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REVIEW ARTICLE

Avoidance and suppression of plant defenses by herbivores and pathogens

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Plants are nutritious and hence herbivores and phytopathogens have specialized to attack and consume them. In turn, plants have evolved adaptations to detect and withstand these attacks. Such adaptations we call 'defenses' and they can operate either directly between the plant and the plant consumer or indirectly i.e. when taking effect via other organisms such as predators and parasitoids of herbivores. Plant defenses put selection pressure on plant-consumers and, as a result, herbivores and pathogens have evolved counter-adaptations to avoid, resist, or manipulate plant defenses. Here we review how plant consumers have adapted to cope with plant defenses and we will put special emphasis on the phenomenon of suppression of plant defenses.

Keywords: plant defense; herbivory; pathogenesis; resistance; adaptation; defense repression

Introduction

Plants are nutritious and hence herbivores (Awmack and Leather 2002) and phytopathogens (Divon and Fluhr 2007) have specialized to consume them. In turn, plants have evolved diverse adaptations to detect and withstand plant consumers and these adaptations we call 'defenses' and they are divided in so-called 'constitutive' and 'induced' defenses which can operate either 'directly' or 'indirectly': the direct defenses are those that play a role in the antagonistic interactions that involve only the plant and its attacker (Howe and Jander 2008) while the indirect defenses take effect via other organisms such as foraging predators and host-searching parasitoids of herbivores which are attracted and/or arrested to plants with prey and hence liberate plants from their attackers either above ground (Sabelis et al. 2001) or below ground (Rasman et al. 2005).

Induction of plant defenses

The discrimination between constitutive and induced defenses is artificial since many defenses fall in both classes and traits associated with defenses can have roles in other primary and secondary physiological processes as well. Defenses are, in principle, those plant traits that make a plant more palatable for a plant consumer when absent. Constitutive defenses are, for example, wax layers, trichomes, and thorns but can also be secondary metabolites (Strauss et al. 2002) and protective coatings (Shepherd et al. 2005). However, although called 'constitutive,' since they are present also when the plant has not experienced an attack, it was found that sometimes these constitutive barriers are reinforced upon herbivory or pathogenesis as well: for example, upon grazing by large herbivores several Acacia species were found to produce more and longer thorns in their canopy (Milewski and Madden 2006) and black mustard *Brassica nigra* was found to increase its number of trichomes upon herbivore feeding (Traw and Dawson 2002). Hence the difference between constitutive and induced defenses is not absolute. In most cases, induction of defenses is caused by the consumption-related activities of the attacker. However, such an induced reinforcement of preexisting defenses and establishment of novel defenses can occur already before a plant-consumer has actually probed its substrate as it was shown that insect eggs (Hilker and Meiners 2010); insect walking activities (Bown et al. 2002; Peiffer et al. 2009), or insect pheromones (Fatouros et al. 2008) can elicit changes in plants associated with defenses.

Elicitation of plant defenses

Most research has focused on the effect that salivary and/or digestive secretions of plant consumers have on the induction of plant defenses but also other consumption-related substances such as honeydew-sugars secreted by phloem feeding insects can be taken up by plants (Williams and Benson 1996) and have the potency to elicit defenses (Mosblech et al. 2008). Secreted salivary and digestive enzymes come into contact with damaged leaf tissue (Schilmiller and Howe 2005) during insect chewing and regurgitation (Peiffer and Felton 2009); when injected into host tissue via the stylets of, for example, aphids (Miles 1999; Will et al. 2009; Bos et al. 2010) or nematodes (Bellafiore et al. 2008); when deposited onto a leaf surface during stylet-sheath formation (Miles 1972;

Carolan et al. 2009); when injected via pathogen type-III or type-IV secretion apparatus or when deposited onto plant tissue via pathogen type-II secretion (Abramovitch et al. 2006). These secretions have in common that they can contain substances that serve to digest plant material in order to obtain nutrients and energy as well as substances that interact with plant defensive substances. In turn, these substances or conjugates can also be recognized by plants in such a way that they elicit a defense response.

Signal transduction in plant defenses

Plants respond to the combination of plant-consumer-derived elicitors and mechanical damage via sequential accumulation of plant hormones (O'Donnell et al. 2003), i.e. jasmonic acid (JA), ethylene (Et), and salicylic acid (SA) often accompanied by changes in abscisic acid (ABA) and auxin levels (Pieterse et al. 2009) followed by the accumulation of toxins either synthesized by the plant at the feeding site (Ferrari et al. 2003) or distally in which case they are transported to the feeding site (Baldwin and Karb 1995). Together with these toxins also protective enzymes accumulate at the feeding site (Zhu-Salzman et al. 2008) and in some cases they were found to exhibit synergistic properties as it was found that the toxin nicotine of the wild tobacco species *Nicotiana attenuata* restrains a herbivore's compensatory-feeding response evoked by plant-borne inhibitors of herbivore digestion (Steppuhn and Baldwin 2007). While prolonged pathogen infection can give rise to local cell death, i.e. the hypersensitive response (HR), and thereby prevents further spreading of pathogens (Dangl and Jones 2001) prolonged arthropod feeding was found to result in the production and release of plant volatiles potent in establishing indirect defenses by guiding foraging natural enemies of herbivores to their prey (Kessler and Baldwin 2001). Interestingly, plant-hormone signaling underlying these defense responses appeared often mutually exclusive since SA inhibits the JA-response and vice versa (Pieterse et al. 2009) while, in turn, the JA-response appeared essential for the formation of the endogenous signaling molecule methyl-salicylate in tomato (Ament et al. 2004) and tobacco (Park et al. 2007). Moreover, ABA was found to antagonize the SA-response (Zabala et al. 2009). Although the biological necessity for these antagonisms is unknown it was found that the network of sequential induction and suppression of phytohormones correlated with the presence of different components in herbivore saliva (Diezel et al. 2009).

Avoidance of plant defenses

Plant defenses pose selection pressure on plant-eaters and, as a result, herbivores and pathogens have evolved counter-adaptations to resist or manipulate plant defenses (Karban and Agrawal 2002). Some of these adaptations enable a plant eater to avoid a plant defense. For instance, the mirid bug *Pameridea roridulae* adapted to be able to walk on the sticky surface of the protocarnivorous plant *Roridula gorgonais* (Voigt and Gorb 2010) while the cotton bollworm *Helicoverpa armigera* was found to select *Arabidopsis thaliana* leaf tissue areas where the levels of defensive glucosinolates are the lowest (Shroff et al. 2008). Other insect species were found to remove leaf hairs that hamper feeding (Medeiros and Moreira 2005); to cut leaf veins or latex channels (Delaney and Higley 2006) or to isolate their feeding site via trenching (Chambers et al. 2007) to prevent distally produced defense compounds to be transported to it (Oppel et al. 2009). Gall-forming plant eaters avoid plant defenses by taking control over plant tissue locally forming a gall and forcing it to become a sink for photosynthates such that they can withdraw these substances from it (Tooker et al. 2008) reminiscent of the crown gall bacterium *Agrobacterium tumefaciens* (Deeken et al. 2006). Similarly, some species of nematodes induce the formation of feeding cells in plant roots which are also supplied with photosynthates by the plant and on which the nematode fully depends. These feeding cells are possibly controlled via a local nematode-hijack of plant auxin-metabolism (Gheysen and Fenoll 2002) and they were found to have down-regulated JA-responsiveness (Ithal et al. 2007). Like nematodes, also aphids do not physically remove tissue during feeding. Aphids withdraw phloem sap from the vascular bundle and although not causing much mechanical damage, plants respond to phloem feeders by sealing off the puncture wounds after stylet piercing (Walling 2008). Since these seals can efficiently block the insect's stylet-food channel, the saliva of some aphids (Carolan et al. 2009) contains proteins that antagonize these plant depositions to prevent feeding site occlusion (Giordanengo et al. 2010).

Suppression of plant defenses

Although there are indications that secreted herbivore defense-elicitors are under negative selection (Mori et al. 2001) many plant-consumers have also acquired traits, often in the form of secreted substances, that enable them to interfere with the plant's ability to properly organize its defenses after the plant has detected the herbivore (Zhu-Salzman et al. 2005) or pathogen (Metraux et al. 2009).

Suppression of RNAi

A central defense against viruses is RNA-interference (RNAi). RNAi is a mechanism during which a plant generates virus-specific small RNAs that form duplexes with viral nucleic acids which are subsequently degraded by plant nucleases (Katiyar-Agarwal and Jin 2010). A role for RNAi in direct defenses against

herbivores remains elusive (Pandey et al. 2008). However, some viruses suppress RNAi via production of proteins that scavenge (Bivalkar-Mehla et al. 2010) or modify small RNAs (Vogler et al. 2007) before they can bind to their RNA target. Moreover, the Cucumber Mosaic Virus produces suppressors that block the activity of the RNAi-specific nucleases (Zhang et al. 2006). Virus-induced RNAi augments the HR during which plant tissue is sacrificed in order to prevent pathogens from spreading (Dangl and Jones 2001).

Suppression of local tissue death

Some pathogenic fungi were found to produce so-called supprescins of induced plant defenses: these supprescins are small glycoproteins that delay the transcription of plant-genes involved in the production of toxins such as phytoalexins (Shiraishi et al. 1994). Moreover, at least two plant pathogenic fungi i.e. Fusarium wilt-causing *Fusarium oxysporum* (Pareja-Jaime et al. 2008) and Septoria leaf spot-causing *Septoria lycopersici* (Bouarab et al. 2002), were found to produce and secrete the enzyme tomatinase that converts the defensive alkaloid tomatine of tomato *Solanum lycopersicum* into harmless substances. Surprisingly, these tomatine-hydrolysis products were found to inhibit tomato defense signaling during infection showing that detoxification and suppression can operate in tandem. In addition, many bacterial phytopathogens secrete so-called effector molecules that play diverse but key-roles during the infection process. Effectors can be either associated with implementing the release of nutrients from the host tissue or with the suppression of local cell death and the deposition of callose in infected tissues. Interestingly, some of these effectors were found to operate as transcriptional or post-transcriptional repressors of plant defense genes while other were found to interfere with the activation of early signaling proteins or with their trafficking between different organelles or cells (Nomura et al. 2005; Metraux et al. 2009). In addition, it was found that the saliva of the green peach aphid *Myzus persicae* contains effectors that interfere with plant defenses e.g. by suppressing the flagellin-22 induced oxidative burst. Interestingly, *in planta* over-expression of two of these effectors reduced rather than increased aphid fitness suggesting that their individual action may be not always beneficial or only when produced in the appropriate amounts at the appropriate moment and location (Bos et al. 2010).

Suppression via the jasmonate-salicylate antagonism

Some effector substances appeared to specifically target plant defense-hormone signaling as *F. oxysporum* (Thatcher et al. 2009) and bacterial speck disease-causing *Pseudomonas syringae* DC3000 (Katsir et al. 2008) were found to use the JA-SA antagonism of plants to their own advantage.

P. syringae produces the JA-isoleucine-mimic coronatine that binds to the so-called COI-complex: under normal circumstances this COI-complex is activated by herbivore-induced JA-isoleucine initiating the degradation of transcriptional repressors of JA-dependent defense-genes thereby allowing these genes to be transcribed. Concomitantly, the binding of coronatine forces the plant to switch on its JA-defenses (Melotto et al. 2008) thereby inhibiting the SA-dependent defense responses to which these pathogens are vulnerable (Uppalapati et al. 2007). Herbivores were found to perform similar manipulations although the mechanisms behind these manipulations are still unknown. On Arabidopsis, the phloem-feeding sweetpotato whitefly *Bemisia tabaci* was found to suppress JA-defenses in an SA-dependent manner since they were found to develop slower on plants with a high level of JA-defenses or which had impaired SA-defenses compared to plants with a high SA-responsiveness or impaired in JA-defenses (Zarate et al. 2007). However, in Lima bean *Phaseolus lunatus*, whitefly-feeding inhibited not only Two-spotted spider mite *Tetyranychus urticae*-induced JA-accumulation and release of volatiles but also mite-induced SA-accumulation suggesting here the whitefly-mediated suppression was independent from SA (Zhang et al. 2009).

Suppression of direct and indirect defenses

How whiteflies induce and suppress plant defenses is unknown but it is likely that the inducing and suppressive agents emanate from the saliva they inject into their host plant via their stylets. Spider mites are also stylet feeders but feed from mesophyll cells and it was found that different genotypes of the Tea red spider mite *Tetranychus kanzawai* differentially induce SA-defenses in beans (Matsushima et al. 2006) while different genotypes of *T. urticae* appeared to differentially induce plant volatiles (Takabayashi et al. 2000). Most *T. urticae* genotypes induce simultaneously JA and SA accumulation in tomato leaflets (Kant et al. 2004) albeit possibly both with distinct spatio-temporal dynamics within leaflets. It was found that some *T. urticae* genotypes are resistant to the JA-dependent tomato defenses they induce (Ament et al. 2004) while others appeared susceptible to these. However, not all of these susceptible genotypes suffered from tomato defenses since some of them were found to suppress the induction of JA- and SA-dependent direct and indirect defenses such that they could uphold a high fitness. This suppression was strong enough to increase the fitness of non-suppressor genotypes co-inhabiting the feeding site (Kant et al. 2008). Recently it was discovered that yet another species from this genus, the Tomato red spider mite *T. evansi*, also suppresses both SA and JA-defenses in tomatoes but to a magnitude that is unprecedented since proteinase-inhibitor gene expression-levels as well as

their associated enzyme activities were lower in mite-infested plants compared to uninfested control plants (Sarmento et al. 2011). The mite suppresses the induction of JA-related volatiles but, surprisingly, its natural enemies the predaceous mites *Phytoseiulus longipes* and *P. macropilis* still responded to the odors of infested plants (Sarmento 2011). Possibly spider mites secrete suppressors via their saliva which was also suggested for some herbivores with haustellate mouthparts such as the Hessian fly *Mayetiola destructor* (Wu et al. 2008) and for some chewing herbivores such as the Colorado potato beetle *Leptinotarsa decemlineata* (Lawrence et al. 2008); the larvae of the Beet armyworm *Spodoptera exigua* (Weech et al. 2008) and those of the Corn earworm *Helicoverpa zea* (Musser et al. 2002). *H. zea* larvae produce the enzyme glucose oxidase which protects the herbivore against pathogens but also suppresses induced nicotine accumulation in tobacco *N. tabacum* (Eichenseer et al. 2010). In addition, *S. exigua* larvae with impaired salivary secretions induced stronger JA-levels and associated activity of defensive plant-enzymes than intact larvae on Arabidopsis (Weech et al. 2008) and it was shown that in *S. exigua* saliva distinct molecular modifications, such as protein phosphorylation, occur (Thivierge et al. 2010). Interestingly, it was shown that not only herbivore oral secretions but also their eggs, such as those of the Cotton leafworm *S. littoralis*, can be a source of agents that after active or passive release into plant tissue cause local suppression of the JA-pathway via induction of the SA-pathway such that newly hatched larvae benefit from leaf tissue being locally pre-suppressed (Bruessow et al. 2010). Finally, it was suggested that the mesophyll feeding western flower thrips *Frankliniella occidentalis* may abuse the JA-SA antagonism in an indirect fashion by vectoring viruses via their saliva that induce SA-responses and hence suppress JA-responses (Belliure et al. 2005). As a consequence, this appeared to make the plant also more suitable for competitors such as *T. urticae* (Belliure et al. 2010). This justifies the question if and when the benefits of adapting to resist plant defenses will outweigh the benefits of adapting to suppress these defenses, since such suppression may create equal opportunities for competitors.

To suppress or to resist plant defenses?

There are indications that herbivores that suppress plant defenses indeed put special efforts in monopolizing their feeding site. *T. evansi,* for example, produces massive amounts of webbing onto the leaf surface impenetrable to other herbivores such as *T. urticae* (Sarmento 2011). This raises the question whether we can predict under which circumstances the selective advantage for traits that enable herbivores to suppress defenses will be greater than that of traits making them resistant to defensive products and vice versa. Resistance to toxins often results from mutations that lead to target site insensitivity i.e. mutations that disrupt the negative interaction between target proteins of the herbivore and toxins but leave the basal function of that specific protein intact (Li et al. 2004; Despres et al. 2007; Van Leeuwen et al. 2008). We found that the fitness advantage of JA-defense-suppression appeared somewhat lower than the fitness advantage of JA-defense-resistance while, in turn, the fitness advantage of suppression was also observed on host plants other than tomato while that of the tomato-resistant genotype was not (Kant et al. 2008). This implies that the ability to resist host defenses may have a larger positive impact on a consumer's fecundity than the ability to suppress those but may also cause its host range to become narrower while the ability to suppress defenses may have a smaller positive impact on a consumer's fecundity while expanding its host range, provided that the mechanism of suppression targets conserved traits of the plant-defense machinery. Hence there may be host-range related trade-offs between the ability to suppress defenses and the ability to resist defenses that balance the persistence of these two traits within local herbivore populations living in a homogeneous or a heterogeneous plant environment as, for example, the species *T. urticae* was reported feeding from over 900 plant species comprising 124 different plant families (Egas et al. 2003) as a result from intraspecific variation since, clearly, not all the individuals of this species have the same host range (Kant et al. 2008). Hence, the challenge for the future is to determine how the fitness landscape of specialist and generalist herbivores is affected by their abilities to deal with host-plant defenses with regard to the spatial heterogeneity in the environment and to identify some of the basal signal-transduction networks and their genetic components that are targets for natural selection and give rise to directional and disruptive adaptive changes in how local herbivore populations cope with plant defenses.

References

Abramovitch RB, Anderson JC, Martin GB. 2006. Bacterial elicitation and evasion of plant innate immunity. Nature Rev Mol Cell Biol. 7:601–611.

Ament K, Kant MR, Sabelis MW, Haring MA, Schuurink RC. 2004. Jasmonic acid is a key regulator of spider mite-induced volatile terpenoid and methyl salicylate emission in tomato. Plant Physiol. 135:2025–2037.

Awmack CS, Leather SR. 2002. Host plant quality and fecundity in herbivorous insects Annu Rev Entomol. 47:817–844.

Baldwin IT, Karb MJ. 1995. Plasticity in allocation of nicotine to reproductive parts in *Nicotiana attenuata*. J Chem Ecol. 21:897–909.

Bellafiore S, Shen ZX, Rosso MN, Abad P, Shih P, Briggs SP. 2008. Direct identification of the *Meloidogyne incognita* secretome reveals proteins with host cell reprogramming potential. PLOS Pathogens. 4: Art. No. e1000192.

Belliure B, Janssen A, Maris PC, Peters D, Sabelis MW. 2005. Herbivore arthropods benefit from vectoring plant viruses. Ecol Lett. 8:70–79.

Belliure B, Sabelis MW, Janssen A. 2010. Vector and virus induce plant responses that benefit a non-vector herbivore. Basic Appl Ecol. 11:162–169.

Bivalkar-Mehla S, Vakharia J, Mehla R, Abreha M, Kanwar JR, Tikoo A, Chauhan A. 2010. Viral RNA silencing suppressors (RSS): novel strategy of viruses to ablate the host RNA interference (RNAi) defense system. Virus Res. 155:1–9. doi:10.1016/j.virusres.2010.10.003

Bos JIB, Prince D, Pitino M, Maffei ME, Win J, Hogenhout SA. 2010. A functional genomics approach identifies candidate effectors from the aphid species *Myzus persicae* (green peach aphid). PLoS Genet. 6(11): e1001216.

Bouarab K, Melton R, Peart J, Baulcombe D, Osbourn A. 2002. A saponin-detoxifying enzyme mediates suppression of plant defences. Nature. 418:889–892.

Bown AW, Hall DE, MacGregor KB. 2002. Insect footsteps on leaves stimulate the accumulation of 4-aminobutyrate and can be visualized through increased chlorophyll fluorescence and superoxide production. Plant Physiol. 129:1430–1434.

Bruessow F, Gouhier-Darimont C, Buchala A, Metraux JP, Reymond P. 2010. Insect eggs suppress plant defence against chewing herbivores. Plant J. 62:876–885.

Carolan JC, Fitzroy CIJ, Ashton PD, Douglas AE, Wilkinson TL. 2009. The secreted salivary proteome of the pea aphid *Acyrthosiphon pisum* characterised by mass spectrometry. Proteomics. 9:2457–2467.

Chambers JLE, Berenbaum MR, Zangerl AR. 2007. Benefits of trenching behavior in the context of an inducible defense. Chemoecol. 17:125–130.

Dangl JL, Jones JDG. 2001. Plant pathogens and integrated defence responses to infection. Nature. 411:826–833.

Deeken R, Engelmann JC, Efetova M, Czirjak T, Muller T, Kaiser WM, Tietz O, Krischke M, Mueller MJ, Palme K, et al. 2006. An integrated view of gene expression and solute profiles of Arabidopsis tumors: a genome-wide approach. Plant Cell. 18:3617–3634.

Delaney KJ, Higley LG. 2006. An insect countermeasure impacts plant physiology: midrib vein cutting, defoliation and leaf photosynthesis. Plant Cell Environ. 29:1245–1258.

Despres L, David JP, Gallet C. 2007. The evolutionary ecology of insect resistance to plant chemicals. Trends Ecol Evol. 22:298–307.

Diezel C, von Dahl CC, Gaquerel E, Baldwin IT. 2009. Different lepidopteran elicitors account for cross-talk in herbivory-induced phytohormone signaling. Plant Physiol. 150:1576–1586.

Divon HH, Fluhr R. 2007. Nutrition acquisition strategies during fungal infection of plants. FEMS Microbiol Lett. 266:65–74.

Eichenseer H, Mathews MC, Powell JS, Felton GW. 2010. Survey of a salivary effector in caterpillars: glucose oxidase variation and correlation with host range. J Chem Ecol. 36:885–897.

Egas M, Norde DJ, Sabelis MW. 2003. Adaptive learning in arthropods: spider mites learn to distinguish food quality. Exp Appl Acarol. 30:233–247.

Fatouros NE, Broekgaarden C, Bukovinszkine'Kiss G, van Loon JJA, Mumm R, Huigens ME, Dicke M, Hilker M. 2008. Male-derived butterfly anti-aphrodisiac mediates induced indirect plant defense. Proc Natl Acad Sci USA. 105:10033–10038.

Ferrari S, Plotnikova JM, De Lorenzo G, Ausubel FM. 2003. Arabidopsis local resistance to *Botrytis cinerea* involves salicylic acid and camalexin and requires EDS4 and PAD2, but not SID2, EDS5 or PAD4. Plant J. 35:193–205.

Gheysen G, Fenoll C. 2002. Gene expression in nematode feeding sites. Annu Rev Phytopathol. 40:191–219.

Giordanengo P, Brunissen L, Rusterucci C, Vincent C, van Bel A, Dinant S, Girousse C, Faucher M, Bonnemain JL. 2010. Compatible plant-aphid interactions: how aphids manipulate plant responses. C R Biologies. 333:516–523.

Hilker M, Meiners T. 2010. How do plants 'notice' attack by herbivorous arthropods? Biol Rev. 85:267–280.

Howe GA, Jander G. 2008. Plant immunity to insect herbivores. Annu Rev Plant Biol. 59:41–66.

Ithal N, Recknor J, Nettleton D, Maier T, Baum TJ, Mitchum MG. 2007. Developmental transcript profiling of cyst nematode feeding cells in soybean roots. Mol Plant-Microbe Int. 20:510–525.

Kant MR, Sabelis MW, Haring MA, Schuurink RC. 2004. Differential timing of spider mite-induced direct and indirect defenses in tomato plants. Plant Physiol. 135:483–495.

Kant MR, Sabelis MW, Haring MA, Schuurink RC. 2008. Intraspecific variation in a generalist herbivore accounts for induction and impact of host-plant defenses. Proc Royal Soc B: Biol Sci. 275:443–452.

Karban R, Agrawal AA. 2002. Herbivore offense. Annu Rev Ecol Syst. 33:641–664.

Katiyar-Agarwal S, Jin HL. 2010. Role of small RNAs in host-microbe interactions. Annu Rev Phytopathol. 48:225–246.

Katsir L, Schilmiller AL, Staswick PE, He SY, Howe GA. 2008. COI1 is a critical component of a receptor for jasmonate and the bacterial virulence factor coronatine. Proc Natl Acad Sci USA. 105:7100–7105.

Kessler A, Baldwin IT. 2001. Defensive function of herbivore-induced plant volatile emissions in nature. Science. 291:2141–2144.

Lawrence SD, Novak NG, Ju CJT, Cooke JEK. 2008. Potato, *Solanum tuberosum*, defense against colorado potato beetle, *Leptinotarsa decemlineata* (Say): microarray gene expression profiling of potato by colorado potato beetle regurgitant treatment of wounded leaves. J Chem Ecol. 34:1013–1025.

Li XC, Baudry J, Berenbaum MR, Schuler MA. 2004. Structural and functional divergence of insect CYP6B proteins: from specialist to generalist cytochrome P450. Proc Natl Acad Sci USA. 101:2939–2944.

Matsushima R, Ozawa R, Uefune M, Gotoh T, Takabayashi J. 2006. Intraspecies variation in the Kanzawa spider mite differentially affects induced defensive

response in lima bean plants. J Chem Ecol. 32:2501–2512.

Medeiros L, Moreira GRP. 2005. Larval feeding behavior of *Gratiana spadicea* (Klug) (Coleoptera: chrysomelidae: cassidinae) on its host plant, *Solanum sisymbriifolium* Lamarck (Solanaceae): interaction with trichomes. Coleopterists Bull. 59:339–350.

Melotto M, Mecey C, Niu Y, Chung HS1, Katsir L, Yao J, Zeng WQ, Thines B, Staswick P, Browse J, et al. 2008. A critical role of two positively charged amino acids in the Jas motif of Arabidopsis JAZ proteins in mediating coronatine- and jasmonoyl isoleucine-dependent interactions with the COI1F-box protein. Plant J. 55:979–988.

Metraux JP, Jackson RW, Schnettler E, Goldbach RW. 2009. Plant pathogens as suppressors of host defense. Plant Innate Immunity. 51:39–89.

Miles PW. 1972. The saliva of Hemiptera. Adv Insect Physiol. 9:183–240.

Miles PW. 1999. Aphid saliva. Biol Rev. 74:41–85.

Milewski AV, Madden D. 2006. Interactions between large African browsers and thorny Acacia on a wildlife ranch in Kenya. African J Ecol. 44:515–522.

Mori N, Alborn HT, Teal PEA, Tumlinson JH. 2001. Enzymatic decomposition of elicitors of plant volatiles in *Heliothis virescens* and *Helicoverpa zea*. J Insect Physiol. 47:749–757.

Mosblech A, Konig S, Stenzel I, Grzeganek P, Feussner I, Heilmann I. 2008. Phosphoinositide and inositolpolyphosphate signalling in defense responses of *Arabidopsis thaliana* challenged by mechanical wounding. Mol. Plant. 1:249–261.

Musser RO, Hum-Musser SM, Eichenseer H, Peiffer M, Ervin G, Murphy JB, Felton GW. 2002. Herbivory: caterpillar saliva beats plant defences – A new weapon emerges in the evolutionary arms race between plants and herbivores. Nature. 416:599–600.

Nomura K, Melotto M, He SY. 2005. Suppression of host defense in compatible plant-*Pseudomonas syringae* interactions. Curr Opin Plant Biol. 8:361–368.

O'Donnell PJ, Schmelz E, Block A, Miersch O, Wasternack C, Jones JB, Klee HJ. 2003. Multiple hormones act sequentially to mediate a susceptible tomato pathogen defense response. Plant Physiol. 133:1181–1189.

Oppel CB, Dussourd DE, Garimella U. 2009. Visualizing a plant defense and insect counterploy: alkaloid distribution in lobelia leaves trenched by a Plusiine caterpillar. J Chem Ecol. 35:625–634.

Pandey SP, Shahi P, Gase K, Baldwin IT. 2008. Herbivory-induced changes in the small-RNA transcriptome and phytohormone signaling in *Nicotiana attenuata*. Proc Natl Acad Sci USA. 105:4559–4564.

Pareja-Jaime Y, Roncero MIG, Ruiz-Roldan MC. 2008. Tomatinase from *Fusarium oxysporum* f. sp *lycopersici* is required for full virulence on tomato plants. Mol Plant-Microbe Int. 21:728–736.

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

Peiffer M, Tooker JF, Luthe DS, Felton GW. 2009. Plants on early alert: glandular trichomes as sensors for insect herbivores. New Phytol. 184:644–656.

Peiffer M, Felton GW. 2009. Do caterpillars secrete 'Oral Secretions'? J Chem Ecol. 35:326–335.

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

Rasman S, Köllner TG, Degenhardt J, Hiltpold I, Toepfer S, Kuhlmann U, Gershenzon J, Turlings TCJ. 2005. Recruitment of entomopathogenic nematodes by insect-damaged maize roots. Nature. 434:732–737.

Sabelis MW, Janssen A, Kant MR. 2001. The enemy of my enemy is my ally. Science. 291:2104–2105.

Sarmento RA. 2011. An acarine herbivore interferes with direct and indirect plant defenses [Ph.D. thesis]. [Amsterdam (NL)]: University of Amsterdam.

Sarmento RA, Lemos F, Bleeker PM, Schuurink RC, Pallini A, Oliveira MGA, Lima GR, Kant MR, Sabelis MW, Janssen A. 2011 (Forthcoming). A herbivore that manipulates plant defence. Ecol Lett. doi: 10.1111/j.1461-0248.2010.01575.x

Schilmiller AL, Howe GA. 2005. Systemic signaling in the wound response. Curr Opin Plant Biol. 8:369–377.

Shepherd RW, Bass WT, Houtz RL, Wagner GJ. 2005. Phylloplanins of tobacco are defensive proteins deployed on aerial surfaces by short glandular trichomes. Plant Cell. 17:2421–2421.

Shiraishi T, Yamada T, Saitoh K, Kato T, Toyoda K, Yoshioka H, Kim HM, Ichinose Y, Tahara M, Oku H. 1994. Suppressors: determinants of specificity produced by plant pathogens. Plant Cell Physiol. 35:1107–1119.

Shroff R., Vergara F, Muck A, Svatos A, Gershenzon J. 2008. Nonuniform distribution of glucosinolates in *Arabidopsis thaliana* leaves has important consequences for plant defense. Proc Natl Acad Sci USA. 105:6196–6201.

Steppuhn A, Baldwin IT. 2007. Resistance management in a native plant: nicotine prevents herbivores from compensating for plant protease inhibitors. Ecol Lett. 10:499–511.

Strauss SY, Rudgers JA, Lau JA, Irwin RE. 2002. Direct and ecological costs of resistance to herbivory. Trends Ecol Evol. 17:278–285.

Takabayashi J, Shimoda T, Dicke M, Ashihara W, Takafuji A. 2000. Induced response of tomato plants to injury by green and red strains of *Tetranychus urticae*. Exp Appl Acarol. 24:377–383.

Thatcher LF, Manners JM, Kazan K. 2009. *Fusarium oxysporum* hijacks COI1-mediated jasmonate signaling to promote disease development in Arabidopsis. Plant J. 58:927–939.

Thivierge K, Prado A, Driscoll BT, Bonneil E, Thibault P, Bede JC. 2010. Caterpillar- and salivary-specific modification of plant proteins. J Proteome Res. 9:5887–5895.

Tooker JF, Rohr JR, Abrahamson WG, De Moraes CM. 2008. Gall insects can avoid and alter indirect plant defenses. New Phytol. 178:657–671.

Traw MB, Dawson TE. 2002. Differential induction of trichomes by three herbivores of black mustard. Oecol. 131:526–532.

Uppalapati SR, Ishiga Y, Wangdi T, Kunkel BN, Anand A, Mysore KS, Bender CL. 2007. The phytotoxin coronatine contributes to pathogen fitness and is required for suppression of salicylic acid accumulation in tomato inoculated with *Pseudomonas syringae* pv. tomato DC3000. Mol Plant-Microbe Int. 20:955–965.

Van Leeuwen T, Vanholme B, Van Pottelberge S, Van Nieuwenhuyse P, Nauen R, Tirry L, Denholm I. 2008. Mitochondrial heteroplasmy and the evolution of insecticide resistance: non-Mendelian inheritance in action. Proc Natl Acad Sci USA. 105:5980–5985.

Vogler H, Akbergenov R, Shivaprasad PV, Dang V, Fasler M, Kwon MO, Zhanybekova S, Hohn T, Heinlein M. 2007. Modification of small RNAs associated with suppression of RNA silencing by tobamovirus replicase protein. J Virol. 81:10379–10388.

Voigt D, Gorb S. 2010. Locomotion in a sticky terrain. Arthropod-Plant Int. 4:69–79.

Walling LL. 2008. Avoiding effective defenses: strategies employed by phloem-feeding insects. Plant Physiol. 146:859–866.

Weech MH, Chapleau M, Pan L, Ide C, Bede JC. 2008. Caterpillar saliva interferes with induced *Arabidopsis thaliana* defence responses via the systemic acquired resistance pathway. J. Exp Bot. 59:2437–2448.

Will T, Kornemann SR, Furch ACU, Tjallingii WF, van Bel AJE. 2009. Aphid watery saliva counteracts sieve-tube occlusion: a universal phenomenon? J Exp Biol. 212:3305–3312.

Williams MW, Benson NR. 1966. Transfer of C14 components from *Psylla pyricola* (Foer.) to pear seedlings. J Insect Physiol. 12:251–254.

Wu JX, Liu XM, Zhang SZ, Zhu YC, Whitworth RJ, Chen MS. 2008. Differential responses of wheat inhibitor-like genes to Hessian fly, *Mayetiola destructor*, attacks during compatible and incompatible interactions. J Chem Ecol. 34:1005–1012.

Zabala MD, Bennett MH, Truman WH, Grant MR. 2009. Antagonism between salicylic and abscisic acid reflects early host-pathogen conflict and moulds plant defence responses. Plant J. 59:375–386.

Zarate SI, Kempema LA, Walling LL. 2007. Silverleaf whitefly induces salicylic acid defenses and suppresses effectual jasmonic acid defenses. Plant Physiol. 143:866–875.

Zhang XR, Yuan YR, Pei Y, Lin SS, Tuschl T, Patel DJ, Chua NH. 2006. Cucumber mosaic virus-encoded 2b suppressor inhibits Arabidopsis Argonaute1 cleavage activity to counter plant defense. Gen Dev. 20:3255–3268.

Zhang PJ, Zheng SJ, van Loon JJA, Boland W, David A, Mumm R, Dicke M. 2009. Whiteflies interfere with indirect plant defense against spider mites in Lima bean. Proc Natl Acad Sci USA. 106:21202–21207.

Zhu-Salzman K, Bi J-L, Lui T-X. 2005. Molecular strategies of plant defense and insect counter-defense. Insect Sci. 12:3–15.

Zhu-Salzman K, Luthe DS, Felton GW. 2008. Arthropod-inducible proteins: broad spectrum defenses against multiple herbivores. Plant Physiol. 146:852–858.