliage, stems and other organs. The induced oleoresin functions as a direct toxin by readily interacting with the cell membranes of the invasive organism, which can lead to uncontrolled cell leakage, finally resulting in cell death. Invaders are also expelled from the tree in the flow of the oleoresin or trapped within the exudate as the wound is sealed by crystallization.

Phenolic compounds such as stilbenes, flavonoids, lignans and tannins are a major class of inducible defence compounds in many woody species (Witzell & Martin, 2008), including angiosperms (e.g. *Eucalyptus* spp., Eyles *et al.*, 2003; *Populus* spp., Tsai *et al.*, 2006; *Betula* spp., Ruuhola *et al.*, 2008) and conifers (e.g. *Pinus nigra*, Blodgett *et al.*, 2007; see review by Franceschi *et al.*, 2005). However, evidence for a direct role of phenolic compounds in IR is limited in conifers and apparently unconvincing, at least against herbivores (Mumm & Hilker, 2006). This ambiguity is also reported for *Populus* species (cottonwoods, aspens and poplars) where phenolic compounds are the major class of secondary metabolites in defence (Tsai *et al.*, 2006). Recent evidence suggests that correlations between insect performance and concentrations of phenolic compounds may not produce consistent results without also measuring the pro-oxidant activity of the phenolic compounds (Ruuhola *et al.*, 2008). Oxidation of phenolic compounds produces reactive oxygen species, resulting in oxidative stress in midgut tissues (Barbehenn *et al.*, 2009).

2. Inducible protein-based defences

Families of soluble pathogenesis-related proteins (PR proteins) include proteins (molecular mass < 100 kDa) involved in inducible protein-based defence. Of the 17 PR protein families that are now classified (Anonymous, 2009), the majority have been shown to be rapidly induced, both locally and systemically. The function and significance of many PR proteins in actual resistance, however, have yet to be fully elucidated (van Loon *et al.*, 2006). Members of the PR-3 family (chitinases) exhibit antimicrobial activities *in vitro* by affecting fungal cell wall or membrane integrity. Other proteins, such as PR-6 proteins (proteinase inhibitors), may target nematodes and herbivorous insects as well as pathogens by impairing their digestive enzyme activity (Jongsma & Beekwilder, 2008).

There have been relatively few studies on PR protein accumulation in conifers. The majority of them have focused on localized induced accumulation of PR proteins (e.g. thaumatin-like proteins (PR-5), Piggott *et al.*, 2004; ribonucleases (PR-10), Liu *et al.*, 2003). Nagy *et al.* (2004) showed that infection with the root rot fungus *Rhizoctonia* sp. resulted in local and systemic increases in peroxidase (PR-9) and chitinase (PR-3) activity in 6-wk-old *Picea abies* (Norway spruce) seedlings.

Wound-induced proteins triggered by and accumulated after insect attack include proteinase inhibitors, cysteine proteases, lectins, lipoxygenases and polyphenol oxidases (PPOs) (Ruuhola *et al.*, 2008). Each defensive protein may exert toxic and antifeedant activities (reviewed by Howe & Jander, 2008). In particular, PPOs apparently decrease the absorption of amino acids by catalysing the oxidation of orthodiphenolic compounds to quinones, which cross-link proteins in the insect gut and render them indigestible (Felton *et al.*, 1992). The defensive activities of inducible proteins, including PPOs, endochitinases and the Kunitz protease inhibitors, against various insect pests have been well studied in poplar (Philippe & Bohlmann, 2007). The larval growth rate of *Malacosoma disstria* (forest tent caterpillars) was shown to decrease on transgenic *Populus* overexpressing the induced leaf PPO gene compared with larvae feeding on control leaves, although this depended in part on the timing of egg hatching (Wang & Constabel, 2004). A more recent study by Barbehenn *et al.* (2007) concluded that the efficacy of PPO as a direct defence against caterpillars may be much weaker than was previously believed, particularly against two species of caterpillars (*Lymantria dispar* and *Orgyia leucostigma*) that feed on poplar.

3. Inducible anatomical defences

Immediately following attack, plants often respond locally by modifying cell walls (Huckelhoven, 2007). Papillae (and their closely related lignitubers) are relatively simple, local cell wall fortifications built at the site of attempted pathogen penetration (Huckelhoven, 2007). Papillae are usually made up of callose, but there are examples of pectin-like matrices (Bonello *et al.*, 1991) and their composition can be modified by deposition of phenolic compounds, lignin, suberin and/or silicon oxides, and enrichment with hydroxyproline-rich glycoproteins (Hammerschmidt & Nicholson, 1999). Induced lignification and suberization of cell walls are known to contribute to cell wall strengthening and form a zone of water-impervious tissue, isolating the wound from neighbouring undamaged cells (Eyles *et al.*, 2003).

A well-recognized form of anatomical modification is the formation of traumatic resin ducts (TRDs) in the xylem and/or phloem of many conifer species (Krokene *et al.*, 2008). TRDs are associated with induction of terpene biosynthesis and increased resin flow within 2–3wk after attack (Luchi *et al.*, 2005).

The formation of the wound (necrophylactic) periderm, particularly its rate of formation, is considered to be a critical resistance mechanism to phloem-feeding borers (e.g. buprestid beetles; Dunn *et al.*, 1990), phloem-invading microorganisms (e.g. stem cankers; Eyles *et al.*, 2003) and fungal leaf spot diseases (e.g. *Mycosphaerella* leaf disease; Smith *et al.*, 2007). Forming at the boundaries of the invaded or damaged region, the wound periderm serves to wall off the wound, inhibit the spread of the colonizing organism (e.g. by encapsulation of insect larvae) and re-establish a continuous impervious surface barrier (Robinson *et al.*, 2004).

4. Inducible ecological or indirect defences

Induced indirect plant defences protect plants via the promotion of tritrophic interactions (Heil, 2008). They include morphological and chemical host modifications that attract and support the natural enemies (predators or parasitoids) of herbivorous insects. A major form of induced indirect defence is the emission of plant volatile organic compounds (VOCs; < 300 Da). Released in response to attack by insect herbivores and pathogens, VOCs act as infochemicals in plant–plant and plant–carnivore communication (Dicke *et al.*, 2009). Interestingly, some of the earliest studies of induced host volatiles were with trees such as *Populus* × *euroamericana*, *Acer saccharum* (sugar maple) and *Alnus glutinosa* (alder) (Baldwin & Schultz, 1983; Tscharntke *et al.*, 2001).

Induced VOCs can serve as plant 'hormones' (compounds serving as within-plant signals) to elicit a defensive response in undamaged parts of the same plant individual (Frost *et al.*, 2008). Frost *et al.* (2007, 2008) showed that VOCs (e.g. cis-3-hexenyl acetate) released either by herbivore-wounded leaves or naturally wounded leaves of hybrid *Populus deltoides* × *nigra* saplings primed defensive responses in neighbouring undamaged leaves of the same individual plant.

Induced VOCs can also function as plant 'pheromones' (compounds serving as between-plant signals) to prime neighbouring, unharmed/noninfested plants to respond faster to future herbivore attack. Primed plants do not show detectable expression of defence traits in the absence of a challenge. Instead, they respond more rapidly or more intensely once they are attacked (Conrath *et al.*, 2006). Following attack, primed plants show a range of amplified defence responses; for example, increased expression of defence-related genes in *Nicotiana attenuata* (wild tobacco) (Karban *et al.*, 2000). Evidence of this tritrophic signalling has also been found for trees. Rhoades (1983) reported that undamaged *Salix sitchensis* (Sitka willow) trees growing close to herbivore-infested conspecifics mounted a higher

chemical defence to *Hyphantria cunea* (fall webworm) larvae than controls from a more distant site. Field studies found that herbivory rates on *Alnus glutinosa* trees were lower when growing close to damaged conspecifics (Dolch & Tscharntke, 2000).

Finally, induced VOCs contribute to indirect defences by attracting natural enemies such as predators (Shepherd *et al.*, 2005) and parasitoids (Hilker *et al.*, 2002). An example of a below-ground interaction is the release of VOCs from the roots of *Thuja occidentalis* when attacked by *Otiorhynchus sulcatus* (black vine weevil) larvae. These VOCs have been shown to attract *Heterorhabditis megidis* (entomopathogenic nematodes) which are predators of *O. sulcatus* (van Tol *et al.*, 2001). An example of an above-ground interaction is the release of VOCs from of the needles of *Pinus sylvestris* (Scots pine) following egg deposition by *Diprion pini* (pine sawfly) (Hilker *et al.*, 2002). These oviposition-induced VOCs, characterized by larger quantities of the sesquiterpene (*E*)-β-farnesene than their controls, have been shown to attract egg parasitoids (Mumm *et al.*, 2003).

5. Inducible civilian defences

Plants can minimize the negative fitness consequences of tissue lost to herbivory or pathogen attack by activating physiological processes that allow the plant to compensate for the reduction in total photosynthetic capacity. These are termed 'civilian' defences. Unlike host resistance, civilian defences do not directly affect the biotic agent's performance. For example, in insect–host interactions, host tolerance allows plants to support herbivore populations similar to a susceptible host without a concomitant reduction in plant fitness (Karban & Baldwin, 1997; Haukioja & Koricheva, 2000).

Mechanisms of tolerance appear common in cases of attack by both leaf-feeding herbivores and foliar pathogens. These include up-regulation of photosynthetic rates in remaining uninfected/undamaged leaves (Quentin *et al.*, 2010), alteration in growth patterns to favour development of leaf area (Frost & Hunter, 2008) and shifts in resource allocation patterns within and between the above- and below-ground organs of a tree. The latter can be achieved by remobilization of reserves from storage tissues or by mobilization of resources to temporary storage in organs that are less susceptible to damage (e.g. the root system) (Babst *et al.*, 2008; Frost & Hunter, 2008). Collectively, these changes enhance the plant's ability to tolerate subsequent pathogen and herbivore attack. However, in some cases such reallocation of resources can be counterproductive. For example, in some insect–plant interactions, herbivory can induce compensatory regrowth that often has a positive effect on other herbivorous insects by increasing the quality or quantity of food available (Utsumi & Ohgushi, 2008). Thus, insect herbivory that stimulates regrowth in host plants may increase their susceptibility to insect herbivores that emerge at a later time.

III. Systemic induction of resistance

Induced resistance can occur at the site of the initial attack (local defence) or be functional in distant parts of the plant

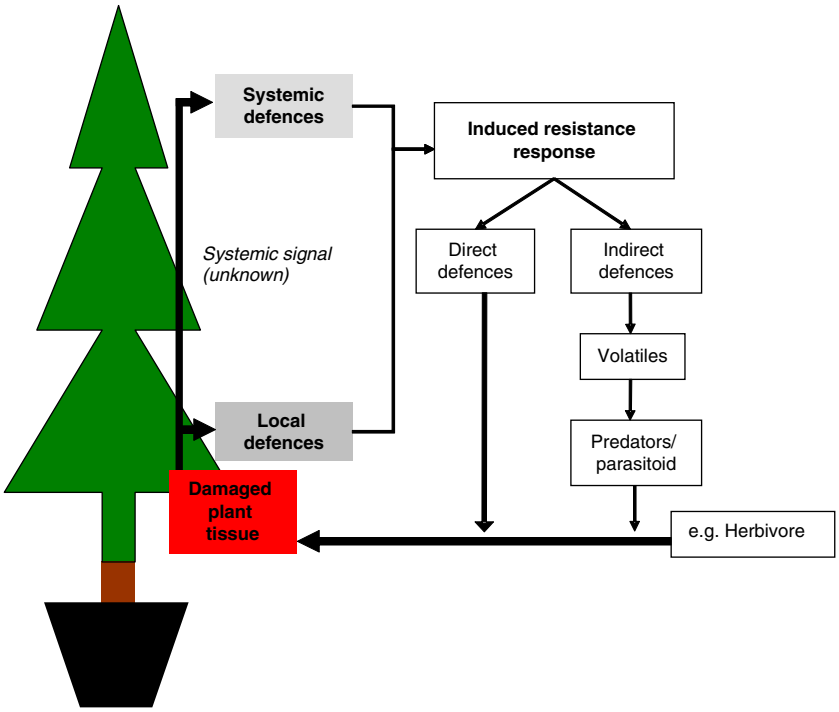

Fig. 2 Overview of local and systemic defences against biotic damaging agents, in this case an insect herbivore in conifers.

Table 1 Different forms of systemic induced resistance (SIR) in plant–pathogen and plant–insect interactions

Inducing agent	Type of SIR	Plant type	Major endogenous signalling molecules	Reference
Pathogens causing HR	SAR	Herbaceous species	SA	van Loon *et al.* (1998); Durrant & Dong (2004)
Necrotizing pathogens	SIR	Conifer species	Unknown	Bonello *et al.* (2001)
Plant growth-promoting rhizobacteria	ISR	Herbaceous species	JA and ET	van Loon (2007)
Plant growth-promoting fungi	ISR	Herbaceous species	JA and ET	van Wees *et al.* (2008)
Mycorrhizosphere/ actinomycete	Unknown	Herbaceous species	Unknown	Lehr *et al.* (2008)
Wounding	Wound-induced IR	Herbaceous species	Unknown	Chassot *et al.* (2008)
Herbivores	Herbivore-induced direct and indirect resistance	Herbaceous and tree species	JA and ET	Kessler & Baldwin (2002)

HR, hypersensitive response; SAR, systemic acquired resistance; ISR, induced systemic resistance; IR, induced resistance; SA, salicylic acid; JA, jasmonic acid; ET, ethylene.

or throughout the entire plant (systemic defence) (Fig. 2). Systemic host responses are thought to be activated through the plant via one or more signalling molecules and may result in systemic induced resistance (SIR) (Kuc, 1983). To date, at least seven types of SIR have been described (Table 1). In the case of necrotic lesion-inducing pathogens that cause a hypersensitive response and the systemic expression of PR genes, the phenomenon is known as systemic acquired resistance, or SAR (Durrant & Dong, 2004). In trees, SIR also develops in response to necrogenic pathogens, but given that nothing is known about the signalling system involved, this type of SIR is viewed differently from SAR (Bonello *et al.*, 2001, 2006). SIR can also be induced by rhizosphere microorganisms, in which case it is known as induced systemic resistance (ISR), but unlike SAR, ISR is not associated with induction of PR genes (van Loon, 2007). ISR is activated by colonization of plant roots by selected strains of free-living, nonpathogenic, plant growth-promoting rhizobacteria (PGPR) (van Loon, 2007) and the less well studied root-colonizing plant growth-promoting fungi (PGPF), such as *Trichoderma* spp. (Vinale *et al.*, 2008), *Penicillium* sp. GP16-2 and *Phoma* sp. GS8-1 (Hossain *et al.*, 2008). Insect herbivores are also known to induce SIR, but in the case of insects, this type has historically been subsumed into the general definition of IR and includes both direct and indirect resistance (Kessler & Baldwin, 2002). Other less well studied types of SIR have been reported, including those induced by wounding (Chassot *et al.*, 2008) and mycorrhizal fungi or rhizosphere actinomycetes (Lehr *et al.*, 2008) (Table 1). Finally, another possible outcome of host–pathogen/pest interaction is one of systemic induced susceptibility (SIS) and, in contrast to SIR, SIS phenotypes are characterized by reduced resistance to subsequent attacks on distal parts of a plant. SIS phenotypes have been reported in herbaceous plants and trees (Simon & Hilker, 2003; Cui *et al.*, 2005; Blodgett *et al.*, 2007; Bonello *et al.*, 2008).

1. SIR and SIS in trees

It is worth noting that the characterization of SIR against stem and branch pathogens in trees has been largely based on coniferous species, and equivalent information for angiospermous species is lacking (Table 2). Bonello *et al.* (2001) demonstrated sustained SIR (as indicated by decreasing lesion size) against the pitch canker pathogen, *Fusarium circinatum*, in *Pinus radiata* (Monterey pine) over a 1.5 yr period. At these same sites, *P. radiata* trees that had been severely affected by pitch canker were shown to be free of disease several years later despite constantly elevated pathogen pressure (Gordon *et al.*, 2001). Subsequently, SIR to pitch canker was confirmed in a subset of apparently healthy trees by direct challenge with the pathogen (Gordon, 2006).

Systemic induced resistance has also been demonstrated in the tree model pathosystem, *Pinus nigra* (Austrian pine) – *Diplodia pinea*. Young Austrian pine trees inoculated in the stem phloem with the necrogenic canker pathogen *D. pinea* or the less aggressive species, *D. scrobiculata* (Bonello & Blodgett, 2003), became more resistant to subsequent inoculations in the stem or branches with *D. pinea* (Blodgett *et al.*, 2007; Wallis *et al.*, 2008). The phenomenon is bidirectional, suggesting that molecular signals move both acropetally and basipetally in the tree to elicit the SIR response (Blodgett *et al.*, 2007). Furthermore, it was shown that the trajectory and resistance outcome of these host-mediated interactions may be organ-dependent (Blodgett *et al.*, 2007). Inoculation of young Austrian pine saplings at

Table 2 Selected examples of induced systemic resistance (ISR), systemic induced resistance (SIR) or systemic induced susceptibility (SIS) against insect herbivores and pathogens in trees (in chronological order)

Host species	Inducing biotic agent	Responses	Reference
Picea abies	*Ceratocystis polonica*	SIR to *C. polonica*	Christiansen *et al.* (1999)
Pinus taeda	*Bacillus pumilus* and *Serratia marcescens* (PGPR)	ISR to *Cronartium quercuum* f. sp. *fusiforme*	Enebak & Carey (2000)
Pinus radiata	*Fusarium circinatum*	SIR to *F. circinatum*	Bonello *et al.* (2001)
Pinus ponderosa	*Heterobasidion annosum*	SIR to *Ips paraconfusus* (in logs)	McNee *et al.* (2003)
Salix × *cuspidata* (hybrid)	*Plagiodera versicolora*	SIS to *Melampsora allii-fragilis*	Simon & Hilker (2003)
Pinus nigra	*Diplodia pinea* and *D. scrobiculata*	SIR in main stem but SIS in shoots to *D. pinea*	Blodgett *et al.* (2007)
Pinus nigra	*Diplodia pinea* and *Neodiprion sertifier*	Cross-induction of SIR to *D. pinea*	Eyles *et al.* (2007)
Picea abies	*Heterobasidion parviporum*	SIR to *H. parviporum*	Swedjemark *et al.* (2007)
Pinus pinea	*Heterobasidion annosum*	SIS to *D. pinea*	Bonello *et al.* (2008)
Picea abies	*Streptomyces* sp. GB 4-2	SIR to *Heterobasidion abietinum* 331	Lehr *et al.* (2008)

the stem base with *D. pinea* or *D. scrobiculata* resulted in contrasting systemic phenotypes with SIR of stem tissues but SIS of shoot tips (Blodgett *et al.*, 2007). Other studies of this model pathosystem also demonstrated that the SIR observed in stems was associated with an integrated host defence response manifested by distinct anatomical and biochemical changes, including enhanced lignin deposition, accumulation of certain soluble phenolic compounds (Blodgett *et al.*, 2007; Wallis *et al.*, 2008), proteins (Wang *et al.*, 2006), and induction of TRDs and resin flow (Luchi *et al.*, 2005).

Historically, plant–herbivore and plant–microbe interactions have been investigated by separate disciplines. There is increasing attention directed to an overlap of disciplines and the examination of induced plant responses against two or even multiple biotic agents spanning different kingdoms (van Oosten *et al.*, 2008). Interference between molecular signalling pathways can result in what is known as cross-talk and this can result in negative or positive interactions (Bostock, 2005). For example, conifers visibly suffering from root disease (i.e. symptomatic trees) are known to be more susceptible to colonization and immediate mortality caused by bark beetles (Erbilgin & Raffa, 2000), providing field evidence of SIS. Basal stem inoculation with the root and butt-rot fungal pathogen, *Heterobasidion annosum*, also elicited SIS against *D. pinea* in *P. pinea* (Italian stone pine) shoots (Bonello *et al.*, 2008). Whole-plant SIR induced in a tree by a fungal pathogen against a defoliating insect and vice versa, under variable nutrient availability, was reported for the first time by Eyles *et al.* (2007). In this 2 yr study, the *P. nigra*/*D. pinea*/*Neodiprion sertifer* (European pine sawfly) model system demonstrated that fungal infection elicited SIR against the defoliating insect across all nutrient environments in the first year only. By contrast, insect defoliation induced SIR against subsequent fungal challenge in the second year only. However, fungal infection elicited SIR against the same fungus in both years. These results suggest that the cross-induction of SIR in *P. nigra* to these biotic agents can be asymmetric within a year and variable between years. Other studies have examined the direct effect of pathogen-induced SIR on insects, and vice versa, in woody plants and have reported contrasting results. Krause & Raffa (1992) found that infection of *Larix decidua* (larch) with the fungal pathogen *Mycosphaerella laricina* induced a systemic reduction in host quality for *Pristiphora erichsonii* (larch sawfly). Simon & Hilker (2003) provided some evidence that feeding by *Plagiodera versicolora* larvae increased the systemic susceptibility towards infection by the rust pathogen, *Melampsora allii-fragilis*.

2. Herbivore-induced IR

Previous attack by an insect herbivore can induce systemic protection in a plant against a second attacker, even when the initial attack has no discernible effects on plant growth or fitness (Karban & Baldwin, 1997; Howe & Jander, 2008). Herbivore-induced changes in plant resistance can occur within hours, days or weeks of initial attack (rapid induced resistance, RIR) or, in long-lived species such as trees, over more extended timescales (delayed induced resistance, DIR) (Haukioja, 1982). Studies investigating herbivory-induced responses on subsequent herbivore resistance of tree species have been inconsistent. Defoliation-induced RIR and DIR to folivores have been documented for *Betula* spp. (birch) (Haukioja & Neuvonen, 1985; Ruuhola *et al.*, 2008), *Larix decidua* (larch) (Krause & Raffa, 1992), *Pinus resinosa* (red pine) (Krause & Raffa, 1995), *Pinus contorta* (lodgepole pine) (Trewhella *et al.*, 1997), *Quercus rubra* (red oak) (Roden & Mattson, 2008), *Populus tremuloides* (trembling aspen) (Roden & Mattson, 2008) and *Eucalyptus globulus* (blue gum) (Rapley *et al.*, 2008). In other cases, previous defoliation had no effect or enhanced the host quality for folivores (Lyytikainen, 1994). Other factors cited as sources of variation in expression of induced responses of tree species

include timing and severity of defoliation, age of foliage and trees, as well as herbivore phenology and behaviour.

Herbivory causes large-scale changes in gene expression. For example, in hybrid poplar *Populus trichocarpa* × *deltoides*, it is estimated that 11% of the transcriptome is modified by forest tent caterpillar (Ralph *et al.*, 2006). A common feature of insect feeding (unlike pathogen attack) is some degree of mechanical damage, and many studies have focused on the regulatory signals generated as a result of artificial wounding. However, such investigations may not yield results that reflect an actual response to an insect. Plants may discriminate insect herbivory from experimental wounding by the different temporal and spatial patterns of natural and artificial injuries (Mithofer *et al.*, 2005). Moreover, the type of feeding (e.g. chewing vs sucking) and blend of oral secretions (saliva or regurgitant of the attacker) may determine the specific response signature of the host plant. In particular, insect-derived elicitors produced during feeding have been shown to trigger direct and indirect defence responses, as detected by changes in gene and protein expression, and production of VOCs and other secondary metabolites (Felton, 2008). To date, work on oral secretions and saliva has been confined to lepidopteran larvae and the chemical characterization of this group, and research into other insect groups has barely commenced. Several groups of elicitors have been found in oral secretions of lepidopteran larvae, such as lytic enzymes like β-glucosidase (Mattiacci *et al.*, 1995), fatty acid-amino conjugates, for example, volicitin (*N*-(17-hydroxylinolenoyl)-L-glutamine) (Alborn *et al.*, 1997; Major & Constabel, 2006), and, more recently, chloroplastic peptide fragments called inceptins (Schmelz *et al.*, 2006). However, another lytic enzyme, glucose oxidase, found in high concentrations in the oral secretions of *Helicoverpa* spp., may function in defence suppression as a counter-defence strategy (Musser *et al.*, 2006).

3. Endophyte-mediated IR

In addition to fungal pathogens, symbiotic fungal species have also been shown to mediate IR in host plants (van Wees *et al.*, 2008). Mutualistic fungi, such as mycorrhizal fungi and PGPF, have been shown to be involved in ISR (Table 1) and, similarly, endophytic fungal associates have been shown to confer enhanced host resistance (Kogel *et al.*, 2006; Rodriguez & Redman, 2008). Endophytic fungi (symbionts that live within the plant without causing disease) have been ubiquitously found in all plant species studied to date (Kogel *et al.*, 2006) and could play a critical role in priming plants for IR, or conversely, in some cases, induced susceptibility.

Endophytic fungi have been shown to enhance host resistance in several tree species (Arnold *et al.*, 2003; Ganley *et al.*, 2008; Saravesi *et al.*, 2008). Arnold *et al.* (2003) showed that inoculation of endophyte-free leaves with endophytes isolated frequently from naturally infected, asymptomatic hosts significantly decreased both leaf necrosis and leaf mortality in *Theobroma cacao* (cocoa plant) when challenged with a pathogenic foliar *Phytophthora* sp. Similarly, Ganley *et al.* (2008) showed that fungal endophytes from *Pinus monticola* (western white pine) were effective at increasing survival in host plants attacked by the exotic pathogen *Cronartium ribicola*, the causal agent of white pine blister rust. Specifically, seedlings previously inoculated with fungal endophytes lived longer than endophyte-free seedlings and also showed some reduction in disease severity. This endophyte-mediated resistance was found to be effective over time, indicating persistence (Ganley *et al.*, 2008).

IV. Defence signalling networks

The nature of SIR clearly suggests that a signal must be generated locally as a consequence of attack and then transmitted throughout the plant, thus activating the expression or priming of inducible factors at distant sites in undamaged host tissues. The model that has emerged from studies in herbaceous plants is that of a nonlinear network of overlapping, synergistic or antagonistic, interactive signalling pathways linked by positive and negative feedback loops that coordinate host responses to diverse biotic threats. These regulatory pathways appear to be mediated by jasmonic acid (JA) (Balbi & Devoto, 2008), salicylic acid (SA) (Grant & Lamb, 2006) and ethylene (ET) (Broekaert *et al.*, 2006; von Dahl & Baldwin, 2007) (Table 1). Other hormones, such as abscisic acid, can also play a role in IR, but their significance is less well established (Mauch-Mani & Mauch, 2005). There are several excellent reviews on these hormone signals, which show that they vary greatly in quantity, composition and timing and result in the activation of different sets of defence responses (Pieterse *et al.*, 2009; Volt *et al.*, 2009). Many questions still remain about the precise nature of the mobile signal(s) involved. For instance, does systemic accumulation of the molecular mediators listed earlier result from *de novo* synthesis in undamaged leaves or from transport from injured source leaves? The phloem mobility and systemic signalling activity of exogenous phytohormones support the latter hypothesis. Recent grafting experiments using salicylic acid–binding protein 2 silenced rootstock implicated methyl salicylate (MeSA) as the SAR mobile signal in tobacco (Park *et al.*, 2007), although in *Arabidopsis*, MeSA seems not to play a role (Attaran *et al.*, 2009).

1. Signalling molecules in trees

The mechanistic understanding of signalling has been largely derived from studies using mutational screens of herbaceous systems. Little is known about the endogenous

Table 3 Selected examples of induced resistance (IR) by exogenous application of elicitor in tree species (in chronological order)

Host species	Elicitor/ signalling molecule	Responses	Reference
Pseudotsuga menziesii *Picea pungens* *Larix occidentalis* *Pinus monticola* *Taxus brevifolia*	MeJA	Induced similar anatomical responses to those from wounding	Hudgins *et al.* (2003); Hudgins *et al.* (2004)
Salix viminalis	SA	Decreased larval survival frequency of *Dasineura marginemtorquens*	Ollerstam & Larsson (2003)
Pseudotsuga menziesii and *Sequoiadendron giganteum*	MeJA ET MeSA	MeJA -induced ET production elicited conifer phloem defences	Hudgins & Franceschi (2004)
Pinus radiata	Chitosan	Increased resistance to artificial inoculation with *Fusarium circinatum*	Reglinski *et al.* (2004)
Populus tremuloides	JA	Induced rapid changes in carbon transport and partitioning	Babst *et al.* (2005)
Pinus sylvestris	MeJA	Increased resistance to *Hylobius abietis*	Heijari *et al.* (2005)
Pseudotsuga menziesii	MeJA	Induced changes in anatomy and terpene chemistry in roots	Huber *et al.* (2005)
Picea sitchensis	MeJA	Induced similar but not identical terpenoid defence responses to attack by *Pissodes strobi*	Miller *et al.* (2005)
Picea abies	MeJA	Reduced *Ips typographus* colonization and reproduction	Erbilgin *et al.* (2006)
Picea abies	MeJA	Increased resistance to *C. polonica*	Zeneli *et al.* (2006) Krokene *et al.* (2008)
Populus hybrid	MeJA	Demonstrated shoot–root systemic defence signalling	Major & Constabel (2007)
Pinus halepensis	5-chloro-SA	Increased resistance to infection by *Diplodia pinea* and *D. scrobiculata*	Moret & Munoz (2007)
Castanea dentata *Castanea mollissima*	JA	Reduced relative growth rate of *Lymantria dispar* on *Castanea dentata* only	Cooper & Rieske (2008)
Pinus radiata	MeJA	Transiently increased resistance to *D. pinea*	Gould *et al.* (2008)
Pinus sylvestris	MeJA	Growth rates of *Neodiprion sertifer* and *Diprion pini* larvae were overall lower on needles of MeJA-treated plants but not across all seed origins	Heijari *et al.* (2008)
Eucalyptus grandis	MeJA	No effect on herbivore performance (*Paropsis atomari*) and foliar chemistry	Henery *et al.* (2008)
Picea abies	Oxalic acid	Increased resistance to *Ceratocystis polonica*	Krokene *et al.* (2008)

MeJA, methyl jasmonate; SA, salicylic acid; MeSA, methyl salicylate; ET, ethylene; JA, jasmonic acid.

signalling activity for SIR in trees and this may be, in part, because biosynthetic mutants are not as readily available for woody species. The current, limited evidence suggests that SIR in conifers could be mediated by signalling molecules that are at least partly different from those of herbaceous systems (Bonello & Blodgett, 2003; Bonello *et al.*, 2006). For example, the accumulation of SA was not associated with changes in the phenolic composition of *P. sylvestris* seedling needles (local and systemic) (Bonello *et al.*, 1993) or a response to a root pathogen in *P. ponderosa* (Bonello *et al.*, 2003) or to *D. pinea* infection in *P. nigra* (Bonello & Blodgett, 2003). Endogenous JAs have been shown to accumulate in *P. pinaster* (maritime pine) in response to cold and water stress (Pedranzani *et al.*, 2008), but no information is available with regard to a possible role of endogenous JAs in conifer SIR.

However, numerous studies have shown that, in the absence of wounding conifer, defence mechanisms can be induced by exogenous application of certain hormones, particularly JAs (Table 3), suggesting a potential role of these molecules in IR. Exogenous JAs induced enhanced localized resistance to biotic agents such as *Hylobius abietis* (large pine weevil) (Heijari *et al.*, 2005), *Ceratocystis polonica* (blue-stain fungus) (Zeneli *et al.*, 2006; Krokene *et al.*, 2008) and *Pythium ultimum* (white root rot fungus) (Kozlowski *et al.*, 1999). Exogenous applications of methyl jasmonate (MeJA) induced the formation of TRDs in various conifer species such as *Picea abies* (Norway spruce) (Erbilgin *et al.*, 2006; Zeneli *et al.*, 2006) and *Picea sitchensis* (Sitka spruce) (Miller *et al.*, 2005). In ongoing work, application of MeJA to the bark of *Fraxinus mandshurica* (Manchurian ash) and *F. americana* (white ash) induced accumulation of phloem

phenolic compounds (Justin Whitehill *et al.*, unpublished), while foliar application of MeJA failed to enhance host resistance against *Phytophthora cinnamomi* in several *Eucalyptus* spp. (McComb *et al.*, 2008).

V. Future prospects: what does IR offer for the management of forest systems?

Trees growing in urban and agricultural landscapes or in natural forest systems provide a full suite of goods and services that are vital to human well-being, such as timber products, biodiversity, watershed services, emissions reduction, carbon storage, scenic landscapes, recreation and aesthetics. With rapid global change, the provision of such ecosystem services by forest trees is increasingly subject to threats such as pollution, drought and damage from both native and invasive alien pests (Dukes *et al.*, 2009). The latter threat is often amplified by the first two. A major challenge is to develop novel pest management options for forest tree systems that are effective, environmentally sustainable and adaptable to the needs of an uncertain environment.

Tree protection strategies based on the manipulation of IR are in the early stages of conceptual development and there are large knowledge gaps surrounding mechanisms and outcomes. In agriculture, the discovery of natural and synthetic inducers that mimic the action of the natural signals prompted a strong interest in IR as a strategy for crop protection (Vallad & Goodman, 2004; Goellner & Conrath, 2008). For example, the synthetic compound, acibenzolar-S-methyl (sold commercially as Actiguard or Bion), a functional analogue of SA, can induce priming for enhanced activation of defence responses and resistance to fungal and bacterial pathogens in various crops (Leadbeater & Staub, 2007). Many plant-protecting compounds have been identified that combine both direct action on the pathogen and priming-inducing activity in the plant, and several have given rise to new commercial formulations (Table 4). Similarly, identification of the VOCs involved in induced indirect defences or characterization of the signalling molecule pathways in trees could provide potential targets for the commercial development of bioactive small metabolites. These discoveries could, in the long term, be patented, developed and marketed for application in forest systems worldwide as environmentally friendly tree health promoters. These synthetic IR-inducing molecules could also be used as a screening tool in traditional breeding programmes, especially those that target the matching of species or genotype to site conditions.

In order to achieve these objectives, characterization of the endogenous signalling pathway is the most critical step to the development of IR for trees. Whereas investigations in herbaceous crops were facilitated by the use of biosynthetic mutants, these are not readily available for most trees but this challenge may be overcome by employing alternative techniques. For example, monitoring the metabolome following exogenous application of elicitors (Kontunen-Soppela *et al.*, 2007; Robinson *et al.*, 2007) may offer one way to identify the small molecule(s) involved in systemic signalling. The availability of the poplar genome (Tuskan *et al.*, 2006) has already enabled new research approaches in angiosperm tree defence biology (Rinaldi *et al.*, 2007; Azaiez *et al.*, 2009; Barakat *et al.*, 2009). Furthermore, RNAi technology may also become one of the most important tools available for functional analysis of possible signalling pathways, although, presently, such technology is probably only feasible with species like poplar that are readily propagated *in vitro* (Coleman *et al.*, 2008). Such advances will substantially accelerate the rate of discovery and functional analysis of genes associated with defence and resistance against biotic agents (Major & Constabel, 2006; Ralph *et al.*, 2006; Miranda *et al.*, 2007; Philippe & Bohlmann, 2007).

The trade-off between disease resistance and the high costs of defence activation involved in IR must also be considered; energy resources that could otherwise be used for growth, development and reproduction are diverted to defence (Björkman *et al.*, 2008). For instance, exogenous applications of MeJA on *P. sylvestris* seedlings and *P. abies* trees resulted in 30% less radial sapwood growth than in control trees (Heijari *et al.*, 2005; Krokene *et al.*, 2008). Elevated resistance of *P. radiata* to *D. pinea* induced by foliar applications of MeJA was accompanied by a reduction in seedling growth rate in the second week following treatment (Gould *et al.*, 2008), although the seedlings recovered and eventually their growth rate exceeded that of control seedlings. All three studies concluded that the observed reduction in growth is likely to be a transient effect and will probably have little impact on long-term tree growth, but recovery may be linked directly to the duration of the heightened IR state.

Induced resistance does not provide complete pest control (Walters, 2009) and therefore it will have to be deployed in a smart manner. The critical question facing conventional agriculture is not 'Will IR work in the field?' but 'How well will IR work in the field?' There are many environmental factors, such as nutrient supply, water availability and temperature, that will influence the efficacy and effectiveness of the IR responses, regardless of whether IR is induced by elicitors, synthetic compounds, pest/pathogens or other microorganisms. Other factors that are likely to be critical include: the timing of application, the risk of rendering the treated plant more susceptible to other pathogens or insect herbivores, and the duration of the induced resistance. Such risks need further investigation and it is likely that individual cost–benefit assessments will be required for each host/pest/IR system.

Table 4 Selected examples of natural and synthetic compound elicitors

Type of elicitor	Protected plant	Comment
Brotomax® Agrométodos, S.A, Madrid, Spain	*Citrus* spp.	Micronutrient complex composed of urea, copper lignosulphonate, manganese lignosulphonate and zinc lignosulphonate Induces resistance to *Phytophthora parasitica*
Fungal cell wall components	*Pinus radiata* Herbaceous species	Chitin is the main cell wall component of many filamentous fungi, while chitosan is the deacetylated derivative of chitin Commercial product: Elexa™ GlycoGenesys, Boston, Massachusetts, USA (4% chitosan active ingredient) Induces resistance and/or direct antifungal activity
Imidacloprid	Tree and herbaceous species	Commercial product: Admire® Bayer Crop Science Inc, Alberta, Canada, Confidor® Bayer Crop Science, Ptd Ltd, Victoria, Australia, Merit® Bayer Crop Science, Ptd Ltd, Victoria, Australia Induces resistance and has direct insecticidal activity. One of its major degradation products, 6- chloronicotinic acid, has a structure very similar to INA
Jasmonates	Tree and herbaceous species	*cis*-jasmone (structurally related to JA and MeJA) Released naturally from insect-damaged plants Insect semiochemical MeJA Induces resistance to insects and pathogens
Plant VOCs	Tree and herbaceous species	For example, terpene alcohols, MeJA, MeSA, and GLVs Induce indirect resistance
Phosphonates	Tree and herbaceous species	Commercial product: Phytoguard (58% potassium phosphonate active ingredient), ProPhyt® Luxembourg-Pamol Inc, Memphis, Tennessee, USA, Phostrol® Nufarm Americas Inc, Burr Ridge, Illinois, USA, Fosphite® JH Biotech Inc. Ventura, California, USA, Agriphos® J and H Bunn Ltd, UK Direct antifungal activity and weakly induced resistance
SA and functional analogues	Tree and herbaceous species	Acibenzolar-S-methyl (ASM or BTH) (syn. benzo(123)thiadiazole-7-carbothioic acid(S) methyl ester) Commercial product: BION® (in Europe), Actiguard® (in the USA) Effective against a wide range of pathogens on a range of crops Probenazole and its active metabolite 1,2-benzisothiazole-1,1-dioxide Commercial product: Oryzemate® Academichem Co., Ltd., Beijing, China Protects rice crops against rice blast caused by *Magnaporthe grisea* Methyl salicylate (MeSA) (biosynthetically related to SA)
β-aminobutyric acid	Herbaceous species	BABA: a nonprotein amino acid that is a potent inducer of resistance in plants with broad-spectrum activity, effective against microbial pathogens, nematodes, insects and drought and stress
Reactive oxygen species (ROS)	Herbaceous species	Commercial product: Oxycom™ Redox Chemicals, Burley, Idaho, USA (registered in North America for management of pathogens from the *Pythium* genus, downy and powdery mildews)
Harpin	Herbaceous species	Originally isolated from *Erwinia amylovora*, harpin is an acidic, heat-stable, cell envelope-associated protein with a molecular mass of *c.* 40 kD. Harpin induces HR and in some cases provides broad-spectrum activity against a wide range of pathogens Commercial product: Messenger® Eden Bioscience, Bothell, Washington, USA (released in North America and Europe)
Pyraclostrobin	Herbaceous species	Commercial product: Cabrio® and Headline® BASF, Florham Park, New Jersey, USA Induces resistance and has direct antimicrobial activity
Silicon	Herbaceous species	Increase resistance via unknown mechanism Reduces disease development
Vitamin B1	Herbaceous species	Increases disease resistance

GLV, green leafy volatile; JA, jasmonic acid; MeJA, methyl jasmonate; MeSA, methyl salicylate; SA, salicylic acid; VOC, volatile organic compounds.

Future research on IR in forest systems provides opportunities to explore mechanisms of local and systemic host defence that may be unique to large and long-lived organisms like trees. Clearly, many critical questions remain unanswered and these will need to be addressed if we are to develop and exploit IR as an alternative, eco-friendly solution for mitigating pest impacts in trees, including those arising from biological invasions. Results from recent studies of IR in trees (Heijari *et al.*, 2005; Blodgett *et al.*, 2007; Ganley *et al.*, 2008; Krokene *et al.*, 2008) are highly encouraging and indicate that the prospect of using IR as a future management option in forest systems is a plausible goal.

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