Plant-Mediated Systemic Interactions Between Pathogens, Parasitic Nematodes, and Herbivores Above- and Belowground

Arjen Biere[1] and Aska Goverse[2]

[1]Department of Terrestrial Ecology, Netherlands Institute of Ecology, NIOO-KNAW, 6708 PB Wageningen, The Netherlands; email: a.biere@nioo.knaw.nl

[2]Lab of Nematology, Department of Plant Sciences, Wageningen University, 6700 PB Wageningen, The Netherlands

Annu. Rev. Phytopathol. 2016. 54:499–527

First published online as a Review in Advance on June 17, 2016

The *Annual Review of Phytopathology* is online at phyto.annualreviews.org

This article's doi: 10.1146/annurev-phyto-080615-100245

Keywords

root-shoot interactions, plant defense, hormone signaling pathways, defense suppression, defense loop

Abstract

Plants are important mediators of interactions between aboveground (AG) and belowground (BG) pathogens, arthropod herbivores, and nematodes (phytophages). We highlight recent progress in our understanding of within- and cross-compartment plant responses to these groups of phytophages in terms of altered resource dynamics and defense signaling and activation. We review studies documenting the outcome of cross-compartment interactions between these phytophage groups and show patterns of cross-compartment facilitation as well as cross-compartment induced resistance. Studies involving soilborne pathogens and foliar nematodes are scant. We further highlight the important role of defense signaling loops between shoots and roots to activate a full resistance complement. Moreover, manipulation of such loops by phytophages affects systemic interactions with other plant feeders. Finally, cross-compartment–induced changes in root defenses and root exudates extend systemic defense loops into the rhizosphere, enhancing or reducing recruitment of microbes that induce systemic resistance but also affecting interactions with root-feeding phytophages.

INTRODUCTION

Plant-Mediated Interactions Between Plant Feeders Have Community-Wide Implications

Plants are a prime source of carbon and/or nutrients for a wealth of different organisms, including plant pathogens, herbivorous arthropods, and plant-feeding nematodes (phytophages). Interactions between plants and these consumers induce changes in the plant's phenotype that can subsequently affect the attraction, behavior, performance, and abundance of any of the other biota sharing the host plant, either simultaneously or later in time. Such indirect, plant-mediated interactions have important consequences for both the plant and its associated community. From the perspective of the individual plant, interactions between its consumers are important, as they can result in synergistic or antagonistic effects on plant fitness (51). From the perspective of the consumers, such interactions are important because the presence of other consumers affects their success in colonizing and exploiting the plant. For instance, early season herbivory modulates plant defense responses to later arriving herbivores, thereby significantly affecting their population dynamics (129). Recent studies have shown that indirect, plant-mediated interactions can in fact be an important force in structuring the plant-associated community connected by this indirect interaction web (122). These effects are not restricted to within-kingdom interactions. For example, there is substantial evidence that plant pathogens can structure plant-associated insect communities (151), indicating that community-wide effects of plant-mediated interactions operate at the cross-kingdom level.

Plant-Mediated Interactions Between Above- and Belowground Phytophages

Plant-mediated indirect interactions are not restricted to organisms feeding within the same plant compartment (roots or shoots). Owing to the tight physiological integration of roots and shoots and the systemic (within- as well as cross-compartment) nature of some of the plant responses to attack, plants mediate interactions between aboveground (AG) and belowground (BG) phytophages. Since the initial recognition of the important role of plants as mediators of interactions between AG and BG communities more than two decades ago, the research field studying AG-BG interactions has seen enormous progress in understanding both the underlying mechanisms and the ecological implications (e.g., 13, 106, 161, 165). Still, there are many gaps in our understanding.

First, recent molecular and biochemical studies have provided revolutionary insight into the mechanisms underlying local and systemic plant responses to different types of plant invaders as well as into how plant invaders subsequently modulate these responses for their own benefit. However, most of our knowledge on systemic responses is based on systemic responses observed in distal parts of the same compartment in which the response was induced, i.e., within roots or shoots (within compartment). These responses can substantially differ from the systemic responses in the other compartment (cross-compartment) (e.g., 7). However, although the latter are the most relevant to understand AG-BG interactions, they are also the least studied.

Second, mechanisms underlying AG-BG interactions are diverse, including alterations of primary metabolism, resource allocation, hydraulics, source-sink relationships, morphology, and local and systemic induction of defenses. The mechanisms considered as dominant explanations for AG-BG interactions have shifted over time. Whereas early studies of AG-BG interactions between herbivores tended to focus on explanations based on resource alterations (e.g., 106), the advent of the molecular era resulted in a stronger emphasis on explanations based on specificity of induction and specificity of response to systemically induced defenses (e.g., 144). Studies simultaneously considering resource- and defense-based mechanisms (e.g., 104) are now emerging and

promise to better explain and predict patterns of outcomes of plant-mediated AG-BG interactions among different types of phytophages.

Third, there are strong indications that defense responses initiated by roots can substantially differ from those initiated by shoots [organ-specific immune responses (8)]. This means that the plant compartment in which the interaction is initiated (root or shoot) is important for the outcome of the interaction, which can contribute to the asymmetry in AG-BG interactions, depending on the locations of the inducer and responder. However, there is a strong research bias toward responses initiated in either the roots or the shoots, depending on the organisms studied, e.g., pathogen, herbivore, or nematode.

The Tight Integration of Roots and Shoots in Plant Responses to Attack

Root and shoot responses to abiotic or biotic stress need to be tightly interconnected because appropriate plant responses in terms of growth, resource allocation, and defense responses depend on a coordinated regulation of the production, mobilization, and transport of carbon and nutrient sources as well as defense compounds at the whole-plant level. This coordinated regulation requires long-distance communication between roots and shoots and vice versa. The plant vascular system provides a unique infrastructure for the transport of RNAs, peptides, phytohormones, and other molecules involved in such long-distance communication, in addition to its role in the transport of water, nutrients, and photosynthates (102).

In recent years, it has become clear that long-distance communication is involved not only in the regulation of growth and interactions with symbionts (121) but also in interactions with phytophages. There are numerous examples of root-to-shoot-to-root (R-S-R) or shoot-to-root-to-shoot (S-R-S) loops necessary to reach a full resistance complement toward phytophages in either roots or shoots. The important role of roots in plant responses to AG herbivores is now well recognized (35, 116). Roots are an important and sometimes exclusive site for the synthesis of toxins and digestibility-reducing proteins or metabolites such as particular cysteine proteases, nicotine, and precursors of pyrrolizidine alkaloids. Studies in *Nicotiana attenuata* (42) and maize (97, 99) have shown that attack by shoot herbivores recruits a root defense signaling pathway that activates production and transport of nicotine and a protease inhibitor, respectively, necessary to launch the full complement of defenses against leaf-chewing as well as sap-sucking herbivores (**Figure 1*a***). Similarly, grafting studies in potato have shown that following shoot infection with the oomycete pathogen *Phytophthora infestans*, roots mobilize signals or metabolites to achieve full resistance to the pathogen in the shoots, mediated by antimicrobial compounds (124) (**Figure 1*a***).

Focus of the Review

Here, we review recent progress in our understanding of local, systemic, within-compartment (AG or BG), and cross-compartment (AG-BG, BG-AG) plant responses to different feeding guilds of pathogens, arthropod herbivores, and nematodes, both in terms of altered resource dynamics and defense signaling and activation. BG-AG interactions involving beneficial soil microbes, such as arbuscular mycorrhizal fungi (AMF) and plant growth–promoting rhizobacteria (PGPR) or plant growth–promoting fungi (PGPF), that trigger induced systemic resistance (ISR) have been well studied and have been the subject of a number of excellent reviews (e.g., 175). By contrast, only a few of the BG-AG interactions involving combinations of nonbeneficial pathogens, arthropods, and nematodes have been well studied. Therefore, our review focuses on interactions among these phytophages. We start with a short recapitulation of the plant immune system and the activation and manipulation of systemic defense within compartments by different phytophages. We then

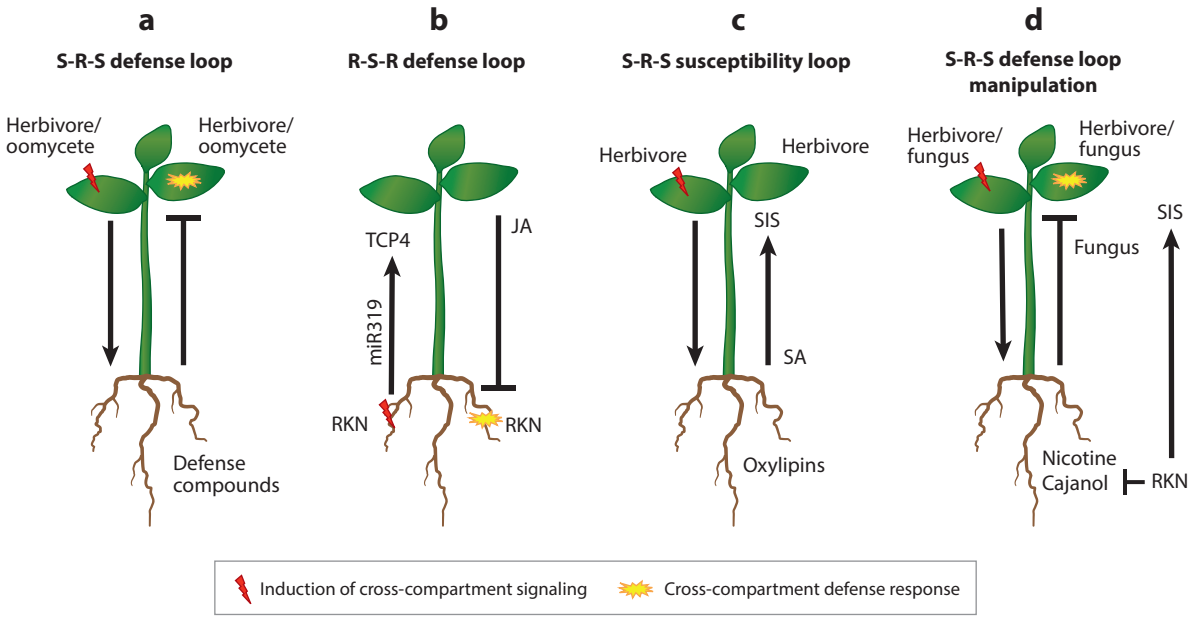

Figure 1

Cross-compartment loops that involve shoot-to-root-to-shoot (S-R-S) or root-to-shoot-to-root (R-S-R) signaling are frequently involved in the activation of a full defense complement in a single compartment. (*a*) Plants attacked by a shoot pathogen (124) or a herbivore (42, 100) recruit root defense signaling pathways to activate synthesis of defense compounds contributing to aboveground defense (S-R-S). (*b*) Plants attacked by a root-knot nematode (RKN) (176) recruit shoot defense signaling pathways to repress root feeding by the RKN (R-S-R). Conversely, R-S-R or S-R-S defense signaling may result in systemic induced susceptibility (SIS). (*c*) An aphid induces biosynthesis of root oxylipins that target a transcriptional activator of salicylic acid (SA) in the shoot, eventually enhancing aphid performance aboveground (115). Manipulation of defense loops by phytophages in one compartment can also affect the performance of phytophages across compartments. (*d*) An RKN suppresses the induction of a root-produced phytoalexin defense metabolite by an aboveground fungal pathogen, resulting in SIS to the pathogen (105). Likewise, RKNs interfere with the induction of root-produced defense chemicals by a shoot herbivore, resulting in systemic induced herbivore susceptibility aboveground (68, 69). Abbreviation: JA, jasmonic acid.

review studies of cross-compartment plant responses activated by exogenous application of plant hormones involved in defense to examine the extent to which such responses are elicited across compartments. Finally, we review the outcome of (bitrophic) plant-mediated systemic interactions among these phytophages when they interact between compartments. This allows us to assess the extent to which patterns of systemic responses and plant-mediated interactions between different phytophage groups observed within compartments can also be observed across compartments. We also emphasize the necessary level of involvement of signaling between roots and shoots to reach full resistance against attack in a single compartment (defense loops) and how such loops are manipulated by phytophages.

INDUCTION AND MANIPULATION OF SYSTEMIC DEFENSE UPON ATTACK

Local Recognition and Initiation of Defense Responses upon Attack

The zigzag model (65) describes a layered plant immune system shaped by a four-stage process of continuous, ongoing coevolution between plants and microbial pathogens. First, plant

pattern recognition receptors (PRRs) recognize conserved microbe-associated molecular patterns (MAMPs). Recognition leads to MAMP-triggered immunity (MTI), which provides broad-spectrum resistance against microbes. Second, microbes evolve effector molecules that allow them to overcome MTI and successfully invade the host [effector-triggered susceptibility (ETS)]. Third, plants evolve specific extra- and intracellular receptors, e.g., resistance (R) proteins, that recognize their cognate effector molecules either directly (receptor-ligand model) or indirectly by sensing effector-induced changes to host virulence targets (guard model) or their mimics (decoy model). Upon recognition, R proteins activate effector-triggered immunity (ETI), which is often stronger than MTI and, when involving pathogens feeding on living tissue, often associated with a hypersensitivity response (HR) characterized by the formation of necrotic lesions that limit microbial proliferation in host tissue. Fourth, pathogens evolve new effectors or new variants of effector molecules that evade recognition or suppress ETI, thereby restoring pathogen virulence. The last two steps are then continuously reiterated during plant-pathogen coevolution. In the past decade, two important new insights have emerged. First, principles underlying the model are not restricted to interactions of plants with microbial pathogens but apply as well to interactions with other organisms, including beneficial microbes (128), insects (1, 36, 58), and plant-parasitic nematodes (46). Moreover, the conserved patterns that are being recognized are not restricted to exogenous, invader-derived molecules but can also be endogenous ligands associated with the breaching of cell wall integrity upon invasion of the host by various pathogens and invaders [damage-associated molecular patterns (DAMPs)] (53). Second, elicitors and receptors involved in MTI and ETI share more commonalities than previously thought. To resolve the limitations of the MTI-ETI dichotomy, Cook et al. (23) recently developed a more generic model of plant immunity (the invasion model) in which invasion pattern receptors (IPRs), including PRRs and R proteins, detect invasion patterns (IPs), including MAMPs, DAMPs, and effectors, to activate an IP-triggered response (IPTR). This model nicely accommodates the whole range of ligands, irrespective of their function, as well as the whole range of immune receptors, from narrow to broad with respect to the range of invaders recognized and from weak to strong with respect to immune response.

Hormone Signaling and Defense Pathways

The perception of highly diverse molecular patterns and effectors produced by a wide range of plant invaders results in the activation of intracellular pathways, which are interconnected to form a signaling network, ultimately resulting in the induction of local and systemic defense responses (72). Major players in systemic induced defense are the plant hormones salicylic acid (SA), jasmonic acid (JA), and ethylene (ET), which are produced in response to plant attack. Upon recognition by their cognate receptors, they orchestrate specific defense pathways as part of the plant's core defense system. Transcription factors are key components in the transcriptional reprogramming of the host during the activation of hormone-regulated defense pathways (157). In SA-mediated signaling, biosynthesis of SA is induced and detected by the central regulator NPR1 (NONEXPRESSOR of PATHOGENESIS RELATED 1), which results in the activation of SA-regulated defense gene expression by the derepression of TGA transcription factors (172). In JA-mediated signaling, biosynthesis of the JA conjugate JA-Ile promotes the interaction between COI1 (CORONATIN INSENSITIVE 1) and JAZ (JASMONATE ZIM-DOMAIN) proteins, resulting in the proteolytic degradation of JAZs and derepression of transcription factors, which activate JA-mediated defense gene expression (22, 166). JA signaling is regulated by two antagonistic branches. ET synergizes the JA-ERF (ETHYLENE RESPONSE FACTOR) branch, in which transcriptional activation is mediated by AP2 (APETALA2)/ERF transcription factors. By contrast, abscisic acid (ABA) synergizes the JA-MYC branch in which transcriptional

activation is mediated by the basic helix-loop-helix (bHLH) domain–containing transcription factor MYC2 (157). In the core defense system, SA- and JA-mediated signaling are interconnected and act antagonistically with each other, depending on dose and location. The SA/JA/ET core defense system is further antagonized by other hormones (134), including the growth hormones auxin (AUX) and cytokinin (CK), reflecting the trade-off between growth and defense in plant performance. Cross talk between pathways allows the integration of defense signaling networks to induce systemic resistance to different plant-feeding organisms (20, 152).

Systemic Induction and Manipulation of Transcriptional Changes in Defense

The induction of plant hormone biosynthesis following attack also results in the induction and priming of noninfected plant parts to confer broad-spectrum resistance upon future attack. Accumulation of SA triggers systemic acquired resistance (SAR), which results in long-lasting and even transgenerational priming of systemic tissue. Upon attack, systemic induced defense requires long-distance communication between the site of infection and distant plant tissues. The underlying principles and signals responsible for the systemic induction of the SA/JA/ET core defense system are not yet fully understood. For SA-mediated systemic defense responses, methyl salicylate (MeSA) and a number of other small molecules have been implicated as long-distance signals. Perception of the signals results in a feedback amplification mechanism of defense responses in systemic leaves (43). SA-induced defense gene expression results in systemic induction and accumulation of SA markers, such as the pathogenesis-related (PR) proteins PR-1, PR-2, and PR-5 (118). For JA-mediated systemic defense, the bioactive form JA-Ile is thought to be the most likely candidate to act as a mobile component in long-distance JA signaling (166), which in *Arabidopsis thaliana* results in the induction of JA-mediated defense responses and the expression of *VSP2* (*VEGETATIVE STORAGE PROTEIN 2*) as a marker of the JA-MYC branch and *PDF1.2* (*PLANT DEFENSIN 1.2*) as a marker of the JA-ERF branch.

To counteract induced plant defense responses, the use of effectors that modulate host immunity is common practice across plant invaders from different kingdoms. Along with pathogens (73), nematodes (46, 119), insects (58, 167), and/or their associated microbes (30) also secrete effectors to suppress immune responses either outside or inside the cell. Pathogen effectors target a variety of phytohormone receptors, transcriptional activators, repressors, and other components of phytohormone signaling (73, 98). Some pathogens, such as *Pseudomonas syringae* pv. *tomato*, have an effector repertoire that enables them to interact with all known major signaling pathways to activate the SA signaling pathway by sabotaging its activators and stimulating its repressors (171). In addition, some root-feeding nematodes secrete chorismate mutase (CM) involved in SA biosynthesis and manipulate lipid signaling involved in JA-induced defense (47, 93). Consequently, local manipulation of defense hormone signaling pathways by phytophages may attenuate systemic induced defense responses to other plant feeding organisms, either within or across compartments.

INDUCED SYSTEMIC DEFENSE RESPONSES WITHIN COMPARTMENTS

Systemic defense responses are induced both within and across plant compartments. In this section, we first focus on the induction of hormone-regulated defense pathways by different phytophage groups with distinct feeding strategies and how this affects other organisms feeding on distant plant parts within the same compartment.

Pathogen-Induced Systemic Defense Responses

Biotrophic pathogens, that feed on living tissue, generally induce SA signaling, resulting in systemic activation of SAR accompanied by the production of PR proteins, whereas necrotrophic pathogens, which kill host tissue and feed on dead cell contents, induce JA-ERF signaling (45, 128). Indeed, biotrophic and hemibiotrophic pathogens, which in addition have a necrotrophic phase, are generally resisted by SAR, and necrotrophs by JA- and ET-mediated defense, whereas some are affected by both (153, 156). Owing to antagonism between the SA and JA/ET pathways, pathogens with a similar lifestyle are predicted to have negative effects on each other, whereas pathogens with opposite lifestyles are predicted to facilitate each other. However, SA-JA antagonism is not unconditional. In *A. thaliana*, a virulent strain of the hemibiotroph *P. syringae* suppresses JA signaling and increases susceptibility to the necrotroph *Alternaria brassicicola* in local tissue (150), but the necrotroph is not affected in more distant leaf tissue. Moreover, an avirulent strain of the hemibiotroph *P. syringae* that induces a hypersensitive response (HR) does not suppress JA signaling (150). Furthermore, SA can only suppress JA/ET signaling when it is induced prior to or simultaneously with JA and ET (92). Pathogen infection also affects herbivores on the shared host plant. For instance, the biotroph *Hyaloperonospora arabidopsidis* suppresses JA-mediated defenses activated by the leaf-chewing herbivore *Pieris rapae* (75). A review by Lazebnik et al. (85) confirms that biotrophic pathogens generally facilitate chewing herbivores through SA-JA antagonism unless plants exhibit ETI, whereas necrotrophs inhibit phloem feeders as well as chewing herbivores by inducing JA, although there are numerous exceptions that can point at a role of factors other than hormone-mediated interactions or hormonal modulation by effector molecules.

Herbivore-Induced Systemic Defense Responses

Chewing arthropods disrupt the integrity of plant tissue, leading to production of DAMPs such as oligogalacturonides (OGs), as well as to wound-induced resistance (WIR). In addition, chewing arthropods produce herbivore-associated molecular patterns (HAMPs) such as fatty acid–amino conjugates (FACs) that elicit HAMP-triggered immunity (HTI) (1, 36, 137). Although HAMP receptors have not yet been identified, detection of HAMPs can amplify the wound-induced defense response. This response is characterized by the induction of the MYC branch of the JA signaling pathway that is synergized by ABA, which rapidly increases after feeding by chewing arthropods. Given that the JA-MYC branch antagonizes SA signaling that activates defenses against biotrophic pathogens, as well as the JA-ERF branch that activates defenses against necrotrophic pathogens and phloem-feeding insects, chewing herbivores are expected to facilitate biotrophic pathogens while effects on phloem-feeding insects and necrotrophic pathogens may depend on additional signaling activation. Surprisingly, predicted effects of leaf-chewing arthropods on biotrophic pathogens are not well supported by data (85), whereas effects on aphids vary from facilitation (e.g., 142) to inhibition (e.g., 2). In contrast to chewing herbivores, phloem-feeding insects induce both the JA/ET and SA pathway, although enhanced JA signaling is not strongly supported for the whitefly *Bemisia tabaci* (41, 100, 155). Whereas the enhanced JA signaling induced by the green peach aphid (GPA) results in the accumulation of shoot defense compounds and contributes to GPA resistance in *A. thaliana*, its enhancement of SA signaling does not (100) and can even promote infestation, most likely by antagonizing JA signaling. Several studies confirm that aphids can facilitate leaf-chewing herbivores because of aphid-mediated attenuation of the induction of JA defenses (2, 76, 142). Phloem-feeding insects further generally inhibit both biotrophic and necrotrophic pathogens, consistent with their induction of both the SA and JA-ERF signaling pathways (85).

Nematode-Induced Systemic Defense Pathways

In contrast to resistance induced by pathogens (SAR) and herbivores [herbivore-induced resistance (HIR)], information about hormone signaling and systemic defense responses induced by plant-parasitic nematodes in roots is still fragmentary. However, a picture emerges showing that root feeding by nematodes activates an integrated signaling network, which is highly regulated by plant hormones. Root feeding by sedentary endoparasitic nematodes results in the production and accumulation of SA, JA, and ET at the site of infection (67). Subsequently, transcriptional reprogramming results in the activation of the core defense system involving SA/JA/ET-mediated pathways (80, 93, 168). For example, in *A. thaliana*, cyst nematode–induced JA-mediated defense depends on the protein phosphatase AP2C1, which is a negative regulator of the mitogen-activated protein kinases MPK3 and MPK6, two positive regulators of plant defense responses controlling ET and JA biosynthesis (140). Infection of the *Arabidopsis* mutants *mpk3* and *mpk6* resulted in higher nematode infection rates, consistent with a reduced wound-induced defense phenotype of these plants. In addition, the upregulation of *VSP2*, a marker for the MYC2 branch in JA defense signaling, was detected upon infection of soybean roots by the cyst nematode *Heterodera glycines* (131). The tissue damage caused by intracellular migration of cyst nematodes may account for the activation of damage-triggered immune responses (necrotrophic phase) before the establishment of a permanent feeding site (biotrophic phase). In contrast to cyst nematodes, root-knot nematodes (RKNs) migrate intercellularly without a noticeable wound response. However, expression of proteinase inhibitors and increased enzyme activity of phospholipase A_2 (PLA_2) indicate that RKN invasion of tomato roots also results in JA-mediated defense signaling responses despite their stealthy host migration strategy (39).

CROSS-COMPARTMENT EFFECTS OF EXOGENOUS HORMONE APPLICATION

Despite their limitations, cross-compartment studies of exogenous application of hormones or their mimics allow assessment of the effects of increased phytohormone levels in shoots on plant responses in the roots and vice versa. In addition, the effect of hormone-induced responses is then tested in the absence of defense-modulating phytophages. Although the core phytohormone signaling system functions in both shoots and roots, details of the signaling cascades can substantially differ between compartments, with important consequences for the activation of downstream defenses. This is illustrated by studies of exogenous JA application to roots versus shoots. In potato, root JA application results in the activation of a root-specific homolog of the proteinase inhibitor PI-II in roots, as well as a shoot-specific PI-II homolog in the shoots. However, whereas JA-mediated PI-II induction in shoots requires the activity of a protein phosphatase in shoots, it does not in roots. Moreover, none of the ABA-mediated pathways for activation of wound-inducible genes that are functional in potato leaves operate in the roots (25). This suggests that the complex signal transduction network that regulates effects of JA and ABA on gene expression in response to wounding is organ specific. Similarly, shoot and root JA application to feral *Brassica oleracea* results in differential expression of JA biosynthesis genes and JAZ proteins that repress the transcriptional activation of JA-responsive genes in both of the compartments (158). This suggests that roots and shoots have a different JA signaling cascade and that the signal that elicits systemic responses contains information about the site of induction, providing plants with a mechanism to tailor their responses specifically to the organ that is damaged. The differential regulation results in organ-specific responses in both primary and secondary metabolism, including glucosinolate and terpene biosynthesis.

Pathogens. Exogenous application of SA to roots generally results in cross-compartment SAR. For instance, root SA application to broccoli results in systemic upregulation of the pathogenesis-related genes *PR-1* and *PR-2* in leaves in a SAR-like response as early as 24-h post-treatment (101). Likewise, root SA application results in increased leaf levels of SA and PR proteins in tomato and a strong reduction in leaf lesions caused by the pathogen *Alternaria solani* (149). In rice, root SA application results in higher leaf concentrations of phytoalexins such as oryzalexins and momilactone A (26). Similarly, exogenous root application of MeJA (methyl jasmonate) induces expression of defense- associated genes, including a 13-lipoxygenase gene (*LOX*), *PR-3*, and *PR-4* in wheat crowns and reduces crown symptoms of the pathogen *Fusarium culmorum* (114). Exogenous shoot application of both SA and JA contributes to resistance to clubroot disease caused by the protist *Plasmodiophora brassicae* in *A. thaliana* (90).

Herbivores. Exogenous application of SA or SA analogs to roots generally increases resistance to phloem-feeding insects. SA application to broccoli roots decreases the abundance of the specialist aphid *Brevicoryne brassicae*, although it does not affect the abundance of the generalist aphid *Myzus persicae* (127). Exogenous root application of the SAR-inducing, nonproteinogenic amino acid BABA (β-aminobutyric acid) also enhances leaf levels of defense-related genes, including those encoding PR proteins in several plant species, enhancing aphid resistance in soybean (177) and resistance against both aphids and leaf-chewing herbivores in *A. thaliana* (56). In addition, exogenous application of SA to roots can prime or inhibit AG tissue for the expression of JA responses upon elicitation by biotic agents, depending on concentration (31). Exogenous root application of JA can also affect leaf-chewing as well as phloem-feeding insects AG, but the responses can be markedly weaker than upon leaf JA application. Root JA application to Chinese broccoli reduces the performance of the phloem-feeding whitefly *B. tabaci* but less strongly than does shoot JA application (94). Similarly, root JA application to feral *B. oleracea* reduces survival and growth of leaf-chewing larvae of *Mamestra brassicae* but less strongly than does shoot JA application (162). Interestingly, whereas root JA application yields a systemic response in shoot glycosinolates (GLSs), shoot JA application does not in roots (164). Given that total GLS levels in roots also do not respond to root JA, this may indicate a general low responsiveness of root GLSs to JA application.

Nematodes. Foliar treatment with JA results in induced resistance to RKNs in tomato roots and JA-induced gene expression profiles (24, 44). Also application of SA on tomato shoots results in systemic resistance to the RKNs *Meloidogyne javanica* (19, 120) and *Meloidogyne incognita* on cowpea (117), but it enhances root infestation by sedentary nematodes in barley (147). In addition, foliar application of BABA, which acts through SA and ABA signaling, results in systemic inhibition of RKN infection by activation of basal defense responses, including the production of reactive oxygen species (ROS), callose, and lignin in rice roots (63). *Arabidopsis* SA-deficient mutants are more susceptible to cyst nematodes, whereas foliar application of SA results in reduced susceptibility (e.g., 48, 170). These studies show that effective SA- and JA-mediated defense responses are induced from shoots to roots, which results in systemic resistance to sedentary endoparasitic nematodes.

CROSS-COMPARTMENT EFFECTS OF DEFENSE MANIPULATION

Given the systemic nature of hormone signaling, it is expected that not only their activation but also their manipulation by pathogens will have cross-compartment effects. So far, this has mainly been documented for hemibiotrophs that can colonize both roots and shoots. The root-infecting hemibiotroph *Fusarium oxysporum* hijacks COI1-mediated jasmonate signaling in *Arabidopsis* by

secreting the JA-Ile mimic effector coronatine, which promotes JA signaling and disease development by antagonizing the SA pathway (154). Plants defective in JA perception (*coi*) that cannot be manipulated this way are highly resistant at later stages to *F. oxysporum*. Grafting studies show that the enhanced resistance in the leaves occurs through *coi1* in the rootstock (154), suggesting a cross-compartment effect in shoots of effector-triggered hormone modulation in the roots. Repression of systemic defense responses in the shoots is observed upon nematode root infections of rice (81). For the migratory nematode *Hirschmanniella oryzae*, a more delayed defense suppression is observed compared with that of the sedentary nematode *Meloidogyne graminicola*, which may indicate that defense suppression is less critical for nematodes with a migratory lifestyle as they move from cell to cell to feed on their cytoplasmic contents, leaving a necrotic track behind (81). Whether systemic suppression of defense in the shoots is just an off-target effect of nematode root feeding or that BG nematode defense requires the shoot compartment for an effective defense response, which is in turn systemically repressed by root nematodes, needs further analysis.

CROSS-COMPARTMENT CHANGES IN RESOURCE DYNAMICS

Infection by pathogens, arthropod herbivores, and nematodes results not only in systemic induction of defense pathways but also in strong local and systemic modulation of host primary metabolism (11, 178), which can profoundly affect phytophages present in the other compartment. Microbial pathogens turn assimilate-producing tissues from source into sink tissues (91) through induction of plant and microbial cell wall invertases (CW-Inv) (15). These invertases catalyze the cleavage of sucrose, the main form in which sugars are transported, into hexoses (glucose and fructose). High hexose-to-sucrose ratios in the apoplast decrease export of assimilates from the tissue; hence, they are key enzymes for supplying sink organs with carbohydrates. This secures local resources for the phytophage but can have negative effects on carbon supply in systemic tissue. Moreover, these sugars function as priming molecules leading to MTI (sweet priming) (113), making it sometimes difficult to distinguish whether systemic within- or cross-compartment effects of phytophages are mediated by modulation of defense signaling pathways or by modulation of source-sink relationships (130). Some pathogens have also evolved effectors that suppress CW-Inv activity to prevent sugar-mediated defense signaling (148). Cell wall invertases also play an important role in interactions with nematodes (21) and insect herbivores. *N. attenuata* plants upregulate CW-Inv in response to leaf herbivory, which is JA dependent and associated with both a reduced depletion of carbohydrates and with increased capacity to enhance secondary metabolite pools (40). JA-dependent inhibition of invertase activity results in a depletion of hexose levels but nevertheless enhances the performance of a leaf-chewing insect (104). Phytophages thus modulate source-sink relationships, which are intertwined with defense signaling.

In general, a wide range of biotic stresses upregulates defense genes but simultaneously downregulates photosynthesis genes (16). However, cross-compartment changes in carbon dynamics following AG biotic challenge are complex. Despite the downregulation of photosynthesis genes and the direct removal of the photosynthetic area, foliar herbivory generally results in increased resource allocation to roots (123). A functional explanation for this so-called herbivore-induced resource sequestration has been that photosynthates are diverted away from the site of tissue damage into tissues inaccessible to foraging herbivores and are stored there to allow regrowth after herbivory has ceased. Indeed, several studies have shown that following AG herbivory, root storage is favored over lateral root growth (35). However, the fate of the diverted resources is subject to regulatory processes and can be allocated not only to storage but also to defense or growth of sink tissues (123). Therefore, phytophages may either benefit or suffer from the increased resource allocation to roots following foliar herbivory, depending on the fate of the diverted resources. For

instance, nematodes benefit from leaf herbivory when it is associated with an increase in root sink strength for photoassimilates following leaf herbivory (68), but BG phytophages may suffer when these resources are invested in BG defenses. Not only foliar herbivores but also phloem-sucking herbivores such as aphids constitute a sink that can modify assimilate allocation at the whole-plant level (91) and induce changes in gene expression involved in sugar and nitrogen metabolism (29). Generalist aphids induce dramatic increases in sucrose and starch contents in source tissues, inhibition of sugar export to other sinks, and increase trehalose, promoting reallocation of C into starch instead of sucrose (141).

BG phytophages can also have a strong impact on AG phytophages through modulation of water and resource dynamics in the AG compartment. Root herbivory can induce systemic water deficits and elicit a hydraulic response in shoots that precedes ABA signaling and stomatal closure and that can result in transient fluxes in leaf and phloem concentrations of nitrogen and amino acids, from which AG phytophages such as aphids can benefit [the pulsed stress hypothesis (62)]. Root herbivory can also reduce foliar expression of root-derived secondary metabolites such as nicotine, whereas they can increase foliar expression of other, leaf-synthesized, compounds (69). This corresponds with a general trend that root herbivory elicits strong responses in both roots and shoots, in contrast to foliar herbivory that elicits strong responses in leaves but weaker responses in roots. Similarly, nematodes suppress constitutive and inducible foliar expression of the root-produced nicotine in tobacco (**Figure 1*d***) (49, 69) and hence increase susceptibility to leaf-feeding insects (68).

SYSTEMIC INTERACTIONS INDUCED BY PATHOGENS ACROSS COMPARTMENTS

Cross-Compartment Signaling and Interactions Induced by Aboveground Pathogens

Indications for shoot-to-root SAR induced by AG pathogens comes from leaf inoculation with hemibiotrophic fungi, which results in systemic responses in roots associated with increased SA (169). For instance, inoculation of cucumber leaves with the anthracnose pathogen *Colletotrichum lagenarium* results in a more than threefold higher accumulation of SA in the roots (77). A shoot-to-root SAR is not always observed. Leaf inoculation of maize variant Jubilee with the hemibiotrophic fungus *Colletotrichum graminicola* does not strongly affect SAR-related gene expression in roots. Although it strongly enhances SA levels and expression of pathogenesis-related proteins typical for SAR in systemic leaves, it upregulates only oxylipin-related genes in the roots (7). The results are asymmetrical, as root infection does strongly enhance SA and ABA levels and expression of PR proteins in systemic leaves. This suggests that shoot-to-root SAR induced by hemibiotrophs may be weaker than root-to-shoot SAR. Below, we summarize effects of AG pathogens on the different groups of BG phytophages.

Against belowground pathogens. Perhaps not surprisingly, many of the studies investigating cross-compartment interactions between AG and BG pathogens have used pathogens that can colonize both roots and shoots. In Cavendish banana (*Musa* AAA Cavendish subgroup), AG inoculation with an incompatible race of the hemibiotrophic fungus *F. oxysporum* f. sp. *cubense* (*Foc* race 1) enhances resistance to a compatible strain of the fungus (*Foc* race 4) when that strain is subsequently inoculated in the roots (169). The enhanced resistance to *Foc* race 4 is associated with elevated levels of SA and expression of *PR-1* and several other defense-related genes in roots compared to those found in plants not preinoculated with *Foc* race 1. The results suggest that the increased resistance is based on a shoot-to-root SAR.

Against belowground herbivores. Whereas numerous studies show SAR against AG herbivores, reports on SAR against BG herbivores are virtually absent. Based on the detection of systemic changes in roots upon SAR induced by foliar pathogens, it is anticipated that the foliar pathogens may negatively affect BG herbivores responsive to SA-mediated defenses and facilitate BG chewing herbivores through antagonism of the JA pathway.

Against belowground nematodes. Information on the effect of AG pathogens on root-feeding nematodes is virtually absent. Stolon inoculation of white clover with the fungus *Drechslera halodes* significantly increased densities of the nematodes *Helicotylenchus dihystera* and *Heterodera trifolii*, and suppressed negative effects of the root pathogen *Pythium irregulare* on the nematode *Meloidogyne trifoliophila* (174). This contrasts with the general negative effects of root pathogens on these nematodes. However, AG elicitor-induced ROS and related scavenging enzymes enhance plant defense to *M. javanica* infection in the roots (120).

Cross-Compartment Signaling and Interactions Induced by Belowground Pathogens

Root infection with BG pathogens generally results in considerable transcriptional changes in the shoots. For instance, root inoculation of *A. thaliana* with the hemibiotroph *F. oxysporum* alters the expression (> twofold) of 512 genes in the roots but an even larger number of genes (1,763) in the leaves at 1 dpi (day post-infection) before any colonization of the fungus in the shoots can be observed (103). The genes that are upregulated in shoots following root inoculation include the pathogenesis-related protein PR-1, characteristic of SAR, as well as the defensin PDF1.2, characteristic of JA/ET signaling (103). The former is later strongly downregulated in leaves. Also, in beech there is a massive transcriptional reprogramming in the shoots during the first, biotrophic growth phase of the root pathogen *Phytophthora citrocola* that is interpreted as a response resulting from the increased sink strength of roots to support growth of the oomycete (136). Whereas expression of PR proteins typically associated with SAR is enhanced in roots shortly after infection, they are strongly downregulated in shoots as early as 24 hours post-infection (136). Below, we summarize effects of BG pathogens on different groups of AG phytophages.

Against aboveground pathogens. Transcriptional changes in shoots following infection by root pathogens have consequences for shoot pathogens. Within 1 dpi, root infection of maize by the hemibiotrophic fungus *C. graminicola* results in a significant increase of ABA and SA in leaves, and enhanced expression in leaves of genes involved in the biosynthesis of benzoxazinoids and PR proteins, and this lasts until at least 4 dpi (7). Consequently, preinoculation of roots with *C. graminicola* strongly reduces fungal growth of the same pathogen when it is inoculated in the leaves six days after root preinoculation (7), indicating an operational root-to-shoot SAR. Interestingly, effects of the fungus on JA are more locally restricted. Root inoculation results in initial suppression and later enhancement of root JA but does not affect JA levels in the shoots. As far as we know, studies showing effects of BG pathogen infection on heterospecific AG pathogens have not yet been completed.

Against aboveground herbivores. Studies of the effects of BG pathogens on AG herbivores are scarce. The root pathogen *Phytophthora plurivora* enhances the performance of *Lymantria dispar* on trees (110). Older studies show that forage legumes infected by *Fusarium* fungi in roots have reduced aphid densities (86).

These studies show that there are only a few cross-compartment studies involving pathogens. The available studies show that (hemi-)biotrophic pathogens can induce SAR across compartments, perhaps more strongly from root to shoot than in the opposite direction, but cross-compartment studies for necrotrophs are scant. AG pathogens may further induce susceptibility to nematodes, and BG pathogens can affect performance of AG herbivores.

SYSTEMIC INTERACTIONS INDUCED BY HERBIVORES ACROSS COMPARTMENTS

Cross-Compartment Signaling and Interactions Induced by Aboveground Herbivores

AG herbivory results in massive cross-compartment induction of transcriptional changes in the roots. The leaf-chewing herbivore *Spodoptera frugiperda* alters expression of 270 transcripts in shoots and 308 in roots of maize, of which only 135 are shared (3). *S. frugiperda* enhances the expression of JA/ET biosynthesis genes in the shoot but upregulates only genes involved in ET perception and signaling in the roots (3). This is consistent with the relatively weak herbivore-induced JA burst observed in roots compared to shoots (33) and suggests that AG herbivore-mediated induction and suppression of the JA/ET signaling are tissue specific. Also, phloem feeders, such as aphids (87) and whiteflies (126), and cell content feeders, such as thrips and mites (66), induce large cross-compartment transcriptional changes. In pepper (*Capsicum annuum*), infestation by the aphid *M. persicae* results in systemic upregulation of both SA and JA signaling and priming of systemic defense responses in roots (87). AG feeding by whitefly (*B. tabaci*) induces almost twice as many transcriptional changes in roots as in shoots of pepper plants (126). It induces SA as well as JA/ET signaling in both shoots and roots, but details differ between compartments. Whereas it induces the SAR-associated PR-protein PR-1 in both shoots and roots, it enhances expression of the SAR- and ISR (induced systemic resistance)-associated PR-10 and PR-4 in roots only (173). The authors suggest that part of the observed changes may be mediated by whitefly-induced recruitment of ISR-inducing rhizosphere bacteria (**Figure 2*a***). Below, we summarize effects of AG herbivores on different groups of BG phytophages.

Against belowground pathogens. Studies of the effects of AG herbivores, mechanical leaf damage, and defoliation on BG pathogens have yielded mixed results. Repeated mechanical leaf wounding of *Medicago truncatula* induces JA in the roots, reducing growth of the oomycete root-rot pathogen *Aphanomyces euteiches* (83). However, repeated defoliation has no effect on levels of root infection by *F. oxysporum* in *Medicago sativa* (89) and even increases the incidence of *Fusarium* root rot in *Trifolium pratense* in the lab and field (138). For true AG herbivory, early studies suggested that feeding by AG sap-sucking insects enhances BG disease. Feeding by nymphs of the three-cornered alfalfa hopper *Spissistilus festinus* enhances crown rot severity caused by *F. oxysporum* in alfalfa roots (111). Similarly, the aphid *Acyrthosiphon pisum* enhances *F. oxysporum* root-rot infection in partly defoliated red clover and alfalfa (86). However, later studies have also shown negative effects of AG sap-sucking insects on BG pathogens. In pepper (*C. annuum*), the upregulation root SA and JA signaling following infestation by the aphid *M. persicae* is accompanied by increased resistance against the bacterial root pathogen *Ralstonia solanacearum* (87). The upregulation in this system of PR proteins by the whitefly *B. tabaci* (173) is also associated with enhanced resistance to *R. solanacearum*. Whitefly infestation of *Nicotiana benthamiana* also leads to enhanced resistance to a BG pathogen (146), e.g., a twofold reduction in gall formation in both stems and crown roots by the root pathogen *Agrobacterium tumefaciens*. Interestingly, whitefly-infested plants have

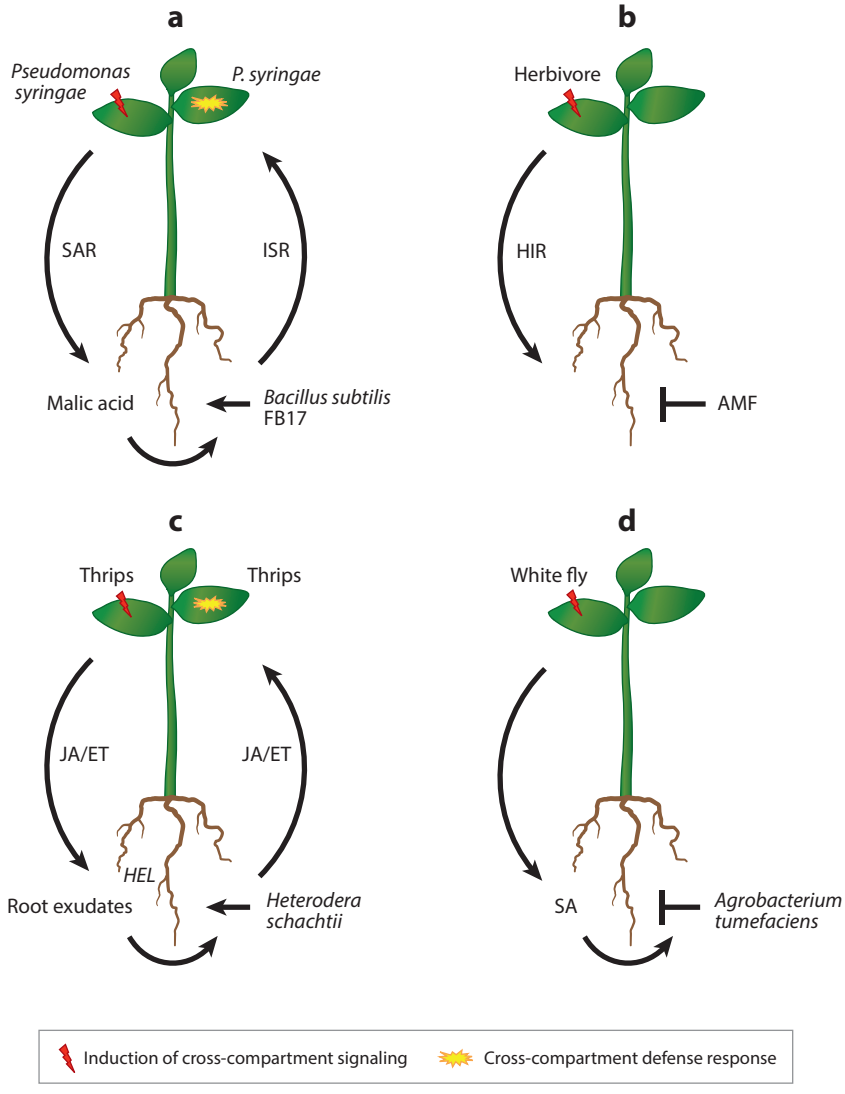

Figure 2

Cross-compartment shoot-to-root-to-shoot (S-R-S) defense loops are often extended by changes in root defense or root exudates induced by aboveground phytophages that can either enhance or reduce the recruitment of beneficial microbes and/or phytophages in the rhizosphere. (*a*) Plants attacked by an aboveground pathogen induce systemic acquired resistance (SAR) and the production of malic acid in root exudates. This results in the recruitment of beneficial microbes that trigger induced systemic resistance (ISR) (82, 135). ISR confers broad-spectrum resistance against not only the aboveground inducer but also other phytophages. Likewise, aboveground herbivores can enhance recruitment of beneficial soil microbes (173). (*b*) Plants attacked by an aboveground herbivore induce systemic defense responses [herbivore-induced resistance (HIR)] in roots, which have a negative impact on arbuscular mycorrhizal fungi (AMF), reducing the scope for ISR (6, 9, 28). In addition, systemic induced changes in root exudates can either facilitate or inhibit infection by belowground phytophages. (*c*) Plants attacked by a herbivore induce jasmonic acid (JA)- and ethylene (ET)-mediated defense pathways and changes in root exudates, which promote the attraction of infective nematodes to the roots that subsequently induce JA/ET-mediated defense to an aboveground herbivore (66). (*d*) Plants attacked by an aboveground herbivore produce root exudates with increased salicylic acid (SA) levels, which reduce root infection by a bacterial pathogen (146).

threefold higher SA levels in the root exudates, inhibiting *A. tumefaciens*–mediated transformation (**Figure 2*d***), which may have wider implications for the recruitment of beneficial rhizosphere microbes or for interactions with soilborne pathogens. AG leaf-chewing insects can also enhance resistance to BG pathogen infection. Prior defoliation by larvae of the soybean looper *Pseudoplusia includens* reduces infection levels of crown rot caused by the fungus *Calonectria crotalariae* in soybean, a pathogen involved in a disease complex with the nematode *H. glycines* (125). However, defoliation by *Spodoptera ornithogalli* does not affect crown rot severity caused by strains of *Fusarium solani* and *F. oxysporum* in *M. sativa* (88).

Against belowground herbivores. For overviews of plant-mediated effects of AG herbivores on BG herbivores, we refer to the following excellent reviews: see References 13, 38, 64, 69, 144, and 159–161. A recent meta-analysis shows that AG herbivores that arrive prior to BG herbivores generally reduce the performance of these root feeders (64). For instance, foliar herbivory by *Pieris brassicae* reduces the survival of the root-feeding dipteran *Delia radicum* by more than 50%, possibly mediated by increased levels of root indole glucosinolates (143). Similarly, in maize, foliar feeding by *S. frugiperda* reduces colonization and growth of root-feeding *Diabrotica virgifera virgifera*, provided that foliar herbivory is initiated prior to root feeding (37). In cucumber, foliar herbivory by the beetle *Acalymma vittatum* and the lepidopteran *S. frugiperda* does not induce changes in leaf or root cucurbitacins but does reduce root biomass as well as the performance of the beetle's root-feeding larvae (109). Some studies indicate that AG herbivores may also interfere with the ability of plants to induce compounds in response to root herbivores (priming/inhibition). In *Triadica sebifera*, adults of the chrysomelid beetle *Bikasha collaris* feed on leaves, whereas the larvae feed on roots. Larval feeding induces root tannins. However, when adults are feeding on the plant as well, they prevent induction of root tannins, resulting in increased larval survival (61). Interestingly, this intraspecific facilitation is asymmetric; larval feeding amplifies the induction of leaf tannins in response to adult feeding. Effects of AG phloem-feeding insects on root herbivores have been less well studied but suggest that in addition to inhibitory effects facilitation might occur through aphid-induced compensatory growth (108).

Against belowground nematodes. Studies on systemic interactions between shoot herbivores and root parasitic nematodes predominantly focus on nematode-induced effects on herbivores rather than herbivore-induced effects on nematodes (168). An increased number of sedentary nematode infections is generally observed on plants defoliated by the chewing herbivores (168). This is explained by the increased reallocation of resources from the shoots to the roots upon caterpillar attack, promoting the development of RKNs that form a permanent metabolic active feeding cell (70). By contrast, sap-sucking herbivores such as aphids, which are less likely to provoke such herbivore-induced resource reallocation (123), indeed have weaker or even negative effects on the performance of root-feeding nematodes (52, 59, 70, 78). Interestingly, when considering two cell content feeders on *A. thaliana*, the thrips *Frankiniella occidentalis* increases root infection by the cyst nematode *Heterodera schachtii*, whereas the spider mite *Tetranychus urticae* does not (66). In roots, expression of the ET/JA marker gene HEL (PR-4) is enhanced by thrips feeding but decreased by mite feeding. The study suggests that thrips-induced susceptibility is achieved by systemic induction of the ET/JA pathway, promoting the attractiveness of the roots for infective juveniles of cyst nematodes, probably due to changes in root exudates (**Figure 2*c***).

In summary, AG phloem-feeding herbivores can induce both SA and JA/ET signaling in roots and have mixed effects on BG (hemi-)biotrophic pathogens and chewing herbivores; they tend to have negative effects on nematodes. Chewing AG herbivores induce at least ET signaling in roots and generally enhance resistance to BG herbivores; they can also affect resistance against

BG pathogens and enhance susceptibility to sedentary nematodes. Further, roots and shoots show differences in the specific sets of defense genes that are induced by AG herbivores, indicating organ specificity of induction. Too few studies are available to test whether AG chewing arthropods facilitate BG necrotrophs and impede BG biotrophs through cross-compartment induction of JA/ET and antagonism with SA.

Cross-Compartment Signaling and Interactions Induced by Belowground Herbivores

Root damage induces large transcriptional changes in shoots. Leaves of *A. thaliana* plants exposed to razorblade-induced root damage show a large number of transcriptional changes, predominantly associated with systemic induction of JA and ET signals, interpreted as transcripts involved in root-to-shoot communication (50). The same is observed for true root herbivory, such as root feeding by larvae of corn rootworms (32, 34). The Southern corn rootworm *Diabrotica undecimpunctata howardi* induces transcriptional changes in shoots of *A. thaliana* involved in JA signaling even when leaves are highly senescent (84). Below, we summarize effects of BG herbivores on the performance of AG phytophages.

Against aboveground pathogens. Root herbivory can significantly affect the performance of AG pathogens. Root infestation of maize with the Western corn rootworm *D. virgifera virgifera* results in enhanced resistance against the necrotrophic fungal pathogen Northern corn leaf blight (*Setosphaeria turcica*) (32). The root herbivore induces a local increase in root levels of oxylipins and ABA. Systemically, it results in upregulation of shoot levels of ABA but not of JA or SA, even though two pathogenesis-related proteins, PR-1 and PR-5, are upregulated in the shoot (34). Furthermore, the root herbivore reduces shoot water content and induces a systemic increase in shoot levels of the benzoxazinoid DIMBOA, which has toxicity toward both herbivores and fungi. Root herbivory by larvae of another Chrysomelid beetle, the striped cucumber beetle *A. vittatum*, does not affect infection of cucumber by powdery mildew caused by the biotrophic leaf pathogen *Sphaerotheca fuliginea* (55) but reduces infection of cucumber by downy mildew caused by the oomycete biotrophic leaf pathogen *Pseudoperonospora cubensis* (10). These results indicate that root herbivores can enhance resistance against both biotrophic and necrotrophic leaf pathogens.

Against aboveground herbivores. There are several excellent reviews on plant-mediated interactions between AG and BG herbivores that illustrate the strong impacts of BG herbivory on AG herbivores (13, 17, 38, 64, 145, 159–161). However, surprisingly few studies have linked observed BG-AG interactions to changes in defense hormone signaling. A recent meta-analysis shows that BG herbivores generally enhance the performance of simultaneously arriving AG herbivores. This effect appears to be mainly due to the positive effects of BG feeding beetle larvae (Coleoptera) on AG aphids and other Hemiptera, which may be explained by the flux of shoot amino acids and nitrogen following induced hydraulic stress [pulsed stress hypothesis (62)]. By contrast, AG chewing insects often suffer from root herbivore–induced water stress. Root herbivory of maize by the Western corn rootworm *D. virgifera virgifera* enhances shoot levels of ABA and the feeding deterrent benzoxazinoid DIMBOA and primes plants for a stronger leaf induction of the phenolic acid chlorogenic acid (CGA) upon attack by the AG chewing herbivore *Spodoptera littoralis* (32). Even though root herbivory results in increased levels of shoot defense metabolites, the increased resistance to *S. littoralis* following root herbivory appears to be mainly mediated by changes in the insect's feeding strategy caused by shoot hydraulic changes resulting

from root herbivore–induced water stress (34). However, several other studies show that AG chewing herbivores do suffer from leaf defenses induced by BG herbivores, including terpenoids, pyrrolizidine alkaloids, and glucosinolates (38). For instance, root feeding by wireworms increases leaf terpenoid defenses in cotton, reducing leaf consumption by AG chewing *Spodoptera exigua* by half (14). Interestingly, whereas AG herbivory induces these terpenoids only in young leaves, BG herbivory induces them in all leaves, illustrating the advantage of BG-induced leaf defense compared to AG-induced leaf defense through its better vascular connections (14). Root herbivory furthermore induces shoot resource reallocation. Maize plants attacked by the Western corn rootworm *D. virgifera virgifera* allocate more new photosynthates from source leaves to stems but not to roots (133), indicating that resource sequestration (123) can also occur from root to shoot.

These studies indicate that BG chewing herbivores generally have positive effects on phloem-feeding herbivores, presumably mediated by enhanced nutritional status, but not on leaf-chewing AG herbivores, through induction of ABA-mediated or other defenses. Likewise, BG herbivory can enhance resistance against both biotrophic and necrotrophic pathogens.

SYSTEMIC INTERACTIONS INDUCED BY NEMATODES ACROSS COMPARTMENTS

Cross-Compartment Signaling and Interactions Induced by Aboveground Nematodes

Although the vast majority of plant-parasitic nematodes are soil-dwelling nematodes, many are able to infect AG plant organs. Foliar pathogenic nematodes such as *Aphelenchoides* spp. and *Ditylenchus* spp. are migratory nematodes that move systemically on water films in the shoot and feed as ectoparasites on buds, leaves, and seeds (80). This could dramatically affect the response to other plant feeders. Indeed, rice plants infected with *Aphelenchoides besseyi* and *Ditylenchus angustus* are more susceptible to rice blast caused by *Magnaporthe oryzae* (112) and stem rot caused by *Sclerotinium oryzae* (107). This may be due to facilitation of fungal infection by tissue damage caused by these migrating nematodes. However, it cannot be excluded that this phenomenon is caused by systemic responses induced by shoot-feeding nematodes in the same compartment.

Cross-Compartment Signaling and Interactions Induced by Belowground Nematodes

Transcriptome analyses reveal that root feeding by migratory and sedentary endoparasitic nematodes induces systemic changes in gene expression and defense responses in the shoots of both dicots and monocots (74, 81). Moreover, nematodes induce species-specific changes in glucosinolate profiles in shoots (57, 59, 96). Various other defense compounds also accumulate in the shoots upon nematode root infection, including PR proteins (170), peroxidases, and catalases (4). This may be due to the tissue damage caused by nematodes that migrate intracellularly. However, the local and systemic accumulation of the wound-responsive proteinase inhibitors and other proteins upon infection of tomato roots by RKNs (39, 44) suggest that even nematodes with a stealthy host invasion strategy cause local injury, which subsequently activates defense signaling across compartments. Recently, the screening of miRNA libraries from wild type and the JA-deficient tomato mutant *spr2* showed that miR319 targets TCP4 (TEOSINTE BRANCHED1/CYCLOIDAE/PROLIFERATING CELL FACTOR 4) upon RKN infection of the roots (176). TCP4 targeting results in systemic changes in JA biosynthesis in the leaves, which subsequently affect induced defense responses to RKN in the roots. The authors postulate

that miR319 acts as a systemic signal responder and regulator that connects roots and shoots via its transport in the phloem (**Figure 1*b***). Below we summarize the effects of BG nematodes on the performance of AG phytophages.

Against aboveground pathogens. Despite numerous reports on disease complexes between root-feeding nematodes and soilborne plant pathogens (5), only a few cross-compartment interactions between nematodes and AG pathogens have been described. Rice plants preinfested with the RKN *M. graminicola* three days prior to AG pathogen infection show increased susceptibility to the fungal pathogen *M. oryzae* (79). Root layering and grafting experiments in tomato show that upon nematode infection a systemic signal is translocated from the roots to the shoot and results in the breaking of *F. oxysporum* resistance (race 1) in nematode-susceptible tomato upon stem inoculation with the fungus (139). Similarly, in a split root experiment, RKN infestation increases the severity of *F. oxysporum* wilting (54). Another interesting example is that RKNs suppress the induction of the root-produced phytoalexin cajanol in the xylem of pigeonpea (*Cajanus cajan*) in response to stem inoculation with the wilt fungus *Fusarium udum*, which strongly enhances fungal wilt symptoms AG (105) (**Figure 1*d***).

Against aboveground herbivores. Several studies have addressed systemic interactions between root-feeding nematodes and shoot-feeding insects (168). Studies testing systemic effects of sedentary RKNs on leaf-chewing herbivores generally show an increase in insect performance, whereas the migratory endoparasitic nematode *Pratylenchus penetrans* shows a reduction in performance. Likewise, the sedentary cyst nematode *H. schachtii* enhances the attractiveness of *A. thaliana* for the cell content–feeding spider mite *Tetranynchus uricae* and enhances its development (66), whereas the migratory root-feeding nematode *P. penetrans* reduces its fecundity on *Phaseolus vulgaris* (18). From such observations, it is tempting to speculate that sedentary nematodes have stronger facilitating effects on AG chewing herbivores than migratory nematodes. By contrast, sedentary cyst nematodes have consistent negative effects on phloem feeders, similar to what is observed for the migratory nematode *P. penetrans*, suggesting that there is no correlation between nematode feeding strategies and the performance of phloem feeders.

Root feeding by nematodes results in systemic changes in both defense compounds and nutrient allocation. Root feeding by *P. penetrans* enhances the production of leaf phenolics and glucosinolates, which could account for reduced caterpillar performance (163). Root feeding by the sedentary cyst nematode *H. schachtii* reduces the population size of the aphid *B. brassicae* and induces changes in the composition of glucosinolates, although a causal link cannot be established (59). The reduction in the performance of the cell content–feeding thrips (*F. occidentalis*) on shoots of *Arabidopsis* upon root feeding by *H. schachtii* (66) is associated with elevated levels of JA and expression of JA-regulated defense genes in shoots. JA-mediated defense is known to have a negative impact on thrips performance, and therefore it was proposed that the observed reduction in thrips performance was in part due to nematode-induced systemic JA defense responses (**Figure 2*c***). Interestingly, positive effects of RKN infection on leaf-chewing herbivores have been observed in tobacco despite RKN-induced increases in shoot phenolics and terpenoids (69). The enhanced AG herbivore performance on RKN-infected plants is caused by the fact that the nematode suppresses the induction of the root-produced defense chemical nicotine in response to foliar herbivory, resulting in low leaf nicotine levels and enhanced caterpillar performance (**Figure 1*d***).

In addition to the manipulation of defense responses, nematode root feeding also results in changes in resource allocation. The establishment of a local feeding relationship with the host results in systemic induced changes in the amino acid and carbohydrate content of the areal parts

of the plant, which can influence the performance of shoot-feeding herbivores. Studies using *Arabidopsis* mutants impaired in sucrose-cleaving enzymes have revealed that cyst nematodes and RKNs induce activity of these enzymes in roots to enhance local accumulation of sugars but also systemically induce enzyme activity and accumulation of sugars—particularly starch—in shoots. It is not clear whether such enhanced levels of shoot resources contribute to the enhanced performance observed for some AB chewing insects, but clearly these are not large enough to benefit aphids, which are generally reduced by nematode feeding. It has been postulated (71) that the extent to which aphids and nematodes increase the AG and BG sink strength, respectively, could be a determinant of their competitive interaction, i.e., that stronger sink strength of nematodes could explain their negative effects on aphids, but this was not supported by data. In contrast to sedentary nematodes, the migratory nematode *Hirschmaniella* sp., which does not form a permanent feeding cell and is less strongly dependent on such sink-source relationships, induces a substantial reduction in shoot soluble sugar and amino acid content in rice (60). This could account for the negative impact of root-feeding migratory nematodes on aphid performance (12). Thus, differences in sink-source relationship between nematodes with different feeding strategies could determine differences in nutrient quality of the shoot and drive systemic interactions with AG interacting organisms such as insects.

In conclusion, root-feeding nematodes can inhibit induction of root-produced defense chemicals but enhance induction of shoot-produced defense metabolites. They generally induce susceptibility to shoot pathogens but increase resistance to shoot phloem-feeding herbivores, whereas their effects on leaf-chewing insects may depend on nematode life strategy.

CONCLUDING REMARKS

Our review illustrates that cross-compartment loops, which involve S-R-S or R-S-R signaling, are frequently involved in the activation of a full defense complement in a single compartment for pathogens, herbivores, and nematodes (**Figure 1**). Subsequently these loops are, not surprisingly, also exploited by phytophages to suppress defenses against them. For example, root feeding by BG phytophages can result in the facilitation of AG phytophages by suppressing the induction and transport of root-synthesized secondary defense metabolites (**Figure 1*d***). Both root herbivores and nematodes can be the facilitators, and both pathogens and herbivores can be the beneficiaries. Conversely, AG herbivores can facilitate BG phytophages by suppressing the BG induction of root-produced secondary metabolites (61). These studies indicate that facilitation between phytophages from either the same or different kingdoms can occur both within and across compartments. Interestingly, it has been shown that R-S-R feedbacks loops, such as the autoregulation of nodulation (AON) to restrict symbiosis, have wider effects on pathogens and nematodes (95), and it has recently been suggested that such autoregulation strategies with broad-spectrum induced resistance may underlie both ISR and SAR (175).

Interestingly, S-R-S defense loops are often even more extended, including effects mediated by altered recruitment of BG phytophages or beneficial microbes that results from changes in root defense or root exudates induced by AG pathogens or herbivores (**Figure 2**). The altered recruitment of ISR-inducing microbes in roots or rhizosphere can confer broad-spectrum resistance against not only the AG inducer but also other phytophages. This is in fact a more extended loop than the recruitment of phytophage enemies such as parasitoids in the aerial compartment, as they directly interact with the AG phytophages, whereas effects of the recruitment of ISR-inducing rhizosphere microorganisms are mediated by changes in the plant's resistance. The role of such extended loops involving changes in the rhizosphere microbiome in AG-BG interactions is an exciting avenue for future studies.

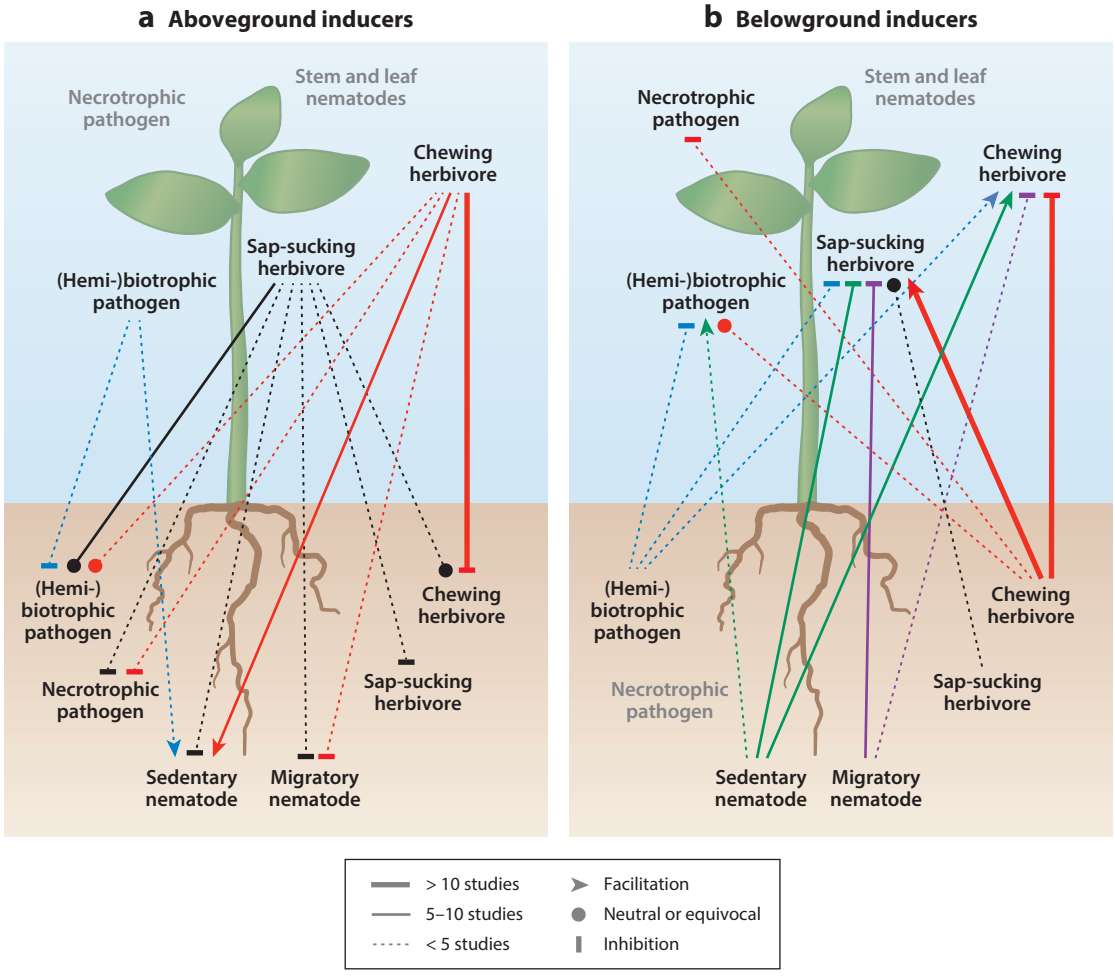

Figure 3

Schematic overview of plant-mediated cross-compartment interactions between aboveground and belowground phytophages based on literature discussed in this review and others (64, 168). (*a*) Interactions between aboveground inducers and belowground responders. (*b*) Interactions between belowground inducers and aboveground responders. The line type indicates a conservative estimate of the number of studies currently available for each of the interactions. Thick solid line: >10; thin solid line: 5–10; dotted line: <5. The arrow type indicates the most frequently observed outcome of the interaction. Pointed arrow, facilitation; perpendicular bar, inhibition; circle, effects are either predominantly neutral or equivocal, i.e., a mix of facilitation and inhibition with no predominance of either of these effects. Note that since the arrows only indicate the most commonly observed outcome of an interaction, they may in fact represent a wider range of outcomes; for details, see text and other reviews (64, 168). To our knowledge, no cross-compartment interactions have been reported for the organism groups highlighted in grey.

Our review shows a strong imbalance in the number of studies that have addressed AG-BG interactions between different combinations of the feeding guilds of the three phytophage groups (**Figure 3**). After the emergence of a research agenda for studying interactions involving BG herbivores (132), interactions between BG insect herbivores or BG nematodes and AG herbivores have been relatively well studied, but surprisingly few studies have addressed interactions between AG pathogens and BG nematodes and interactions involving root pathogens (27). For instance, although facilitation between biotrophic and necrotrophic pathogens through SA-JA antagonism

has been well described within shoots, it awaits cross-compartment testing. Likewise, AG-BG interactions involving AG nematodes are a virtually unexplored area. For the groups that have been well studied, some interesting patterns arise. For example, BG insect herbivores generally enhance AG aphid performance, whereas BG nematodes generally reduce AG aphid performance. It will be interesting to test why this is.

Predicting the outcome of interactions between phytophages sharing a host plant based on the knowledge of the specific induction of particular signaling pathways by the organism groups and feeding guilds involved is challenging, even within compartments. Temporal patterns of hormonal changes following attack are often complex, especially in response to phytophages with lifestyles that include a biotrophic and necrotrophic phase, and measurements of hormonal balances at one point in time have limited value for predicting effects on other phytophages. Perhaps most important is the challenge that other phytophages are affected by not only induction of systemic defense but also changes in resource dynamics. These two processes are tightly interconnected, as illustrated by the importance of sugar signaling in defense and the role of defense hormones in the regulation of source-sink relationships. Predicting cross-compartment interactions faces some extra challenges. Although exogenous hormone application and phytophage inoculation studies show that SAR and JA/ET defense signaling are broadly activated across compartments, details of the induced systemic hormonal changes can significantly differ between the attacked and nonattacked compartment, limiting predictions of cross-compartment effects from observed systemic hormonal changes within the attacked compartment. An area that especially deserves attention is therefore the extent to which local effector-based suppression of induced systemic signaling by phytophages in the attacked compartment also suppresses systemic signaling across compartments.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

We apologize to all the investigators whose research could not be cited because of space limitations. Furthermore, we would like to thank Ian Kaplan for critical reading of the manuscript. This work benefited from interactions funded through COST Actions FA1208 and FA1405. Publication 6073 of NIOO-KNAW.

LITERATURE CITED

1. Acevedo FE, Rivera-Vega LJ, Chung SH, Ray S, Felton GW. 2015. Cues from chewing insects: the intersection of DAMPs, HAMPs, MAMPs and effectors. *Curr. Opin. Plant Biol.* 26:80–86
2. Ali JG, Agrawal AA. 2014. Asymmetry of plant-mediated interactions between specialist aphids and caterpillars on two milkweeds. *Funct. Ecol.* 28:1404–12
3. Ankala A, Kelley RY, Rowe DE, Williams WP, Luthe DS. 2013. Foliar herbivory triggers local and long distance defense responses in maize. *Plant Sci.* 199:103–12
4. Aryal SK, Davis RF, Stevenson KL, Timper P, Ji PS. 2011. Induction of systemic acquired resistance by *Rotylenchulus reniformis* and *Meloidogyne incognita* in cotton following separate and concomitant inoculations. *J. Nematol.* 43:160–65
5. Back MA, Haydock PPJ, Jenkinson P. 2002. Disease complexes involving plant parasitic nematodes and soilborne pathogens. *Plant Pathol.* 51:683–97

6. Ballhorn DJ, Younginger BS, Kautz S. 2014. An aboveground pathogen inhibits belowground rhizobia and arbuscular mycorrhizal fungi in *Phaseolus vulgaris*. *BMC Plant Biol.* 14:321
7. Balmer D, de Papajewski DV, Planchamp C, Glauser G, Mauch-Mani B. 2013. Induced resistance in maize is based on organ-specific defence responses. *Plant J.* 74:213–25
8. Balmer D, Mauch-Mani B. 2013. More beneath the surface? Root versus shoot antifungal plant defenses. *Front. Plant Sci.* 4:256
9. Barber NA, Adler LS, Theis N, Hazzard RV, Kiers ET. 2012. Herbivory reduces plant interactions with above- and belowground antagonists and mutualists. *Ecology* 93:1560–1570
10. Barber NA, Milano NJ, Kiers ET, Theis N, Bartolo V, et al. 2015. Root herbivory indirectly affects above- and below-ground community members and directly reduces plant performance. *J. Ecol.* 103:1509–18
11. Berger S, Sinha AK, Roitsch T. 2007. Plant physiology meets phytopathology: plant primary metabolism and plant-pathogen interactions. *J. Exp. Bot.* 58:4019–26
12. Bezemer TM, De Deyn GB, Bossinga TM, van Dam NM, Harvey JA, Van der Putten WH. 2005. Soil community composition drives aboveground plant-herbivore-parasitoid interactions. *Ecol. Lett.* 8:652–61
13. Bezemer TM, van Dam NM. 2005. Linking aboveground and belowground interactions via induced plant defenses. *Trends Ecol. Evol.* 20:617–24
14. Bezemer TM, Wagenaar R, van Dam NM, Wackers FL. 2003. Interactions between above- and below-ground insect herbivores as mediated by the plant defense system. *Oikos* 101:555–62
15. Biemelt S, Sonnewald U. 2006. Plant-microbe interactions to probe regulation of plant carbon metabolism. *J. Plant Physiol.* 163:307–18
16. Bilgin DD, Zavala JA, Zhu J, Clough SJ, Ort DR, DeLucia EH. 2010. Biotic stress globally downregulates photosynthesis genes. *Plant Cell Environ.* 33:1597–613
17. Blossey B, Hunt-Joshi TR. 2003. Belowground herbivory by insects: influence on plants and aboveground herbivores. *Annu. Rev. Entomol.* 48:521–47
18. Bonte D, De Roissart A, Vandegehuchte ML, Ballhorn DJ, Van Leeuwen T, de la Pena E. 2010. Local adaptation of aboveground herbivores towards plant phenotypes induced by soil biota. *PLOS ONE* 5:e11174
19. Branch C, Hwang CF, Navarre DA, Williamson VM. 2004. Salicylic acid is part of the Mi-1-mediated defense response to root-knot nematode in tomato. *Mol. Plant-Microbe Interact.* 17:351–56
20. Caarls L, Pieterse CMJ, Van Wees SCM. 2015. How salicylic acid takes transcriptional control over jasmonic acid signaling. *Front. Plant Sci.* 6:170
21. Cabello S, Lorenz C, Crespo S, Cabrera J, Ludwig R, et al. 2014. Altered sucrose synthase and invertase expression affects the local and systemic sugar metabolism of nematode-infected *Arabidopsis thaliana* plants. *J. Exp. Bot.* 65:201–12
22. Campos ML, Kang JH, Howe GA. 2014. Jasmonate-triggered plant immunity. *J. Chem. Ecol.* 40:657–75
23. Cook DE, Mesarich CH, Thomma B. 2015. Understanding plant immunity as a surveillance system to detect invasion. *Annu. Rev. Phytopathol.* 53:541–63
24. Cooper WR, Jia L, Goggin L. 2005. Effects of jasmonate-induced defenses on root-knot nematode infection of resistant and susceptible tomato cultivars. *J. Chem. Ecol.* 31:1953–67
25. Dammann C, Rojo E, Sanchez-Serrano JJ. 1997. Abscisic acid and jasmonic acid activate wound-inducible genes in potato through separate, organ-specific signal transduction pathways. *Plant J.* 11:773–82
26. Daw BD, Zhang LH, Wang ZZ. 2008. Salicylic acid enhances antifungal resistance to *Magnaporthe grisea* in rice plants. *Austral. Plant Pathol.* 37:637–44
27. De Coninck B, Timmermans P, Vos C, Cammue BPA, Kazan K. 2015. What lies beneath: belowground defense strategies in plants. *Trends Plant Sci.* 20:91–101
28. De Roman M, Fernandez I, Wyatt T, Sahrawy M, Heil M, Pozo MJ. 2011. Elicitation of foliar resistance mechanisms transiently impairs root association with arbuscular mycorrhizal fungi. *J. Ecol.* 99:36–45
29. de Vos M, Kim JH, Jander G. 2007. Biochemistry and molecular biology of *Arabidopsis*-aphid interactions. *BioEssays* 29:871–83
30. Diezel C, von Dahl CC, Gaquerel E, Baldwin IT. 2009. Different lepidopteran elicitors account for cross-talk in herbivory-induced phytohormone signaling. *Plant Physiol.* 150:1576–86
31. Engelberth J, Viswanathan S, Engelberth MJ. 2011. Low concentrations of salicylic acid stimulate insect elicitor responses in *Zea mays* seedlings. *J. Chem. Ecol.* 37:263–66

32. Erb M, Flors V, Karlen D, de Lange E, Planchamp C, et al. 2009. Signal signature of aboveground-induced resistance upon belowground herbivory in maize. *Plant J.* 59:292–302
33. Erb M, Glauser G, Robert CAM. 2012. Induced immunity against belowground insect herbivores: activation of defenses in the absence of a jasmonate burst. *J. Chem. Ecol.* 38:629–40
34. Erb M, Kollner TG, Degenhardt J, Zwahlen C, Hibbard BE, Turlings TCJ. 2011. The role of abscisic acid and water stress in root herbivore-induced leaf resistance. *New Phytol.* 189:308–20
35. Erb M, Lenk C, Degenhardt J, Turlings TCJ. 2009. The underestimated role of roots in defense against leaf attackers. *Trends Plant Sci.* 14:653–59
36. Erb M, Meldau S, Howe GA. 2012. Role of phytohormones in insect-specific plant reactions. *Trends Plant Sci.* 17:250–59
37. Erb M, Robert CAM, Hibbard BE, Turlings TCJ. 2011. Sequence of arrival determines plant-mediated interactions between herbivores. *J. Ecol.* 99:7–15
38. Erb M, Ton J, Degenhardt J, Turlings TCJ. 2008. Interactions between arthropod-induced aboveground and belowground defenses in plants. *Plant Physiol.* 146:867–74
39. Fan JW, Hu CL, Zhang LN, Li ZL, Zhao FK, Wang SH. 2015. Jasmonic acid mediates tomato's response to root knot nematodes. *J. Plant Growth Regul.* 34:196–205
40. Ferrieri AP, Arce CCM, Machado RAR, Meza-Canales ID, Lima E, et al. 2015. A *Nicotiana attenuata* cell wall invertase inhibitor (NaCWII) reduces growth and increases secondary metabolite biosynthesis in herbivore-attacked plants. *New Phytol.* 208:519–30
41. Foyer CH, Verrall SR, Hancock RD. 2015. Systematic analysis of phloem-feeding insect-induced transcriptional reprogramming in *Arabidopsis* highlights common features and reveals distinct responses to specialist and generalist insects. *J. Exp. Bot.* 66:495–512
42. Fragoso V, Rothe E, Baldwin IT, Kim SG. 2014. Root jasmonic acid synthesis and perception regulate folivore-induced shoot metabolites and increase *Nicotiana attenuata* resistance. *New Phytol.* 202:1335–45
43. Fu ZQ, Dong XN. 2013. Systemic acquired resistance: turning local infection into global defense. *Annu. Rev. Plant Biol.* 64:839–63
44. Fujimoto T, Tomitaka Y, Abe H, Tsuda S, Futai K, Mizukubo T. 2011. Expression profile of jasmonic acid–induced genes and the induced resistance against the root-knot nematode (*Meloidogyne incognita*) in tomato plants (*Solanum lycopersicum*) after foliar treatment with methyl jasmonate. *J. Plant Physiol.* 168:1084–97
45. Glazebrook J. 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annu. Rev. Phytopathol.* 43:205–27
46. Goverse A, Smant G. 2014. The activation and suppression of plant innate immunity by parasitic nematodes. *Annu. Rev. Phytopathol.* 52:243–65
47. Haegeman A, Mantelin S, Jones JT, Gheysen G. 2012. Functional roles of effectors of plant-parasitic nematodes. *Gene* 492:19–31
48. Hamamouch N, Li CY, Seo PJ, Park CM, Davis EL. 2011. Expression of *Arabidopsis* pathogenesis-related genes during nematode infection. *Mol. Plant Pathol.* 12:355–64
49. Hanounik SB, Osborne WW. 1975. Influence of *Meloidogyne incognita* on content of amino acids and nicotine in tobacco grown under gnotobiotic conditions. *J. Nematol.* 7:332–36
50. Hasegawa S, Sogabe Y, Asano T, Nakagawa T, Nakamura H, et al. 2011. Gene expression analysis of wounding-induced root-to-shoot communication in *Arabidopsis thaliana*. *Plant Cell Environ.* 34:705–16
51. Hauser TP, Christensen S, Heimes C, Kiaer LP. 2013. Combined effects of arthropod herbivores and phytopathogens on plant performance. *Funct. Ecol.* 27:623–32
52. Heeren JR, Steffey KL, Tinsley NA, Estes RE, Niblack TL, Gray ME. 2012. The interaction of soybean aphids and soybean cyst nematodes on selected resistant and susceptible soybean lines. *J. Appl. Entomol.* 136:646–55
53. Heil M. 2009. Damaged-self recognition in plant herbivore defence. *Trends Plant Sci.* 14:356–63
54. Hillocks RJ. 1986. Localized and sytemic effects of root-knot nematode on incidence and severity of *Fusarium* wilt in cotton. *Nematologica* 32:202–8
55. Hladun KR, Adler LS. 2009. Influence of leaf herbivory, root herbivory, and pollination on plant performance in *Cucurbita moschata*. *Ecol. Entomol.* 34:144–52

56. Hodge S, Pope TW, Holaschke M, Powell G. 2006. The effect of β-aminobutyric acid on the growth of herbivorous insects feeding on Brassicaceae. *Ann. Appl. Biol.* 148:223–29
57. Hofmann J, El Ashry A, Anwar S, Erban A, Kopka J, Grundler F. 2010. Metabolic profiling reveals local and systemic responses of host plants to nematode parasitism. *Plant J.* 62:1058–71
58. Hogenhout SA, Bos JIB. 2011. Effector proteins that modulate plant-insect interactions. *Curr. Opin. Plant Biol.* 14:422–28
59. Hol WHG, De Boer W, Termorshuizen AJ, Meyer KM, Schneider JHM, et al. 2013. *Heterodera schachtii* nematodes interfere with aphid-plant relations on *Brassica oleracea*. *J. Chem. Ecol.* 39:1193–203
60. Huang JH, Liu MQ, Chen XY, Chen J, Li HX, Hu F. 2015. Effects of intraspecific variation in rice resistance to aboveground herbivore, brown planthopper, and rice root nematodes on plant yield, labile pools of plant, and rhizosphere soil. *Biol. Fertil. Soils* 51:417–25
61. Huang W, Siemann E, Yang XF, Wheeler GS, Ding JQ. 2013. Facilitation and inhibition: Changes in plant nitrogen and secondary metabolites mediate interactions between above-ground and below-ground herbivores. *Proc. R. Soc. Lond. B* 280:20131318
62. Huberty AF, Denno RF. 2004. Plant water stress and its consequences for herbivorous insects: a new synthesis. *Ecology* 85:1383–98
63. Ji HL, Kyndt T, He W, Vanholme B, Gheysen G. 2015. β-aminobutyric acid–induced resistance against root-knot nematodes in rice is based on increased basal defense. *Mol. Plant-Microbe Interact.* 28:519–33
64. Johnson SN, Clark KE, Hartley SE, Jones TH, McKenzie SW, Koricheva J. 2012. Aboveground-belowground herbivore interactions: a meta-analysis. *Ecology* 93:2208–15
65. Jones JDG, Dangl JL. 2006. The plant immune system. *Nature* 444:323–29
66. Kammerhofer N, Egger B, Dobrev P, Vankova R, Hofmann J, et al. 2015. Systemic above- and below-ground cross talk: hormone-based responses triggered by *Heterodera schachtii* and shoot herbivores in *Arabidopsis thaliana*. *J. Exp. Bot.* 66:7005–17
67. Kammerhofer N, Radakovic Z, Regis JMA, Dobrev P, Vankova R, et al. 2015. Role of stress-related hormones in plant defence during early infection of the cyst nematode *Heterodera schachtii* in *Arabidopsis*. *New Phytol.* 207:778–89
68. Kaplan I, Halitschke R, Kessler A, Rehill BJ, Sardanelli S, Denno RF. 2008. Physiological integration of roots and shoots in plant defense strategies links above- and belowground herbivory. *Ecol. Lett.* 11:841–51
69. Kaplan I, Halitschke R, Kessler A, Sardanelli S, Denno RF. 2008. Constitutive and induced defenses to herbivory in above- and belowground plant tissues. *Ecology* 89:392–406
70. Kaplan I, Sardanelli S, Denno RF. 2009. Field evidence for indirect interactions between foliar-feeding insect and root-feeding nematode communities on *Nicotiana tabacum*. *Ecol. Entomol.* 34:262–70
71. Kaplan I, Sardanelli S, Rehill BJ, Denno RF. 2011. Toward a mechanistic understanding of competition in vascular-feeding herbivores: an empirical test of the sink competition hypothesis. *Oecologia* 166:627–36
72. Katagiri F, Tsuda K. 2010. Understanding the plant immune system. *Mol. Plant-Microbe Interact.* 23:1531–36
73. Kazan K, Lyons R. 2014. Intervention of phytohormone pathways by pathogen effectors. *Plant Cell* 26:2285–309
74. Klink VP, Overall CC, Alkharouf NW, MacDonald MH, Matthews BF. 2007. Laser capture microdissection (LCM) and comparative microarray expression analysis of syncytial cells isolated from incompatible and compatible soybean (*Glycine max*) roots infected by the soybean cyst nematode (*Heterodera glycines*). *Planta* 226:1389–409
75. Koornneef A, Pieterse CMJ. 2008. Cross talk in defense signaling. *Plant Physiol.* 146:839–44
76. Kroes A, van Loon JJA, Dicke M. 2015. Density-dependent interference of aphids with caterpillar-induced defenses in *Arabidopsis*: involvement of phytohormones and transcription factors. *Plant Cell Physiol.* 56:98–106
77. Kubota M, Nishi K. 2006. Salicylic acid accumulates in the roots and hypocotyl after inoculation of cucumber leaves with *Colletotrichum lagenarium*. *J. Plant Physiol.* 163:1111–17
78. Kutyniok M, Muller C. 2013. Plant-mediated interactions between shoot-feeding aphids and root-feeding nematodes depend on nitrate fertilization. *Oecologia* 173:1367–77
79. Kyndt T, Denil S, Bauters L, Van Criekinge W, De Meyer T. 2014. Systemic suppression of the shoot metabolism upon rice root nematode infection. *PLOS ONE* 9:e106858

80. Kyndt T, Fernandez D, Gheysen G. 2014. Plant-parasitic nematode infections in rice: molecular and cellular insights. *Annu. Rev. Phytopathol.* 52:135–53
81. Kyndt T, Nahar K, Haegeman A, De Vleesschauwer D, Hofte M, Gheysen G. 2012. Comparing systemic defence-related gene expression changes upon migratory and sedentary nematode attack in rice. *Plant Biol.* 14:73–82
82. Lakshmanan V, Castaneda R, Rudrappa T, Bais HP. 2013. Root transcriptome analysis of *Arabidopsis thaliana* exposed to beneficial *Bacillus subtilis* FB17 rhizobacteria revealed genes for bacterial recruitment and plant defense independent of malate efflux. *Planta* 238:657–68
83. Landgraf R, Schaarschmidt S, Hause B. 2012. Repeated leaf wounding alters the colonization of *Medicago truncatula* roots by beneficial and pathogenic microorganisms. *Plant Cell Environ.* 35:1344–57
84. Lawrence SD, Novak NG, El Kayal W, Ju CJT, Cooke JEK. 2012. Root herbivory: molecular analysis of the maize transcriptome upon infestation by Southern corn rootworm, *Diabrotica undecimpunctata howardi*. *Physiol. Plant.* 144:303–19
85. Lazebnik J, Frago E, Dicke M, van Loon JJA. 2014. Phytohormone mediation of interactions between herbivores and plant pathogens. *J. Chem. Ecol.* 40:730–41
86. Leath KT, Byers RA. 1977. Interaction of *Fusarium* root-rot with pea aphid and potato leafhopper feeding on forage legumes. *Phytopathology* 67:226–29
87. Lee B, Lee S, Ryu CM. 2012. Foliar aphid feeding recruits rhizosphere bacteria and primes plant immunity against pathogenic and non-pathogenic bacteria in pepper. *Ann. Bot.* 110:281–90
88. Lee JW, Colyer PD, Quisenberry SS. 1990. The effect of insect defoliation on the presence and severity of *Fusarium* crown-rot in alfalfa. *J. Entomol. Sci.* 25:253–60
89. Lee JW, Quisenberry SS, Colyer PD. 1991. Development of *Fusarium* crown-rot in alfalfa stressed by multiple defoliations by the yellowstriped armyworm (Lepidoptera, Noctuidae). *J. Entomol. Sci.* 26:85–94
90. Lemarie S, Robert-Seilaniantz A, Lariagon C, Lemoine J, Marnet N, et al. 2015. Both the jasmonic acid and the salicylic acid pathways contribute to resistance to the biotrophic clubroot agent *Plasmodiophora brassicae* in *Arabidopsis*. *Plant Cell Physiol.* 56:2158–68
91. Lemoine R, La Camera S, Atanassova R, Deedaldeechamp F, Allario T, et al. 2013. Source-to-sink transport of sugar and regulation by environmental factors. *Front. Plant Sci.* 4:272
92. Leon-Reyes A, Du YJ, Koornneef A, Proietti S, Korbes AP, et al. 2010. Ethylene signaling renders the jasmonate response of *Arabidopsis* insensitive to future suppression by salicylic acid. *Mol. Plant-Microbe Interact.* 23:187–97
93. Li RJ, Rashotte AM, Singh NK, Weaver DB, Lawrence KS, Locy RD. 2015. Integrated signaling networks in plant responses to sedentary endoparasitic nematodes: a perspective. *Plant Cell Rep.* 34:5–22
94. Li SJ, Xue X, Ren SX, Cuthbertson AGS, van Dam NM, Qiu BL. 2013. Root and shoot jasmonic acid induced plants differently affect the performance of *Bemisia tabaci* and its parasitoid *Encarsia formosa*. *Basic Appl. Ecol.* 14:670–79
95. Lohar DP, Bird DM. 2003. *Lotus japonicus*: a new model to study root-parasitic nematodes. *Plant Cell Physiol.* 44:1176–84
96. Lohmann M, Scheu S, Muller C. 2009. Decomposers and root feeders interactively affect plant defence in *Sinapis alba*. *Oecologia* 160:289–98
97. Lopez L, Camas A, Shivaji R, Ankala A, Williams P, Luthe D. 2007. Mir1-CP, a novel defense cysteine protease accumulates in maize vascular tissues in response to herbivory. *Planta* 226:517–27
98. Lo Presti L, Lanver D, Schweizer G, Tanaka S, Liang L, et al. 2015. Fungal effectors and plant susceptibility. *Annu. Rev. Plant Biol.* 66:513–45
99. Louis J, Basu S, Varsani S, Castano-Duque L, Jiang V, et al. 2015. Ethylene contributes to maize insect resistance1-mediated maize defense against the phloem sap-sucking corn leaf aphid. *Plant Physiol.* 169:313–24
100. Louis J, Shah J. 2013. *Arabidopsis thaliana–Myzus persicae* interaction: shaping the understanding of plant defense against phloem-feeding aphids. *Front. Plant Sci.* 4:213
101. Lovelock DA, Donald CE, Conlan XA, Cahill DM. 2013. Salicylic acid suppression of clubroot in broccoli (*Brassicae oleracea* var. *italica*) caused by the obligate biotroph *Plasmodiophora brassicae*. *Austral. Plant Pathol.* 42:141–53

102. Lucas WJ, Groover A, Lichtenberger R, Furuta K, Yadav SR, et al. 2013. The plant vascular system: evolution, development and functions. *J. Integr. Plant Biol.* 55:294–388
103. Lyons R, Stiller J, Powell J, Rusu A, Manners JM, Kazan K. 2015. *Fusarium oxysporum* triggers tissue-specific transcriptional reprogramming in *Arabidopsis thaliana*. *PLOS ONE* 10:e0121902
104. Machado RAR, Arce CCM, Ferrieri AP, Baldwin IT, Erb M. 2015. Jasmonate-dependent depletion of soluble sugars compromises plant resistance to *Manduca sexta*. *New Phytol.* 207:91–105
105. Marley PS, Hillocks RJ. 1994. Effect of root-knot nematodes on cajanol accumulation in the vascular tissues of pigeonpea after stem inoculation with *Fusarium udum*. *Plant Pathol.* 43:172–76
106. Masters GJ, Brown VK, Gange AC. 1993. Plant mediated interactions between aboveground and belowground insect herbivores. *Oikos* 66:148–51
107. McGawley EC, Rush MC, Hollis JP. 1984. Occurrence of *Aphelenchoides besseyi* in Louisiana rice seed and its interaction with *Sclerotium oryzae* in selected cultivars. *J. Nematol.* 16:65–68
108. McKenzie SW, Vanbergen AJ, Hails RS, Jones TH, Johnson SN. 2013. Reciprocal feeding facilitation between above- and below-ground herbivores. *Biol. Lett.* 9:20130341
109. Milano NJ, Barber NA, Adler LS. 2015. Conspecific and heterospecific aboveground herbivory both reduce preference by a belowground herbivore. *Environ. Entomol.* 44:317–24
110. Milanovic S, Lazarevic J, Karadzic D, Milenkovic I, Jankovsky L, et al. 2015. Belowground infections of the invasive *Phytophthora plurivora* pathogen enhance the suitability of red oak leaves to the generalist herbivore *Lymantria dispar*. *Ecol. Entomol.* 40:479–82
111. Moellenbeck DJ, Quisenberry SS, Colyer PD. 1992. *Fusarium* crown-rot development in alfalfa stressed by threecornered alfalfa hopper (Homoptera, Membracidae) feeding. *J. Econ. Entomol.* 85:1442–49
112. Mondal AH, Rahman L, Ahmed HJ, Miah SA. 1986. The cause of increasing blast susceptibility of ufra infected rice plants. *Bangladesh J. Agric.* 111:77–79
113. Morkunas I, Ratajczak L. 2014. The role of sugar signaling in plant defense responses against fungal pathogens. *Acta Physiol. Plant* 36:1607–19
114. Motallebi P, Niknam V, Ebrahimzadeh H, Hashemi M, Pisi A, et al. 2015. Methyl jasmonate strengthens wheat plants against root and crown rot pathogen *Fusarium culmorum* infection. *J. Plant Growth Regul.* 34:624–36
115. Nalam VJ, Keeretaweep J, Sarowar S, Shah J. 2012. Root-derived oxylipins promote green peach aphid performance on *Arabidopsis* foliage. *Plant Cell* 24:1643–53
116. Nalam VJ, Shah J, Nachappa P. 2013. Emerging role of roots in plant responses to aboveground insect herbivory. *Insect Sci.* 20:286–96
117. Nandi B, Kundu K, Banerjee N, Babu SPS. 2003. Salicylic acid–induced suppression of *Meloidogyne incognita* infestation of okra and cowpea. *Nematology* 5:747–52
118. Navarova H, Bernsdorff F, Doring AC, Zeier J. 2012. Pipecolic acid, an endogenous mediator of defense amplification and priming, is a critical regulator of inducible plant immunity. *Plant Cell* 24:5123–41
119. Nguyen PV, Bellafiore S, Petitot AS, Haidar R, Bak A, et al. 2014. *Meloidogyne incognita*–rice (*Oryza sativa*) interaction: a new model system to study plant-root-knot nematode interactions in monocotyledons. *Rice* 7:23
120. Nikoo FS, Sahebani N, Aminian H, Mokhtarnejad L, Ghaderi R. 2014. Induction of systemic resistance and defense-related enzymes in tomato plants using *Pseudomonas fluorescens* CHAO and salicylic acid against root-knot nematode *Meloidogyne javanica*. *J. Plant Prot. Res.* 54:383–89
121. Notaguchi M, Okamoto S. 2015. Dynamics of long-distance signaling via plant vascular tissues. *Front. Plant Sci.* 6:161
122. Ohgushi T. 2005. Indirect interaction webs: herbivore-induced effects through trait change in plants. *Annu. Rev. Ecol. Evol. Syst.* 36:81–105
123. Orians CM, Thorn A, Gomez S. 2011. Herbivore-induced resource sequestration in plants: why bother? *Oecologia* 167:1–9
124. Orlowska E, Basile A, Kandzia I, Llorente B, Kirk HG, Cvitanich C. 2012. Revealing the importance of meristems and roots for the development of hypersensitive responses and full foliar resistance to *Phytophthora infestans* in the resistant potato cultivar Sarpo Mira. *J. Exp. Bot.* 63:4765–79

125. Padgett GB, Russin JS, Snow JP, Boethel DJ, Berggren GT. 1994. Interactions among the soybean looper (Lepidoptera, Noctuidae), threecornered alfalfa hopper (Homoptera, Membracidae), stem canker, and red crown rot in soybean. *J. Entomol. Sci.* 29:110–19
126. Park YS, Ryu CM. 2014. Understanding cross-communication between aboveground and belowground tissues via transcriptome analysis of a sucking insect whitefly-infested pepper plants. *Biochem. Biophys. Res. Commun.* 443:272–77
127. Pierre SP, Dugravot S, Herve MR, Hassan HM, van Dam NM, Cortesero AM. 2013. Belowground induction by *Delia radicum* or phytohormones affect aboveground herbivore communities on field-grown broccoli. *Front. Plant Sci.* 4:305
128. Pieterse CMJ, Van der Does D, Zamioudis C, Leon-Reyes A, Van Wees SCM. 2012. Hormonal modulation of plant immunity. *Annu. Rev. Cell Dev. Biol.* 28:489–521
129. Poelman EH, Broekgaarden C, Van Loon JJA, Dicke M. 2008. Early season herbivore differentially affects plant defence responses to subsequently colonizing herbivores and their abundance in the field. *Mol. Ecol.* 17:3352–65
130. Proels RK, Huckelhoven R. 2014. Cell-wall invertases, key enzymes in the modulation of plant metabolism during defence responses. *Mol. Plant Pathol.* 15:858–64
131. Puthoff DP, Nettleton D, Rodermel SR, Baum TJ. 2003. *Arabidopsis* gene expression changes during cyst nematode parasitism revealed by statistical analyses of microarray expression profiles. *Plant J.* 33:911–21
132. Rasmann S, Agrawal AA. 2008. In defense of roots: a research agenda for studying plant resistance to belowground herbivory. *Plant Physiol.* 146:875–80
133. Robert CAM, Ferrieri RA, Schirmer S, Babst BA, Schueller MJ, et al. 2014. Induced carbon reallocation and compensatory growth as root herbivore tolerance mechanisms. *Plant Cell Environ.* 37:2613–22
134. Robert-Seilaniantz A, Grant M, Jones JDG. 2011. Hormone crosstalk in plant disease and defense: more than just JASMONATE-SALICYLATE antagonism. *Annu. Rev. Phytopathol.* 49:317–43
135. Rudrappa T, Czymmek KJ, Pare PW, Bais HP. 2008. Root-secreted malic acid recruits beneficial soil bacteria. *Plant Physiol.* 148:1547–56
136. Schlink K. 2010. Down-regulation of defense genes and resource allocation into infected roots as factors for compatibility between *Fagus sylvatica* and *Phytophthora citricola*. *Funct. Integr. Genom.* 10:253–64
137. Schmelz EA. 2015. Impacts of insect oral secretions on defoliation-induced plant defense. *Curr. Opin. Insect Sci.* 9:7–15
138. Siddiqui WM, Halisky PM, Lund S. 1968. Relationship of clipping frequency to root and crown deterioration in red clover. *Phytopathology* 58:486–88
139. Sidhu G, Webster JM. 1977. Predisposition of tomato to wilt fungus (*Fusarium oxysporum lycopersici*) by root knot nematode (*Meloidogyne incognita*). *Nematologica* 23:436–42
140. Sidonskaya E, Schweighofer A, Shubchynskyy V, Kammerhofer N, Hofmann J, et al. 2016. Plant resistance against the parasitic nematode *Heterodera schachtii* is mediated by MPK3 and MPK6 kinases, which are controlled by the MAPK phosphatase AP2C1 in *Arabidopsis*. *J. Exp. Bot.* 67:107–18
141. Singh V, Louis J, Ayre BG, Reese JC, Shah J. 2011. TREHALOSE PHOSPHATE SYNTHASE11–dependent trehalose metabolism promotes *Arabidopsis thaliana* defense against the phloem-feeding insect *Myzus persicae*. *Plant J.* 67:94–104
142. Soler R, Badenes-Perez FR, Broekgaarden C, Zheng SJ, David A, et al. 2012. Plant-mediated facilitation between a leaf-feeding and a phloem-feeding insect in a brassicaceous plant: from insect performance to gene transcription. *Funct. Ecol.* 26:156–66
143. Soler R, Bezemer TM, Cortesero AM, Van der Putten WH, Vet LEM, Harvey JA. 2007. Impact of foliar herbivory on the development of a root-feeding insect and its parasitoid. *Oecologia* 152:257–64
144. Soler R, Erb M, Kaplan I. 2013. Long distance root-shoot signalling in plant-insect community interactions. *Trends Plant Sci.* 18:149–56
145. Soler R, Van der Putten WH, Harvey JA, Vet LEM, Dicke M, Bezemer TM. 2012. Root herbivore effects on aboveground multitrophic interactions: patterns, processes and mechanisms. *J. Chem. Ecol.* 38:755–67
146. Song GC, Lee S, Hong J, Choi HK, Hong GH, et al. 2015. Aboveground insect infestation attenuates belowground *Agrobacterium*-mediated genetic transformation. *New Phytol.* 207:148–58

147. Sonnemann I, Finkhaeuser K, Wolters V. 2002. Does induced resistance in plants affect the belowground community? *Appl. Soil Ecol.* 21:179–85
148. Sonnewald S, Priller JPR, Schuster J, Glickmann E, Hajirezaei MR, et al. 2012. Regulation of cell wall–bound invertase in pepper leaves by *Xanthomonas campestris* pv. *vesicatoria* type three effectors. *PLOS ONE* 7:e51763
149. Spletzer ME, Enyedi AJ. 1999. Salicylic acid induces resistance to *Alternaria solani* in hydroponically grown tomato. *Phytopathology* 89:722–27
150. Spoel SH, Johnson JS, Dong X. 2007. Regulation of tradeoffs between plant defenses against pathogens with different lifestyles. *PNAS* 104:18842–47
151. Tack AJM, Dicke M. 2013. Plant pathogens structure arthropod communities across multiple spatial and temporal scales. *Funct. Ecol.* 27:633–45
152. Thaler JS, Humphrey PT, Whiteman NK. 2012. Evolution of jasmonate and salicylate signal crosstalk. *Trends Plant Sci.* 17:260–70
153. Thaler JS, Owen B, Higgins VJ. 2004. The role of the jasmonate response in plant susceptibility to diverse pathogens with a range of lifestyles. *Plant Physiol.* 135:530–38
154. Thatcher LF, Manners JM, Kazan K. 2009. *Fusarium oxysporum* hijacks COI1-mediated jasmonate signaling to promote disease development in *Arabidopsis*. *Plant J.* 58:927–39
155. Thompson GA, Goggin FL. 2006. Transcriptomics and functional genomics of plant defence induction by phloem-feeding insects. *J. Exp. Bot.* 57:755–66
156. Ton J, Van Pelt JA, Van Loon LC, Pieterse CMJ. 2002. Differential effectiveness of salicylate-dependent and jasmonate/ethylene-dependent induced resistance in *Arabidopsis*. *Mol. Plant-Microbe Interact.* 15: 27–34
157. Tsuda K, Somssich IE. 2015. Transcriptional networks in plant immunity. *New Phytol.* 206:932–47
158. Tytgat TOG, Verhoeven KJF, Jansen JJ, Raaijmakers CE, Bakx-Schotman T, et al. 2013. Plants know where it hurts: root and shoot jasmonic acid induction elicit differential responses in *Brassica oleracea*. *PLOS ONE* 8:e65502
159. van Dam NM. 2009. Belowground herbivory and plant defenses. *Annu. Rev. Ecol. Evol. Syst.* 40:373–91
160. van Dam NM, Harvey JA, Wackers FL, Bezemer TM, van der Putten WH, Vet LEM. 2003. Interactions between aboveground and belowground induced responses against phytophages. *Basic Appl. Ecol.* 4:63–77
161. van Dam NM, Heil M. 2011. Multitrophic interactions below and above ground: en route to the next level. *J. Ecol.* 99:77–88
162. van Dam NM, Oomen MWAT. 2008. Root and shoot jasmonic acid applications differentially affect leaf chemistry and herbivore growth. *Plant Signal. Behav.* 3:91–98
163. van Dam NM, Raaijmakers CE, van der Putten WH. 2005. Root herbivory reduces growth and survival of the shoot feeding specialist *Pieris rapae* on *Brassica nigra*. *Entomol. Exp. Appl.* 115:161–70
164. van Dam NM, Witjes L, Svatos A. 2004. Interactions between aboveground and belowground induction of glucosinolates in two wild *Brassica* species. *New Phytol.* 161:801–10
165. Van der Putten WH, Vet LEM, Harvey JA, Wackers FL. 2001. Linking above- and belowground multitrophic interactions of plants, herbivores, pathogens, and their antagonists. *Trends Ecol. Evol.* 16:547–54
166. Wasternack C, Hause B. 2013. Jasmonates: biosynthesis, perception, signal transduction and action in plant stress response, growth and development. An update to the 2007 review in *Annals of Botany*. *Ann. Bot.* 111:1021–58
167. Will T, Tjallingii WF, Thonnessen A, van Bel AJE. 2007. Molecular sabotage of plant defense by aphid saliva. *PNAS* 104:10536–41
168. Wondafrash M, Van Dam NM, Tytgat TOG. 2013. Plant systemic induced responses mediate interactions between root parasitic nematodes and aboveground herbivorous insects. *Front. Plant Sci.* 4:87
169. Wu YL, Yi GJ, Peng XX, Huang BZ, Liu E, Zhang JJ. 2013. Systemic acquired resistance in Cavendish banana induced by infection with an incompatible strain of *Fusarium oxysporum* f. sp. *cubense*. *J. Plant Physiol.* 170:1039–46
170. Wubben MJE, Jin J, Baum TJ. 2008. Cyst nematode parasitism of *Arabidopsis thaliana* is inhibited by salicylic acid (SA) and elicits uncoupled SA-independent pathogenesis-related gene expression in roots. *Mol. Plant-Microbe Interact.* 21:424–32

171. Xin X-F, He SY. 2013. *Pseudomonas syringae* pv. *tomato* DC3000: a model pathogen for probing disease susceptibility and hormone signaling in plants. *Annu. Rev. Phytopathol.* 51:473–98
172. Yan SP, Dong XN. 2014. Perception of the plant immune signal salicylic acid. *Curr. Opin. Plant Biol.* 20:64–68
173. Yang JW, Yi HS, Kim H, Lee B, Lee S, et al. 2011. Whitefly infestation of pepper plants elicits defence responses against bacterial pathogens in leaves and roots and changes the below-ground microflora. *J. Ecol.* 99:46–56
174. Zahid MI, Gurr GM, Nikandrow A, Hodda M, Fulkerson WJ, Nichol HI. 2002. Effects of root and stolon-infecting fungi on root-colonizing nematodes of white clover. *Plant Pathol.* 51:242–50
175. Zamioudis C, Pieterse CMJ. 2012. Modulation of host immunity by beneficial microbes. *Mol. Plant-Microbe Interact.* 25:139–50
176. Zhao WC, Li ZL, Fan JW, Hu CL, Yang R, et al. 2015. Identification of jasmonic acid–associated microRNAs and characterization of the regulatory roles of the miR319/TCP4 module under root-knot nematode stress in tomato. *J. Exp. Bot.* 66:4653–67
177. Zhong YP, Wang B, Yan JH, Cheng LJ, Yao LM, et al. 2014. DL-β-aminobutyric acid-induced resistance in soybean against *Aphis glycines* Matsumura (Hemiptera: Aphididae). *PLOS ONE* 9:e85142
178. Zhou SQ, Lou YR, Tzin V, Jander G. 2015. Alteration of plant primary metabolism in response to insect herbivory. *Plant Physiol.* 169:1488–98