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Role of phytohormones in insect-specific plant reactions

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Abstract

The capacity to perceive and respond is integral to biological immune systems, but to what extent can plants specifically recognize and respond to insects? Recent findings suggest that plants possess surveillance systems that are able to detect general patterns of cellular damage as well as highly specific herbivore-associated cues. The jasmonate (JA) pathway has emerged as the major signaling cassette that integrates information perceived at the plant–insect interface into broad-spectrum defense responses. Specificity can be achieved via JA-independent processes and spatio-temporal changes of JA-modulating hormones, including ethylene, salicylic acid, abscisic acid, auxin, cytokinins, brassinosteroids and gibberellins. The identification of receptors and ligands and an integrative view of hormone-mediated response systems are crucial to understand specificity in plant immunity to herbivores.

Know your enemy – a golden rule of plant defense?

“If you know your enemies and know yourself, you can win a hundred battles without a single loss”, states Sun Tzu in his ancient military treatise *The Art of War*. Plants, as primary producers of organic matter in terrestrial ecosystems, must continuously resist a multitude of attackers and, unlike the armies of Sun Tzu, do not have the option of retreating to safe ground. Have plants nevertheless evolved the capacity to “know” the attacking enemies and adjust their defenses accordingly? In this review we use the paradigm of molecular specificity in plant–pathogen interactions as a framework to discuss potential mechanisms by which plants specifically recognize and respond to insect herbivores.

Plants recognize herbivores via mechanical and chemical cues

An appropriate defense response to a biotic threat requires initial recognition. Pathogens are recognized when conserved patterns of microbial molecules called microbe- or pathogen-associated molecular patterns (MAMPs or PAMPs) are detected by pattern recognition receptors (PRRs) on the surface of the host plant cell, leading to PAMP-triggered immunity (PTI; Figure 1). Damage-associated molecular patterns (DAMPs), which are endogenous

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molecules that are produced by the plant after infection, are also recognized by PRRs to trigger defensive reactions [1]. Pathogens can evade this innate immune response through the action of effector proteins that, upon delivery into the host cell, suppress PTI. Some plant genotypes again contain disease resistance (R) proteins that specifically recognize pathogen effectors, resulting in effector-triggered immunity (ETI) [2]. Although the PTI/ETI model is sometimes regarded as an oversimplification [3], the molecular identification of the involved ligands and receptors has enabled conclusions to be drawn about the specificity of recognition in plant–pathogen interactions: in general, PTI is based on non-specific recognition of common microbial molecules, whereas ETI is triggered by highly pathogen-specific compounds [4].

To what extent can the PTI/ETI model inform research aimed at elucidating the specificity of recognition in plant–herbivore interactions? In comparison to pathogens, insects are highly complex multicellular organisms with various lifestyles and behavioral patterns. Cues emanating from these patterns may be used by the plant to recognize the threat of herbivory and to mount appropriate defensive responses [5] (Figure 1).

The first contact with the herbivore often occurs at the leaf surface when the tarsi of an arriving insect touch the leaf surface. Landing and walking on a plant will exert pressure, break trichomes, and deposit chemicals from tarsal pads on the leaf [5]. Plants have evolved mechanisms to sense pressure. The Venus fly trap (*Dionaea muscipula*), for example, closes immediately when its sensory hairs are stimulated by insects [6]. Non-carnivorous plants are also highly sensitive to touch [7]. In at least some cases, mechanostimulation by repeated touching is sufficient to induce the accumulation of jasmonic acid (JA) [8], the precursor of the defense hormone jasmonoyl-L-isoleucine (JA-Ile). Breaking of tomato (*Solanum lycopersicum*) leaf trichomes by adult moths or caterpillars induces hydrogen peroxide (H₂O₂) formation and expression of defensive proteinase inhibitors [9]. To date, there is no indication that this type of 'early warning' response is specific for particular insect species, and the observed effects may be mostly related to DAMP-like effects (see below).

Oviposition represents another opportunity for plants to detect insect herbivores. The formation of necrotic zones following egg deposition has been observed in black mustard (*Brassica nigra*) and certain potato (*Solanum* spp.) clones [10, 11]. In pea (*Pisum sativa*) plants, long-chain alpha,omega-diols (bruchins) deposited during oviposition by pea weevils (*Bruchus pisorum*) on pea pods trigger the formation of undifferentiated cells beneath the eggs, which increase plant resistance by hindering the larvae when they try to burrow into the pod [12]. Oviposition can be accompanied by wounding, and in the interaction between the elm leaf-beetle (*Xanthogaleruca luteola*) and the field elm (*Ulmus minor*), for example, oviduct secretions induce defenses only when they are released into oviposition wound sites [13]. Overall, some oviposition-associated cues seem to act as MAMP-like molecular patterns that can be used by plants to recognize and predict herbivore attack. Consistent with the PTI/ETI framework, oviposition effectors may be produced by herbivores to suppress the plant immune response (Box 1). Taken together, these findings suggest that oviposition events trigger plant defense reactions in an insect- and potentially even species-specific manner.

Herbivory disrupts the integrity of plant tissue, and many plant defense responses can be triggered by mechanical wounding alone [14–16], leading to wound-induced resistance (WIR). Extensive studies of the wound response in model plants such as tomato and Arabidopsis (*Arabidopsis thaliana*) have identified plant-derived compounds that trigger anti-insect defense responses. Such compounds are potentially recognized by PRRs and, thus, can be defined conceptually as DAMPs [17]. Among the DAMPs shown to activate anti-insect defenses in tomato are cell wall-derived oligosaccharides and the peptide signal

systemin [18]. These studies lend support to the idea that many plant defense responses against herbivores are mediated by recognition of the plant's 'damaged self' [17, 19]. Analogous to danger signal models in the vertebrate immune system, when plant tissue suffers mechanical damage this is likely to disrupt intracellular compartmentalization in ways that lead to the production of molecules that trigger general plant immune responses.

It is becoming clear that a second layer of perception in addition to WIR can enable plants to detect herbivores more specifically: plants seem to recognize compounds that are released by herbivores during feeding. Extensive genetic analysis of the Hessian fly (*Mayetiola destructor*)–wheat (*Triticum* spp.) interaction [20, 21] and the cloning of receptor-like resistance (*R*) genes have demonstrated that there is a high degree of specificity of perception in this case [22–24]. The recognition systems for hemipteran and dipteran parasites, together with the identification of possible effectors (Box 1), appear to conform to the general PTI/ETI theory [25]. Less is known about the mechanisms by which plants perceive chewing insects such as beetles and caterpillars, which constitute the vast majority of plant herbivore species. Numerous studies have shown that insect oral secretions, when applied to artificial wound sites, amplify the wound response of the plant [26–29]. Identified elicitors include fatty acid-amino acid conjugates (FACs), sulfur-containing fatty acids (caeliferins), peptides from digested plant proteins, and lipases [30–34]. Based on their eliciting activity, at least the insect-derived compounds can be conceptually classified as herbivore-associated molecular patterns (HAMPs), which presumably are recognized by PRRs at the cell surface [35, 36] to trigger HAMP-induced immunity (HTI). Even though HAMP receptors have not been identified, several trends have emerged concerning the specificity of elicitor-mediated recognition of chewing herbivores by plants. First, herbivore-derived elicitors boost the amplitude of wound-induced defense responses [30, 31, 33, 34, 37]. Second, different herbivore species produce qualitatively and quantitatively different elicitor combinations [38, 39]. Third, the activity of the different known elicitors varies between plant species [37]. Taken together, these observations suggest the potential for elicitor-mediated, species-specific recognition of chewing insects by plants.

Plant hormone regulatory networks integrate different herbivore recognition cues

Following the recognition of an attacker, plants use different signaling cascades to reprogram their phenotype. Extended PTI/ETI models in plant–pathogen interactions suggest that although the recognition of pathogens can be very specific, plants have a “common downstream signaling machinery” [40] that is activated upon recognition of many different attackers. To what extent is this paradigm valid for plant–insect interactions? The jasmonate signaling cascade, including the wound hormone JA-Ile, is widely considered to be a master regulator of plant resistance to arthropod herbivores as well as various pathogens [15, 17, 41–45], and jasmonates may therefore represent the core signaling pathway for activating resistance to insects.

Disruption of plant tissue integrity during insect feeding triggers the production of JA-Ile and the activation of a well-defined signal transduction chain leading to transcriptional activation of defense responses. Thus, it is the defining feature of most, if not all herbivores, namely the need to obtain nutrition from plant tissues, which betrays the presence of the attacker to the host. A major unresolved question is the extent to which herbivore-induced production of JA-Ile is promoted by signals originating from the plant's (i.e. DAMPs, “self”) versus signals from the herbivores (i.e. HAMPs, “non-self”) [19]. Mechanical wounding is sufficient to trigger robust local and systemic increases in JA-Ile levels within minutes of leaf injury, which indicates that herbivore-associated cues are not strictly required to activate the response [17, 46, 47]. However, the severity of crushing-type

wounds typically used in these studies may bypass a requirement for HAMPs in the elicitation of herbivore-induced responses. Research aimed at identifying herbivore-derived elicitors has therefore relied on the application of insect oral secretions to wound sites created by mild wounding regimens that do not elicit a strong defense response in the absence of oral secretion [35, 36, 48–50]. Importantly, defense responses are attenuated in leaf-feeding lepidopteran herbivores that lack known elicitors in their oral secretions, lending support to the concept that wound-induced responses at an ecologically relevant intensity are potentiated by recognition factors [51].

We argue here that the JA pathway represents a conserved core-signaling machinery that is activated by both non-specific and specific recognition patterns following herbivore attack. But how do plants fine tune their defense machinery to appropriately mount herbivore-specific responses beyond jasmonates? We propose two potential answers to this question. First, plants may use JA-independent, parallel pathways to create distinct response patterns. Second, specificity may be mediated through the activation of spatio-temporal modulators of the JA-response (Figure 2). Evidence for the first concept comes from studies on the recognition and response system of tomato to the potato aphid *Macrosiphum euphorbiae*. *Mi-1*, a putative receptor, triggers salicylic acid (SA)-mediated signaling [52] and resistance independently of the JA pathway [53]. Plant recognition of and response to many other hemipterans seems to follow a similar pattern [54], which suggests that plants use JA-independent hormone response pathways to achieve specific resistance against phloem feeders.

However, most herbivores inflict much greater cell damage than phloem feeders, and will activate JA signaling and resistance. In this case, specificity may be achieved via cross-talk with other hormones (Figure 3). Indeed, JA-induced changes in gene expression typically depend on the context in which the hormone is perceived [55],[56]. The best-studied hormones that alter JA-mediated defense responses and herbivore resistance are SA and ethylene (ET). In general, SA antagonizes JA-induced resistance, whereas ET can have both positive and negative effects. For ET, some of the transcriptional responses that are modulated by crosstalk with JA were recently shown to be mediated by the ET-stabilized transcriptional regulator EIN3 [57]. Several studies have shown that SA and ET are specifically modulated by different herbivore elicitors [29, 58] and may thereby provide a degree of hormone-mediated specificity. A striking example of how herbivore-induced SA-signaling can modulate JA-dependent defenses comes from research on *A. thaliana*. Oviposition by the cabbage butterfly (*Pieris brassicae*) induces SA accumulation and reduces the induction of JA-responsive genes, leading to reduce plant resistance against *S. littoralis* [59]. SA–JA–ET crosstalk has been reviewed in detail elsewhere [60, 61] (see also other reviews in this special issue). However, abscisic acid (ABA), auxins, gibberellins (GB), cytokinins (CK) and brassinosteroids (BR) have received less attention as potential factors that modulate herbivore resistance. The following discussion highlights examples from the recent literature that indicate that these hormones play an important role in mediating specificity in herbivory-induced defense responses as well.

Absciscic acid

ABA levels in maize (*Zea mays*) are increased during attack by the specialist root herbivore western corn rootworm (*Diabrotica virgifera virgifera*), but not by mechanical wounding alone [27, 62], and in *Arabidopsis* after induction with wounding and the oral secretions of the desert locust (*Schistocerca gregaria*), a generalist herbivore [34]. ABA levels also increased in a goldenrod species (*Solidago altissima*) after induction by the tobacco budworm (*Heliothis virescens*) caterpillar, but not by the gall-inducing caterpillar *Gnorimoschema gallaesolidaginis* [63]. ABA synthesis and signaling affect herbivore-induced transcript levels and JA biosynthesis in *Arabidopsis* [64, 65], JA-inducible defense

responses in maize [27] and resistance to herbivores in tomato [66]. ABA and JA synergistically induce MYC2-dependent gene expression during wound responses. MYC2 encodes a nuclear localized basic helix-loop-helix-type transcription factor which acts as both activator and repressor of JA-mediated gene expression and serves as an integration point between ABA and JA signaling [67–69]. In tobacco (*Nicotiana tabacum*), JA also regulates the expression of *NtPYL4*, a gene belonging to the PYR/PYL/RCAR family, which encodes an ABA receptor protein thereby affecting ABA-induced levels of root alkaloids [70]. The same study demonstrated that *AtPYL4* and *AtPYL5* mutants in *Arabidopsis* are more sensitive to JA-induced growth inhibition and less sensitive to JA-induced anthocyanin accumulation. Although the molecular mechanisms behind ABA–JA crosstalk are still elusive, recent findings suggest that both pathways share similar regulatory proteins. The co-repressor TOPLESS (TPL) interacts with EAR-motif (ethylene-responsive element binding factor-associated amphiphilic repression) proteins to repress transcription of genes involved in several hormone pathways [71]. The EAR-motif protein NINJA (Novel Interactor of JAZ) connects TPL to the JAZ complex, thereby mediating repression of genes demarcated by JAZ-bound transcription factors such as MYC2. TPL also interacts with NINJA-related proteins that are part of a complex that mediates ABA-induced degradation of negative transcriptional regulators [72]. Taken together, these findings indicate that ABA and JA are tightly interconnected and that regulation of ABA levels in response to herbivory can modulate JA-driven defense responses (Figure 3). However, because ABA is also an important signal for responses to desiccation, which is an effect that accompanies herbivore attack in many cases, it remains to be determined to what extent this stress hormone is involved in recognition-mediated responses to insect feeding. The application of Egyptian cotton leafworm (*Spodoptera littoralis*) regurgitant to *Arabidopsis* can reduce wound-induced stomatal closure and water loss [73], whereas *Schistocerca gregaria* oral secretions induce ABA levels [34], which suggests specific elicitor-mediated regulation of this hormone.

Auxin

Levels of the auxin indole-3-acetic acid (IAA) are elevated in plants attacked by gall-feeding insects [63, 74]. By contrast, IAA levels in the leaves of a species of wild tobacco (*Nicotiana attenuata*) are reduced within three days after simulated herbivory [75]. It is known that plant resistance to pathogens can be modulated through changes in auxin sensitivity. For example, the perception of the bacterial elicitor flagellin decreases auxin sensitivity, thereby elevating resistance to *Pseudomonas syringae* [76]. Concomitantly, *P. syringae* suppresses host defense by promoting auxin production via delivery of effectors into the plant cell [77, 78]. Treatments with synthetic auxin directly suppress SA-induced defense responses [79], which can be linked to SA-mediated resistance to phloem-feeding insects [52]. Whether insects that are negatively affected by SA-mediated defenses can alter auxin homeostasis or signaling to suppress host defense is not known. In addition to regulation of SA signaling, studies have suggested that there is an intimate molecular interplay between auxin and JA signaling: auxin formation in *Arabidopsis* roots is enhanced by JA-mediated induction of genes involved in auxin biosynthesis and transport [69, 80]. In *N. attenuata* leaves, JA negatively regulates wound-induced decreases in auxin content [75], demonstrating that the effects of JA on auxin biosynthesis are tissue specific and possibly also species specific [55]. Importantly, these data suggest that herbivore-induced JA levels might affect auxin homeostasis. Conversely, there is evidence to indicate that auxin enhances JA biosynthesis and signaling [81–83]. JAZ1 and MYC2 are co-regulated by auxin and JA, demonstrating the potential for crosstalk between both hormones [82, 83]. Analogous to ABA signaling, EAR-motif containing AUX/IAA proteins, which are negative regulators of auxin-induced responses, also interact with TPL [72], suggesting that TPL acts as an integrator of multiple hormone pathways. Another protein that links auxin and JA responses is SGT1 (suppressor

of G-two allele of SKP1), which connects chaperone-mediated protein assembly and ubiquitin-mediated protein degradation. *SGT1* mutants of *Arabidopsis* are compromised in their sensitivity to both auxin and JA [84], and silencing *SGT1* in *N. attenuata* attenuates JA levels, defense metabolite accumulation, and resistance to the tobacco hornworm (*Manduca sexta*) caterpillar [85]. Auxin can also regulate plant defense responses independently of SA and JA [86, 87]. These findings demonstrate that auxin is a potent modifier of herbivore-relevant defense responses and indicate that plants may modulate auxin levels to mediate attacker specificity.

Gibberellins

Studies with plants altered in gibberellin (GB) signaling have suggested a role for GB in herbivore-induced defense responses. DELLA proteins are negative transcriptional regulators of gibberellic acid (GA)-induced gene expression and are considered to play key roles in integrating plant responses to diverse developmental and environmental stimuli [88]. Remarkably, GAs affect JA signaling through competitive binding of DELLAs to the JAZ proteins, thereby preventing JAZ–MYC2 interaction and promoting MYC2-induced transcriptional responses [89]. GA perception leads to degradation of DELLAs, which ultimately leads to inhibition of MYC2 and diminished JA responses. Accordingly, alteration of DELLA levels affects JA biosynthesis and signaling [90, 91].

Cytokinins

In *N. attenuata*, cytokinin (CK)-related transcripts are among the genes that are most strongly regulated by FAC elicitors [92, 93], suggesting that CK has a role in the hormonal regulatory network. In addition, gall-forming insects and possibly some leaf miners modulate plant CK levels, presumably to maintain the sink status of the infected tissues [63, 74, 94]. Isopentenyltransferases (IPT) represent the rate-limiting step in CK biosynthesis, and IPT overexpression increases resistance of common tobacco to the lepidopteran herbivore *M. sexta* [95]. Several lines of evidence also support an important role for CK in the activation of JA biosynthesis. Transgenic tobacco (*Nicotiana tabacum* cv. Xanthi nc) plants that overexpress a small GTP-binding protein accumulate high levels of CK, resulting in increased rates of JA production after wounding, a response that can be mimicked by long-term CK treatments [96]. Furthermore, CK treatments of hybrid poplar (*Populus* sp.) leaves increase the wound-induced JA burst and the expression of genes involved in JA biosynthesis [97]. The same study also shows that wounding and CK treatments of sink but not source leaves impairs gypsy moth (*Lymantria dispar*) larval performance, suggesting that CK-mediated resistance to insects depends on leaf ontogeny. CK levels in leaves are thought to be regulated by ontogenic constraints because the hormone accumulates to high levels in younger leaves, whereas reduced CK levels promote leaf senescence [98, 99]. Because CKs modulate herbivore-induced defenses, the CK status of a given tissue might determine the intensity of the defense response of that particular tissue after perception of herbivory and, thus, contribute to tissue-specific responses in herbivory-induced signaling [100] (Figure 2).

Brassinosteroids

Recent findings have also suggested important roles for brassinosteroids (BR) in herbivore resistance. BRs antagonize JA-mediated trichome density and defense metabolite accumulation in tomato [101]. BRs are also known to repress JA-governed inhibition of root growth [102]. BR are perceived by BR insensitive 1 (BRI1), a leucine-rich repeat receptor-like kinase [103, 104]. BRI1-associated kinase 1 (BAK1) interacts with BRI1 and plays an essential role in BR signaling [105]. Apart from BR signaling, BAK1 also interacts with the flagellin receptor FLS2 and is required for multiple MAMP-elicited responses [106]. Silencing BAK1 in *N. attenuata* reduces wound- and herbivory-induced JA and JA-Ile levels

and JA-induced trypsin proteinase inhibitor (TPI) activity [107]. Whether or not BAK regulates HAMP or DAMP perception to modulate JA levels, or whether the effects in BAK1-silenced plants are due to changes in BR perception requires further analysis [107].

Taken together, these examples illustrate the many possibilities plants have to modulate the JA-pathway to achieve specific responses. However, apart from JA/SA crosstalk (see Whitham and colleagues in this special issue), clear examples of specific induction of JA-modulators following herbivore recognition remain scarce. The identification of herbivore receptors followed by in-depth analysis of their downstream targets should help to fill this gap of knowledge. Another open question is whether the JA-pathway and its modulating factors function in a similar manner in different plant species. Inter-specific comparative approaches would be important to be construct generalized hormonal networks that mediate specificity.

Do recognition-induced plant hormone networks trigger specific and appropriate defense responses?

The recognition systems used by plants to perceive herbivore attack are integrated with hormone response pathways that reprogram the plant. However, what evidence is there that the resulting responses are specific and appropriate (see Glossary) for defense against the attacking herbivore? Again, for hemipterans, compelling examples of gene-for-gene resistance link specific recognition to both specific and appropriate responses [54]. In the case of chewing herbivores, many studies have demonstrated the differential responses of plants to different insect species as well [108–111], but to date evidence that these responses provide specific resistance is rare. Given the complexity of host transcriptional responses to herbivory, some of these differences may be attributed to a variety of experimental factors that influence the response. Plant growth conditions, plant and insect developmental stage, herbivore density and treatment duration are among the parameters that are expected to have major effects on transcript profiles. Also, in some cases, different insects from the same feeding guild elicit similar or converging responses via the general JA-signaling cassette [112–115]. The question thus arises whether from an adaptive point of view plants benefit from tailoring their response to different chewing herbivores, or whether a generalized response following recognition is the most pertinent strategy?

There is evidence to indicate that among the multitude of defenses induced by one herbivore species, some responses target specific types of herbivores, even within the same feeding guild. One example comes from work on the JA-regulated defensive enzyme threonine deaminase (TD2), which degrades the essential amino acid Thr in the lepidopteran gut [116]. Although TD2 expression in tomato leaves is induced in response to attack by both beet armyworm (*Spodoptera exigua*) and cabbage looper (*Trichoplusia ni*) caterpillars and by the Colorado potato beetle (*Leptinotarsa decemlineata*), the defensive activity of the enzyme is only activated in the gut of lepidopteran herbivores, not in the gut of the coleopteran herbivore [117]. Levels of the JA-regulated non-protein amino acid *N*^δ-acetylornithine in *Arabidopsis* increases in response to feeding by larvae of the small white (*Pieris rapae*) and the diamondback moth (*Plutella xylostella*) as well as the green peach aphid (*Myzus persicae*), but this defense appears to target only the aphids [45]. Also, distinct patterns of volatile compounds have been shown to be induced different herbivores, leading to specific attraction of natural enemies [118, 119] (see McCormick and colleagues in this special issue). However, also in this case, it remains open whether the differences in the plants reaction are based on specific recognition patterns.

Overall, generalist and specialist herbivores might be susceptible to different types of defenses, and plants may benefit from detecting highly adapted herbivores and adjusting

their regulation of quantitative and qualitative direct and indirect defenses. Specialist herbivores in particular may have found ways to suppress plant defenses (Box1) or to circumvent them via behavioral adaptations, which, in analogy to the PTI/ETI model in plant-pathogen interactions, may have led to the counter-evolution of specifically adapted defense mechanisms in plants [120, 121]. Until today, few mechanistic examples of plant-counter adaptations to specialists are known (but see other articles in this special issue), and further research is required to disentangle whether differential responses of plants to chewing herbivores are truly specific, and whether plants have evolved to tailor their response to different chewing attackers.

Conclusions and future directions: piecing together the recognition–response puzzle

A recurring theme in all spheres of plant–herbivore biology is the ability of each player to perceive and respond to cues generated by the other; this exchange of information provides an excellent focal point for elucidating basic chemical and molecular principles of plant–herbivore interactions. Understanding the mechanisms behind perception and response may also inform studies about their evolutionary history. In contrast to well-established models describing the evolution of plant–pathogen interactions [122], our understanding of how molecular recognition and response systems shape plant–herbivore relationships is still in its infancy, and many important questions remain unanswered (see Box 2). Nevertheless, the literature supports several general conclusions about specificity in plant–herbivore interactions. First, plants perceive different arthropods by integrating various environmental cues, ranging from mechanostimulation by insects walking on plant surfaces to contact with salivary components during feeding. Second, the perception of herbivores triggers regulatory responses that include different phytohormones, with the JA pathway playing a dominant role in host resistance. Third, although JA signaling is highly conserved, it is becoming increasingly clear that multiple hormone response pathways interact to translate initial perception events into appropriate responses that increase plant fitness in the presence of hostile aggressors.

Box 1. Herbivore effectors

Just as plants recognize a variety of herbivore-derived cues, there is evidence that herbivores can use effector molecules to suppress plant defenses:

- Oviposition fluids trigger the SA pathway in *Arabidopsis*, which increases the growth of Egyptian cotton leafworm (*Spodoptera littoralis*) larvae [124, 125]. Oviposition can also suppress herbivore-induced plant volatiles in maize [126].
- During feeding, insects secrete effector-like compounds to suppress plant immunity. The best known example is glucose oxidase produced by the salivary glands of various lepidopteran insects [26]. Many aphids also produce effector-like compounds [127], and other examples of herbivore-mediated suppression of plant defenses via as yet unknown mechanisms have also been documented [36, 73].
- Herbivores can produce plant hormones or hormone mimics to manipulate the host defense responses [128].
- Insect herbivores are hosts to microorganisms (e.g. endosymbionts) and surface-dwelling parasites that produce compounds that potentially interfere or otherwise affect plant immunity [36]. For example, a recent study suggests that *Wolbachia* endosymbionts suppress the induction of maize genes involved in defense against the Western corn rootworm (*Diabrotica virgifera*), which feeds

on roots [129]. Bacterial symbionts are also involved in the production of cytokinins that are secreted by the larvae of a leaf-miner moth (*Phyllonorycter blancardella*) to inhibit leaf senescence to maintain a source of food for the larvae [130].

The PTI/ETI paradigm indicates that plants have evolved various ways to recognize and respond defensively to pathogen effectors. Have plants acquired the capacity to recognize insect effectors as well? Although there is evidence to suggest that this might indeed be the case for hymenopterans [131], the ligands and receptors that constitute this form of recognition have yet to be identified and characterized [127]. Future research focusing on the identification of insect effectors and their mechanism of action is likely to mark a new phase in plant–insect interaction research.

Box 2. Outstanding questions

- Which receptors are involved in plant perception of herbivores?
- How prevalent are herbivore effectors and how do they act to suppress the plant immune system?
- Are herbivore effectors recognized by plant *R* genes in accordance with the ETI/PTI model in plant–pathogen interactions?
- How do plants integrate information derived from multiple herbivore- and plant-derived cues?
- Which JA-independent processes mediate specific plant responses to herbivore attack?
- What is the precise role of growth hormones (gibberellins, cytokinins, auxin and brassinosteroids) in modulating plant immunity to herbivores?
- Is the recognition of a specific chewing herbivore translated into a distinct defense response?

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Glossary

Appropriate response	a phenotypical change following herbivory that provides a benefit to the plant. This benefit can be realized either by increasing resistance and fending off the attacker, or by changing the primary metabolism to enable a more effective regrowth after attack. Appropriate responses are not necessarily based on specific recognition and specific metabolic changes, as plants can use general mechanisms to defend themselves against a variety of attackers. Different chewing herbivores are likely to be susceptible to the same defensive mechanisms. On the other hand, phloem feeders might require different measures of protection because they only feed on specialized cells. From an adaptive point of view, truly specific responses can be expected to be appropriate.
Direct crosstalk	a phenomenon in which two or more hormone pathways either share a common signaling component e.g. the use of co-repressor TOPLESS (TPL) by both the jasmonic acid and auxin pathways, or contain components that physically interact to modify the signal output (e.g. the JAZ–DELLA interaction). The biological significance of direct crosstalk in shaping the outcome of plant–insect interactions remains to be demonstrated.

Hormone crosstalk	a phenomenon in which a signal transmitted through one hormone pathway stimulates or represses signal output (e.g., a physiological or defense-related response) from another signaling pathway. Interactions between the hormone signals can be direct or indirect (see direct and indirect crosstalk).
Indirect crosstalk	a common phenomenon in which two or more hormone pathways are integrated at the hormone response gene-network level rather than at the upstream level of signal transduction. One example is the jasmonic acid-induced expression of <i>NtPYL4</i> , which affects the ability of abscisic acid (ABA) to regulate alkaloid production in tobacco.
Specificity of recognition	the extent to which a plant can discriminate the presence of and/or attack by different herbivores. Specific recognition of arthropod herbivores can occur at different levels, ranging from phyla (i.e. distinct detection of arthropods compared with vertebrates) to species (i.e. distinct detection of two different herbivore species). Little is known about the molecular mechanisms underlying plant recognition of herbivores; however, generalized examples based on the pathogen-associated molecular pattern-triggered immunity (PTI)/ effector-triggered immunity (ETI) paradigm are informative. For example, a high degree of specificity in recognition could be achieved by <i>R</i> gene products that evolved to recognize effector molecules in adapted insects. Low-level specificity might involve the action of mechanosensors that detect insect movement on the leaf surface. Receptor-mediated recognition of herbivore-associated molecular patterns (HAMPs) and/or damage-associated molecular patterns (DAMPs) produced at the site of insect feeding is expected to provide an intermediate level of specificity because these signals are common in plant interactions with multiple insect species.
Specificity of response	the extent to which plant physiological and/or metabolic changes elicited by the specific perception of a given herbivore are distinct from changes elicited by the perception of other attackers. Unlike the adaptive immune system in animals, which creates an immunological memory of a specific invading pathogen, the recognition of many insects (e.g. chewing herbivores) is channeled into a general defense response. Many measured differences in responses are not based on specific perception but are likely to be artifacts of secondary stress factors. These responses are referred to as 'distinct' or 'different' but not 'specific'. Examples of specific responses are the different phenotypical changes triggered by different putative aphid receptors [123].

Phytohormone signaling and induced resistance

Figure 1.

Molecular recognition of pathogens and herbivores by plants. 1. Microbe-, pathogen- and damage-associated molecular patterns (MAMPs, PAMPs and DAMPs) are recognized by pattern recognition receptors (PRRs) and lead to PAMP-triggered immunity (PTI). 2. Pathogen effectors suppress PTI. 3. Resistance gene products recognize effectors and lead to effector-triggered immunity (ETI). 4. Oviposition-associated compounds are recognized by unknown receptors and trigger defensive responses. 5. Putative herbivore-associated molecular patterns (HAMPs) are recognized by receptors and lead to herbivore-triggered immunity (HTI). 6. Wounding leads to the release of DAMPs and to wound-induced resistance (WIR). 7. Effector-like molecules from insects can suppress HTI and WIR. Uncharacterized elements are indicated by broken lines.

Figure 2.

Perception triggers herbivore- and tissue-specific hormonal network responses. **(a–d)** Conceptual kinetics of three hypothetical phytohormones are shown by solid and broken, black and gray lines. Panels **(a–c)** with a white background represent the same tissue, whereas panel **(d)** with a darker background represents a different tissue or tissue age. **(a,b)** Different herbivores can elicit different hormonal responses. **(c,d)** Hormonal responses to the same herbivore can show tissue- or age-specific differences. A hypothetical hormone-responsive transcriptional network is then triggered by the different hormones. This network is represented here by specific transcripts (black circles) which differ in their expression intensity (different sizes of the circles) and interact in space and time (solid black lines represent strong interactions and dashed black lines represent weak interactions). Grey ellipses denote specific groups of transcripts that are functionally related. The integration of spatiotemporal changes of hormone signaling into the downstream transcriptional network can lead to herbivore-specific plant responses.

Figure 3.

The jasmonate core pathway and its modulating factors. A conceptual, non-exhaustive overview is presented. General and specific herbivore-associated patterns, including HAMPs, DAMPs and wounding, activate the jasmonate pathway (blue area). Increased accumulation of JA-Ile promotes the interaction of JAZ proteins with the SCF ubiquitin ligase SCF^{COI1}. Ubiquitin-dependent degradation of JAZs by the 26S proteasome releases transcription factors from their JAZ-bound repressed state, thereby activating the expression of transcriptional regulons that promote defense and inhibit vegetative growth. JA-independent hormonal pathways are also induced (purple area), and several hormones, including salicylic acid (SA), ethylene (ET), auxin, gibberellins (GA), cytokinins (CK) and brassinosteroids (BR) modulate JA metabolism and signaling (light-blue area). Herbivory also leads to oxidative stress, changes in intracellular pH and dessication, which modulate

the JA pathway either directly or indirectly through other hormones. Together, this leads to complex phenotypic changes that comprise both specific and general responses, the majority of which can be linked back to the jasmonate pathway.