Plant Immunity to Insect Herbivores

Gregg A. Howe[1] and Georg Jander[2]

[1]Department of Energy-Plant Research Laboratory and Department of Biochemistry and Molecular Biology, Michigan State University, East Lansing, Michigan 48824; email: howeg@msu.edu

[2]Boyce Thompson Institute for Plant Research, Cornell University, Ithaca, New York 14853; email: gj32@cornell.edu

Annu. Rev. Plant Biol. 2008. 59:41–66

The *Annual Review of Plant Biology* is online at plant.annualreviews.org

This article's doi: 10.1146/annurev.arplant.59.032607.092825

1543-5008/08/0602-0041$20.00

Key Words

plant-insect interaction, plant defense, jasmonate, COI1, secondary metabolism

Abstract

Herbivorous insects use diverse feeding strategies to obtain nutrients from their host plants. Rather than acting as passive victims in these interactions, plants respond to herbivory with the production of toxins and defensive proteins that target physiological processes in the insect. Herbivore-challenged plants also emit volatiles that attract insect predators and bolster resistance to future threats. This highly dynamic form of immunity is initiated by the recognition of insect oral secretions and signals from injured plant cells. These initial cues are transmitted within the plant by signal transduction pathways that include calcium ion fluxes, phosphorylation cascades, and, in particular, the jasmonate pathway, which plays a central and conserved role in promoting resistance to a broad spectrum of insects. A detailed understanding of plant immunity to arthropod herbivores will provide new insights into basic mechanisms of chemical communication and plant-animal coevolution and may also facilitate new approaches to crop protection and improvement.

Supplemental Videos

Contents

Secondary metabolites: compounds that are not required for normal plant growth or reproduction and are often unique to, or characteristic of, specific plant lineages

INTRODUCTION

Terrestrial plants are a food source for an estimated one million or more insect species from diverse taxonomic groups. Insects use various feeding strategies to obtain nutrients from all above- and belowground plant parts. Although all phytophagous insects inflict mechanical damage on plant tissues, the quantity and quality of injury vary greatly, depending on the feeding tactic. Approximately two-thirds of all known herbivorous insect species are leaf-eating beetles (Coleoptera) or caterpillars (Lepidoptera) that cause damage with mouthparts evolved for chewing, snipping, or tearing (116). Piercing-sucking herbivores such as thrips and spider mites use tube-like structures to suck the liquid content from lacerated cells, whereas leafminers develop and feed on soft tissue between epidermal cell layers. Aphids, whiteflies, and other Hemiptera insert specialized stylets between cells to establish a feeding site in the phloem. In each of these insect-plant relationships, both partners send and receive chemical cues that determine the outcome of the interaction. Contact chemoreceptors on the insect mouthparts, antennae, and tarsi (feet), for example, gauge the suitability of the host as a food source. Conversely, plant cells recognize and respond to insect movement, wound trauma inflicted by feeding, and compounds in insect oral secretions.

A recurring theme in all spheres of plant-insect biology is variation in the extent to which herbivores exercise dietary specialization. Generalist (i.e., polyphagous) insects feed on many hosts from different plant families. Specialists (i.e., monophagous and oligophagous insects) subsist on one or a few plants from the same family. An insect's decision to accept or reject a host is determined in large part by a myriad of chemical deterrents and attractants. There is good reason to believe that much of the extraordinary diversity of specialized plant compounds, so-called secondary metabolites, results from the coevolutionary struggle of herbivores and plants to eat or not be eaten (13). As sessile organisms, plants rely heavily on chemical defenses to thwart insect attack. Compounds that exert repellent, antinutritive, or toxic effects on herbivores are commonly referred to as direct defenses. Physical barriers such as leaf toughness and trichomes that increase plant fitness in the presence of herbivores are also direct defenses. A second layer of indirect

protection is afforded by herbivore-induced plant volatiles and nectar rewards that attract natural enemies of the herbivore (67). The combined effects of direct and indirect defense provide durable resistance to a broad spectrum of arthropod herbivores in natural ecosystems (33, 68).

Plant traits that confer resistance to insect pests may also be classified according to the manner in which they are regulated. Some traits are expressed constitutively under the control of hard-wired developmental programs, irrespective of the herbivore threat level. Reproductive tissues, for example, typically accumulate large amounts of defensive proteins and metabolites. In contrast to these preformed barriers, herbivore-challenged plants mount active defense responses at the site of tissue damage and, in many cases, systemically in undamaged tissues (14). This highly dynamic form of induced resistance has been documented in species throughout the plant kingdom (61). Evidence indicates that induced defenses evolved because they have lower resource allocation costs than constitutive resistance traits (10, 60). In addition to induced defensive traits, plants can minimize the fitness consequences of tissue loss by activating physiological processes, such as sequestration of sugars in below-ground tissues, that allow the plant to better tolerate herbivory (118).

The ability of plants to recognize and respond defensively to insect attack constitutes a form of immunity. Unlike the highly specific adaptive immune system of vertebrates in which mobile defender cells recognize and eliminate pathogenic challenges, plant immunity to insects relies on the innate ability of each cell to perceive "danger" signals, to transmit this information systemically to fend off future attacks, and to mount direct and indirect defenses that reduce insect performance. Our current understanding of the mechanisms and evolutionary origins of immune recognition in plants comes mainly from studies of plant-pathogen interactions (55). Basal resistance to pathogen infection is triggered by transmembrane receptors that recognize pathogen-associated molecular patterns. These ancient microbial molecules, which include fragments of bacterial cell walls, flagellin, and EF-Tu (elongation factor Tu), alert the host to the presence of intruding microorganisms (37, 148). As a second line of defense, the plant immune system relies on disease resistance (R) proteins to detect effector molecules (i.e., virulence factors) that pathogens secrete into plant cells to counteract or weaken host defense. A unifying theme in plant immunity to pathogens is the involvement of receptors that recognize pathogen-derived molecules or, in the case of most R proteins, pathogen-modified host proteins.

Relatively little is known about the molecular recognition events that trigger plant immunity to insect herbivores. However, plants appear to use multiple surveillance systems to recognize insects with a wide range of lifestyles and feeding behaviors. One of these recognition systems is conceptually similar to pathogen-triggered immunity because it involves the perception of exogenous molecules that, when delivered to plant cells via insect secretions, elicit a host defense response. These elicitors can be insect-derived molecules or plant compounds that are modified by the insect. The notion that plants recognize insect-modified compounds of plant origin is consistent with the so-called guard hypothesis, which postulates that R proteins recognize damage to endogenous plant proteins and subsequently initiate a defense response (55). That plants activate many anti-insect defenses in response to mechanical tissue damage indicates that endogenous signals produced by distressed cells also play a critical role in plant perception of herbivory; the concept of wound trauma as a trigger for defense is analogous to danger signal models of the vertebrate immune system (86). The plant hormone jasmonic acid (JA) and related signaling compounds (collectively referred to as jasmonates) are ubiquitous signals for tissue injury and for the subsequent activation of defense responses to many, if not most, insect herbivores.

Direct defense: a toxin, antifeedant, physical barrier, or other plant defensive trait that deters herbivory

Indirect defense: a plant defensive trait that protects against herbivory by enhancing the attraction of predators of the herbivore

Constitutive defense: toxins and other defensive barriers that are produced irrespective of whether herbivores are present

Induced defense: toxins and other defensive traits that are only expressed in response to herbivory

Elicitor: insect- or plant-derived compound that, upon recognition by the host plant, activates a defensive response

JA: jasmonic acid

Here, we review recent advances in our understanding of the molecular and biochemical mechanisms of plant immunity to insect herbivores. First, we discuss early signaling events at the plant-insect interface and their involvement in insect recognition. Second, we describe the central role of jasmonates in the regulation of defense responses to herbivory and discuss important new developments regarding the mechanism of jasmonate action. Finally, we highlight specific examples of direct and indirect defensive traits that impact host-plant selection by, or resistance to, insect herbivores.

EARLY SIGNALING EVENTS AT THE PLANT-INSECT INTERFACE

Successful implementation of an induced defense response requires that plants respond to herbivory both rapidly and accurately. Early signaling events at the plant-insect interface, which occur well before changes in host plant gene expression and defense-related metabolism, are critical for the process of herbivore recognition (83). Several studies have identified insect elicitors that allow plants to distinguish herbivory from mechanical damage. In the case of hemipteran herbivores, there is evidence for the involvement of *R* genes in the control of host plant resistance.

Mechanical Wounding Versus Herbivory

Although all herbivory results in plant tissue damage, tissue disruption per se is not always a reliable indicator of insect attack. Therefore, to avoid wasting defensive resources, plants must differentiate insect feeding and simple mechanical damage, such as that caused by hail or wind in natural settings. Some responses, including the upregulation of genes required for cell repair and response to osmotic stress, would likely occur as a result of either herbivory or mechanical wounding. However, the production of toxic secondary metabolites and other defensive responses would presumably benefit only herbivore-challenged plants.

Changes in gene expression underlie the induced synthesis of most defensive secondary metabolites and proteins, as well as other changes in plant metabolism that occur during herbivory. Microarray experiments with several plant species, including *Arabidopsis thaliana* (Arabidopsis) (23, 102), *Nicotiana attenuata* (coyote tobacco) (137), *Populus trichocarpa* × *Populus deltoides* (hybrid poplar) (84, 99), and *Picea sitchensis* (Sitka spruce) (100), compared gene expression patterns induced by mechanical wounding with that induced by insect feeding or simulated herbivory. Although there is considerable overlap in the induced expression patterns, there are also transcriptional responses that appear to be specific to insect feeding or the application of insect oral secretions to wound sites. In some cases, these responses have been associated with the production of specific insect-deterrent compounds such as nicotine (52) and glucosinolates (88).

There are two main theories to explain how plants discriminate insect herbivory from mechanical damage. The first is that plants recognize compounds in insect oral secretions. This view is supported by the identification of several insect-derived factors that elicit defense responses when applied to artificial wounds (see below). Plants may also differentiate mechanical wounding from herbivory through the use of as yet unknown mechanisms that gauge the quantity and quality of tissue damage. Caterpillar feeding, for example, involves the action of specialized mandibles that remove similarly sized pieces of leaf tissue in a highly choreographed and predictable manner. Most studies showing that mechanical wounding and herbivory (or simulated herbivory) elicit different responses have relied on wound treatments that do not approximate tissue injury caused by insect grazing (90). Recognizing this limitation, researchers have developed novel approaches to disentangle the effects of mechanical damage

from the effects of oral secretions. One approach in studying the role of insect saliva in plant-lepidopteran interactions, for example, is to challenge plants with larvae in which the labial salivary gland is removed. Such experiments with *Helicoverpa zea* (corn earworm) provided evidence that salivary secretions qualitatively affect plant defense responses to caterpillar feeding (93). A second approach is to challenge plants with mechanical caterpillar devices that more accurately mimic tissue injury caused by caterpillar feeding (90). These experiments have shown that repetitive mechanical wounding of *Phaseolus lunatus* (lima bean) leaves elicits a pattern of volatile emission that is qualitatively similar to that induced by caterpillar attack. It is thus clear that the temporal and spatial patterns of mechanical injury are a critical determinant of the host defense response.

Insect Oral Secretions

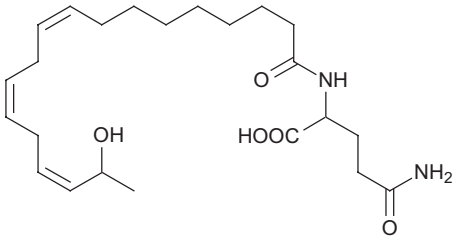
One of the best-studied plant responses to herbivory is the elevated release of volatiles, which include terpenes, green leafy volatiles, ethylene, and other volatile organic compounds. Studies with several plant-insect combinations have demonstrated that insect feeding or application of oral secretions to wound sites elicits a different or more intense volatile response than mechanical damage alone (7, 8, 22, 112, 135). Plants can benefit from herbivory-specific volatile production through direct deterrent effects on the herbivores, attraction of predators to the site of insect feeding, and intraplant signaling events that poise uninfested tissue for more rapid defense induction. Because of the relative ease of volatile collection and the nondestructive nature of the assay, induced volatile production has been used to identify insect-derived elicitors of plant defense responses.

N-17-hydroxylinolenoyl-L-glutamine (volicitin) (**Figure 1**) was identified in *Spodoptera exigua* (beet armyworm) oral secretions through its ability to induce volatile release in *Zea mays* (maize) seedlings (5). This compound was the first example of what appears to be a widely prevalent production of fatty acid–amino acid conjugates (FACs) (**Figure 1**) by lepidopteran larvae (3, 40, 92, 98). Specific responses to FACs in lepidopteran oral secretions, including changes in gene expression, alteration of the plant proteome, and induced production of nicotine and protease inhibitors, have been studied most extensively in *N. attenuata* (35, 39). Selective binding of volicitin to plasma membrane preparations from maize suggests the existence of an FAC receptor (134). Other workers have suggested that the detergent-like properties of FACs may account for some of the biological activities of these amphiphilic compounds (81).

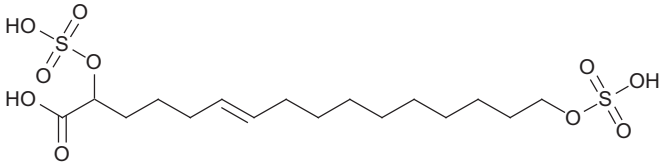
FAC: fatty acid–amino acid conjugate

Some plants, including *P. lunatus* and *Gossypium hirsutum* (cotton), do not respond to FACs (120). However, the release of volatiles by FAC-insensitive plants in response to caterpillar feeding indicated that other elicitors are present in the oral secretions. A bioassay-guided search for elicitors of ethylene production in *Vigna unguiculata* (cowpea) led to the identification of proteolytic fragments of plastidic ATP synthase γ-subunit in the oral secretions of *Spodoptera frugiperda* (fall armyworm) (112). At least four of these disulfide-bonded peptides, called inceptins (**Figure 1**), are produced through the digestion of plant proteins in the *S. frugiperda* gut (113). Although *Phaseolus vulgaris* (common bean) responded similarly to inceptin, *Z. mays* and *Nicotiana tabacum* (cultivated tobacco) did not (112). The proposed mechanism of action of inceptin is consistent with the guard hypothesis of plant immunity, but plant receptors for these elictors remain to be identified.

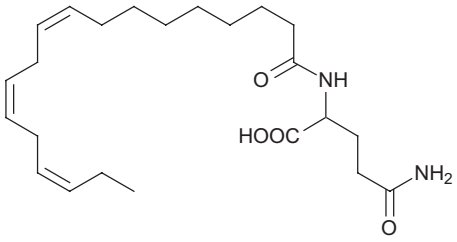
Oral secretions from orthopteran insects also use an FAC-independent mechanism to elicit volatile release in the host plants of these insects. Recently, a new class of sulfated fatty acids called caeliferins (**Figure 1**) was identified in the oral secretions of *Shistocerca americana* (American bird grasshopper) and other grasshopper species (4). Like FACs, caeliferins elicit the release of volatile terpenes



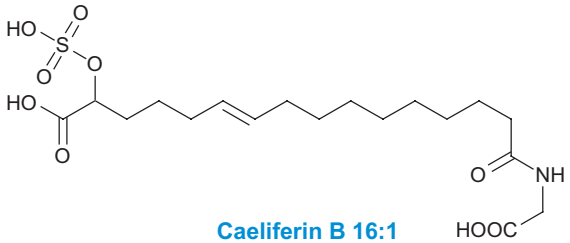
N-17-Hydroxylinolenoyl-L-glutamine (volicitin)



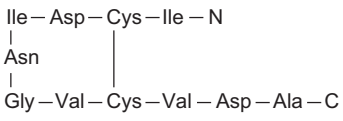
Caeliferin A 16:1



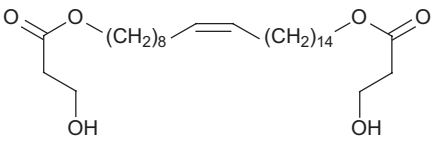
N-Linolenoyl-L-glutamine



Caeliferin B 16:1



Inceptin



Bruchin C

Figure 1

Insect-derived elicitors of host plant defense responses. Volicitin and *N*-linolenoyl-L-glutamine belong to the family of fatty acid–amino acid conjugates (FACs) found in oral secretions of lepidopteran larvae. The fatty acid and amino acid moieties of FACs are derived from the insect and host plant, respectively (95). Inceptin, which was also isolated from oral secretions of lepidopteran larvae, is produced by proteolytic degradation of chloroplast ATP synthase in the insect gut. FACs and inceptin thus represent examples of elicitors that are produced by modification of plant compounds within the insect. Caeliferins were isolated from the oral secretions of the grasshopper species *Schistocerca americana*. Bruchins, which are produced by pea weevils and related bruchids, stimulate neoplastic growth at the site of weevil oviposition.

from maize seedlings. It is not known whether this response functions to deter grasshopper feeding directly, attracts predators, or provides some other benefit to the host plant. Further research is also necessary to determine whether the abundantly produced caeliferins provide an essential defensive or digestive benefit to the grasshoppers.

Given the numerous insect herbivores that trigger defense responses and the comparatively small number of plant-insect combinations that have been examined in detail, the discovery of additional components of insect oral secretions that elicit host defense responses can be anticipated. Other types of insect secretions also elicit defense responses. For instance, plants can respond to insect oviposition fluids, either in anticipation of imminent herbivory or to attract egg-eating predators (48). Bruchins (**Figure 1**) in the

oviposition fluid of *Bruchis pisorum* (pea weevil) elicit tumor-like growths beneath the egg on *Pisum sativum* (pea), which inhibits entry of the larvae into the pod. Oviposition by the sawfly *Diprion pini* on *Pinus sylvestris* (Scots pine) increases the production of terpenoid volatiles and decreases ethylene release (117). Similarly, oviposition by *Pieris brassicae* (large white butterfly) on *A. thaliana* triggers the expression of defense-related genes (80). The plant signaling compounds JA and salicylic acid (SA) accumulate in insect eggs and may contribute to the elicitation of defense responses (133).

R Genes Mediate Aphid Resistance

Insect-derived elicitors have not yet been identified for aphids, whiteflies, or other phloem-feeding Hemiptera. Although these insects cause comparatively little tissue damage when feeding from phloem sieve elements, plants are nevertheless able to mount distinctive metabolic and transcriptional responses to hemipteran attack (23, 65, 88, 137). Aphid salivary enzymes such as peroxidase and pectinase may be elicitors of plant defense responses (89), but this hypothesis remains to be tested rigorously.

Genetic evidence from several monocot and dicot crop species supports the idea that *R* gene products mediate resistance to phloem-feeding insects (119). In two cases, specific plant NBS-LRR (nucleotide binding site–leucine rich repeat) proteins that contribute to the recognition of hemipteran herbivores have been identified. The tomato *Mi-1* gene provides resistance to some isolates of *Macrosiphum euphorbiae* (potato aphid) and *Bemisia tabaci* (silverleaf whitefly), although not to *Myzus persicae* (green peach aphid) (94, 106). Another NBS-LRR protein, encoded by the melon *Vat* gene, confers increased resistance to both *Aphis gossypii* (cotton aphid) and the transmission of plant viruses by this aphid species (26). By analogy to plant defense against pathogens, these findings suggest a gene-for-gene interaction between the plant and the aphid. However, the presumed avirulence proteins in aphid saliva have not yet been identified.

SA: salicylic acid

MAPK: mitogen-activated protein kinase

Calcium Flux, Membrane Potential, and Mitogen-Activated Protein Kinases

Relatively little is known about the signal transduction pathways that connect insect-specific elicitors to the plant defense responses they evoke. The calcium ion (Ca^{2+}) has been implicated as a second messenger in many plant signaling pathways, including responses to herbivory (83). Under normal conditions, the cytosolic Ca^{2+} content is several orders of magnitude lower than that in organelles or apoplastic fluid. Transient increases in cytosolic Ca^{2+} levels activate calmodulin and other calcium-sensing proteins that subsequently promote downstream signaling events, including protein phosphorylation and transcriptional responses. Feeding by *Spodoptera littoralis* (Egyptian cotton worm) on *P. lunatus* causes a transient increase in cytosolic Ca^{2+} in cells adjacent to the insect bite (82). In other experiments with *P. lunatus*, treatment with a Ca^{2+} chelator prevented defense gene induction in response to feeding by *Tetranychus urticae* (two-spotted spider mite) and volatiles from mite-infested neighboring plants (9). In *A. thaliana*, the nuclear protein IQD1 binds calmodulin in a Ca^{2+}-dependent manner and thereby affects the transcription of genes involved in glucosinolate biosynthesis (75). IQD1 overexpression reduces herbivory by *M. persicae* and *Trichoplusia ni* (cabbage looper), suggesting that this protein is involved in perceiving Ca^{2+} signals to modulate plant defense responses.

R gene–mediated resistance to pathogens and other plant responses to environmental stress involve mitogen-activated protein kinase (MAPK) signaling cascades (42). Although no complete MAPK pathway leading to insect resistance has been identified, there is evidence that such pathways play a role in some plant-insect interactions. In

Jasmonate pathway: a hormone signaling pathway that plays an essential and central role in regulating plant responses to herbivory

Priming: metabolic preparation for a more rapid or robust response to subsequent herbivory

JA-Ile: jasmonoyl-isoleucine

JAC: jasmonoyl–amino acid conjugate

Solanum lycopersicum (tomato), *Mi-1* mediated resistance was attenuated when expression of certain MAPKs and MAPK kinases was reduced by virus-induced gene silencing (VIGS) (78). VIGS studies in tomato also showed that at least three MAPKs are required for systemin-mediated defense responses to *Manduca sexta* (tobacco hornworm) (58). FACs in *M. sexta* oral secretions increase wound-induced expression of SA-induced MAPK and wound-induced MAPK in *N. attenuata* (142). Furthermore, reducing expression of these two kinases by VIGS demonstrated that they are important for characteristic responses of *N. attenuata* to caterpillar oral secretions. In the wild rice *Oryza minuta*, expression of a putative MAPK kinase, *OmMKK1*, is induced by *Nilaparvata lugens* (brown planthopper) feeding, as well as by treatment with JA and SA (144).

REGULATION OF DEFENSE RESPONSES BY JASMONATES

Evidence has accumulated over the past few years to indicate that the jasmonate family of signaling compounds functions in endogenous regulation of plant resistance to arthropod herbivores. Below, we discuss the various roles of jasmonates in anti-insect defense, the mechanism by which herbivore-induced jasmonate synthesis promotes global reprogramming of defense gene expression, and the regulation of this response.

Jasmonates Serve Multiple Roles in Plant Immunity to Insects

Jasmonates play a central role in regulating defense responses to herbivores that inflict various types of tissue damage. This conclusion is based on numerous laboratory and field studies showing that jasmonate mutants are compromised in resistance to a wide range of arthropod herbivores, including caterpillars (Lepidoptera), beetles (Coleoptera), thrips (Thysanoptera), leafhoppers (Homoptera), spider mites (Acari), fungal gnats (Diptera), and mirid bugs (Heteroptera) (14, 50, 67). DNA microarray studies show that the jasmonate pathway has a dominant role in regulating global changes in gene expression in response to both mechanical wounding and herbivory (23, 24, 39, 84, 99, 100, 102, 103). Jasmonates are also involved in the regulation of tritrophic interactions (28, 128, 136), host plant resistance to phloem-feeding insects (27, 32, 87, 145), trichome-based defenses (15, 77), priming of direct and indirect defenses (28, 132), pathogen resistance (36), and systemic transmission of defense signals (109). In addition to these defense-related processes, jasmonates regulate several aspects of plant development; the hormone generally promotes defensive and reproductive processes while inhibiting the growth and photosynthetic output of vegetative tissues (25, 35). These juxtaposing activities suggest a broader role for jasmonates in managing the "dilemma of plants to grow or defend" (47) in rapidly changing and hostile environments.

Wound trauma inflicted by chewing insects or mechanical damage results in rapid (<30 min) accumulation of JA at the site of wounding. In higher plants, JA is synthesized via the octadecanoid pathway (**Figure 2**). Genes encoding nearly all jasmonate biosynthetic enzymes have been identified (108). It is now clear that further metabolism of newly synthesized JA plays a critical role in regulating downstream transcriptional responses. Among the routes of JA metabolism that modulate plant responses to biotic stress are (*a*) synthesis of the volatile compound methyl-JA (MeJA) by JA-carboxymethyl transferase (108) and (*b*) formation of jasmonoyl-isoleucine (JA-Ile) and other JA–amino acid conjugates (JACs) by JASMONATE RESISTANT 1 (JAR1) and related conjugating enzymes (59, 121) (**Figure 2**). A strict requirement for JA synthesis in anti-insect defense was demonstrated by the use of mutants that are impaired in the β-oxidation stage of the octadecanoid pathway (76, 110). This conclusion is consistent with the fact that conjugation of JA

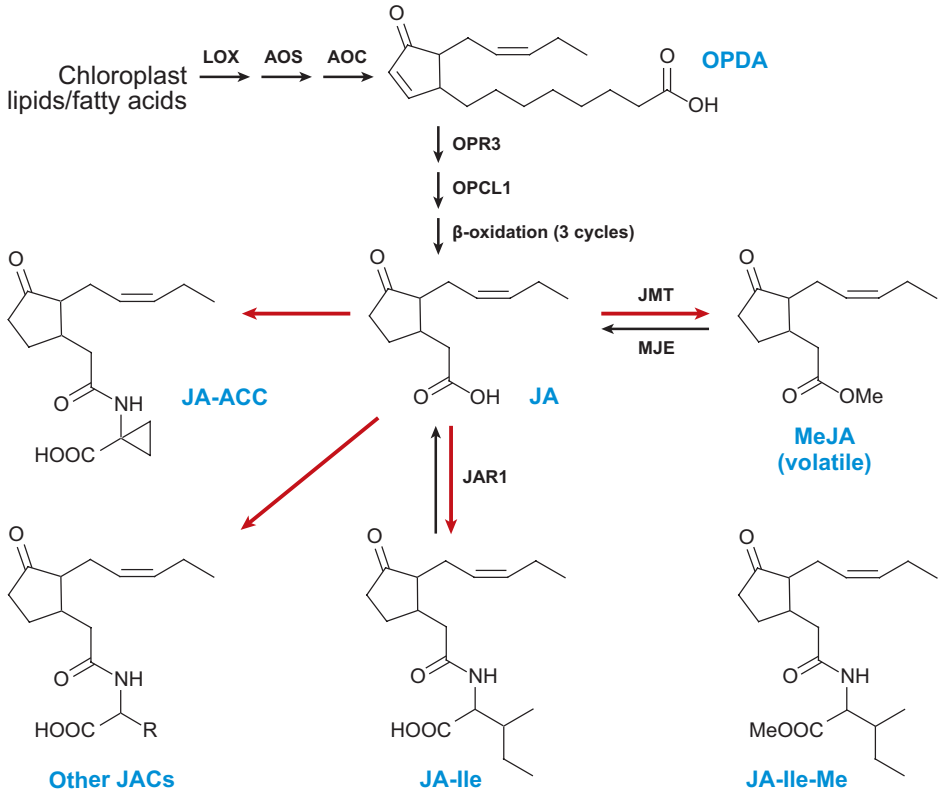


Figure 2

The octadecanoid pathway for the biosynthesis of jasmonic acid (JA) and bioactive conjugates of JA. JA synthesis is initiated in the chloroplast, where linolenic acid is converted to 12-oxo-phytodienoic acid (OPDA) by the sequential action of lipoxygenase (LOX), allene oxide synthase (AOS), and allene oxide cyclase (AOC). Following transport to the peroxisome, OPDA is reduced to OPC-8:0 (not shown) by OPDA reductase 3 (OPR3). OPC-8:0 CoA ligase (OPLC1) converts OPC-8:0 to its corresponding CoA derivative, which is the entry substrate for three cycles of β-oxidation that yield (+)-7-iso-JA (JA). JA is metabolized to several biologically active derivatives (*red arrows*). JA carboxyl methyltransferase (JMT) converts JA to the volatile compound MeJA. The reverse reaction is catalyzed by MeJA esterase (MJE). Conjugation of JA to isoleucine (Ile) by JASMONATE RESISTANT 1 (JAR1) produces JA-Ile, which promotes CORONATINE INSENSITIVE 1 (COI1) interaction with JAZ repressor proteins. JA-Ile can be methylated to produce JA-Ile-Me (44), the biological role of which is not known. As yet unidentified enzymes conjugate JA to ACC (1-aminocyclopropane-1-carboxylic acid, an ethylene biosynthetic intermediate) and other amino acids to generate JA-ACC and other jasmonoyl–amino acid conjugates (JACs), respectively.

to isoleucine (Ile) is required for direct defense against caterpillar feeding (59). Plastid-derived 12-oxo-phytodienoic acid (OPDA) is implicated as a signal per se (i.e., in the absence of its conversion to JA) for a limited range of direct (123) and indirect (71) defense responses to herbivory. Recent studies provide evidence that the mechanism of OPDA signaling is distinct from that involved in the perception of JA-derived signals such as JA-Ile (124, 129).

Jasmonate Perception

Much of our understanding of the role of jasmonates in plant-insect interactions has

COI1: CORONATINE-INSENSITIVE 1

JAZ: jasmonate ZIM domain

come from the analysis of mutants that fail to perceive JA/MeJA (25). In particular, mutants that are defective in the *CORONATINE INSENSITIVE 1* (*COI1*) gene are impaired in all jasmonate-signaled processes and, as a consequence, are highly susceptible to a wide range of arthropod herbivores (18, 77, 87, 96, 102, 123, 145). COI1 is the F-box component of a multi-protein E3-ubiquitin ligase called SCF^{COI1}, which is named for the SKP1-like, cullin, and F-box proteins of the complex. This finding led to the proposal that jasmonate signaling involves SCF^{COI1}-mediated ubiquitination of regulatory proteins that control the transcription of jasmonate-responsive genes (25).

A major step toward elucidating the mechanism of jasmonate action came from the recent discovery of jasmonate ZIM domain (JAZ) proteins that are targeted by SCF^{COI1} for degradation during jasmonate signaling (19, 129). Several lines of evidence indicate that at least some members of the JAZ family act as repressors of jasmonate-responsive genes. First, JAZ proteins are degraded in a COI1- and 26S proteasome–dependent manner in response to JA treatment. Second, dominant mutations in the conserved C-terminal domain of JAZ proteins stabilize them against SCF^{COI1}-mediated degradation and, as a consequence, reduce the plant's responsiveness to JA/MeJA (19, 129, 143). Finally, physical interaction of COI1 and JAZ1 is stimulated in a dose-dependent manner by JA-Ile and, to a lesser extent, by JA-Leu (129). That JA, MeJA, and OPDA failed to promote this interaction indicates that the COI1-dependent biological activity of these compounds requires their conversion to a bioactive JAC (e.g., JA-Ile) or that these JA derivatives promote COI1 interaction with different JAZ substrates. The ability of JA-Ile to stimulate the COI1-JAZ1 interaction in the yeast two-hybrid system (i.e., in the absence of other plant proteins) implicates the COI1-JAZ complex as a receptor for JA-Ile (129). Although ligand-binding studies are needed to test this hypothesis, it is worth noting that the emerging picture of jasmonate action is analogous to the auxin signaling pathway, in which auxin binding to the LRR domain of the TRANSPORT INHIBITOR 1 (TIR1) receptor promotes the degradation of Aux/IAA transcriptional repressors (126). It will be interesting to determine whether the concept of auxin as a molecular glue that promotes TIR1-substrate interactions extends to JA-Ile as a ligand for the LRR domain of COI1.

These new insights into the mechanism of jasmonate action suggest that the core signal transduction chain for wound- and herbivore-induced expression of defensive genes is composed of relatively few links. In healthy undamaged leaves, low JA-Ile levels presumably allow JAZs to accumulate and repress the transcription of target genes (**Figure 3*a***). Establishment of this repressed state involves direct interaction of JAZ proteins with transcription factors, such as MYC2, that promote the expression of jasmonate-responsive genes (19). In response to tissue injury, rapid accumulation of JA-Ile would trigger SCF^{COI1}-mediated degradation of JAZ proteins and subsequent derepression of defense-related genes (**Figure 3*b***). A noteworthy feature of most *JAZ* transcripts is their rapid accumulation in response to jasmonate treatment (129) or wound trauma (H.S. Chung & G.A. Howe, unpublished data). Rapid resynthesis of JAZ repressors presumably provides a mechanism to restrain the expression of energetically demanding and potentially cell-damaging defensive processes when jasmonate levels decline, for example, upon cessation of insect feeding. Such a mechanism of negative feedback control suggests that the expression of jasmonate-based defenses should be viewed more as a continuum than as discrete induced and uninduced states. The key role of JA-Ile in regulating the strength of the response highlights the need to study further the cellular mechanisms of JAC homeostasis in healthy and injured tissues. It can be anticipated that a combination of negative and positive feedback mechanisms allows the plant to mount a defense response that is

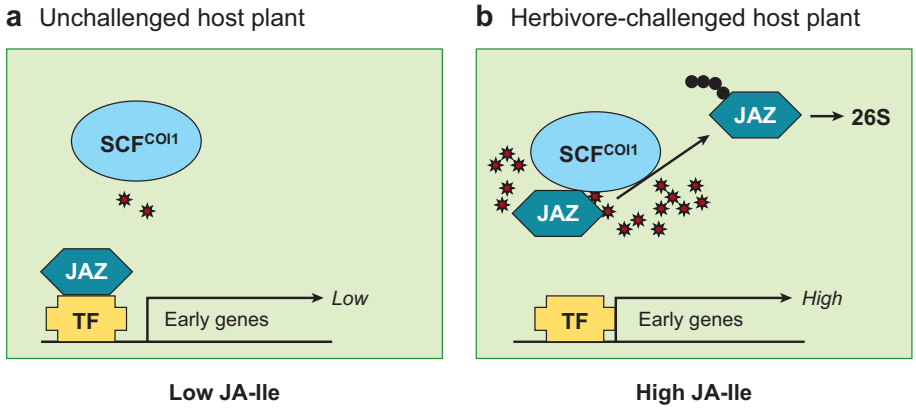


Figure 3

The JAZ repressor model of jasmonate signaling. (*a*) Low intracellular levels of jasmonoyl-isoleucine (JA-Ile) (*red stars*) favor the accumulation of jasmonate ZIM domain (JAZ) proteins, which bind to and repress the activity of transcription factors (TF) such as MYC2 that positively regulate jasmonate-responsive early genes. (*b*) Tissue injury, such as that caused by chewing insects, results in rapid accumulation of JA-Ile. These high levels of JA-Ile promote SCF^{COI1}-mediated ubiquitination (*black circles*) and subsequent degradation of JAZ repressor proteins via the 26S proteasome (26S), resulting in the derepression of transcription factors and the expression of early response genes. See text for more details.

commensurate with the intensity and duration of the attack.

Specificity of Jasmonate-Based Defenses

An important question concerning jasmonate-regulated defenses is whether the response is specific for different herbivores and, if so, how this specificity is achieved. Reymond and colleagues showed that crucifer specialist (*Pieris rapae*, small white butterfly) and generalist (*S. littoralis*) caterpillars elicit nearly identical gene expression patterns in *A. thaliana* (102). Much more divergent patterns were observed in plants challenged with insects from different feeding guilds (23, 45). For example, although the transcriptional response of *A. thaliana* to chewing (*P. rapae*) and piercing-sucking (*Frankliniella occidentalis*, western flower thrips) insects was dominated by jasmonate-regulated genes, the majority of these genes (61%) exhibited an expression pattern that was specific to one of the two attackers (23). Transcript profiles elicited by phloem-feeding insects are markedly different from those induced by attackers from other feeding guilds and are generally associated with the activation of SA-responsive genes and weak expression of JA-responsive genes (23, 32, 65, 131). Induction of the jasmonate pathway by aphid feeding likely reflects cell damage caused by stylet probing (57, 131). The phloem-feeding *B. tabaci*, which causes much less mechanical damage than do aphids, does not activate JA-responsive genes (145). Emerging evidence indicates that phloem feeders actively suppress jasmonate-based defenses (131, 145). In summary, insects from different feeding guilds tend to elicit distinct (but overlapping) patterns of gene expression, whereas attackers from the same guild evoke very similar responses. Because most insects betray their presence by triggering jasmonate synthesis in damaged tissues, jasmonate-signaled defenses may have evolved as a relatively nonspecific strategy to deter a large variety of different herbivores.

Relatively little is known about the early signaling events involved in herbivore-induced production of bioactive jasmonates.

Feeding guild: a group of insect herbivores that use one of various types of feeding behavior (e.g., chewing or piercing-sucking) to obtain nutrients from host plant tissue

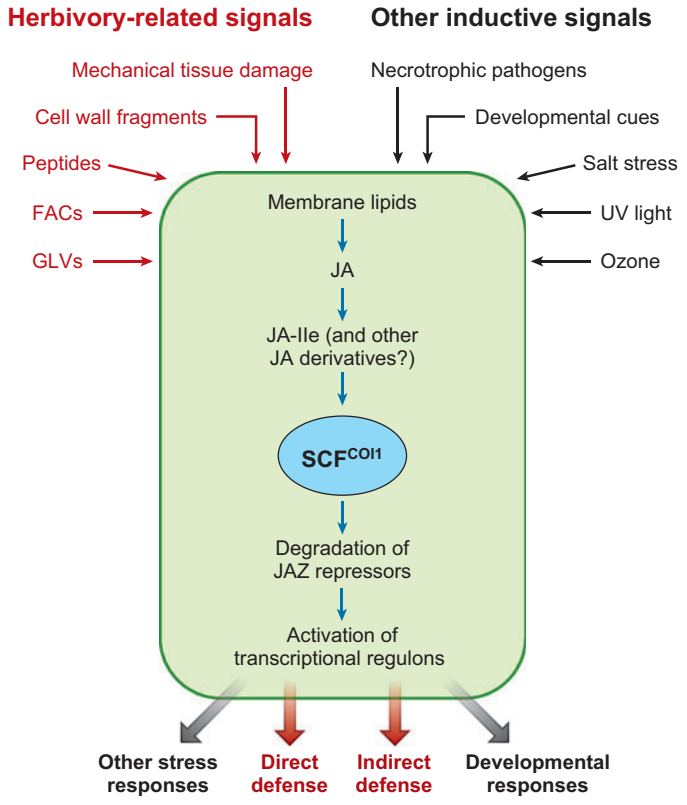


Figure 4

Regulation of jasmonate-based defenses in response to herbivory. Herbivore-induced signals (*red arrows*) activate the production of bioactive jasmonates such as jasmonoyl-isoleucine (JA-Ile). JA-Ile promotes SCF^{COI1}-mediated degradation of jasmonate ZIM domain (JAZ) repressor proteins, resulting in derepression of transcriptional regulons that control direct and indirect defensive traits. The jasmonate signaling pathway also regulates plant responses to developmental cues and other stress conditions (*black arrows*). FACs, fatty acid—amino acid conjugates; GLVs, green leafy volatiles [e.g., (*Z*)-3-hexenal]; JA, jasmonic acid.

That severe mechanical tissue damage, abiotic stress, and developmental cues activate jasmonate synthesis and many associated defenses indicates that insect-derived factors are not strictly required for these responses (**Figure 4**). Rather, it would appear that insect-derived elicitors such as FACs and inceptin (see above), which stimulate JA synthesis when applied to artificial wounds, reinforce or amplify the jasmonate pathway (67, 112). In addition to modulation by herbivore-derived factors, jasmonate-regulated defenses may be positively or negatively modulated by other phytohormones (14, 105). SA, for example, is well known for its ability to antagonize induced defense responses to lepidopteran insects (14, 21). Ethylene also affects the expression of defensive proteins and secondary metabolites (43, 51, 141). In comparison to the jasmonates, however, ethylene production during herbivore attack is considered to play a relatively minor role in the active defense response (138).

The specificity of induced defense responses to herbivory may also be influenced by differential interactions between various bioactive jasmonates, COI1, JAZ proteins, and the downstream transcription factors they regulate. For example, specific members of the JAZ family may control distinct sets of herbivore-responsive genes in different cell types or in response to different ligands. Phosphorylation of JAZ proteins by a MAPK (64) suggests a mechanism to modify the specificity of responses or integrate the jasmonate pathway with other signals. The recent discovery (121) that JA is conjugated to the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) is of interest because JA and ethylene accumulate simultaneously and act synergistically in response to herbivory (49, 111, 138). The hypothesis that JA-ACC (**Figure 2**) is involved in the control of defenses that are coordinately regulated by the jasmonate and ethylene pathways deserves further attention.

Systemic Signaling

Many inducible defenses are expressed rapidly (i.e., within hours) in undamaged leaves of herbivore-challenged plants. This fascinating systemic response, which has been documented in a wide range of plant species, provides effective resistance to future insect attacks (14, 61). Since the discovery of this phenomenon more than 35 years ago (38), considerable research effort has been devoted to the identification of systemic wound signals and the underlying mechanisms by which they are produced, transported, and perceived. Classical grafting experiments indicate that

systemic proteinase inhibitor (PI) expression in *S. lycopersicum* depends both on JA synthesis at the site of wounding and on jasmonate perception in distal undamaged leaves. These and other findings support a model in which JA (or a derivative of JA) acts as a phloem-mobile signal (109). Systemin, which is a potent peptide elicitor of PI expression in *S. lycopersicum*, appears to strengthen systemic defenses by amplifying jasmonate synthesis in damaged leaves (109). Notably, however, the systemin homolog in *Solanum nigrum* (black nightshade) does not mediate PI expression or other direct defense responses (114). The divergent role of systemin in closely related species suggests that different plants may employ distinct mechanisms to regulate jasmonate synthesis and systemic responses to herbivory (50).

Recent studies with *N. attenuata* indicate that FACs in oral secretions of *M. sexta* elicit rapid activation of MAPK activity and defense-related genes in undamaged areas of the attacked leaf (142). FAC binding to a hypothetical receptor was proposed to generate a rapidly acting, short-distance mobile signal that triggers MAPK cascades in the damaged leaf. This intraleaf systemic response is followed by the production of a second mobile signal (e.g., jasmonate) that initiates PI expression in distal undamaged leaves. These findings are consistent with the idea that multiple intercellular signals, acting over a range of distances, mediate the complex spatiotemporal responses of plants to herbivory. That both *S. lycopersicum* systemin and FACs positively regulate jasmonate synthesis via a MAPK cascade (58, 142) suggests that parallel signaling pathways initiated at the plant-insect interface may converge on the jasmonate pathway (**Figure 4**).

DIRECT DEFENSE RESPONSES

Secondary Metabolites

It is likely that all plants exhibit constitutive or induced accumulation of toxic secondary metabolites as part of their defense against herbivory. Research with numerous plant species has revealed a great variety of small molecules with toxic or antifeedant effects on insect herbivores. Many terpenoids, the most metabolically diverse class of plant secondary metabolites (>40,000 known structures), play a role in plant defense (2). The alkaloids, widely distributed secondary metabolites that are best known for their metabolic effects in mammals (e.g., caffeine, nicotine, morphine, strychnine, and cocaine), likely evolved as a defense against insect herbivory. Other well-studied classes of plant secondary metabolites with defensive properties include the furanocoumarins, cardenolides, tannins, saponins, glucosinolates, and cyanogenic glycosides.

PI: proteinase inhibitor

Recent advances in plant molecular biology have made it possible to identify the biosynthetic pathways leading to the production of defensive toxins. For example, Frey and coworkers (30) discovered genes encoding all five enzymes involved in the biosynthesis of 2,4-dihydroxy-1,4-benzoxazin-3-one (DIBOA), a toxin found in maize, wheat, and other Gramineae. Almost all genes required for the production of glucosinolates, a diverse class of metabolites found in the model plant *A. thaliana* and other Cruciferae, have been identified (41). As an example of how such knowledge of biochemical pathways can be applied to change plant immunity to herbivory, *A. thaliana* was engineered with three enzymes from *Sorghum bicolor* (grain sorghum) to produce the cyanogenic glycoside dhurrin, thereby enhancing resistance to *Phyllotreta nemorum* (yellow-striped flea beetle) (127).

Many defensive compounds are potentially toxic to the plants that produce them. Therefore, the storage of relatively benign precursors that are activated by herbivory is a recurring theme in plant biology. For instance, all three of the defensive systems mentioned in the previous paragraph include compounds that are sequestered in plants, but not activated until the onset of herbivory. DIBOA is stored as inactive DIBOA-glucoside,

glucosinolates are enzymatically activated to produce toxic breakdown products, and the respiratory inhibitor hydrogen cyanide is released from cyanogenic glycosides during herbivory.

The complex mixture of toxins found in many plants may provide synergistic effects in defense against herbivory. For instance, a combination of two monoterpenoids is almost ten times more toxic to *Spodoptera litura* (tobacco cutworm) than would have been predicted from a simple additive effect (53). Similarly, exposure of *Callosobruchus maculatus* (cowpea bruchid) to PIs shows synergistic effects; growth inhibition by several PIs is more effective than the sum of the individual inhibitors (6). Although some herbivores can compensate for the presence of PIs by consuming additional plant tissue (140), increased food consumption may be constrained by the deleterious effects of increased exposure to toxins. Steppuhn & Baldwin (122) recently verified this experimentally by silencing PI expression and/or nicotine production in *N. attenuata* and demonstrating that compensatory feeding by *M. sexta* in response to PIs was prevented by the presence of nicotine in the diet.

In addition to possible synergistic effects, metabolic diversity in toxin production by individual plants can also provide defense against multiple herbivores with different feeding styles or resistance mechanisms. Recent work on glucosinolates demonstrates how natural selection for a diverse profile of secondary metabolites can provide defensive specificity. Nearly 40 different glucosinolates have been found in *A. thaliana*, and more than 100 breakdown products are likely formed after activation by the enzyme myrosinase. Experiments with four insect herbivores showed that tryptophan-derived indole and methionine-derived aliphatic glucosinolates have differing effects on Hemiptera and Lepidoptera (87). Indole glucosinolates, which break down in the absence of the activating enzyme myrosinase (11), provide a better defense against *M. persicae* than do the more stable aliphatic glucosinolates (70). The *EPITHIOSPECIFIER* (*ESP*) and *EPITHIOSPECIFIER MODIFIER 1* (*ESM1*) loci, which were identified by means of natural variation in insect resistance (73, 147), control the breakdown of aliphatic glucosinolates to either nitriles or isothiocyanates. Isothiocyanates provide better resistance to *T. ni*, *S. littoralis*, and *P. rapae* than do nitriles (1, 16, 73). Nevertheless, the continued presence of the nitrile-generating ESP protein in many *A. thaliana* land races (73) suggests that nitrile production benefits plants in nature.

Defensive Proteins

Insect feeding triggers the expression of plant defensive proteins that exert direct effects on the attacker. PIs, which impair various mechanistic classes of digestive proteases in the insect midgut, have been thoroughly studied for their role in the active defense response (38, 107). Inhibition of gut proteases by PIs results in amino acid deficiencies that negatively affect the growth and development of the herbivore (79, 146). The effectiveness of PIs as a defense is often thwarted by the insect's adaptive ability to express digestive proteases that are insensitive to the host plant complement of PIs or that inactivate PIs (12, 34, 56, 104). The diversity and rapid evolution of certain *PI* gene families may reflect the evolution of insect counter-adaptations that have led to the chemical arms race between plants and herbivores (125).

The plant's defensive protein arsenal also includes enzymes that disrupt insect digestive physiology and other aspects of food consumption. Members of the cysteine protease family of enzymes, for example, disrupt the chitin-rich peritrophic membrane that protects the gut epithelium (72, 91). Plant lectins and chitinases may also target carbohydrate-containing components of the insect gut (74, 97). Oxidative enzymes such as polyphenol oxidase (PPO) and lipoxygenase (LOX) covalently modify dietary protein through the

production of reactive *o*-quinones and lipid peroxides, respectively (20, 29, 139). Because catalysis by O_2-dependent enzymes is limited by low oxygen levels in the foregut and midgut of some insect species (54), an alternative possibility is that PPO and LOX act rapidly (i.e., within seconds) during tissue mastication by insect mouthparts. This hypothesis is particularly germane in the case of plants such as potato and tomato that express high levels of PPO (130) and LOX (A. Schilmiller, R. Last & C. Wilkerson, unpublished observation) in glandular trichomes.

The discovery of novel defensive proteins has been facilitated by proteomic analysis of gut content and feces (frass) of insect herbivores. This approach is based on the premise that defensive proteins are relatively resistant to gut proteases and, as a consequence, are highly enriched during passage of the food bolus through the insect. Application of this procedure to the tomato-reared *M. sexta* larvae led to the identification of isoforms of arginase and threonine deaminase (TD), which degrade the essential amino acids arginine and threonine, respectively, in the lepidopteran midgut (18). Arginase and TD appear to be components of a multitiered defensive system that functions to deplete amino acid availability in the alkaline environment of the lepidopteran gut; the low protein (i.e., amino acid) content of plant tissue is often a limiting factor for the growth of insect herbivores (85). TD's ability to degrade threonine is activated during herbivore attack by proteolytic removal of the enzyme's C-terminal regulatory domain (17). An emerging concept from this and other recent studies (112, 139) is that limited proteolysis of plant proteins in the insect gut provides a level of regulation in the overall control of induced host defenses. In summary, wound-induced postingestive defenses likely involve synergistic interactions between PIs, oxidative enzymes, amino acid–degrading enzymes, and metabolites that exert a combination of toxic and antifeedant effects. The central role of proteins in this process broadens the traditional view that secondary metabolites are the major determinants of host plant utilization by insects.

Induced expression of many anti-insect proteins is tightly regulated by the jasmonate signaling pathway. Examples of jasmonate-inducible proteins that have an established or putative role in direct defense include PPO, arginase, TD, leucine amino peptidase, acid phosphatase (VSP2), and a broad spectrum of PIs (17, 18, 20, 29, 79). The abundance of many of these proteins in the insect gut correlates with high-level accumulation of the corresponding transcripts in insect-damaged leaves (17, 18). Jasmonate-induced transcription and high protein stability appear to provide complementary mechanisms to maximize the effectiveness of protein-based defenses while minimizing the high allocation costs associated with the production of protein-based defenses. The lack of expression of jasmonate-inducible proteins in *coi1* mutants (18, 77) indicates that these proteins are dispensable for plant vegetative growth in the laboratory. This observation, together with the sporadic occurrence of jasmonate-inducible proteins (e.g., TD) in specific plant lineages, suggests that midgut-active defensive enzymes evolved from preexisting housekeeping enzymes that catabolize essential nutrients during normal plant development.

TD: threonine deaminase

VOLATILE COMMUNICATION DURING HERBIVORY

Volatile-Mediated Direct and Indirect Defenses

The release of volatiles in response to herbivory can provide a direct defensive benefit by deterring further conspecific oviposition (22) or an indirect benefit by attracting predators (66). The attraction of parasitoid wasps by damage-induced volatiles is a well-studied phenomenon in many plant species. Parasitoids associate plant-derived odors with the presence of prey, which provides a defensive benefit to the emitting plants as long as volatile production is a reliable beacon of

herbivory. The specificity of this interaction has been demonstrated with the expression of *Z. mays* TPS10, an herbivore-induced terpene synthase that forms (*E*)-β-farnesene, (*E*)-α-bergamotene, and other sesquiterpenes, in *A. thaliana* (115). Females of the parasitoid *Cotesia marginiventris*, which had learned to associate this odor with their prey, *S. littoralis*, were subsequently attracted to TPS10-producing *A. thaliana*. Because wild-type *A. thaliana* does not produce significant amounts of volatile terpenes, this experiment demonstrates that a single herbivore-induced gene from *Z. mays* is sufficient to elicit this indirect defense.

Other recent work with *Z. mays* shows that the release of volatiles provides an indirect defense against underground herbivory. In response to attack by larvae of *Diabrotica virgifera* (western corn rootworm), maize roots release (*E*)-β-caryophyllene, which attracts *Heterorhabditis megidis* nematodes that feed on the beetle larvae (101). Treatment of nonproducing plants with (*E*)-β-caryophyllene attracted *H. magidis* and reduced herbivory.

Inter- and Intraplant Volatile Communication

In addition to mediating interactions with herbivores and their predators, damage-induced volatiles can provide a signal that allows neighboring plants to prepare for imminent herbivory. This process, called priming, results in a more rapid or a more robust response to subsequent herbivory (9, 28, 62). Although early experiments relied on laboratory setups with unrealistically high volatile concentrations, there are now good examples of interplant signaling through the release of endogenous volatiles. Green leafy volatiles (primarily degradation products of linoleic and linolenic acids) released by *Z. mays* primed neighboring plants to respond more vigorously to subsequent mechanical damage and application of caterpillar oral secretions (28). *N. attenuata* planted adjacent to clipped *Artemisia tridentata* (sagebrush) received a blend of volatile organic compounds that altered gene expression and caused more rapid induction of PI production upon subsequent feeding by *M. sexta* (69).

At first glance, it would appear that eavesdropping on volatile signals should provide a defensive benefit only to the receiving plant. However, in a tree or other large plant, volatiles transferred between branches or leaves of the same individual would potentially allow faster communication of imminent threats than would phloem-mediated propagation of a systemic signal (described above). Recent studies with three plant-insect interaction systems provide evidence of such intraplant volatile-mediated priming. Field experiments with mechanically clipped *A. tridentata* showed that defense priming depends on the movement of an airborne signal between damaged and undamaged branches (63). In laboratory experiments with *P. deltoides* × *P. nigra* (hybrid poplar), volatiles released by *Lymantria dispar* (gypsy moth)-challenged leaves primed a nearby leaf on the same plant to release terpene volatiles more rapidly in response to subsequent attack by *L. dispar* (31). Extrafloral nectar production, which can attract insect predators, was both induced and primed by volatiles released from *P. lunatus* leaves on the same plant under natural conditions (46).

PERSPECTIVES AND FUTURE DIRECTIONS

As described in this review, there has been considerable recent progress in deciphering the molecular basis of plant immunity to insect herbivores. Nevertheless, knowledge of how plants perceive and respond to herbivory lags far behind our understanding of plant responses to pathogen invasion. Although both insect-derived elicitors (**Figure 1**) and NBS-LRR receptors involved in insect recognition have been identified independently, there are as yet no known receptor-ligand interactions or direct links from these

to the induction of plant defense pathways. Calcium flux, phosphorylation cascades, and other early signaling events are necessary for full defense induction, but the order of these signals, feedback loops that augment or attenuate responses, and connections to downstream transcriptional and metabolic changes remain relatively uninvestigated. Identification of the JAZ family of proteins will permit experiments that lead to a deeper mechanistic understanding of jasmonate signaling and its role in controlling the outcome of a myriad of plant-pest interactions. Rather than being a set of linear pathways, jasmonate and other signals that regulate the expression of plant defenses clearly involve a complex mesh of interactions that provide the flexibility needed to respond to multiple herbivores and pathogens in a natural setting. The inevitable tradeoffs that occur during plant responses to simultaneous attacks and the possible manipulation of the plant defense network (e.g., phytohormone synthesis and action) by herbivores are important research areas that deserve further attention.

Research on plant-pathogen interactions provides a good model for approaches that can also be used to study plant interactions with herbivores. Many of the key pathogen defense genes in plants were discovered by means of mutant screens. Similarly, genetic mapping of plant mutations that alter herbivore resistance, or perhaps responses to purified insect elicitors, will almost certainly lead to the identification of previously unknown defense pathways. Research on plant-pathogen interactions has also benefited greatly from experimental systems in which it is possible to study responses and perform genetic manipulations on both sides of the interaction (e.g., *A. thaliana* and *Pseudomonas syringae*). Ongoing genome projects for *Medicago truncatula* (barrel medic) and *Acyrthosiphon pisum* (pea aphid) will provide the first plant-insect system in which it is possible to study broad-scale gene expression on both sides of the interaction. Beyond genome sequencing, additional effort should be placed on identifying insect genetic markers, studying natural variation in host plant utilization, and developing methods such as RNA interference for manipulating insect gene expression. The development of such research tools will facilitate studies on both sides of the plant-insect interaction and thereby achieve a more complete understanding of plant immunity to insect herbivores.

SUMMARY POINTS

1. Terrestrial plants use a combination of constitutive and inducible defensive traits to resist challenge by herbivorous insects; in natural ecosystems, any given plant species is consumed by only a small fraction of the herbivores in that environment.
2. Initial signaling events at the plant-insect interface (i.e., the bite zone), which include rapid changes in Ca^{2+} flux, membrane potential, and phosphorylation status, play an important role in the control of defensive processes but remain poorly understood.
3. Defense responses to insect attack are elicited by compounds in insect oral secretions. In the case of plant interactions with some hemipteran insects, there is evidence for the involvement of *R* genes in the control of host plant resistance.
4. Temporal and spatial patterns of mechanical tissue injury resulting from herbivory play a critical role in the production of endogenous signals that promote host defense responses.
5. The jasmonate signaling pathway is an evolutionarily conserved mechanism to regulate the expression of direct and indirect defenses. As relatively nonspecific sentinels of cellular injury, jasmonates promote resistance to a wide variety of biotic aggressors.

6. Herbivore attack results in rapid accumulation of JA and its bioactive conjugate, JA-Ile. Recent studies support a model in which binding of JA-Ile to COI1 triggers the degradation of JAZ repressor proteins and subsequent derepression of defensive genes.
7. Plant defensive metabolites and proteins thwart herbivory by exerting direct repellent, antifeedant, and toxic effects on the insect. Synergistic interactions between these compounds strengthen the host defense response.
8. Herbivore-induced plant volatiles serve several important functions in plant immunity to insect herbivores, including the attraction of insect predators and priming of defense responses.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

This review is dedicated to the memory of Clarence "Bud" Ryan, whose pioneering research inspired several generations of plant biologists to pursue the study of plant-insect interactions. The authors wish to thank Eric Schmelz, Peter Constabel, Martin de Vos, and Gary Felton for helpful comments on the manuscript. We also thank Jim Tumlinson, Tony Schilmiller, Rob Last, and Curtis Wilkerson for sharing unpublished information. Plant-insect interaction research in G.H.'s laboratory is currently supported by the National Institutes of Health (R01GM57795), the U.S. Department of Energy (DE-FG02-91ER20021), the U.S. Department of Agriculture (2007-35604-17791), and the National Science Foundation (DBI-0604336). Plant-insect interaction research in G.J.'s laboratory is supported by the U.S. Department of Agriculture (2005-35604-15446) and the National Science Foundation (IOS-0718733, DBI-0500550, and OISE-0436554).

LITERATURE CITED

1. Agrawal AA, Kurashige NS. 2003. A role for isothiocyanates in plant resistance against the specialist herbivore *Pieris rapae*. *J. Chem. Ecol.* 29:1403–15
2. Aharoni A, Jongsma MA, Bouwmeester HJ. 2005. Volatile science? Metabolic engineering of terpenoids in plants. *Trends Plant Sci.* 10:594–602
3. Alborn HT, Brennan EB, Tumlinson JH. 2003. Differential activity and degradation of plant volatile elicitors in regurgitant of tobacco hornworm (*Manduca sexta*) larvae. *J. Chem. Ecol.* 29:1357–72
4. Alborn HT, Hansen TV, Jones TH, Bennett DC, Tumlinson JH, et al. 2007. Novel disulfoxy fatty acids from the American bird grasshopper *Shistocerca americana*, elicitors of plant volatiles. *Proc. Natl. Acad. Sci. USA* 104:12976–81
5. Alborn HT, Turlings TCJ, Jones TH, Stenhagen G, Loughrin JH, Tumlinson JH. 1997. An elicitor of plant volatiles from beet armyworm oral secretion. *Science* 276:945–49

6. Amirhusin B, Shade RE, Koiwa H, Hasegawa PM, Bressan RA, et al. 2007. Protease inhibitors from several classes work synergistically against *Callosobruchus maculatus*. *J. Insect Physiol.* 53:734–40
7. Arimura G, Huber DP, Bohlmann J. 2004. Forest tent caterpillars (*Malacosoma disstria*) induce local and systemic diurnal emissions of terpenoid volatiles in hybrid poplar (*Populus trichocarpa* × *deltoides*): cDNA cloning, functional characterization, and patterns of gene expression of (-)-germacrene D synthase, *PtdTPS1*. *Plant J.* 37:603–16
8. Arimura G, Ozawa R, Kugimiya S, Takabayashi J, Bohlmann J. 2004. Herbivore-induced defense response in a model legume. Two-spotted spider mites induce emission of (*E*)-β-ocimene and transcript accumulation of (*E*)-β-ocimene synthase in *Lotus japonicus*. *Plant Physiol.* 135:1976–83
9. Arimura G, Ozawa R, Shimoda T, Nishioka T, Boland W, Takabayashi J. 2000. Herbivory-induced volatiles elicit defence genes in lima bean leaves. *Nature* 406:512–15
10. Baldwin IT. 1998. Jasmonate-induced responses are costly but benefit plants under attack in native populations. *Proc. Natl. Acad. Sci. USA* 95:8113–18
11. Barth C, Jander G. 2006. Arabidopsis myrosinases TGG1 and TGG2 have redundant function in glucosinolate breakdown and insect defense. *Plant J.* 46:549–62
12. Bayes A, Comellas-Bigler M, de la Vega MR, Maskos K, Bode W, et al. 2005. Structural basis of the resistance of an insect carboxypeptidase to plant protease inhibitors. *Proc. Natl. Acad. Sci. USA* 102:16602–7
13. Becerra JX. 2007. The impact of herbivore-plant coevolution on plant community structure. *Proc. Natl. Acad. Sci. USA* 104:7483–88
14. Bostock RM. 2005. Signal crosstalk and induced resistance: straddling the line between cost and benefit. *Annu. Rev. Phytopathol.* 43:545–80
15. Boughton AJ, Hoover K, Felton GW. 2005. Methyl jasmonate application induces increased densities of glandular trichomes on tomato, *Lycopersicon esculentum*. *J. Chem. Ecol.* 31:2211–16
16. Burow M, Muller R, Gershenzon J, Wittstock U. 2006. Altered glucosinolate hydrolysis in genetically engineered *Arabidopsis thaliana* and its influence on the larval development of *Spodoptera littoralis*. *J. Chem. Ecol.* 32:2333–49
17. Chen H, Gonzales-Vigil E, Wilkerson CG, Howe GA. 2007. Stability of plant defense proteins in the gut of insect herbivores. *Plant Physiol.* 143:1954–67
18. Chen H, Wilkerson CG, Kuchar JA, Phinney BS, Howe GA. 2005. Jasmonate-inducible plant enzymes degrade essential amino acids in the herbivore midgut. *Proc. Natl. Acad. Sci. USA* 102:19237–42
19. Chini A, Fonseca S, Fernández G, Adie B, Chico JM, et al. 2007. The JAZ family of repressors is the missing link in jasmonate signalling. *Nature* 448:666–71
20. Constabel CP, Bergey DR, Ryan CA. 1995. Systemin activates synthesis of wound-inducible tomato leaf polyphenol oxidase via the octadecanoid defense signaling pathway. *Proc. Natl. Acad. Sci. USA* 92:407–11
21. Cui JP, Jander G, Racki LR, Kim PD, Pierce NE, Ausubel FM. 2002. Signals involved in Arabidopsis resistance to *Trichoplusia ni* caterpillars induced by virulent and avirulent strains of the phytopathogen *Pseudomonas syringae*. *Plant Physiol.* 129:551–64
22. De Moraes CM, Mescher MC, Tumlinson JH. 2001. Caterpillar-induced nocturnal plant volatiles repel conspecific females. *Nature* 410:577–80
23. De Vos M, Van Oosten VR, Van Poecke RMP, Van Pelt JA, Pozo MJ, et al. 2005. Signal signature and transcriptome changes of *Arabidopsis* during pathogen and insect attack. *Mol. Plant-Microbe Interact.* 18:923–37

24. Devoto A, Ellis C, Magusin A, Chang HS, Chilcott C, et al. 2005. Expression profiling reveals COI1 to be a key regulator of genes involved in wound- and methyl jasmonate-induced secondary metabolism, defence, and hormone interactions. *Plant Mol. Biol.* 58:497–513
25. Devoto A, Turner JG. 2005. Jasmonate-regulated Arabidopsis stress signalling network. *Physiol. Plant.* 123:161–72
26. Dogimont C, Bendahmane A, Pitrat M, Burget-Bigeard E, Hagen L, et al. 2007. *U.S. Patent No. 20,070,016,977*
27. Ellis C, Karafyllidis I, Turner JG. 2002. Constitutive activation of jasmonate signaling in an *Arabidopsis* mutant correlates with enhanced resistance to *Erysiphe cichoracearum*, *Pseudomonas syringae*, and *Myzus persicae*. *Mol. Plant-Microbe Interact.* 15:1025–30
28. Engelberth J, Alborn HT, Schmelz EA, Tumlinson JH. 2004. Airborne signals prime plants against insect herbivore attack. *Proc. Natl. Acad. Sci. USA* 101:1781–85
29. Felton GW, Bi JL, Summers CB, Mueller AJ, Duffey SS. 1994. Potential role of lipoxygenases in defense against insect herbivory. *J. Chem. Ecol.* 20:651–66
30. Frey M, Chomet P, Glawischnig E, Stettner C, Grun S, et al. 1997. Analysis of a chemical plant defense mechanism in grasses. *Science* 277:696–99
31. Frost CJ, Appel HM, Carlson JE, De Moraes CM, Mescher MC, Schultz JC. 2007. Within-plant signalling via volatiles overcomes vascular constraints on systemic signalling and primes responses against herbivores. *Ecol. Lett.* 10:490–98
32. Gao LL, Anderson JP, Klingler JP, Nair RM, Edwards OR, Singh KB. 2007. Involvement of the octadecanoid pathway in bluegreen aphid resistance in *Medicago truncatula*. *Mol. Plant-Microbe Interact.* 20:82–93
33. Gatehouse JA. 2002. Plant resistance towards insect herbivores: a dynamic interaction. *New Phytol.* 156:145–69
34. Giri AP, Harsulkar AM, Deshpande VV, Sainani MN, Gupta VS, Ranjekar PK. 1998. Chickpea defensive proteinase inhibitors can be inactivated by podborer gut proteinases. *Plant Physiol.* 116:393–401
35. Giri AP, Wunsche H, Mitra S, Zavala JA, Muck A, et al. 2006. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. VII. Changes in the plant's proteome. *Plant Physiol.* 142:1621–41
36. Glazebrook J. 2005. Contrasting mechanisms of defense against biotrophic and necrotrophic pathogens. *Annu. Rev. Phytopathol.* 43:205–27
37. Gómez-Gómez L, Boller T. 2002. Flagellin perception: a paradigm for innate immunity. *Trends Plant Sci.* 7:251–56
38. Green TR, Ryan CA. 1972. Wound-induced proteinase inhibitor in plant leaves: a possible defense mechanism against insects. *Science* 175:776–77
39. Halitschke R, Gase K, Hui D, Schmidt DD, Baldwin IT. 2003. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. VI. Microarray analysis reveals that most herbivore-specific transcriptional changes are mediated by fatty acid-amino acid conjugates. *Plant Physiol.* 131:1894–902
40. Halitschke R, Schittko U, Pohnert G, Boland W, Baldwin IT. 2001. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. III. Fatty acid-amino acid conjugates in herbivore oral secretions are necessary and sufficient for herbivore-specific plant responses. *Plant Physiol.* 125:711–17
41. Halkier BA, Gershenzon J. 2006. Biology and biochemistry of glucosinolates. *Annu. Rev. Plant Biol.* 57:303–33

42. Hamel LP, Nicole MC, Sritubtim S, Morency MJ, Ellis M, et al. 2006. Ancient signals: comparative genomics of plant MAPK and MAPKK gene families. *Trends Plant Sci.* 11:192–98
43. Harfouche AL, Shivaji R, Stocker R, Williams PW, Luthe DS. 2006. Ethylene signaling mediates a maize defense response to insect herbivory. *Mol. Plant-Microbe Interact.* 19:189–99
44. Hause B, Stenzel I, Miersch O, Maucher H, Kramell R, et al. 2000. Tissue-specific oxylipin signature of tomato flowers: Allene oxide cyclase is highly expressed in distinct flower organs and vascular bundles. *Plant J.* 24:113–26
45. Heidel AJ, Baldwin IT. 2004. Microarray analysis of salicylic acid- and jasmonic acid-signalling in responses of *Nicotiana attenuata* to attack by insects from multiple feeding guilds. *Plant Cell Environ.* 27:1362–73
46. Heil M, Silva Bueno JC. 2007. Within-plant signaling by volatiles leads to induction and priming of an indirect plant defense in nature. *Proc. Natl. Acad. Sci. USA* 104:5467–72
47. Herms DA, Mattson WJ. 1992. The dilemma of plants: to grow or defend. *Q. Rev. Biol.* 67:283–335
48. Hilker M, Meiners T. 2006. Early herbivore alert: Insect eggs induce plant defense. *J. Chem. Ecol.* 32:1379–97
49. Horiuchi J, Arimura G, Ozawa R, Shimoda T, Takabayashi J, Nishioka T. 2001. Exogenous ACC enhances volatiles production mediated by jasmonic acid in lima bean leaves. *FEBS Lett.* 509:332–36
50. Howe GA. 2004. Jasmonates as signals in the wound response. *J. Plant Growth Regul.* 23:223–37
51. Hudgins JW, Franceschi VR. 2004. Methyl jasmonate-induced ethylene production is responsible for conifer phloem defense responses and reprogramming of stem cambial zone for traumatic resin duct formation. *Plant Physiol.* 135:2134–49
52. Hui DQ, Iqbal J, Lehmann K, Gase K, Saluz HP, Baldwin IT. 2003. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. V. Microarray analysis and further characterization of large-scale changes in herbivore-induced mRNAs. *Plant Physiol.* 131:1877–93
53. Hummelbrunner LA, Isman MB. 2001. Acute, sublethal, antifeedant, and synergistic effects of monoterpenoid essential oil compounds on the tobacco cutworm, *Spodoptera litura* (Lep., Noctuidae). *J. Agric. Food Chem.* 49:715–20
54. Johnson KS, Barbehenn RV. 2000. Oxygen levels in the gut lumens of herbivorous insects. *J. Insect Physiol.* 46:897–903
55. Jones JD, Dangl JL. 2006. The plant immune system. *Nature* 444:323–29
56. Jongsma MA, Bakker PL, Peters J, Bosch D, Stiekema WJ. 1995. Adaptation of *Spodoptera exigua* larvae to plant proteinase inhibitors by induction of gut proteinase activity insensitive to inhibition. *Proc. Natl. Acad. Sci. USA* 92:8041–45
57. Kaloshian I, Walling LL. 2005. Hemipterans as plant pathogens. *Annu. Rev. Phytopathol.* 43:491–521
58. Kandoth PK, Ranf S, Pancholi SS, Jayanty S, Walla MD, et al. 2007. Tomato MAPKs LeMPK1, LeMPK2, and LeMPK3 function in the systemin-mediated defense response against herbivorous insects. *Proc. Natl. Acad. Sci. USA* 104:12205–10
59. Kang JH, Wang L, Giri A, Baldwin IT. 2006. Silencing threonine deaminase and JAR4 in *Nicotiana attenuata* impairs jasmonic acid-isoleucine-mediated defenses against *Manduca sexta*. *Plant Cell* 18:3303–20
60. Karban R, Agrawal AA, Mangel M. 1997. The benefits of induced defenses against herbivores. *Ecology* 78:1351–55

61. Karban R, Baldwin IT. 1997. *Induced Responses to Herbivory*. Chicago, IL: Univ. Chicago Press
62. Karban R, Baldwin IT, Baxter KJ, Laue G, Felton GW. 2000. Communication between plants: induced resistance in wild tobacco plants following clipping of neighboring sagebrush. *Oecologia* 125:66–71
63. Karban R, Shiojiri K, Huntzinger M, McCall AC. 2006. Damage-induced resistance in sagebrush: Volatiles are key to intra- and interplant communication. *Ecology* 87:922–30
64. Katou S, Yoshioka H, Kawakita K, Rowland O, Jones JDG, et al. 2005. Involvement of PPS3 phosphorylated by elicitor-responsive mitogen-activated protein kinases in the regulation of plant cell death. *Plant Physiol.* 139:1914–26
65. Kempema LA, Cui X, Holzer FM, Walling LL. 2007. Arabidopsis transcriptome changes in response to phloem-feeding silverleaf whitefly nymphs. Similarities and distinctions in responses to aphids. *Plant Physiol.* 143:849–65
66. Kessler A, Baldwin IT. 2001. Defensive function of herbivore-induced plant volatile emissions in nature. *Science* 291:2141–44
67. Kessler A, Baldwin IT. 2002. Plant responses to insect herbivory: the emerging molecular analysis. *Annu. Rev. Plant Biol.* 53:299–328
68. Kessler A, Halitschke R, Baldwin IT. 2004. Silencing the jasmonate cascade: induced plant defenses and insect populations. *Science* 305:665–68
69. Kessler A, Halitschke R, Diezel C, Baldwin IT. 2006. Priming of plant defense responses in nature by airborne signaling between *Artemisia tridentata* and *Nicotiana attenuata*. *Oecologia* 148:280–92
70. Kim JH, Jander G. 2007. *Myzus persicae* (green peach aphid) feeding on Arabidopsis induces the formation of a deterrent indole glucosinolate. *Plant J.* 49:1008–19
71. Koch T, Krumm T, Jung V, Engelberth J, Boland W. 1999. Differential induction of plant volatile biosynthesis in the lima bean by early and late intermediates of the octadecanoid-signaling pathway. *Plant Physiol.* 121:153–62
72. Konno K, Hirayama C, Nakamura M, Tateishi K, Tamura Y, et al. 2004. Papain protects papaya trees from herbivorous insects: role of cysteine proteases in latex. *Plant J.* 37:370–78
73. Lambrix V, Reichelt M, Mitchell-Olds T, Kliebenstein DJ, Gershenzon J. 2001. The Arabidopsis epithiospecifier protein promotes the hydrolysis of glucosinolates to nitriles and influences *Trichoplusia ni* herbivory. *Plant Cell* 13:2793–807
74. Lawrence SD, Novak NG. 2006. Expression of poplar chitinase in tomato leads to inhibition of development in Colorado potato beetle. *Biotech. Lett.* 28:593–99
75. Levy M, Wang Q, Kaspi R, Parrella MP, Abel S. 2005. Arabidopsis IQD1, a novel calmodulin-binding nuclear protein, stimulates glucosinolate accumulation and plant defense. *Plant J.* 43:79–96
76. Li C, Schilmiller AL, Liu G, Lee GI, Jayanty S, et al. 2005. Role of β-oxidation in jasmonate biosynthesis and systemic wound signaling in tomato. *Plant Cell* 17:971–86
77. Li L, Zhao Y, McCaig BC, Wingerd BA, Wang J, et al. 2004. The tomato homolog of *CORONATINE-INSENSITIVE1* is required for the maternal control of seed maturation, jasmonate-signaled defense responses, and glandular trichome development. *Plant Cell* 16:126–43
78. Li Q, Xie QG, Smith-Becker J, Navarre DA, Kaloshian I. 2006. *Mi-1*-mediated aphid resistance involves salicylic acid and mitogen-activated protein kinase signaling cascades. *Mol. Plant-Microbe Interact.* 19:655–64
79. Lison P, Rodrigo I, Conejero V. 2006. A novel function for the cathepsin D inhibitor in tomato. *Plant Physiol.* 142:1329–39

80. Little D, Gouhier-Darimont C, Bruessow F, Reymond P. 2007. Oviposition by pierid butterflies triggers defense responses in Arabidopsis. *Plant Physiol.* 143:784–800
81. Maffei M, Bossi S, Spiteller D, Mithöfer A, Boland W. 2004. Effects of feeding *Spodoptera littoralis* on lima bean leaves. I. Membrane potentials, intracellular calcium variations, oral secretions, and regurgitate components. *Plant Physiol.* 134:1752–62
82. Maffei ME, Mithöfer A, Arimura G, Uchtenhagen H, Bossi S, et al. 2006. Effects of feeding *Spodoptera littoralis* on lima bean leaves. III. Membrane depolarization and involvement of hydrogen peroxide. *Plant Physiol.* 140:1022–35
83. Maffei ME, Mithöfer A, Boland W. 2007. Before gene expression: early events in plant-insect interaction. *Trends Plant Sci.* 12:310–16
84. Major IT, Constabel CP. 2006. Molecular analysis of poplar defense against herbivory: comparison of wound- and insect elicitor-induced gene expression. *New Phytol.* 172:617–35
85. Mattson WJ. 1980. Herbivory in relation to plant nitrogen-content. *Annu. Rev. Ecol. System.* 11:119–61
86. Matzinger P. 2002. The danger model: a renewed sense of self. *Science* 296:301–5
87. Mewis I, Appel HM, Hom A, Raina R, Schultz JC. 2005. Major signaling pathways modulate Arabidopsis glucosinolate accumulation and response to both phloem-feeding and chewing insects. *Plant Physiol.* 138:1149–62
88. Mewis I, Tokuhisa JG, Schultz JC, Appel HM, Ulrichs C, Gershenzon J. 2006. Gene expression and glucosinolate accumulation in *Arabidopsis thaliana* in response to generalist and specialist herbivores of different feeding guilds and the role of defense signaling pathways. *Phytochemistry* 67:2450–62
89. Miles PW. 1999. Aphid saliva. *Biol. Rev.* 74:41–85
90. Mithöfer A, Wanner G, Boland W. 2005. Effects of feeding *Spodoptera littoralis* on lima bean leaves. II. Continuous mechanical wounding resembling insect feeding is sufficient to elicit herbivory-related volatile emission. *Plant Physiol.* 137:1160–68
91. Mohan S, Ma PWK, Pechan T, Bassford ER, Williams WP, Luthe DS. 2006. Degradation of the *S. frugiperda* peritrophic matrix by an inducible maize cysteine protease. *J. Insect Physiol.* 52:21–28
92. 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.* 47:749–57
93. Musser RO, Farmer E, Peiffer M, Williams SA, Felton GW. 2006. Ablation of caterpillar labial salivary glands: technique for determining the role of saliva in insect-plant interactions. *J. Chem. Ecol.* 32:981–92
94. Nombela G, Williamson VM, Muniz M. 2003. The root-knot nematode resistance gene *Mi-1.2* of tomato is responsible for resistance against the whitefly *Bemisia tabaci*. *Mol. Plant-Microbe Interact.* 16:645–49
95. Pare PW, Alborn HT, Tumlinson JH. 1998. Concerted biosynthesis of an insect elicitor of plant volatiles. *Proc. Natl. Acad. Sci. USA* 95:13971–75
96. Paschold A, Halitschke R, Baldwin IT. 2007. Co(i)-ordinating defenses: NaCOI1 mediates herbivore-induced resistance in *Nicotiana attenuata* and reveals the role of herbivore movement in avoiding defenses. *Plant J.* 51:79–91
97. Peumans WJ, Vandamme EJM. 1995. Lectins as plant defense proteins. *Plant Physiol.* 109:347–52
98. Pohnert G, Jung V, Haukioja E, Lempa K, Boland G. 1999. New fatty acid amides from regurgitant of Lepidopteran (Noctuidae, Geometridae) caterpillars. *Tetrahedron* 55:11275–80

99. Ralph S, Oddy C, Cooper D, Yueh H, Jancsik S, et al. 2006. Genomics of hybrid poplar (*Populus trichocarpa* × *deltoides*) interacting with forest tent caterpillars (*Malacosoma disstria*): normalized and full-length cDNA libraries, expressed sequence tags, and a cDNA microarray for the study of insect-induced defences in poplar. *Mol. Ecol.* 15:1275–97
100. Ralph SG, Yueh H, Friedmann M, Aeschliman D, Zeznik JA, et al. 2006. Conifer defence against insects: Microarray gene expression profiling of Sitka spruce (*Picea sitchensis*) induced by mechanical wounding or feeding by spruce budworms (*Choristoneura occidentalis*) or white pine weevils (*Pissodes strobi*) reveals large-scale changes of the host transcriptome. *Plant Cell Environ.* 29:1545–70
101. Rasmann S, Köllner TG, Degenhardt J, Hiltpold I, Toepfer S, et al. 2005. Recruitment of entomopathogenic nematodes by insect-damaged maize roots. *Nature* 434:732–37
102. Reymond P, Bodenhausen N, Van Poecke RM, Krishnamurthy V, Dicke M, Farmer EE. 2004. A conserved transcript pattern in response to a specialist and a generalist herbivore. *Plant Cell* 16:3132–47
103. Reymond P, Weber H, Damond M, Farmer EE. 2000. Differential gene expression in response to mechanical wounding and insect feeding in *Arabidopsis*. *Plant Cell* 12:707–20
104. Rivard D, Cloutier C, Michaud D. 2004. Colorado potato beetles show differential digestive compensatory responses to host plants expressing distinct sets of defense proteins. *Arch. Insect Biochem. Physiol.* 55:114–23
105. Robert-Seilaniantz A, Navarro L, Bari R, Jones JD. 2007. Pathological hormone imbalances. *Curr. Opin. Plant Biol.* 10:372–79
106. Rossi M, Goggin FL, Milligan SB, Kaloshian I, Ullman DE, Williamson VM. 1998. The nematode resistance gene *Mi* of tomato confers resistance against the potato aphid. *Proc. Natl. Acad. Sci. USA* 95:9750–54
107. Ryan CA. 1990. Protease inhibitors in plants: Genes for improving defenses against insects and pathogens. *Annu. Rev. Phytopathol.* 28:425–49
108. Schaller F, Schaller A, Stintzi A. 2005. Biosynthesis and metabolism of jasmonates. *J. Plant Growth Regul.* 23:179–99
109. Schilmiller AL, Howe GA. 2005. Systemic signaling in the wound response. *Curr. Opin. Plant Biol.* 8:369–77
110. Schilmiller AL, Koo AJ, Howe GA. 2007. Functional diversification of acyl-CoA oxidases in jasmonic acid biosynthesis and action. *Plant Physiol.* 143:812–24
111. Schmelz EA, Alborn HT, Tumlinson JH. 2003. Synergistic interactions between volicitin, jasmonic acid and ethylene mediate insect-induced volatile emission in *Zea mays*. *Physiol. Plant.* 117:403–12
112. Schmelz EA, Carroll MJ, LeClere S, Phipps SM, Meredith J, et al. 2006. Fragments of ATP synthase mediate plant perception of insect attack. *Proc. Natl. Acad. Sci. USA* 103:8894–99
113. Schmelz EA, Leclere S, Carroll MJ, Alborn HT, Teal PEA. 2007. Cowpea (*Vigna unguiculata*) chloroplastic ATP synthase is the source of multiple plant defense elicitors during insect herbivory. *Plant Physiol.* 144:793–805
114. Schmidt S, Baldwin IT. 2006. Systemin in *Solanum nigrum*. The tomato-homologous polypeptide does not mediate direct defense responses. *Plant Physiol.* 142:1751–58
115. Schnee C, Köllner TG, Held M, Turlings TC, Gershenzon J, Degenhardt J. 2006. The products of a single maize sesquiterpene synthase form a volatile defense signal that attracts natural enemies of maize herbivores. *Proc. Natl. Acad. Sci. USA* 103:1129–34
116. Schoonhoven LM, Jermey T, van Loon JJA. 1998. *Insect-Plant Biology: From Physiology to Evolution*. London: Chapman & Hall

117. Schroder R, Cristescu SM, Harren FJ, Hilker M. 2007. Reduction of ethylene emission from Scots pine elicited by insect egg secretion. *J. Exp. Bot.* 58:1835–42
118. Schwachtje J, Minchin PE, Jahnke S, van Dongen JT, Schittko U, Baldwin IT. 2006. SNF1-related kinases allow plants to tolerate herbivory by allocating carbon to roots. *Proc. Natl. Acad. Sci. USA* 103:12935–40
119. Smith CM, Boyko EV. 2007. The molecular bases of plant resistance and defense responses to aphid feeding: current status. *Entomol. Exp. Appl.* 122:1–16
120. Spiteller D, Pohnert G, Boland G. 2001. Absolute configuration of volicitin, an elicitor of plant volatile biosynthesis from lepidopteran larvae. *Tetrahedron Lett.* 42:1483–85
121. Staswick PE, Tiryaki I. 2004. The oxylipin signal jasmonic acid is activated by an enzyme that conjugates it to isoleucine in Arabidopsis. *Plant Cell* 16:2117–27
122. 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
123. Stintzi A, Weber H, Reymond P, Browse J, Farmer EE. 2001. Plant defense in the absence of jasmonic acid: the role of cyclopentenones. *Proc. Natl. Acad. Sci. USA* 98:12837–42
124. Taki N, Sasaki-Sekimoto Y, Obayashi T, Kikuta A, Kobayashi K, et al. 2005. 12-oxo-phytodienoic acid triggers expression of a distinct set of genes and plays a role in wound-induced gene expression in Arabidopsis. *Plant Physiol.* 139:1268–83
125. Talyzina NM, Ingvarsson PK. 2006. Molecular evolution of a small gene family of wound inducible Kunitz trypsin inhibitors in *Populus*. *J. Mol. Evol.* 63:108–19
126. Tan X, Calderon-Villalobos LI, Sharon M, Zheng C, Robinson CV, et al. 2007. Mechanism of auxin perception by the TIR1 ubiquitin ligase. *Nature* 446:640–45
127. Tattersall DB, Bak S, Jones PR, Olsen CE, Nielsen JK, et al. 2001. Resistance to an herbivore through engineered cyanogenic glucoside synthesis. *Science* 293:1826–28
128. Thaler JS. 1999. Jasmonate-inducible plant defences cause increased parasitism of herbivores. *Nature* 399:686–88
129. Thines B, Katsir L, Melotto M, Niu Y, Mandaokar A, et al. 2007. JAZ repressor proteins are targets of the SCFCOI1 complex during jasmonate signalling. *Nature* 448:661–65
130. Thipyapong P, Joel DM, Steffens JC. 1997. Differential expression and turnover of the tomato polyphenol oxidase gene family during vegetative and reproductive development. *Plant Physiol.* 113:707–18
131. Thompson GA, Goggin FL. 2006. Transcriptomics and functional genomics of plant defence induction by phloem-feeding insects. *J. Exp. Bot.* 57:755–66
132. Ton J, D'Alessandro M, Jourdie V, Jakab G, Karlen D, et al. 2007. Priming by airborne signals boosts direct and indirect resistance in maize. *Plant J.* 49:16–26
133. Tooker JF, De Moraes CM. 2005. Jasmonate in lepidopteran eggs and neonates. *J. Chem. Ecol.* 31:2753–59
134. Truitt CL, Wei HX, Pare PW. 2004. A plasma membrane protein from *Zea mays* binds with the herbivore elicitor volicitin. *Plant Cell* 16:523–32
135. Turlings TCJ, Tumlinson JH, Lewis WJ. 1990. Exploitation of herbivore-induced plant odors by host-seeking parasitic wasps. *Science* 250:1251–53
136. van Poecke RM, Dicke M. 2002. Induced parasitoid attraction by *Arabidopsis thaliana*: involvement of the octadecanoid and the salicylic acid pathway. *J. Exp. Bot.* 53:1793–99
137. Voelckel C, Weisser WW, Baldwin IT. 2004. An analysis of plant-aphid interactions by different microarray hybridization strategies. *Mol. Ecol.* 13:3187–95
138. von Dahl CC, Baldwin IT. 2007. Deciphering the role of ethylene in plant-herbivore interactions. *J. Plant Growth Regul.* 26:201–9

139. Wang JH, Constabel CP. 2004. Polyphenol oxidase overexpression in transgenic *Populus* enhances resistance to herbivory by forest tent caterpillar (*Malacosoma disstria*). *Planta* 220:87–96
140. Winterer J, Bergelson J. 2001. Diamondback moth compensatory consumption of protease inhibitor-transformed plants. *Mol. Ecol.* 10:1069–74
141. Winz RA, Baldwin IT. 2001. Molecular interactions between the specialist herbivore *Manduca sexta* (Lepidoptera, Sphingidae) and its natural host *Nicotiana attenuata*. IV. Insect-induced ethylene reduces jasmonate-induced nicotine accumulation by regulating putrescine *N*-methyltransferase transcripts. *Plant Physiol.* 125:2189–202
142. Wu J, Hettenhausen C, Meldau S, Baldwin IT. 2007. Herbivory rapidly activates MAPK signaling in attacked and unattacked leaf regions but not between leaves of *Nicotiana attenuata*. *Plant Cell* 19:1096–122
143. Yan Y, Stolz S, Chetelat A, Reymond P, Pagni M, et al. 2007. A downstream mediator in the growth repression limb of the jasmonate pathway. *Plant Cell* 19:2470–83
144. You MK, Oh SI, Ok SH, Cho SK, Shin HY, et al. 2007. Identification of putative MAPK kinases in *Oryza minuta* and *O. sativa* responsive to biotic stresses. *Mol. Cells* 23:108–14
145. Zarate SI, Kempema LA, Walling LL. 2007. Silverleaf whitefly induces salicylic acid defenses and suppresses effectual jasmonic acid defenses. *Plant Physiol.* 143:866–75
146. Zavala JA, Patankar AG, Gase K, Hui DQ, Baldwin IT. 2004. Manipulation of endogenous trypsin proteinase inhibitor production in *Nicotiana attenuata* demonstrates their function as antiherbivore defenses. *Plant Physiol.* 134:1181–90
147. Zhang Z, Ober JA, Kliebenstein DJ. 2006. The gene controlling the quantitative trait locus *EPITHIOSPECIFIER MODIFIER1* alters glucosinolate hydrolysis and insect resistance in Arabidopsis. *Plant Cell* 18:1524–36
148. Zipfel C, Kunze G, Chinchilla D, Caniard A, Jones JDG, et al. 2006. Perception of the bacterial PAMP EF-Tu by the receptor EFR restricts *Agrobacterium*-mediated transformation. *Cell* 125:749–60

Annual Review of
Plant Biology
Volume 59, 2008

Contents

Indexes

Errata

An online log of corrections to *Annual Review of Plant Biology* articles may be found at http://plant.annualreviews.org/