Tansley review

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.

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

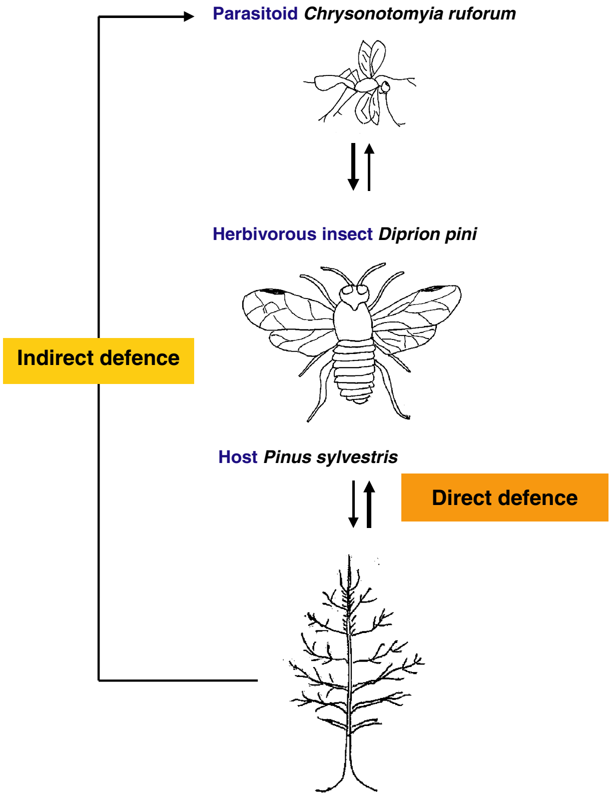

Fig. 1 Direct defence of a tree (in this case, *Pinus sylvestris*) has a direct adverse effect on the invading organism (*Diprion pini*). Indirect defences rely on the attraction of natural enemies (e.g. *Chrysonotomyia ruforum*) of *D. pini*. (Diagram based on findings reported by Hilker *et al.*, 2002.)

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

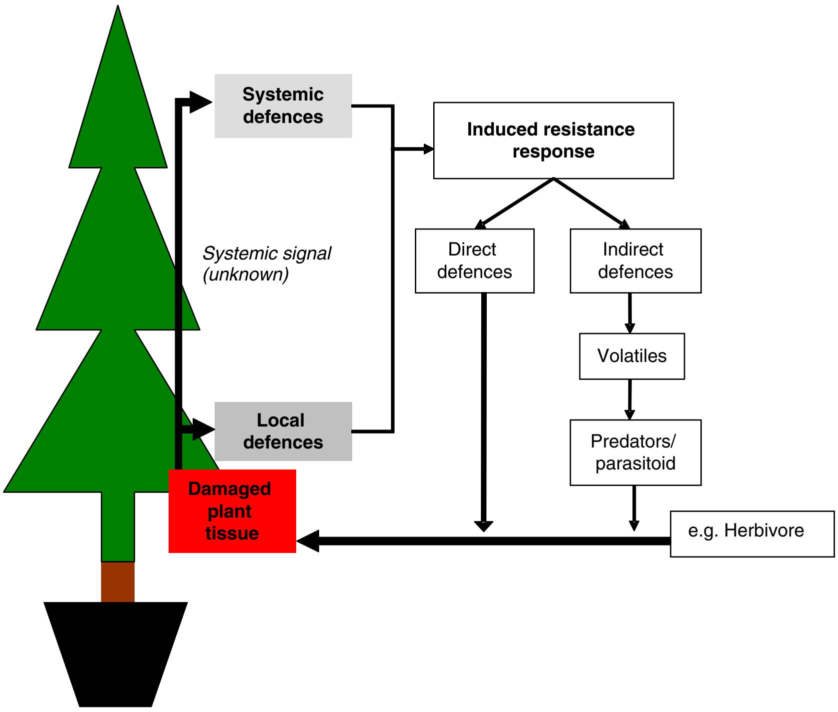

Fig. 2 Overview of local and systemic defences against biotic damaging agents, in this case an insect herbivore in conifers.

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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References

Agrawal AA, Tuzun S, Bent E. 1999. *Induced plant defenses against pathogens and herbivores: biochemistry, ecology, and agriculture*. St Paul, MN, USA: American Phytopathological Society Press, 403.

Alborn HT, Turlings TCJ, Jones TH, Stenhagen G, Loughrin JH, Tumlinson JH. 1997. An elicitor of plant volatiles from beet armyworm oral secretion. *Science* **276**: 945–949.

Anonymous. 2009. Recognized families of pathogenesis-related proteins, accessed 4 August 2009, <http://www.bio.uu.nl/~fytopath/PR-families.htm>.

Arnold AE, Mejia LC, Kyllo D, Rojas EI, Maynard Z, Robbins N, Herre EA. 2003. Fungal endophytes limit pathogen damage in a tropical tree. *Proceedings of the National Academy of Sciences, USA* **100**: 15649–15654.

Attaran E, Zeier TE, Griebel T, Zeierb J. 2009. Methyl salicylate production and jasmonate signaling are not essential for systemic acquired resistance in arabidopsis. *Plant Cell* **21**: 954–971.

Azaiez A, Boyle B, Levée V, Séguin A. 2009. Transcriptome profiling in hybrid poplar following interactions with *Melampsora* rust fungi. *Molecular Plant-Microbe Interactions* **22**: 190–200.

Babst BA, Ferrieri RA, Gray DW, Lerdau M, Schlyer DJ, Schueller M, Thorpe MR, Orians CM. 2005. Jasmonic acid induces rapid changes in carbon transport and partitioning in *Populus. New Phytologist* **167**: 63–72.

Babst BA, Ferrieri RA, Thorpe MR, Orians CM. 2008. *Lymantria dispar* herbivory induces rapid changes in carbon transport and partitioning in *Populus nigra. Entomologia Experimentalis et Applicata* **128**: 117–125.

Balbi V, Devoto A. 2008. Jasmonate signalling network in *Arabidopsis thaliana*: crucial regulatory nodes and new physiological scenarios. *New Phytologist* **177**: 301–318.

Baldwin IT, Schultz JC. 1983. Rapid changes in tree leaf chemistry induced by damage: evidence for communication between plants. *Science* **221**: 277–279.

Barakat A, DiLoretol DS, Zhang Y, Smith C, Baier K, Powell WA, Wheeler N, Sederoff R, Carlson JE. 2009. Comparison of the transcriptomes of American chestnut (*Castanea dentata*) and Chinese chestnut (*Castanea mollissima*) in response to the chestnut blight infection. *BMC Plant Biology* **9**: 51–61.

Barbehenn RV, Jones CP, Yip L, Tran L, Constabel CP. 2007. Limited impact of elevated levels of polyphenol oxidase on tree-feedingcaterpillars: assessing individual plant defenses with transgenic poplar. *Oecologia* **154**: 129–140.

Barbehenn RV, Jaros A, Lee G, Mozola C, Weir Q, Salminen JP. 2009. Tree resistance to *Lymantria dispar* caterpillars: importance and limitations of foliar tannin composition. *Oecologia* **159**: 777–788.

Bernards MA, Bastrup-Spohr L. 2008. Phenylpropanoid metabolism induced by wounding and insect herbivory. In: Schaller A, ed. *Induced plant resistance to herbivory*. New York: Springer, 189–213.

Björkman C, Dalin P, Ahrné K. 2008. Leaf trichome responses to herbivory in willows: induction, relaxation and costs. *New Phytologist* **179**: 176–184.

Blodgett JT, Eyles A, Bonello P. 2007. Organ-dependent induction of systemic resistance and systemic susceptibility in *Pinus nigra* inoculated with *Sphaeropsis sapinea* and *Diplodia scrobiculata. Tree Physiology* **27**: 511–517.

Bolton MD. 2009. Primary Metabolism and Plant Defense – Fuel for the Fire. *Molecular Plant-Microbe Interactions* **22**: 487–497.

Bonello P, Blodgett JT. 2003. *Pinus nigra–Sphaeropsis sapinea* as a model pathosystem to investigate local and systemic effects of fungal infection of pines. *Physiological and Molecular Plant Pathology* **63**: 249–261.

Bonello P, Pearce RB, Watt F, Grime GW. 1991. An induced papilla response in primary roots of Scots pine challenged in vitro with *Cylindrocarpon destructans. Physiological and Molecular Plant Pathology* **39**: 213–228.

Bonello P, Heller W, Sandermann H Jr. 1993. Ozone effects on root disease susceptibility and defence responses in mycorrhizal and non-mycorrhizal seedlings of Scots pine *Pinus sylvestris* L. *New Phytologist* **124**: 653–663.

Bonello P, Gordon TR, Storer AJ. 2001. Systemic induced resistance in Monterey pine. *Forest Pathology* **31**: 99–106.

Bonello P, Storer AJ, Gordon TR, Wood DL, Heller W. 2003. Systemic effects of *Heterobasidion annosum* on ferulic acid glucoside and lignin of pre-symptomatic ponderosa pine phloem, and potential effects on bark beetle-associated fungi. *Journal of Chemical Ecology* **29**: 1167–87.

Bonello P, Gordon TR, Herms DA, Wood DL, Erbilgin N. 2006. Nature and ecological implications of pathogen-induced systemic resistance in conifers: a novel hypothesis. *Physiological and Molecular Plant Pathology* **68**: 95–104.

Bonello P, Capretti P, Luchi N, Martini V, Michelozzi M. 2008. Systemic effects of *Heterobasidion annosum s.s.* infection on severity of *Diplodia pinea* tip blight and terpenoid metabolism in Italian stone pine *Pinus pinea. Tree Physiology* **28**: 1653–1660.

Bostock RM. 2005. Signal crosstalk and induced resistance: straddling the line between cost and benefit. *Annual Review of Phytopathology* **43**: 545–580.

Broekaert WF, Delaure SL, De Bolle MFC, Cammuel BPA. 2006. The role of ethylene in host–pathogn interactions. *Annual Review of Phytopathology* **44**: 393–416.

Chassot C, Buchala A, Schoonbeek H, Metraux JP, Lamotte O. 2008. Wounding of Arabidopsis leaves causes a powerful but transient protection against *Botrytis infection. Plant Journal* **55**: 555–567.

Chester KS. 1933. The problem of acquired physiological immunity in plants. *Quarterly Review of Biology* **8**: 129–154, 275–324.

Christiansen E, Krokene P, Berryman AA, Franceschi VR, Krekling T, Lieutier F, Lonneborg A, Solheim H. 1999. Mechanical injury and fungal infection induce acquired resistance in Norway spruce. *Tree Physiology* **19**: 355–381.

Coleman HD, Park JY, Nair R, Chapple C, Mansfield SD. 2008. RNAi-mediated suppression of p-coumaroyl-CoA 3′-hydroxylase in hybrid poplar impacts lignin deposition and soluble secondary metabolism. *Proceedings of the National Academy of Sciences, USA* **105**: 4501–4506.

Conrath U, Beckers GJM, Flors V, García-Agustín P, Jakab G, Mauch F. 2006. Priming: getting ready for battle. *Molecular Plant-Microbe Interactactions* **19**: 1062–1071.

Cooper WR, Rieske LK. 2008. Differential responses in American (*Castanea dentata* Marshall) and Chinese (*C. mollissima* Blume) chestnut (Falales: Fagaceae) to foliar application of jasmonic acid. *Chemoecology* **18**: 121–127.

Cui J, Bahrami AK, Pringle EG, Hernandez-Guzman G, Bender CL, Pierce NE, Ausube FM. 2005. *Pseudomonas syringae* manipulates systemic plant defenses against pathogens and herbivores. *Proceedings of the National Academy of Sciences, USA* **102**: 1791–1796.

von Dahl CC, Baldwin IT. 2007. Deciphering the role of ethylene in plant-herbivore interactions. *Journal of Plant Growth Regulators* **26**: 201–209.

Develey-Rivière MP, Galiana E. 2007. Resistance to pathogens and host developmental stage: a multifaceted relationship within the plant kingdom. *New Phytologist* 175: 405–416.

Dicke M, Van Loon JJA, Soler R. 2009. Chemical complexity of volatiles from plants induced by multiple attack. *Nature Chemical Biology* 5: 317–324.

Dolch R, Tscharntke T. 2000. Defoliation of alders (*Alnus glutinosa*) affects herbivory by leaf beetles on undamaged neighbours. *Oecologia* 125: 504–511.

Dukes JS, Pontius J, Orwig D, Garnas JR, Rodgers VL, Brazee N, Cooke B, Theoharides KA, Stange EE, Harrington R *et al.* 2009. Responses of insect pests, pathogens, and invasive plant species to climate change in the forests of northeastern North America: what can we predict? *Canadian Journal of Forest Research* 39: 231–248.

Dunn JP, Potter DA, Kimmerer TW. 1990. Carbohydrate reserves, radial growth, and mechanisms of resistance of oak trees to phloem-boring insects. *Oecologia* 83: 458–468.

Durrant WE, Dong X. 2004. Systemic acquired resistance. *Annual Review of Phytopathology* 42: 185–209.

Enebak SA, Carey WA. 2000. Evidence for induced systemic protection to fusiform rust in loblolly pine by plant growth-promoting rhizobacteria. *Plant Disease* 84: 306–308.

Erbilgin N, Raffa K. 2000. Effects of host tree species on attractiveness of tunnelling pine engravers, Ips pini, to conspecifics and insect predators. *Journal of Chemical Ecology* 26: 823–840.

Erbilgin N, Krokene P, Christiansen E, Zeneli G, Gershenzon J. 2006. Exogenous application of methyl jasmonate elicits defenses in Norway spruce (*Picea abies*) and reduces host colonization by the bark beetle *Ips typographus. Oecologia* 148: 426–436.

van Etten HD, Mansfield JW, Bailey JA, Farmer E. 1994. Two classes of plant antibiotics: phytoalexins versus "phytoanticipins". *Plant Cell* 6: 1191–1192.

Eyles A, Davies NW, Yuan ZQ, Mohammed C. 2003. Host responses to natural infection by *Cytonaema* sp. in the aerial bark of *Eucalyptus globulus. Forest Pathology* 33: 317–331.

Eyles A, Chorbadjian R, Wallis C, Hansen R, Cipollini D, Herms D, Bonello P. 2007. Cross-induction of systemic induced resistance between an insect and a fungal pathogen in Austrian pine over a fertility gradient. *Oecologia* 153: 365–374.

Eyles A, Beadle C, Barry K, Francis A, Glen M, Mohammed C. 2008. Management of fungal root-rot pathogens in tropical acacia plantations. *Forest Pathology* 38: 332–355.

Facchini PJ. 2001. Alkaloid biosynthesis in plants: biochemistry, cell biology, molecular regulation, and metabolic engineering applications. *Annual Review of Plant Physiology and Plant Molecular Biology* 52: 29–66.

Felton GW. 2008. Caterpillar secretions and induced plant responses. In: Schaller A, ed. *Induced plant resistance to herbivory.* New York: Springer, 369–389.

Felton GW, Donato KK, Broadway RM, Duffey SS. 1992. Impact of oxidized plant phenolics on the nutritional quality of dietary protein to a noctuid herbivore, *Spodoptera exigua. Journal of Insect Physiology* 38: 277–285.

Franceschi VR, Krokene P, Christiansen E, Krekling T. 2005. Anatomical and chemical defenses of conifer bark against bark beetles and other pests. *New Phytologist* 167: 353–375.

Frost CJ, Hunter MD. 2008. Herbivore-induced shifts in carbon and nitrogen allocation in red oak seedlings. *New Phytologist* 178: 835–845.

Frost CJ, Appel HM, Carlson JE, De Moraes CM, Mescher MC, Schultz JC. 2007. Within-plant signalling via volatiles overcomes vascular constraints on systemic signaling and primes responses against herbivores. *Ecological Letters* 10: 490–498.

Frost CJ, Mescher MC, Dervinis C, Davis JM, Carlson JE, De Moraes CM. 2008. Priming defense genes and metabolites in hybrid poplar by the green leaf volatile *cis*-3-hexenyl acetate. *New Phytologist* 180: 722–734.

Ganley RJ, Sniezko RA, Newcombe G. 2008. Endophyte-mediated resistance against white pine blister rust in *Pinus monticola. Forest Ecology and Management* 255: 2751–2760.

Gatehouse JA. 2002. Plant resistance towards insect herbivores: a dynamic interaction. *New Phytologist* 156: 145–169.

Goellner K, Conrath U. 2008. Priming: it's all the world to induced disease resistance. *European Journal of Plant Pathology* 121: 233–242.

Gordon TR. 2006. Pitch canker disease of pines. *Phytopathology* 96: 657–659.

Gordon TR, Storer AJ, Wood DL. 2001. The pitch canker epidemic in California. *Plant Disease* 85: 1128–1139.

Gould N, Reglinski T, Spiers M, Taylor JT. 2008. Physiological trade-offs associated with methyl jasmonate – induced resistance in *Pinus radiata. Canadian Journal of Forest Research* 38: 677–684.

Grant M, Lamb C. 2006. Systemic immunity. *Current Opinions in Plant Biology* 9: 414–420.

Green TR, Ryan CA. 1972. Wound-induced proteinase inhibitor in plant leaves: a possible defense mechanism against insects. *Science* 175: 776–777.

Hammerschmidt R. 1999. Phytoalexins: what have we learned after 60 years? *Annual Review of Phytopathlogy* 37: 285–306.

Hammerschmidt R. 2006. Host–pathogen interaction in conifers: complicated systems yield interesting possibilities for research. *Physiological and Molecular Plant Pathology* 68: 93–94.

Hammerschmidt R. 2007. Introduction: definitions and some history. In: Walters D, Newton A, Lyon G, eds. *Induced resistance for plant disease control: a sustainable approach to crop protection.* Oxford, UK: Blackwell Publishing, 1–8.

Hammerschmidt R, Nicholson RL. 1999. A survey of plant defense responses to pathogens. In: Agrawal AA, Tuzun S, Bent E, eds. *Induced plant defenses against pathogens and herbivores.* St Paul, MN, USA: American Phytopathological Society Press, 55–71.

Haukioja E. 1982. Inducible defences of white birch to a geometrid defoliator, *Epirrita autumnata.* In: Visser JH, Minks AK, eds. *Proceedings of the 5th international symposium on insect–plant relationships.* Wageningen, the Netherlands: Pudoc, 199–203.

Haukioja E, Koricheva J. 2000. Tolerance to herbivory in woody vs. herbaceous plants. *Evolutionary Ecology* 14: 551–562.

Haukioja E, Neuvonen S. 1985. Induced long-term resistance of birch foliage against defoliators: defensive or incidental? *Ecology* 66: 1303–1308.

Heijari J, Nerg AM, Kainulainen P, Viiri H, Vuorinen M, Holopainen JK. 2005. Application of methyl jasmonate reduces growth but increases chemical defense and resistance against *Hylobius abietis* in Scots pine seedlings. *Entomologia Experimentalis et Applicata* 115: 117–124.

Heijari J, Nerg AM, Kainulainen P, Viiri H, Vuorinen M, Holopainen JK. 2008. Long-term effects of exogenous methyl jasmonate application on Scots pine (*Pinus sylvestris*) needle chemical defence and diprionid sawfly performance. *Entomologia Experimentalis et Applicata* 128: 162–171.

Heil M. 2008. Indirect defence via tritrophic interactions. *New Phytologist* 178: 41–61.

Henery ML, Wallis IR, Stone C, Foley WJ. 2008. Methyl jasmonate does not induce changes in *Eucalyptus grandis* leaves that alter the effect of constitutive defences on larvae of a specialist herbivore. *Oecologia* 156: 847–859.

Hilker M, Kobs C, Varma M, Schrank K. 2002. Insect egg deposition induces *Pinus sylvestris* to attract egg parasitoids. *Journal of Experimental Biology* 205: 455–461.

Hossain MM, Sultana F, Kubota M, Koyama H, Hyakumachi M. 2008. Systemic resistance to bacterial leaf speck pathogen in *Arabidopsis* *thaliana* induced by the culture filtrate of a plant growth-promoting fungus (PGPF) *Phoma* sp GS8-1. *Journal of General Plant Pathology* **74**: 213–221.

Howe GA, Jander G. 2008. Plant immunity to insect herbivores. *Annual Review of Plant Biology* **59**: 41–66.

Huber DPW, Philippe RN, Madilao LL, Sturrock RN, Bohlmann J. 2005. Changes in anatomy and terpene chemistry in roots of Douglas-fir seedlings following treatment with methyl jasmonate. *Tree Physiology* **25**: 1075–1083.

Huckelhoven R. 2007. Cell wall – associated mechanisms of disease resistance and susceptibility. *Annual Review of Phytopathology* **45**: 101–127.

Hudgins JW, Franceschi V. 2004. Methyl jasmonate-induced ethylene production is responsible for conifer phloem defense responses and reprogramming of stem cambial zone for traumatic resin duct formation. *Plant Physiology* **135**: 2134–2149.

Hudgins JW, Christiansen E, Franceschi VR. 2003. Methyl jasmonate induces changes mimicking anatomical defenses in diverse members of the *Pinaceae*. *Tree Physiology* **23**: 361–371.

Hudgins JW, Christiansen E, Franceschi VR. 2004. Induction of anatomically based defense responses in stems of diverse conifers by methyl jasmonate: a phylogenetic perspective. *Tree Physiology* **24**: 251–264.

Jones JD, Dangl JL. 2006. The plant immune system. *Nature* **444**: 323–329.

Jongsma MA, Beekwilder J. 2008. Plant protease inhibitors: functional evolution for defence. In: Schaller A, ed. *Induced plant resistance to herbivory*. New York: Springer, 235–253.

Karban R, Baldwin IT. 1997. *Induced responses to herbivory*. Chicago, IL, USA: University of Chicago Press, 320.

Karban R, Baldwin IT, Baxter KJ, Laue G, Felton GW. 2000. Communication between plants: induced resistance in wild tobacco plants following clipping of neighboring sagebrush. *Oecologia* **125**: 66–71.

Keeling CI, Bohlmann J. 2006. Genes, enzymes and chemicals of terpenoid diversity in the constitutive and induced defence of conifers against insects and pathogens. *New Phytologist* **170**: 657–675.

Kessler A, Baldwin IT. 2002. Plant responses to insect herbivory: the emerging molecular analysis. *Annual Review of Plant Biology* **53**: 299–328.

Kiraly L, Barnaz B, Kiralyz Z. 2007. Plant resistance to pathogen infection: forms and mechanisms of innate and acquired resistance. *Journal of Phytopathology* **155**: 385–396.

Kogel KH, Franken P, Huckelhoven R. 2006. Endophyte or parasite – what decides? *Current Opinions in Plant Biology* **9**: 358–363.

Kontunen-Soppela S, Ossipov V, Ossipova S, Oksanen E. 2007. Application of metabolomics to genotype and phenotype discrimination of birch trees grown in a long-term open-field experiment. *Global Change Biology* **13**: 1053–1067.

Kozlowski G, Buchala A, Metraux JP. 1999. Methyl jasmonate protects Norway spruce (*Picea abies* L. Karst.) seedlings against Pythium ultimum Trow. *Physiological and Molecular Plant Pathology* **55**: 53–58.

Krause SC, Raffa KF. 1992. Comparison of insect, fungal, and mechanically induced defoliation of larch – effects on plant productivity and subsequent host susceptibility. *Oecologia* **90**: 411–416.

Krause SC, Raffa KF. 1995. Defoliation intensity and larval age interact to affect sawfly performance on previously injured *Pinus resinosa*. *Oecologia* **102**: 24–30.

Krokene P, Nagy NE, Solheim H. 2008. Methyl jasmonate and oxalic acid treatment of Norway spruce: anatomically based defense responses and increased resistance against fungal infection. *Tree Physiology* **28**: 29–35.

Kuc J. 1983. Induced systemic resistance in plants to diseases caused by fungi and bacteria. In: Bailey JA, Deverall BJ, eds. *The dynamics of host defence*. Sydney, Australia: Academic Press, 191–221.

Leadbeater A, Staub T. 2007. Exploitation of induced resistance: a commercial perspective. In: Walters D, Newton A, Lyon G, eds. *Induced resistance for plant defence: a sustainable approach to crop protection*. Oxford, UK: Blackwell Publishing, 229–241.

Lehr NA, Schrey SD, Hampp R, Tarkka MT. 2008. Root inoculation with a forest soil streptomycete leads to locally and systemically increased resistance against phytopathogens in Norway spruce. *New Phytologist* **177**: 965–976.

Liu JJ, Ekramoddoullah AKM, Yu XS. 2003. Differential expression of multiple PR10 proteins in western white pine following wounding, fungal infection and cold-hardening. *Physiology Plantarum* **119**: 544–553.

van Loon LC. 2007. Plant responses to plant growth-promoting rhizobacteria. *European Journal of Plant Pathology* **119**: 243–254.

van Loon LC, Bakker PAHM, Pieterse CMJ. 1998. Systemic resistance induced by rhizosphere bacteria. *Annual Review of Phytopathology* **36**: 453–483.

van Loon LC, Rep M, Pieterse CMJ. 2006. Significance of inducible defense-related proteins in infected plants. *Annual Review of Phytopathology* **44**: 135–162.

Luchi N, Ma R, Capretti P, Bonello P. 2005. Systemic induction of traumatic resin ducts and resin flow in Austrian pine by wounding and inoculation with *Sphaeropsis sapinea* and *Diplodia scrobiculata*. *Planta* **221**: 75–84.

Lyytikainen P. 1994. Effects of natural and artificial defoliations on sawfly performance and foliar chemistry of Scots pine saplings. *Annales Zoologici Fennici* **31**: 307–318.

Major IT, Constabel CP. 2006. Molecular analysis of poplar defense against herbivory. Comparison of wound- and insect elicitor induced gene expression. *New Phytologist* **172**: 617–635.

Major IT, Constabel CP. 2007. Shoot–root defense signaling and activation of root defense by leaf damage in poplar. *Canadian Journal of Botany* **85**: 1171–1181.

Mattiacci L, Dicke M, Posthumus MA. 1995. Beta-glucosidase – an elicitor of herbivore-induced plant odor that attracts host-searching parasitic wasps. *Proceedings of the National Academy of Sciences, USA* **92**: 2036–2040.

Mauch-Mani B, Mauch F. 2005. The role of abscisic acid in plant–pathogen interactions. *Current Opinions in Plant Biology* **8**: 409–414.

McComb JA, O'Brien P, Calver M, Staskowski P, Jardine N, Eshraghi L, Ellery J, Gilovitz J, Scott P, O'Brien J *et al.* 2008. Research into natural and induced resistance in Australian native vegetation of *Phytophthora cinnamomi* and innovative methods to contain and/or eradicate within localised incursions in areas of high biodiversity in Australia. Enhancing the efficacy of phosphite with the addition/supplementation of other chemicals such as those known to be involved in resistance. Prepared by the Centre for Phytophthora Science and Management for the Australian Government Department of the Environment, Water, Heritage and the Arts. 92 p.

McNee WR, Bonello P, Wood DL, Storer AJ, Gordon TR. 2003. Feeding response of *Ips paraconfusus* to phloem and phloem metabolites of *Heterobasidion annosum*-inoculated ponderosa pine, *Pinus ponderosa*. *Journal of Chemical Ecology* **29**: 1183–1202.

Miller B, Madilao LL, Ralph S, Bohlmann J. 2005. Insect-induced conifer defense. White pine weevil and methyl jasmonate induce traumatic resinosis, de novo formed volatile emissions, and accumulation of terpenoid synthase and putative octadecanoid pathway transcripts in Sitka spruce. *Plant Physiology* **137**: 369–382.

Miranda M, Ralph SG, Mellway R, Heath M, Bohlmann J, Constabel P. 2007. The transcriptional response of hybrid poplar (*Populus trichocarpa* × *P-deltoides*) to infection by *Melampsora medusae* leaf rust involves induction of flavonoid pathway genes leading to the accumulation of proanthocyanidins. *Molecular Plant-Microbe Interactions* **20**: 816–831.

Mithofer A, Wanner G, Boland W. 2005. Effects of feeding *Spodoptera littoralis* on lima bean leaves. II. Continuous mechanical wounding resembling insect feeding is sufficient to elicit herbivory-related volatile emission. *Plant Physiology* **137**: 1160–1168.

Moret A, Munoz Z. 2007. Control of *Diplodid pinea and D-scrobiculata* in *Pinus halepensis* by 5-chloro-salicylic acid. *Phytopathologia Mediterranea* **46**: 150–156.

Mumm R, Hilker M. 2006. Direct and indirect chemical defence of pine against folivorous insects. *Trends in Plant Science* **11**: 351–358.

Mumm R, Schrank K, Wegener R, Schulz S, Hilker M. 2003. Chemical analysis of volatiles emitted by *Pinus sylvestris* after induction by insect oviposition. *Journal of Chemical Ecology* **29**: 1235–1252.

Musser RO, Farmer E, Peiffer M, Williams SA, Felton GW. 2006. Ablation of caterpillar labial salivary glands: technique for determining the role of saliva in insect–plant interactions. *Journal of Chemical Ecology* **32**: 981–992.

Nagy NE, Fossdal CG, Krokene P, Krekling T, Lønneborg A, Solheim H. 2004. Induced responses to pathogen infection in Norway spruce phloem: changes in polyphenolic parenchyma cells, chalcone synthase transcript levels and peroxidase activity. *Tree Physiology* **24**: 505–515.

Ollerstam O, Larsson S. 2003. Salicylic acid mediates resistance in the willow *Salix viminalis* against the gall midge *Dasineura marginemtorquens*. *Journal of Chemical Ecology* **29**: 63–174.

van Oosten VR, Bodenhausen N, Reymond P, Van Pelt JA, Van Loon LC, Dicke M, Pieterse CMJ. 2008. Differential Effectiveness of Microbially Induced Resistance Against Herbivorous Insects in *Arabidopsis*. *Molecular Plant-Microbe Interactions* **21**: 919–930.

Park SW, Kaimoyo E, Kumar D, Mosher S, Klessig DF. 2007. Methyl salicylate is a critical mobile signal for plant systemic acquired resistance. *Science* **318**: 113–116.

Pearce RB. 1996. Antimicrobial defences in the wood of living trees. *New Phytologist* **132**: 203–233.

Pedranzani H, Sierra-de-Grado R, Vigliocco A, Miersch O, Abdala G. 2008. Cold and water stresses produce changes in endogenous jasmonates in two populations of *Pinus pinaster* Ait. *Plant Growth Regulator* **52**: 111–116.

Philippe RN, Bohlmann J. 2007. Poplar defense against insect herbivores. *Canadian Journal of Botany* **85**: 1111–1126.

Pieterse CMJ, Leon-Reyes A, Van der Ent A, Van Wees SCM. 2009. Networking by small-molecule hormones in plant immunity. *Nature Chemical Biology* **5**: 308–316.

Piggott N, Ekramoddoullah AKM, Liu JJ, Yu XS. 2004. Gene cloning of a thaumatin-like (PR-5) protein of western white pine (*Pinus monticola* D. Don) and expression studies of members of the PR-5 group. *Physiological and Molecular Plant Pathology* **64**: 1–8.

Quentin A, Pinkard EA, Beadle C, Wardlaw TJ, O'Grady AP, Paterson S, Mohammed CL. 2010. Do artificial and natural defoliation have similar effects on physiology of *Eucalyptus globulus* Labill. seedlings? *Annals of Forest Science* **67**: 203–211.

Ralph S, Yueh H, Friedmann MF, Aeschliman D, Zeznik JA, Nelson CC, ButterWeld YSN, Kirkpatrick R, Liu J, Jones SJM *et al.* 2006. Conifer defense against insects: microarray gene expression profiling of Sitka spruce (Picea sitchensis) induced by mechanical wounding or feeding by spruce budworm (Choristoneura occidentalis) or white pine weevil (Pissodes strobe) reveals large-scale changes of the host transcriptome. *Plant, Cell & Environment* **29**: 545–570.

Rapley L, Allen GR, Potts BR, Davies NW. 2008. Constitutive or induced defences – how does *Eucalyptus globulus* defend itself from larval feeding? *Chemoecology* **17**: 235–243.

Reglinski T, Taylor JT, Dick MA. 2004. Chitosan induces resistance to pitch canker in *Pinus radiata*. *New Zeal Journal of Forest Science* **34**: 49–58.

Rhoades DF. 1983. Responses of alder and willdow to attack by tent caterpillars and webworms: evidence for pheromonal sensitivity of willows. In: Hedin PA, ed. *Plant resistance to insects*. Washington, DC, USA: American Chemical Society, 55–68.

Rinaldi C, Kohler A, Frey P, Duchaussoy F, Ningre N, Couloux A, Wincker P, Le Thiec D, Fluch S, Martin F *et al.* 2007. Transcript profiling of poplar leaves upon infection with compatible and incompatible strains of the foliar rust *Melampsora larici-populina*. *Plant Physiology* **144**: 347–366.

Robinson RM, Jensen GD, Morrison DJ. 2004. Necrophylactic periderm formation in the roots of western larch and Douglas fir trees infected with *Armillaria ostoyae*. I. The response to abiotic wounding in non-infected roots. *Forest Pathology* **34**: 105–118.

Robinson AR, Ukrainetz NK, Kang KY, Mansfield SD. 2007. Metabolite profiling of Douglas-fir (*Pseudotsuga menziesii*) field trials reveals strong environmental and weak genetic variation. *New Phytologist* **174**: 762–773.

Roden DB, Mattson WJ. 2008. Rapid induced resistance and host species effects on gypsy moth, (*Lymantria dispar* L.): Implications for outbreaks on three tree species in the boreal forest. *Forest Ecology and Management* **255**: 1868–1873.

Rodriguez R, Redman R. 2008. More than 400 million years of evolution and some plants still can't make it on their own: plant stress tolerance via fungal symbiosis. *Journal of Experimental Botany* **59**: 1109–1114.

Ruuhola T, Yang SY, Ossipov V, Haukioja E. 2008. Foliar oxidases as mediators of the rapidly induced resistance of mountain birch against *Epirrita autumnata*. *Oecologia* **154**: 725–730.

Saravesi K, Markkola A, Rautio P, Roitto M, Tuomi J. 2008. Defoliation causes parallel temporal responses in a host tree and its fungal symbionts. *Oecologia* **156**: 117–123.

Schmelz EA, Carroll MJ, LeClere S, Phipps SM, Meredith J, Chourey PS, Alborn HT, Teal PEA. 2006. Fragments of ATP synthase mediate plant perception of insect attack. *Proceedings of the National Academy of Sciences, USA* **103**: 8894–8899.

Shepherd WP, Sullivan BT, Goyer RA, Klepzig KD. 2005. Electrophysiological and olfactometer responses of two histerid predators to three pine bark beetle pheromones. *Journal of Chemical Ecology* **31**: 1101–1110.

Simon M, Hilker M. 2003. Herbivores and pathogens on willow: do they affect each other? *Agriculture and Forest Entomology* **5**: 275–284.

Smith AH, Gill WM, Pinkard EA, Mohammed CL. 2007. Anatomical and histochemical defence responses induced in juvenile leaves of *Eucalyptus globulus* and *Eucalyptus nitens* by *Mycosphaerella infection*. *Forest Pathology* **37**: 361–373.

Swedjemark G, Karlsson B, Stenlid J. 2007. Exclusion of *Heterobasidion parviporum* from inoculated clones of *Picea abies* and evidence of systemic induced resistance. *Scandinavian Journal of Forest Research* **22**: 110–117.

van Tol RWHM, Van der Sommen ATC, Boff MIC, Van Bezooijen J, Sabelis MW, Smits PH. 2001. Plants protect their roots by alerting the enemies of grubs. *Ecological Letters* **4**: 292–294.

Trewhella KE, Leather SR, Day KR. 1997. Insect induced resistance in Lodgepole pine: effects on two pine feeding insects. *Journal of Applied Entomology* **121**: 129–136.

Tsai CJ, Harding SA, Tschaplinski TJ, Lindroth RL, Yuan YN. 2006. Genome-wide analysis of the structural genes regulating defense phenylpropanoid metabolism in *Populus*. *New Phytologist* **172**: 47–62.

Tscharntke T, Thiessen S, Dolch R, Boland W. 2001. Herbivory, induced resistance, and interplant signal transfer in *Alnus glutinosa*, *Biochemical Systematics*. *Ecology* **29**: 1025–1047.

Tuskan GA, DiFazio S, Jansson S, Bohlmann J, Grigoriev I, Hellsten U, Putnam N, Ralph S, Rombauts S, Salamov A *et al.* 2006. The genome of black cottonwood, *Populus trichocarpa* Torr. & Gray. *Science* **313**: 1596–1604.

Utsumi S, Ohgushi T. 2008. Host plant variation in plant-mediated indirect effects: moth boring-induced susceptibility of willows to a specialist leaf beetle. *Ecological Entomology* **33**: 250–260.

Vallad GE, Goodman RM. 2004. Systemic acquired resistance and induced systemic resistance in conventional agriculture. *Crop Science* **44**: 1920–1934.

Vinale F, Sivasithamparam K, Ghisalberti EL, Marra R, Woo SL, Lorito M. 2008. *Trichoderma*-plant-pathogen interactions. *Soil Biology & Biochemistry* **40**: 1–10.

Volt AC, Klessig DF, Park SW. 2009. Systemic acquired resistance: the elusive signal(s). *Current Opinion in Plant Biology* **11**: 436–442.

Wallis C, Eyles A, Chorbadjian R, Riedel K, Schwartz S, Hansen R, Cipollini D, Herms D, Bonello P. 2008. Systemic induction of phloem secondary metabolism and its relationship to resistance to a canker pathogen in Austrian pine. *New Phytologist* **177**: 767–778.

Walters D. 2009. Are plants in the field already induced? Implications for practical disease control. *Crop Protection* **28**: 459–465.

Wang J, Constabel CP. 2004. Polyphenol oxidase overexpression in transgenic *Populus* enhances resistance to herbivory by forest tent caterpillar (*Malacosoma disstria*). *Planta* **220**: 87–96.

Wang D, Eyles A, Mandich D, Bonello P. 2006. Systemic aspects of host–pathogen interactions in Austrian pine (*Pinus nigra*): a proteomics approach. *Physiological and Molecular Plant Pathology* **68**: 149–157.

van Wees SCM, Van der Ent S, Pieterse CMJ. 2008. Plant immune responses triggered by beneficial microbes. *Current Opinion in Plant Biology* **11**: 443–448.

Witzell J, Martin JA. 2008. Phenolic metabolites in the resistance of northern forest trees to pathogens – past experiences and future prospects. *Canadian Journal of Forest Research* **38**: 2711–2727.

Zeneli G, Krokene P, Christiansen E, Krekling T, Gershenzon J. 2006. Methyl jasmonate treatment of mature Norway spruce (*Picea abies*) trees increases the accumulation of terpenoid resin components and protects against infection by *Ceratocystis polonica*, a bark beetle-associated fungus. *Tree Physiology* **26**: 977–988.