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The Myriad Plant Responses to Herbivores

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Abstract

Plant responses to herbivores are complex. Genes activated on herbivore attack are strongly correlated with the mode of herbivore feeding and the degree of tissue damage at the feeding site. Phloem-feeding whiteflies and aphids that produce little injury to plant foliage are perceived as pathogens and activate the salicylic acid (SA)-dependent and jasmonic acid (JA)/ethylene-dependent signaling pathways. Differential expression of plant genes in response to closely related insect species suggest that some elicitors generated by phloem-feeding insects are species-specific and are dependent on the herbivore's developmental stage. Other elicitors for defense-gene activation are likely to be more ubiquitous. Analogies to the pathogen-incompatible reactions are found. Chewing insects such as caterpillars and beetles and cell-content feeders such as mites and thrips cause more extensive tissue damage and activate wound-signaling pathways. Herbivore feeding is not equivalent to mechanical wounding. Wound responses are a part of the induced responses that accompany herbivore feeding. Herbivores induce direct defenses that interfere with herbivore feeding, growth and development, fecundity, and fertility. In addition, herbivores induce an array of volatiles that creates an indirect mechanism of defense. Volatile blends provide specific cues to attract herbivore parasites and predators to infested plants. The nature of the elicitors for volatile production is discussed.

Key words: Phloem-feeding insects; Jasmonic acid; Pathogenesis-related proteins; Ethylene; Saliva; Signal transduction; Salicylic acid; Defense; Chewing insects; Wounding

Introduction

In their natural habitat, plants encounter multiple biotic and abiotic challenges simultaneously. Each environmental hazard activates multiple signal transduction pathways to ensure an effective spatial and temporal defense response (Dempsey and others 1999; Genoud and Métraux 1999; Pieterse and van Loon 1999; Ryan 2000). Therefore, plants must be able to identify and prioritize each signaling pathway to mount the most efficacious defense strategy to minimize current and future damage and also to preserve vegetative growth and reproductive success (Karban and Baldwin 1997). These complex biochemical and physiologic responses often result in a tolerance or protection from further environmental challenges (Bostock 1999; Dempsey and others 1999; Karban and Baldwin 1997; Pieterse and van Loon 1999).

To protect themselves from pathogen and herbivore attack, plants use constitutive and induced defenses. These defenses can influence herbivore settling, feeding, oviposition, growth and development, fecundity, and/or fertility. All defenses, whether constantly or transiently expressed, are costly (Baldwin and Preston 1999). Defense responses channel

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carbon and nitrogen resources from vegetative and reproductive growth into protective mechanisms. The plant must attain a balance to ensure survival from immediate and subsequent attacks without sacrificing plant vitality, longevity, or reproduction. The balance of constitutive and induced defense responses appears to supply the plant with the flexibility to achieve these goals.

Constitutive defenses include the physical barriers that impede pathogen ingression or arthropod access to tissues, that is, cell walls, suberin, callose, and cuticles, as well as the stored allelochemicals that have antixenotic (deters herbivore colonization of plant) or antibiotic (deters herbivore growth, reproduction, development, or survival) effects (Conn 1981; Hedin 1983; Paiva 2000; Rosenthal and Janzen 1979). Constitutive defenses, as well as induced defenses, are only effective if the herbivore contacts the defense phytochemical. Because allelochemicals are often species-specific and are expressed in a subset of tissues or cells, the choice of host plant by an herbivore and the mode and site of herbivore feeding determines the defense chemicals that are encountered.

Most of our knowledge of plant responses to herbivores has been gleaned from studies with insects that extensively damage foliage (Karban and Baldwin 1997; Stotz and others 1999). Far less is known about plant responses to herbivores that cause less tissue damage such as arthropods that mine or gall, or herbivores that pierce or lacerate cells to feed on intracellular fluids (Gerling and Mayer 1996; Miles 1999; Needham and others 1928; Raven 1983; Raman 1994). The landmark text by Karban and Baldwin (1997) provides a comprehensive literature on induced responses to damage-inducing herbivores. By contrast, the literature on plant responses to nonchewing insects has emerged more recently. This review will focus primarily on this latter group of herbivores. Herbivores induce several well-characterized plant defense- and wound-response pathways, as well as novel pathways to alter plant gene expression (Figures 1, 2). Herbivores produce novel signals (elicitors) to activate plant gene expression and volatile synthesis (Korth and Dixon 1997a; Páre and others 1998; van de Ven and others 2000). The source of these cues will be discussed.

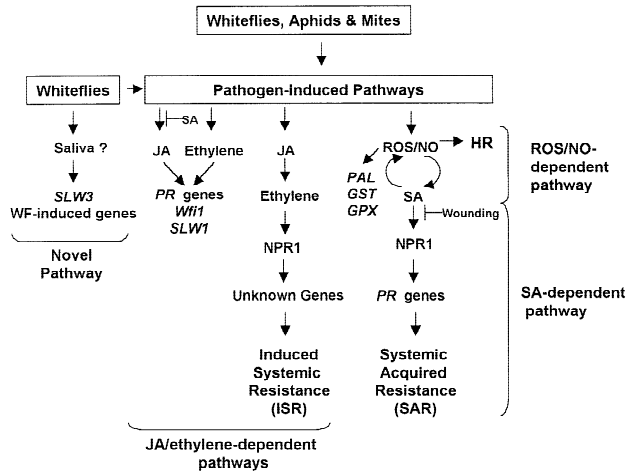

Figure 1. The signal transduction pathways induced by pathogens and herbivores are outlined (see text for details). Genes regulated by these pathways are described in Table 1. Pathogens use four signaling pathways to activate gene expression: the SA-, the ROS-, and two JA/ethylene-dependent pathways. Phloem-feeding whiteflies and aphids activate both the JA/ethylene and SA-dependent pathways. It is unclear whether herbivores activate ISR. Evidence for a novel pathway activated by whitefly (WF) feeding is provided by the *SLW3* gene of squash. The source of the elicitor for *SLW3* may be provided by digestive or sheath saliva (saliva?). This figure is based on data compiled from several plant species (*Arabidopsis,* tomato, and squash).

Responses to Piercing/sucking Insects: Activation of Pathogen-defense Response Pathways

Insects that use a piercing/sucking mode of feeding have an intimate and long-lasting interaction with plant cells. Using a stylet to pierce cells, these herbivores consume large quantities of fluids as a nutritional source. Feeding sites vary. Most aphids, mealy bugs, leafhoppers, psyllids, and whiteflies stylets must traverse the cuticle, epidermis, and mesophyll to establish feeding sites in veins of the phloem (Miles 1999; Raven 1983). Other piercing/sucking insects primarily feed on (1) mesophyll parenchyma (scale insects), (2) both epidermal and mesophyll cells (thrips), or (3) xylem (leafhoppers) (Parker and others 1995; Raven 1983; Rosen 1990). The amount of tissue damage caused by piercing/sucking insects varies tremendously. Some herbivores (like thrips and spider mites) are cell-content feeders; they lacerate cells and consume cellular contents by means of their stylets (Helle and Sabelis 1985; Parker and others 1995). Other cell-content feeders such as the pyrrhocorids and lygaeids (true-bugs) cause more extensive damage along the path to and at their feeding site (Saxena 1963; Taylor and Miles 1994).

The stylets of piercing/sucking insects that feed on phloem are in continuous contact with plant cells. Once feeding sites are established, they can be

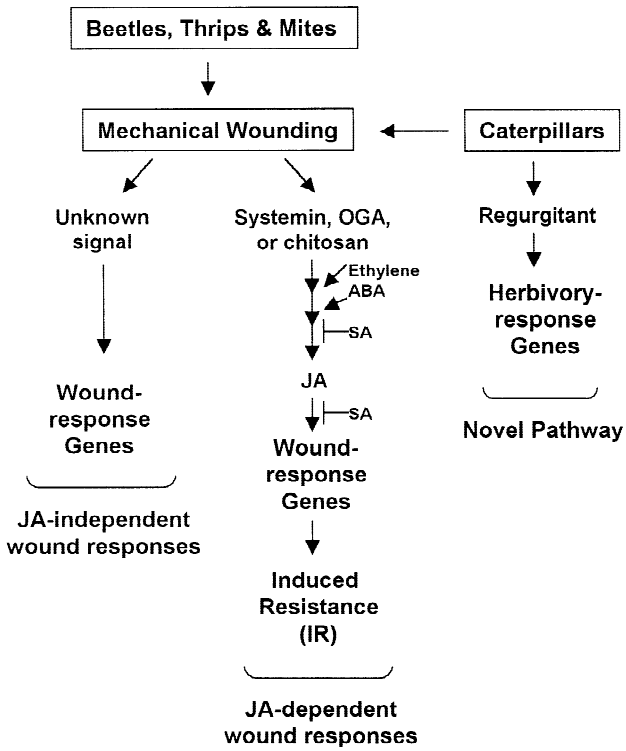

Figure 2. The signal transduction pathways induced by wounding and herbivores in the Solanaceae are illustrated (see text for details). Genes regulated by these pathways are described in Table 1. Chewing insects (beetles and caterpillars) and cell-content feeders (mites and thrips) induce the JA-regulated wound-response pathway. Evidence for a JA-independent pathway in the Solanaceae exists but its regulator is unknown. Caterpillar regurgitant induces novel genes not activated by wounding, providing evidence for a novel herbivory-response pathway.

used for hours to weeks. Given the limited tissue damage and prolonged stylet interactions with plant cells, it is not surprising to find that plant responses to phloem-feeding insects are distinct from that of chewing insects and tissue-damaging cell-content feeders. On the basis of the limited number of studies currently available, some piercing/sucking insects induce the defense-signaling pathways most commonly activated by bacterial, fungal, and viral pathogens (Figure 1).

Plant Responses to Pathogen Attack

Signaling mechanisms activated after pathogen attack have been elegantly dissected using the tools of genetics, molecular biology, and biochemistry (for reviews, see Dempsey and others 1999; Glazebrook 1999; Martin 1999; McDowell and Dangl 2000; Pieterse and van Loon 1999). When pathogens and plants with cognate avirulence (*avr*) and resistance (*R*) genes interact (incompatible interactions), pathogens are rapidly perceived. Plants respond with production of reactive oxygen species (ROS) and nitric oxide (NO), membrane depolarizations, ion fluxes, and activation of signaling cascades that involve the action of kinases and phosphatases (Bolwell and Wojtaszek 1997; Dempsey and others 1999). The signaling cascades result in the accumulation of defense-response RNAs and proteins locally and systemically (Figure 1; Table 1). Defense-response proteins hydrolyze pathogen cell wall polymers, strengthen and modify plant cell walls, turnover proteins, enhance synthesis of secondary metabolites, generate signals to modulate defense-signaling pathways, or have unknown functions in defense (for reviews, see Kombrink and Somssich 1997; Reymond and Farmer 1998). Similar biochemical events are induced in compatible interactions but at a slower pace. Incompatible (fast recognition) and compatible (slow recognition) interactions are also distinguished by the presence or absence of a hypersensitive response (HR), respectively. During incompatible interactions, the HR causes a rapid, localized cell death at the site of infection, which restricts the pathogen to small areas of tissue. Micro-HRs occur systemically and may aid in activation of defense responses at remote locations on the plant (Alvarez and others 1998).

Four signaling pathways are important in responses to pathogens (Figure 1). First, the salicylic acid (SA)-dependent cascade uses SA (Figure 3) and its methyl conjugate (MeSA; Figure 3) to stimulate expression of defense-response genes, including pathogenesis-related protein (*PR*) genes that encode proteins with an apoplastic localization (acidic *PR* genes) (Table 1) (Kombrink and Somssich 1997; Shulaev and others 1997). SA also promotes the development of systemic acquired resistance (SAR), which confers a broad-range resistance to pathogens and some insects (Bostock 1999; Dempsey and others 1999). The second pathway is dependent on ROS and NO, which increase after pathogen attack. These molecules promote an HR, stimulate SA synthesis, and induce some defense-response genes (Figure 1; Table 1) (Lamb and Dixon 1997; McDowell and Dangl 2000).

The remaining two pathways are regulated by jasmonic acid (JA) and ethylene (Figure 1) (Chao and others 1999; Penninckx and others 1998; van Wees and others 1999). Both JA (Figure 3) and ethylene (Figure 3) levels increase after pathogen attack. In *Arabidopsis,* JA and ethylene act concomitantly to induce expression of defensin and *PR* genes that encode vacuolar-localized proteins (basic *PR* genes)

Table 1. List of Genes and Their Roles in Plant Defense or Wound Responses

Genes	Function in Defense or Wounding
Tomato *Chi3*	*Chi3* encodes an acidic chitinase that hydrolyzes chitin in pathogen cells walls and possibly in insect guts. Acidic chitinases are localized in the apoplast.
Tomato *Chi9*	*Chi9* encodes a basic chitinase that hydrolyzes chitin in pathogen cells walls and possibly in insect guts. Basic chitinases are localized in the vacuole.
Tomato *GluAC*	*GluAC* encodes an acidic β-1,3-glucanase that hydrolyzes callose and glucan polymers of pathogen walls. Acidic glucanases are localized to the apoplast.
Tomato *GluB*	*GluB* encodes a basic β-1,3-glucanase that hydrolyzes callose and glucan polymers of pathogen walls. Basic glucanases are stored in the vacuole.
Tomato *LapA*	*LapA* encodes the wound-induced leucine aminopeptidase that removes *N*-terminal amino acids from peptides and proteins. *LAP-A* function in wounding is not known. It is only found in a subset of the Solanaceae (Figure 4).
Tomato *LOX* *Arabidopsis LOX1, LOX2*	LOX encodes the 13-lipoxygenase that synthesizes 13-hydroperoxide-octadecatrienoic acid from linolenic acid (an 18:3 fatty acid), (Figure 4).
Tomato *PAL* *Arabidopsis PAL*	PAL (phenylalanine ammonia lyase) is important for the biosynthesis of SA, flavanoid phytoalexins, lignin, and other cell wall phenolics.
Tomato *PR-1*	The *PR-1* gene of tomato encodes an acidic PR-1 protein also known as P4 and highly related to P6. Its function is not known. Acidic PR-1 has an apoplastic location. Basic *PR-1* genes also exist and encode proteins with a vacuolar location.
Tomato *P2*	The P2 protein is a basic win-like protein with a chitin-binding domain. Its function is unknown and it has a vacuolar location.
Tomato *PR-4*	*PR-4* encodes an acidic win-like protein with a chitin-binding domain. Its function is unknown and it has an apoplastic location.
Tomato *pin2, pin1* *Arabidopsis PIN*	*Pin* genes encode inhibitors of Ser proteases. Pins interfere with insect growth and development by hyper-inducing proteases in the insect gut (Figure 4).
Tomato *PG*	A wound-induced PG (polygalacturonase) hydrolyzes pectin in the cell wall to release OGAs, which are potent signals that activate the tomato octadecanoid pathway (Figure 4).
Tomato *Sys*	*Sys* encodes prosystemin, which is a precursor protein that is proteolytically processed to its bioactive peptide systemin (Figure 3). Systemin is only found in Solanaceous plants. Systemin is a potent activator of the octadecanoid pathway (Figure 4).
Tomato *Wfi1*	Wfi1 is the large subunit of the multi-subunit plasma-membrane complex called NADPH oxidase. NADPH oxidase generates the reactive oxygen species superoxide anion. Only the gp91-*phox* subunit has been cloned in plants (Figure 1).
Arabidopsis ACO	*ACO* encodes an acyl CoA oxidase-like protein. ACO is involved in β-oxidation of fatty acids. Its substrate and function in the wound response are not known (Figure 5B).
Arabidopsis AOS	AOS (allene oxide synthase) converts linolenic acid (18:3) to a 13-hydroperoxide form. This enzyme commits lipids to the octadecanoid pathway and appears to have an important regulatory in regulation of the wound responses in *Arabidopsis* (Figure 5B).
Arabidopsis CHS	*CHS* encodes chalcone synthase important for the synthesis of lignin to strengthen the cell wall and phenylpropanoid compounds that can have antimicrobial activity (Figure 5B).
Arabidopsis CK	*CK* encodes a choline kinase–like protein. It function in the wound-response is not known (Figure 5B).
Arabidopsis CPR1	CPR1 (constitutive expresser of PR genes) function is not yet known. It is a regulator of multiple defense signaling pathways. *cpr1* mutants express *PR* proteins and SAR constitutively (Figure 5B).
Arabidopsis DFR	*DFR* encodes dihydroflavanol reductase. DFR is important for the synthesis of anthocyanin pigments (Figure 5B).
Arabidopsis GST	*GST* encodes glutathione S-transferase. GST is important in detoxifying many compounds and ROS (Figure 5B).
Arabidopsis GPX	*GPX* encodes glutathione peroxidase. GPX is important in scavenging of ROS (Figure 5B).
Arabidopsis JR1	*JR1* (*JA-regulated 1*) encodes a protein of unknown function (Figure 5B).
Arabidopsis JR3	*JR3* (*JA-regulated 3*) encodes a protein similar to the *ILR1* amidohydrolase. ILR1 releases auxin from conjugated forms. The role of JR3 in wounding is not known (Figure 5B).

Table 1. Continued

Genes	Function in Defense or Wounding
Arabidopsis NPR1	*NPR1* (nonexpresser of *PR* genes) encodes protein similar to the transcription factor inhibitor I_KB. This is a critical regulator of both SA-induced responses and responses leading to ISR. *npr1* (also known as *nim1*) mutants cannot induce *PR* gene expression or SAR (Figure 1).
Arabidopsis PDF1.2	*PDF1.2* encodes the small antimicrobial protein defensin. *PDF1.2* is regulated by JA but not by wounding.
Arabidopsis SSI1	The function of SSI1 (suppressor of SA-insensitivity) is not known. The *ssi1* mutant suppresses the *npr1* phenotype. SSI1 is a key regulator of multiple defense signaling pathways.
Arabidopsis TAT	*TAT* encodes a tyrosine aminotransferase that is important for the synthesis of tyrosine. Tyrosine is used for the synthesis of cell wall phenolics, lignins, and flavanoids (Figure 5B).
Arabidopsis Thi2.1	*Thi2.1* encodes thionin, which is a small polypeptide with anti-fungal activity (Figure 5B)
Arabidopsis VSP	*VSP* (vegetative storage protein) encodes a protein that accumulates to high levels in leaves. *VSP* is regulated by JA, but its role in defense is not known (Figure 5B).
Arabidopsis WR3	*WR3 (wound-response 3)* encodes an RNA that accumulates in response to wounding but its function is not known (Figure 5B).
Squash *SLW1*	*SLW1 (silverleaf whitefly-induced 1)* encodes a M20b peptidase. Its function unknown. *SLW1* is preferentially induced by silverleaf whiteflies and is regulated by JA and ethylene (Figure 1).
Squash *SLW3*	*SLW3 (silverleaf whitefly-induced 3)* encodes a β-glucosidase-like protein. Its function unknown. *SLW3* is preferentially expressed in response to silverleaf whiteflies and is regulated by a novel signaling pathway (Figure 1).

[a] *Defense- and wound-response genes mentioned within the review are tabulated. Species are indicated because similar genes in other plants are given different names, although gene products are thought to have similar functions. A comprehensive listing of wound- and defense-response proteins can be found in several recent reviews (Kombrink and Somssich 1997; Reymond and Farmer 1998; Reymond and others 2000).*

(Table 1) (Penninckx and others 1998). In addition, JA and ethylene act sequentially to induce a systemic tolerance to a broad range of pathogens, which is distinct from SAR, and called induced systemic resistance (ISR) (Pieterse and others 1998). Development of ISR is not correlated with the SA- or JA/ethylene-induced genes studied to date (Schweizer and others 1998; van Wees and others 1999). Several other uncharacterized signaling pathways may be active after pathogen attack (Chappell and others 1997; Clarke and others 1998; Thomma and others 1999).

The SA- and JA/ethylene-dependent signaling pathways cross talk. Rises in SA are correlated with down-regulation of the JA/ethylene-regulated defense-response genes, as well as JA-regulated wound responses (Figures 1, 2) (Doares and others 1995; Doherty and others 1988; Peña-Cortés and others 1993; Pieterse and van Loon 1999; Preston and others 1999; van Wees and others 1999). Coordination of these defense pathways with each other and with wound-response pathways is complex and not understood at this time (Figures 1, 2). These signaling mechanisms appear to converge at several regulatory junctions involving wound-induced (WIPK) and SA-activated (SIPK) MAP kinases (Kumar and Klessig 2000; Romeis and others 1999; Sano and others 1994; Seo and others 1995). In addition, some gene products that regulate *PR* gene expression, NPR1 (non-expresser of *PR* genes), SSI1 (suppressor of SA insensitivity), and CPR6 (constitutive expresser of *PR* genes), influence multiple signal transduction pathways (Clarke and others 1998; Shah and others 1999). Convergence of these signaling networks allows plants to prioritize cues and activate the mechanisms that most effectively combat the current invading pathogen or pest.

Activation of Defense-signaling Pathways by Herbivores

Several nonchewing arthropods increase levels of *PR* proteins and activities (Tables 1 and 2). Increases in chitinase or β-1,3-glucanase activities after whitefly, aphid, or mite infestations have been observed (Broderick and others 1997; Bronner and others 1991; Mayer and others 1996; van der Westhuizen and others 1998a; b). Increases in chitinase and β-1,3-glucanase activities are correlated with increases in these proteins (Mayer and others 1996). Furthermore, increases in P2, P4, and PR-10-like proteins are detected (Broderick and others 1997; Mayer and others 1996). As in incompatible plant-pathogen interactions, *PR* proteins and activities increase more

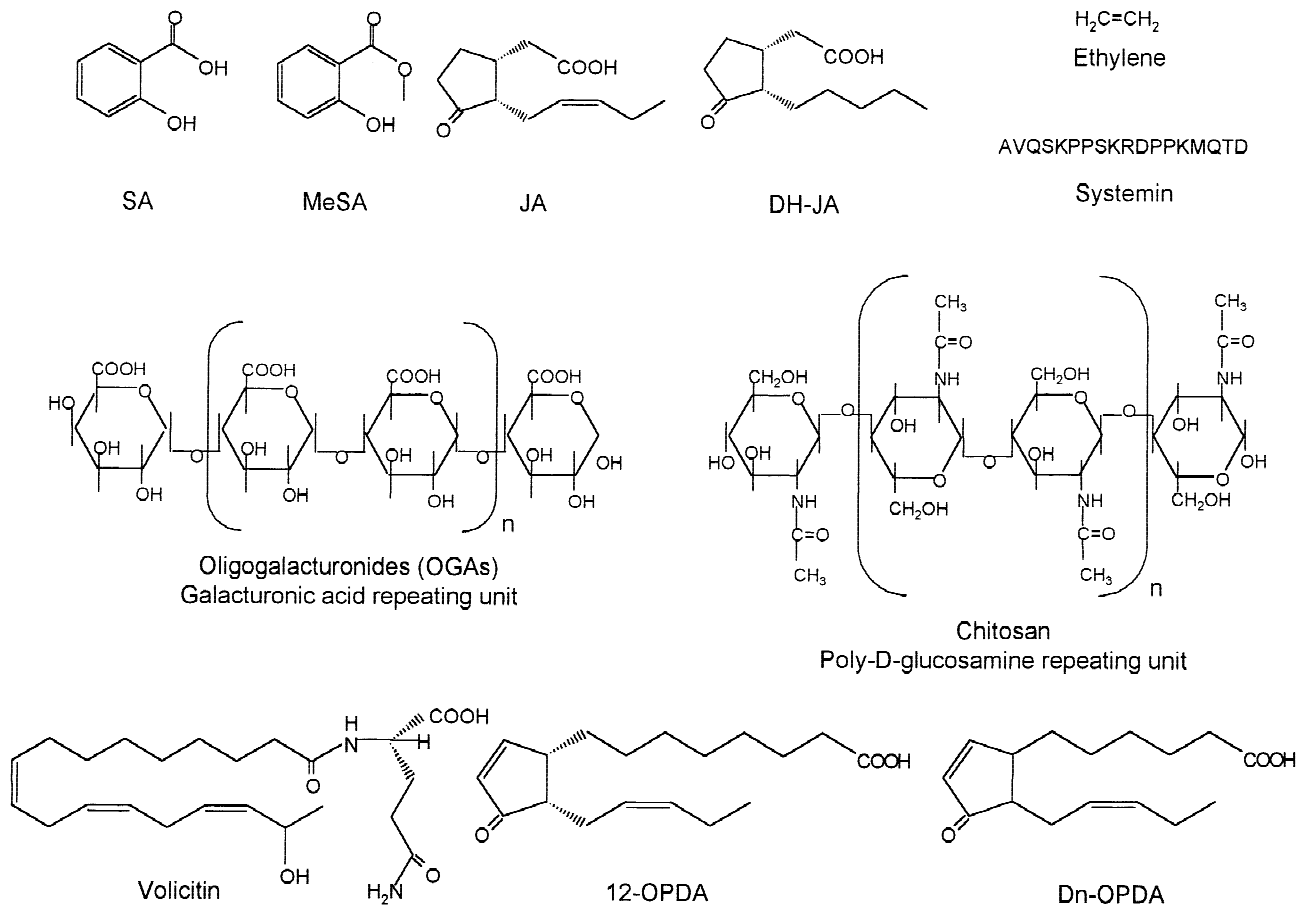

Figure 3. Structures of signaling compounds in herbivore-plant interactions. The structures of salicylic acid (SA), methyl salicylate (MeSA), ethylene, jasmonic acid (7-iso-JA; JA), dihydro-JA (DH-JA); 12-oxo-phytodienoic acid (12-OPDA), dinor-oxo-phytodienoic acid (dn-OPDA), and *N*-(17-hydroxylinolenyl)-L-glutamine (volicitin) are shown. The bioactive peptide systemin is processed from the prosystemin polypeptide and its peptide sequence is indicated in the single letter code. Oligogalacturonides (OGAs) are polymers of galacturonic acid with an α 1→4 linkage. The galacuturonic acid repeating unit is shown. Chitosan is a polymer of D-glucosamine with a β 1→4 linkage. The D-glucosamine repeating unit is shown.

rapidly when herbivores attack resistant plant genotypes (Bronner and others 1991; van der Westhuizen and others 1998a; b).

Feeding by greenhouse whitefly (*Trialeurodes vaporariorum*) and silverleaf whitefly (*Bemisia argentifolii*) nymphs activates the SA- and JA/ethylene-dependent pathways of tomato (Figure 1; Table 1) (Puthoff and others unpublished). However, *PR* gene RNAs do not increase locally or systemically in response to adult whiteflies. Transcripts for *PR* genes regulated by JA and/or ethylene (basic β-1,3-glucanase, basic chitinase, and *PR-1*) accumulate to higher levels than SA-regulated gene RNAs (acidic β-1,3-glucanase and acidic chitinase) (Chao and others 1999; Puthoff and others unpublished; van Kan and others 1995). Similar to whiteflies, the potato aphid (*Macrosiphum euphorbiae*) and green peach aphid (*Myzus persicae*) cause increases in lipoxygenase (*LOX*) and *PR-1* RNAs in infested tomato leaves (Fidantsef and others 1999). In *Arabidopsis,* the cabbage aphid (*Brevicoryne brassicae*) and the cotton aphid (*Aphis gossypii*) induce both the SA-dependent (*PR-1* and acidic β-1,3-glucanase) and JA/ethylene-dependent (defensin and *LOX*) signaling pathways (P. Moran and G.A. Thompson, personal communication).

Consistent with limited tissue damage during whitefly feeding (Walker and Perring 1994), wound-response gene RNAs (leucine aminopeptidase [*LapA*] and proteinase inhibitor [*pin2*]) (Table 1) do not accumulate in response to adult or immature whitefly feeding (Puthoff and others unpublished). Furthermore, analyses of *LapA:GUS* transgenic tomato plants show that the *LapA* promoter is

Table 2. Changes in Defense-Response Protein and Activity Levels in Response to Nonchewing Insects

Insect	Plant	Protein Accumulation[a,b]	Enzymatic Activities[c]	Reference
Silverleaf whitefly *Bemisia argentifolii* (Bellows and Perring)	Pumpkin *Cucurbita pepo* L.	Changes in IF proteins	Reduced apoplastic chitinase[d] Reduced apoplastic β-1,3-glucanase	Jiménez and others 1995
Silverleaf whitefly *Bemisia argentifolii* (Bellows and Perring)	Tomato *Lycopersicon esculentum* L.	Chitinase β-1,3-glucanase P2 P4 (PR-1)	Peroxidase Chitinase β-1, 3-glucanase	Mayer and others 1996
Russian wheat aphid *Diuraphis noxia* (Mordvilko)	Wheat *Triticum aestivum* L.	Changes in IF proteins Apoplastic peroxidase Apoplastic chitinase Apolastic β-1,3-glucanase	Apoplastic peroxidase Apoplastic chitinase Apoplastic β-1,3-glucanase Intracellular β-1,3-glucanase	Van der Westhuizen and others 1998a, b
Greenbugs *Schizaphis graminum* (Rondani)	Sorghum *Sorghum bicolor*	ND	Apoplastic peroxidase Apoplastic chitinase Apoplastic β-1,3-glucanase	Krishnaveni and others 1999
Redlegged earth mite *Halotydeus destructor* (Tucker)	Subterranean clover *Trifolium subterraneum*	PR-10	Apoplastic peroxidase Apoplastic chitinase	Broderick and others 1997
Gall mite *Aceria caldophthirus* (Nalepa)	Bittersweet nightshade *Solanum dulcamara* L.	Changes in IF proteins	β-1, 3-glucanase chitinase	Bronner and others 1991

[a]*Changes in the protein composition of intercellular fluids (IF) were assessed after SDS-polyacrylamide gel electrophoresis and Coomassie blue staining. Immunoblot analyses detected changes in specific PR* proteins (chitinases, β-1,3-glucanases, P2 and P4. PR-10 was identified as an induced protein in a stained gel and its peptide sequence was determined. Changes in protein levels were not determined (ND) in some studies.
[b]*Functions of PR* proteins are listed in Table 1.
[c]*The intracellular location of enzymatic activities were determined by assessing activities in IF versus total extracts. In these cases, the implied subcellular localization of activities is indicated.*
[d]*In the silverleaf whitefly-pumpkin interaction, reduced levels of apoplastic enzyme activities were noted. In all other interactions, herbivore infestation resulted in increased levels of enzymatic activities.*

not activated in cells along the stylet path (Puthoff and others unpublished). Likewise, phloem-feeding aphids do not increase *pin2* RNA levels (Fidantsef and others 1999). This contrasts to the tomato response to the two-spotted spider mite (*Tetranychus urticae*) and western flower thrip (*Frankliniella occidentalis*). Although these arthropods use stylets to feed, they cause substantial cellular damage and induce JA-dependent wound-response genes of tomato (G. Howe, personal communication).

Plant Responses to Whiteflies: Specific Responses to Whitefly Species and Developmental Stages

Studies on the *whitefly-induced 1* (*Wfi1*) gene of tomato and *silverleaf whitefly-induced 1* (*SLW1*) and *SLW3* genes from squash indicate that some plant genes respond to signals generated by specific insect species and specific stages in insect development (Figure 1). *Wfi1* RNAs accumulate locally and systemically in tomato leaves after feeding by whitefly nymphs but not in response to pink potato aphids or whitefly adults (Puthoff and Walling unpublished). These data suggest that some of the signals generated by whitefly and aphid feeding, as well as adult and immature insects, are distinct. *Wfi1* encodes a membrane-bound subunit of NADPH oxidase (a gp91-*phox* homologue) (Table 1) (Puthoff and Walling unpublished). Increases in NADPH oxidase activity are correlated with the ROS burst that accompanies pathogen infection and wounding (Bolwell and Wojtaszek 1997; Ryan 2000). Like basic *PR* genes, *Wfi1* RNAs accumulate after JA and ethylene treatments but not after systemin treatments (a potent inducer of wound-response genes) (Figures 1,

2) (Puthoff and others unpublished; Chao and others 1999; Ryan, 2000). Surprisingly, wounding transiently increases levels of *Wfi1* RNAs. These data indicate that *Wfi1* is primarily regulated by the JA/ethylene defense-signal transduction pathway after whitefly infestation (Figure 1) and may be activated by a systemin-independent pathway after wounding (Figure 2) (Puthoff and others unpublished).

Even closely related insect species, such as the silverleaf whitefly and the sweetpotato whitefly (*B. tabaci* Type A), produce elicitors that differentially regulate plant gene expression (van de Ven and others 2000). Similar to tomato *PR* genes and *Wfi1, SLW1,* and *SLW3* transcripts increase in response to silverleaf whitefly nymph feeding but not to adult feeding (Table 1) (van de Ven and others 2000). More notably, *SLW1* and *SLW3* RNAs accumulate systemically after feeding by the silverleaf whitefly nymphs but not after feeding by sweetpotato whitefly nymphs. The preferential expression of *SLW1* and *SLW3* by the silverleaf whitefly suggests that this insect generates (1) a novel signal, (2) larger amounts of a systemic elicitor, or (3) a more potent signal than is produced by the sweetpotato whitefly. Treatments with defense signals show *SLW1* is regulated by both JA and ethylene (Figure 1). In contrast, *SLW3* RNAs do not accumulate in response to any known defense/wound signals (NO, H_2O_2, NO plus H_2O_2, JA, ethylene, SA, abscisic acid, JA plus ethylene, JA plus SA, or wounding) (van de Ven and others 2000). These data suggest that the silverleaf whitefly activates a novel signaling pathway to induce *SLW3* expression (Figure 1). Interestingly, both *SLW1* and *SLW3* transcripts accumulate after water-deficit stress, suggesting that the signals produced by silverleaf whitefly feeding and water-deficit may share some features (van de Ven and others 2000). This is in contrast to caterpillar feeding in *Arabidopsis,* where genes induced by water-deficit are expressed in response to wounding but not to caterpillar feeding (Reymond and others 2000).

Source of Elicitors in Aphid-, Mite- and Whitefly-plant Interactions

The signals generated by herbivores, which pierce plant cells to remove liquids as a nutrient source, are complex. Some cues, like those that activate *PR* gene expression, are likely to be shared by many herbivores (whiteflies, aphids, and mites). In contrast, some elicitors appear to regulate species-specific responses such as the changes in levels of *Wfi1* and *SLW* RNAs (Puthoff and Walling unpublished; van de Ven and others 2000) and the complex volatile blends that regulate plant-herbivore-herbivore enemy interactions (De Moraes and others 1998). These general or specific signals may derive from physical damage and mechanical stress. During stylet probing for a feeding site, herbivores may inadvertently damage cells along the stylet path and release stored plant signals that stimulate expression of *PR, Wfi1,* and *SLW* genes. Alternatively, movement of the stylet between cells, which disrupts essential cell-to-cell contacts, or puncture of the feeding-site cells and consumption of liquids may be perceived as a physical stress generating signals to activate gene expression. Both hydraulic and electrical signals are implicated in the induction of wound-response genes (Rhodes and others 1999 and references within). However, it is unlikely that these mechanical signaling mechanisms can account for the temporal and spatial differences in *SLW* gene expression induced by the silverleaf and sweetpotato whiteflies (van de Ven and others 2000).

A component of a herbivore's saliva is likely to provide the general and specific elicitors for *PR, Wfi1,* and *SLW* gene expression, respectively. Liquid-imbibing insects, like aphids and whiteflies, secrete two types of saliva along the stylet path and at the feeding site: a rapidly gelling, sheath saliva and a watery, digestive saliva (Miles 1999). Salivas have been characterized in a small number of homopteran and hemipteran insects (Miles 1999). Sheath salivas are composed primarily of protein, phospholipids, and conjugated carbohydrates (Miles 1999). When egested, sheath salivas polymerize around the flexible stylet to form a protective shield, thereby limiting direct contact of the stylet with the plant apoplast. It is not clear whether unpolymerized sheath materials are elicitors in these plant-insect interactions.

The composition of the watery, digestive saliva is more complex and variable containing a wide array of enzymes including pectinases, cellulases, amylases, proteases, lipases, alkaline and acidic phosphatases, and peroxidases (Miles 1999). In addition, chitosan is secreted by gall mites at the feeding site (Bronner and others 1989). The general and species-specific elicitors may correspond to one of the known salivary constituents or may be an uncharacterized component of the saliva. The chitosan (Figure 3), oligogalacturonides created by pectinases (Figure 3), and ROS from peroxidases are known elicitors of wound- and/or defense-signaling pathways (Figures 1, 2).

The general and species-specific elicitors may be directly synthesized by the insect or may be a product of endosymbiotic bacteria (Costa and others 1995; Douglas 1998). An elicitor could also be generated by concerted biochemical activities of the in-

sect and the plant, similar to the synthesis of volicitin (Figure 3), an inducer of terpenoid volatile production in lepidopteran caterpillars (Alborn and others 1997; Páre and others 1998).

Resistance to Piercing/Sucking Herbivores: Role of PR Proteins, Secondary Metabolites, and Resistance Genes

The role of *PR* proteins and other herbivore-induced gene products on plant resistance to herbivores that use a piercing/sucking mode of feeding is unknown (Tables 1 and 2). The accelerated expression of *PR* genes in resistant plant-herbivore interactions (Bronner and others 1991; van der Westhuizen and others 1998b) suggests that one or more components induced by herbivore feeding may function in antixenosis or antibiosis. However, because many *PR* proteins induced after pathogen infection are active against a subset of pathogens, it is unclear whether any *PR* proteins influence resistance to piercing/sucking herbivores. The use of plant mutants that constitutively activate or suppress the JA/ethylene- and SA-dependent defense pathways will be useful for dissecting the impact of these pathways on resistance to phloem-feeding insects (Dempsey and others 1999; Glazebrook 1999; McDowell and Dangl 2000; Penninckx and others 1998). Examination of transgenic plants that up- or down-regulate specific *PR* genes may also provide insight into the roles of individual *PR* proteins in herbivore resistance. Analysis of transgenic plants overexpressing insect and plant chitinases suggests that chitinases have a more limited impact on herbivore interactions (Kramer and Muthukrishnan 1997).

Nonproteinaceous, secondary metabolites appear to influence phloem-feeding insects more profoundly. For example, volatiles derived from SA (MeSA) and lipids (C_6 volatiles) accumulate in response to aphid feeding and actively deter aphid settling and fecundity, respectively (Hardie and others 1994; Hildebrand and others 1993; Shulaev and others 1997). In addition, several secondary metabolites (that is, acyl-sugars, glucosinolates, and hydroxamic acids) have established roles in resistance to phloem-feeding insects. Genes that control the production of these compounds are being used in breeding programs to enhance insect resistance (Blauth and others 1998; Giamoustaris and Mithen 1995; Gianoli and Niemeyer 1998).

In addition to these quantitative traits, single genes that confer resistance to nonchewing insects have been identified (Ponda and Khush 1995). Some *R* genes provide a phloem-mediated resistance that deters aphid feeding (Kaloshian and others 1997; Klingler and others 1998). Several *R* genes confer resistance to a single or a small number of aphid biotypes suggesting that "gene-for-gene"-like mechanisms of resistance are also active against phloem-feeding insects (Glazebrook 1999). Examples include *Nr* in lettuce that confers resistance to a single aphid species, *Nasonovia ribisnigri* (van Helden and others 1993), *Sd1* of apple that mediates resistance to two biotypes of the aphid *Dysaphis devecta,* but not a third biotype (Roche and others 1997), and *Mi1.2* of tomato that confers resistance to the potato aphid (*Macrosiphum euphorbiae*) (Rossi and others 1998).

Mi1.2 was the first insect resistance gene cloned and is a member of the leucine zipper, nucleotide-binding, leucine-rich repeat family of *R* genes that confers resistance to pathogens (Milligan and others 1998; Vos and others 1998). The ability of *Mi1.2* to mediate resistance to the potato aphid and the root-knot nematode (*Meloidigyne incognita*) is intriguing (Rossi and others 1998, see review by Bird and Koltai, this volume). The facts that resistance to aphids and nematodes develops in seedlings of different ages (I. Kaloshian, personal communication) and that *Mi1.2* causes an HR in response to *M. incognita,* but not to *M. euphorbiae,* suggest that *Mi1.2* dual specificity may be complexly regulated.

The parallels to pathogen compatible and incompatible interactions are most compelling in the interactions of the Hessian fly (*Mayetiola destructor*) and cereals. The Hessian fly does not extensively damage leaves (Grover 1995). These larvae appear to secrete substances that stimulate the release of nutrients from cells (Refai and others 1956). Wheat resistance to the Hessian fly is controlled by more than 26 resistance genes, providing resistance to 13 Hessian fly races or biotypes (Dweikat and others 1997). Similar to pathogen/plant incompatible interactions, Hessian fly resistance is accompanied by an HR surrounding the larvae (Grover 1995). Although the temporal and spatial expression of defense-response genes in these interactions are not yet described, there are intensive mapping initiatives to identify and characterize the wheat resistance genes and their cognate avirulence genes from the Hessian fly (Dweikat and others 1997; Schulte and others 1999).

Chewing Insects and Wound Responses

Phytophagous arthropods that cause extensive tissue damage induce changes in plant gene expression and accumulation of secondary metabolites similar

to mechanical wounding (for reviews, see Baldwin and Preston 1999; Karban and Baldwin 1997; Reymond and others 2000; Ryan 2000). Multiple wound signaling pathways are active in plants (Figure 2) (Reymond and others 2000; Rojo and others 2000; Ryan 2000). Wound-signaling pathways control the profound changes in plant cell biochemistry that facilitate recovery and healing at the site of injury (Figure 4) (for reviews; see Bostock and Stermer 1989; Kahl 1982). In addition, wound-response proteins may have a direct or indirect role in (1) limiting damage induced by attacking insects, (2) killing opportunistic pathogens that invade wound sites, and (3) developing an induced resistance (IR) that protects plants from subsequent challenges from pests and pathogens (Bostock and Stermer 1989; Bostock 1999; Felton and others 1999; Karban and Baldwin 1997). IR is distinct from the pathogen-induced SAR, but its relationship to the microbe-induced ISR is not understood (Figures 1, 2).

Wound-response genes may be expressed locally or systemically, and these proteins have diverse roles as outlined in Figure 4 (Duffey and Stout 1996; Karban and Baldwin 1997; Ryan 2000; Wasternack and others 1998a). Many proteins and secondary metabolites that accumulate after wounding and JA-treatments interfere with insect feeding, oviposition, growth and development, and fecundity (Duffey and Stout 1996; Pechan and others 2000) or attract herbivore predators (Dicke 1999; Páre and Tumlinson 1999). These compounds limit plant injury or restrain insect population expansion (Figure 4). Other wound-response proteins have unidentified roles in the defense and/or tissue recovery.

Activation of the Octadecanoid Pathway and JA-mediated Wound Responses

Comprehensive reviews detailing the changes in lipid metabolism and mechanisms used to activate JA-mediated wound responses have been published recently (Blée 1998; León and Sánchez-Serrano 1999; Ryan 2000; Schaller 1999; Wasternack and others 1998a). The octadecanoid pathway is induced by herbivores, such as caterpillars and beetles, that chew and tear tissues (Figure 2) (Ryan 2000). Thrips and spider mites, which lacerate cells and imbibe cellular fluids through stylets, also induce JA-mediated wound responses (G. Howe, personal communication). Herbivores not only damage tissues, but their salivary secretions may directly introduce chitosan (Figure 3) and/or polygalacturonase (PG) into the wound site (Bronner and others 1989; Miles 1999). PG generates oligogalacturonides (OGAs; Figure 3) from the pectin in the plant cell wall. Both chitosan and OGAs are potent inducers of the Solanaceous octadecanoid pathway (Figure 4). OGAs and chitosan act at the site of release or introduction, respectively; these oligosaccharides are not transported throughout the plant (Baydoun and Fry 1988).

Mechanical wounding generates electrical or hydraulic signals that are rapidly propagated from the site of damage (Rhoades and others 1999 and references within). These signals are thought to stimulate the local and systemic release of compounds (OGAs and systemin) that further amplify this signaling cascade throughout the plant (Figures 4, 5A). The increases in plant wound-induced PGs liberate OGAs from pectin (Bergey and others 1999). In addition, the bioactive peptide systemin (Figure 3) is produced and transported through the phloem to mediate both local and systemic activation of the octadecanoid pathway (for details, see Ryan 2000). OGA, chitosan, and systemin treatments cause increases in cytosolic calcium, inactivation of H^+-ATPase, membrane depolarization, K^+ and H^+ fluxes, MAP kinase activity, generation of ROS, and phospholipase A_2 and D activation (for reviews; see Ryan 2000; Schaller 1999).

Phospholipases release linolenic acid (18:3) from membranes (Narvaez-Vasquez and others 1999; Ryu and Wang 1998). LOX converts linolenic acid to a 13-hydroperoxide, which has two possible fates (Figure 4). It is hydrolyzed by hydroperoxide lyase (HPL) to generate C_6 volatiles (See *C_6 Volatiles Herbivore Interactions*) and traumatin, a lipid that stimulates wound healing (Zimmerman and Coudron 1979). Alternatively, the 13-hydroperoxide is committed to the octadecanoid pathway by allene oxide synthase (AOS). After six sequential reactions, the bioactive JA is produced (Figure 4). JA and its methyl ester, amino acid, and glucose conjugates are potent signaling molecules (Kramell and others 1997). These jasmonates activate wound-response genes by a yet undefined mechanism.

ABA, ethylene, auxin, and SA are additional regulators of the octadecanoid pathway (Figure 3). ABA has an early role in activation of the octadecanoid pathway (Carrera and Prat 1998; Chao and others 1999; Peña-Cortés and others 1996). The role of ethylene is more complex. Ethylene is essential for JA-mediated wound-response gene expression (O'Donnell and others 1996) but antagonizes JA-induced nicotine production (Kahl and others 2000). Auxin is a negative regulator of wound responses, and the mechanism of auxin action is not understood (Kernan and Thornberg 1989). SA inhibits both JA synthesis and action (Figure 4)

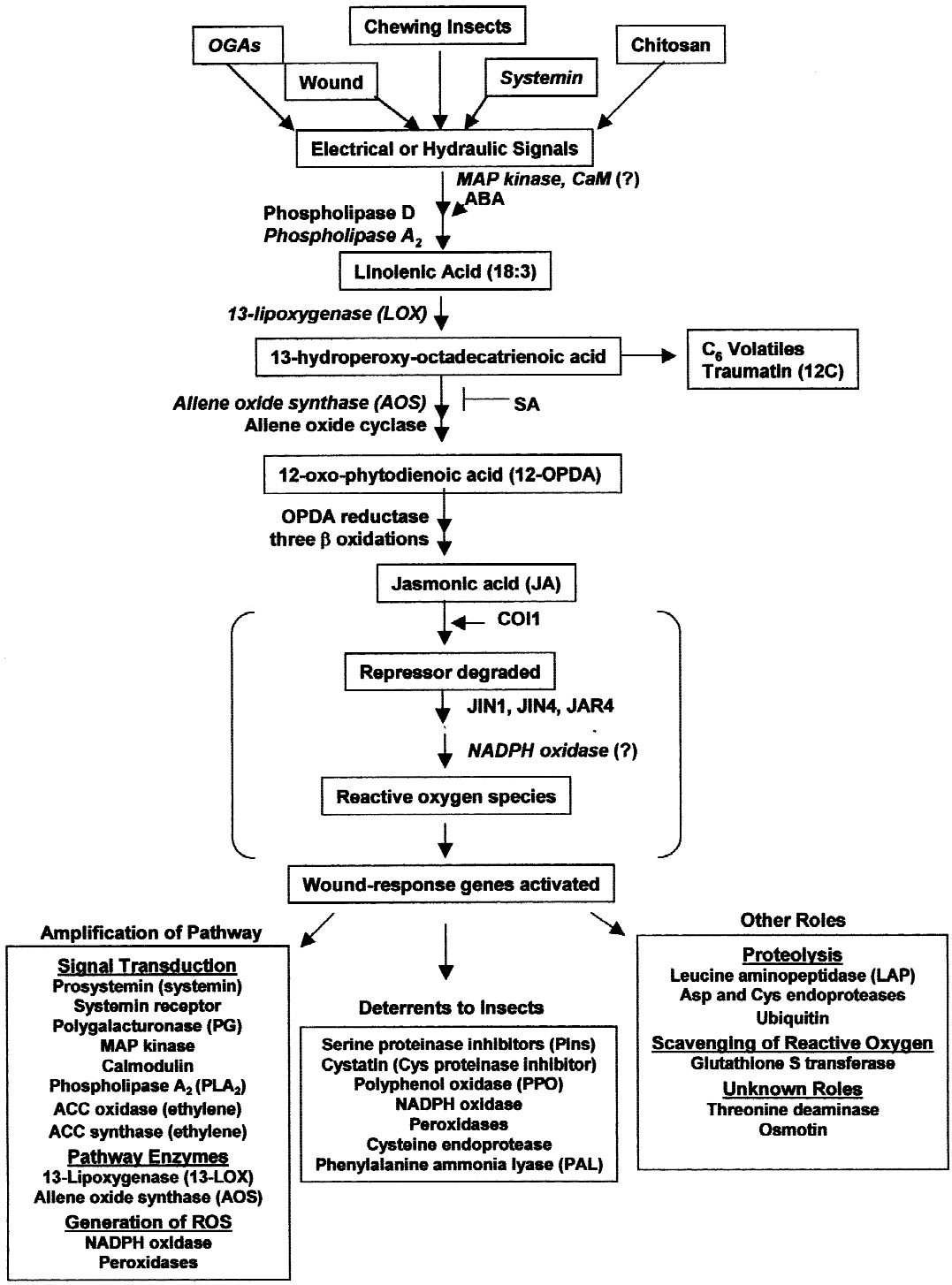

Figure 4. JA-dependent wound-signal transduction pathway of the tomato. This model is based on the octadecanoid signaling pathway of tomato (see text for details). The tomato JA-dependent pathway integrates numerous signals including oligogalacturonides (OGAs), chitosan, systemin, and signals generated by tissue-damaging insects. Linolenic acid is used to synthesize JA or C_6 volatiles and traumatin. Several elicitors, regulatory proteins, and enzymes of the octadecanoid signaling pathway are induced on wounding to amplify the wound-response; these are in red and italicized for emphasis. Several components of this pathway are speculative (area in brackets). The sites of calmodulin [CAM(?)] and NADPH oxidase action are not known. NADPH oxidase appears to act downstream of JA (C.A. Ryan, personal communication). Four *Arabidopsis* genes known to determine JA sensitivity (*COI1, JIN1, JIN4,* and *JAR4*) are included in this scheme, although analogs in tomato have yet to be analyzed. Their order of action is supported by analyses in *Arabidopsis.* The *def1* mutant (not shown) impacts this signaling pathway at an unknown location (G. Howe, personal communication). ABA, ethylene, SA and auxin influence the JA-dependent wound signaling. The sites of action of ethylene, auxin, and the downstream SA are not known (see text for details).

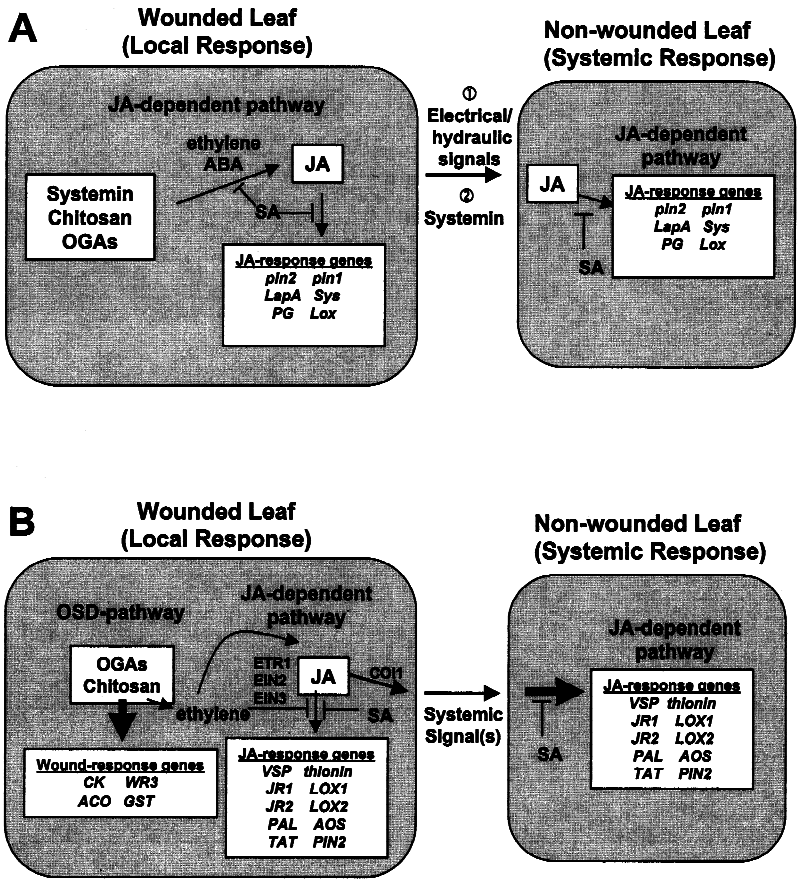

Figure 5. The wound-signaling pathways used in tomato (*panel A*) and *Arabidopsis* (*panel B*) are contrasted and are described in detail in the text. *Panel A,* A single signaling pathway perceives systemin, chitosan, and oligogalacturonides (OGAs) in tomato to increase JA and activate wound-response genes. The initial systemic signal may be electrical or hydraulic (1) and is followed by the phloem-transported peptide systemin (2) (see text for details). JA-independent pathways have not been elucidated in tomato. *Panel B,* OGAs and chitosan stimulate the OSD (oligosaccharide-dependent) signaling pathway in injured arabidopsis cells resulting in expression of wound-response (*WR*) genes, which are described in Table 1. Wounding induces ethylene that blocks expression of the JA-regulated wound signaling pathway but induces AOS (allene oxide synthase), a key enzyme in JA biosynthesis. This provides an important control point for the balancing of the two signaling cascades. Although the nature of the systemic signal(s) is not known in *Arabidopsis, COI1* appears to be a regulator of this process. Wound-response genes that are JA-regulated are presented in Table 1. Only *VSP, JR1,* and *JR2* were examined in the Rojo and others (2000) studies; the regulatory programs for other JA-response genes are made by analogy. The importance of SA or ABA in regulating the OSD- and JA-dependent wound response pathways has not been determined. A systemin analog has not been detected in non-Solanaceous plants to date (C.A. Ryan, personal communication).

(Doares and others 1995; Doherty and others 1988; Peña-Cortés and others 1993).

Lipases also release other fatty acids that are used for the synthesis of additional wound signals: dinor-oxo-phytodienoic acid (dn-ODPA) (Figure 3) and dihydroxy(DH)-JA (Figure 3). The octadecanoid pathway enzymes hydrolyze 16:3 lipids to generate dn-ODPA (Weber and others 1997). The production of DH-JA from linoleic acid (18:2) is not completely understood and may vary in different plants (Gundlach and Zenk 1998). Plants accumulate different levels of JA and its conjugated forms, DH-JA, dn-OPDA, and 12-OPDA (Figure 3). These oxylipins are activators of phytoalexin biosynthesis, alkaloid biosynthesis, or wound-response gene expression in a variety of plants (Gundlach and Zenk 1998; Wasternack and others 1998b; Weber and others 1997).

At present, it is not known whether the balance of these oxylipin-pathway intermediates and products are different after wounding and insect feeding. Furthermore, it is not known whether insect feeding induces novel fatty acid-derived signals, not present in wounded plants. The recent identification of two, previously undescribed 18-carbon divinyl ether fatty acids, colneleic acid and colnelenic acid, which accumulate in response to *Phytophthora infestans* infection (Weber and others 1999), indicates that our understanding of the nature of the lipids that accumulate and their roles in defense are still developing (Blée 1998).

JA-independent Wound-response Pathways

Although JA-mediated wound-responses are best characterized, JA-independent wound responses have been implicated in several studies in tomato (Figure 2) (O'Donnell and others 1998; Pearce and others 1998; Puthoff and Walling unpublished). This contrasts to *Arabidopsis,* where the coordination of the JA-dependent and JA-independent signaling pathways is understood (Figure 5B). At present, wound signaling in *Arabidopsis* appears to be distinct from tomato.

In tomato, a single pathway responds to OGAs,

chitosan, and JA (Figures 4, 5A). In *Arabidopsis* two pathways mediate the spatial and temporal programs of wound-induced gene expression (Figure 5B). A JA-independent signaling pathway is activated by OGAs and chitosan (Rojo and others 2000). This oligosaccharide-dependent (OSD) pathway activates wound-response (*WR*) genes in damaged leaves, consistent with the fact that OGAs are not transported from the site of injury (Baydoun and Fry 1988). The OSD pathway is not influenced by JA or ethylene (Rojo and others 2000).

The expression of JA-regulated (*JR*) wound-response genes is antagonized by OGAs or chitosan in *Arabidopsis* (Figure 5B) (Rojo and others 2000). *JR* RNAs accumulate in apical nonwounded leaves, and at lower levels, in injured leaves. Analysis of mutants that influence ethylene perception or action (*etr1, ein2,* and *ein3*) show that ethylene antagonizes the local expression of *JR* genes (Rojo and others 2000). This is balanced by ethylene increasing levels of allene oxide synthase (AOS), which stimulates the synthesis of JA (Figure 5B) (Laudert and Weiler 1998).

The OSD- and JA-pathways appear to be reciprocally regulated, because they have opposite responses to kinase and phosphatase inhibitors and internal [Ca^{2+}] fluxes (León and others 1998; Rojo and others 1998). A comprehensive analysis of genes that respond to wounding and cabbage butterfly larvae (*Pieris rapae*) shows that these caterpillars induce both JA-dependent and -independent wound-response genes in *Arabidopsis* (Reymond and others 2000). In fact, some *Arabidopsis* genes are regulated by both pathways (Nishiuchi and others 1997; Rojo and others 1998). Collectively, data from *Arabidopsis* indicate that an herbivore will encounter different induced defense molecules in damaged and undamaged leaves. It is unclear whether similar complexity exists in other plants, although it is probable.

Importance of the Octadecanoid Pathway in Herbivore Resistance

Whereas the importance of the JA-independent wound-response in herbivore resistance is not known, activation of the octadecanoid pathway is important for resistance to chewing insects. The *Arabidopsis fad3-2 fad7-2 fad8* mutant abolishes JA and octadecanoid intermediate synthesis and is more susceptible to fungal gnat larvae (*Bradysia impatiens*) (McConn and others 1997). *Arabidopsis* mutants that have an impact on JA sensitivity (*jin1, jar1, jar4,* and *coi1*) have been identified and are impaired in their resistance to several fungal pathogens (Staswick and others 1998; Thomma and others 1998; Vijayan and others 1998). The importance of *jin1, jin4, jar1,* and *coi1* in resistance to herbivores has not been formally tested.

The tomato (*def1*) mutant, which does not induce wound-response gene expression, has a compromised resistance to tobacco hornworm larvae (*Manduca sexta*), thrips (*F. occidentalis*), and spider mites (*T. urticae*) (Howe and others 1996; G. Howe personal communication). Transgenic plants that up- or down-regulate several JA-regulated genes (*LOX, prosystemin,* or *pin2*) enhance or impair resistance, respectively, to lepidopteran caterpillars (Royo and others 1999; for additional references see, Ryan 2000).

Studies using exogenous JA and benzothiadiazole (BTH; a SA mimic) treatments also emphasize the importance of JA-regulated events in resistance to herbivores. Both caterpillar feeding and JA increase the levels of polyphenol oxidase, peroxidase, *pin2,* and *LOX* activities, which interfere with gut function or decrease the nutritional value of food (Stout and others 1994; Thaler and others 1996). JA treatments also increase plant tolerance to challenging insects and pathogens and decrease chewing herbivore performance (Thaler and others 1996; Thomma and others 1998). In a reciprocal fashion, BTH suppresses JA-induced responses, increases susceptibility of plants to beet army worm (*Spodoptera exigua*), and enhances resistance to pathogens (Fidantsef and others 1999). Analysis of PAL over- and under-expressing lines that stimulate SA-regulated and JA-regulated pathways, respectively, supply additional support for the importance of JA-regulated responses in resistance to chewing herbivores (Felton and others 1999).

Wounding and Herbivore Feeding are not Equivalent

Although many responses to chewing insects overlap with mechanical wounding, these processes are not equivalent. First, herbivore feeding or herbivore regurgitant (contents of the foregut) often cause larger increases in JA (Baldwin and others 1997) and wound-response gene RNAs (Korth and Dixon 1997a) than wounding alone. These data imply that the herbivore oral secretions contain elicitors that stimulate the octadecanoid pathway; however, the nature of these elicitors and their site of action in the octadecanoid pathway are unknown. Second, the temporal and spatial increases in polyphenol oxidase, peroxidase, *pin,* and *LOX* activities after wounding and feeding by different tissue-damaging herbivores (caterpillars, mites, and leaf miners) are

distinct (Stout and others 1994). Third, novel genes, which are not induced by wounding alone, are induced by herbivory and tobacco hornworm regurgitant, suggesting novel signaling pathways are induced by chewing insects (Figure 1B) (Korth and Dixon 1997b). Fourth, wounding and herbivore feeding provoke the synthesis and release of different sets of volatiles in most plants (see *Volatiles: Direct and Indirect Defenses*). Fifth, soybean's wound-induced resistance to herbivores is enhanced on insect feeding or application of regurgitant to plants (Lin and Kogan 1990). Sixth, insect feeding and regurgitant suppress selected wound-induced responses such as nicotine production (Kahl and others 2000). Finally, although wounding of *Arabidopsis* induces several genes that respond to water-deficit stress, cabbage butterfly larvae do not induce these genes (Reymond and others 2000). These observations suggest that caterpillar feeding interferes selectively with wound induction of these genes. Collectively these data indicate that there are substantial differences in plant responses to herbivores and wounding, the magnitude of these differences are not currently appreciated. It is presumed that components of herbivore oral secretions are the elicitors for these herbivore-specific responses (See: *Elicitors of Volatile Production*).

Volatiles: Direct and Indirect Defenses

In response to mechanical or herbivore injury, plants release a complex blend of volatiles providing valuable cues for herbivores and their natural enemies (for reviews, see Dicke 1999; Páre and Tumlinson 1999). Volatiles emitted by healthy or infested plants are used by herbivores to discriminate between host and nonhost plants and assess the density of feeding insects on a plant (Bolter and others 1997; Quiroz and others 1997). Volatiles also serve as attractants for herbivore predators and parasites. This indirect defense mechanism provides natural herbivore enemies with reliable, easily detected, and accurate cues to identify plants infested with their host insects. The ability of natural enemies to reduce phytophagous arthropod populations is the basis of biologic control strategies in the field.

Healthy plants release volatiles into the atmosphere, but wounding and herbivore feeding change the volatile blend released by the plant. The constituents of volatile blends are influenced by (1) the herbivore species and its developmental stage; (2) plant species, genotype, and age; and (3) environmental stress (for reviews, see Dicke 1999; Paré and Tumlinson 1999). In response to arthropods or mechanical damage, plants release volatiles in two phases (McCall and others 1994). Some volatiles are released immediately (within 1 h) after injury. Labeling studies indicate that other volatiles are newly synthesized (locally and/or systemically) in response to damage and appear 5 to 6 h later (Páre and Tumlinson 1998). Volatiles have diverse structures and arise from the activities of several biochemical pathways (Figures 6, 7). The most commonly released volatiles include C_6 volatiles (lipoxygenase/hydroperoxide lyase-dependent pathways), indole and MeSA (the shikimic acid/tryptophan pathway), cyclic and acyclic terpenoids (isoprenoid pathway), and oximes and nitriles (derived from amino acids) (Dicke 1999; Dicke and others 1999; Páre and Tumlinson 1999). Glucosinolates are partially volatile and are emitted by a limited number of species including the Brassicacae (Halkier and Du 1997).

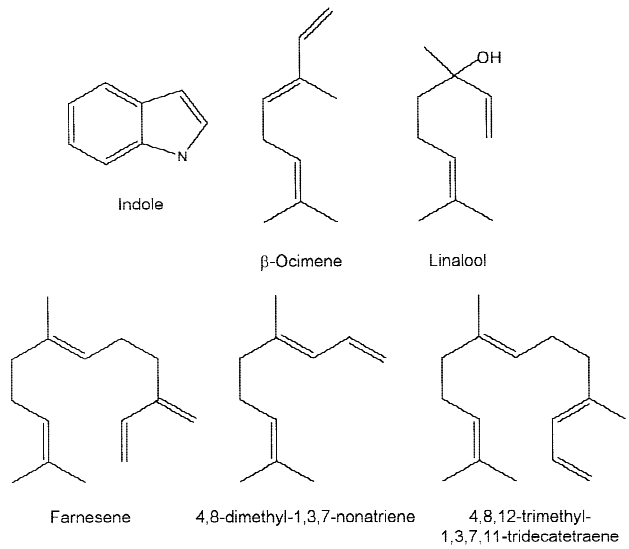

Figure 6. Terpenoid volatiles. The structure for indole and five commonly emitted volatile terpenes are shown.

As with other plant defense responses, wounding and herbivore feeding are not equivalent. In *Brassica* wounding and herbivore feeding release a similar blend of terpenoid volatiles, but increases in C_6 volatiles and indole glucosinolates are enhanced by insect feeding, relative to wounding (Bodnaryk 1994; Mattiacci and others 1994). In contrast, maize, soybean, cotton, lima bean, and cucumber emit distinct arrays of terpenoid volatiles after mechanical wounding and herbivore feeding (Dicke 1999; Páre and Tumlinson 1999), implicating insect-specific elicitors in the volatile production and release (See: *Elicitors of Volatile Production*).

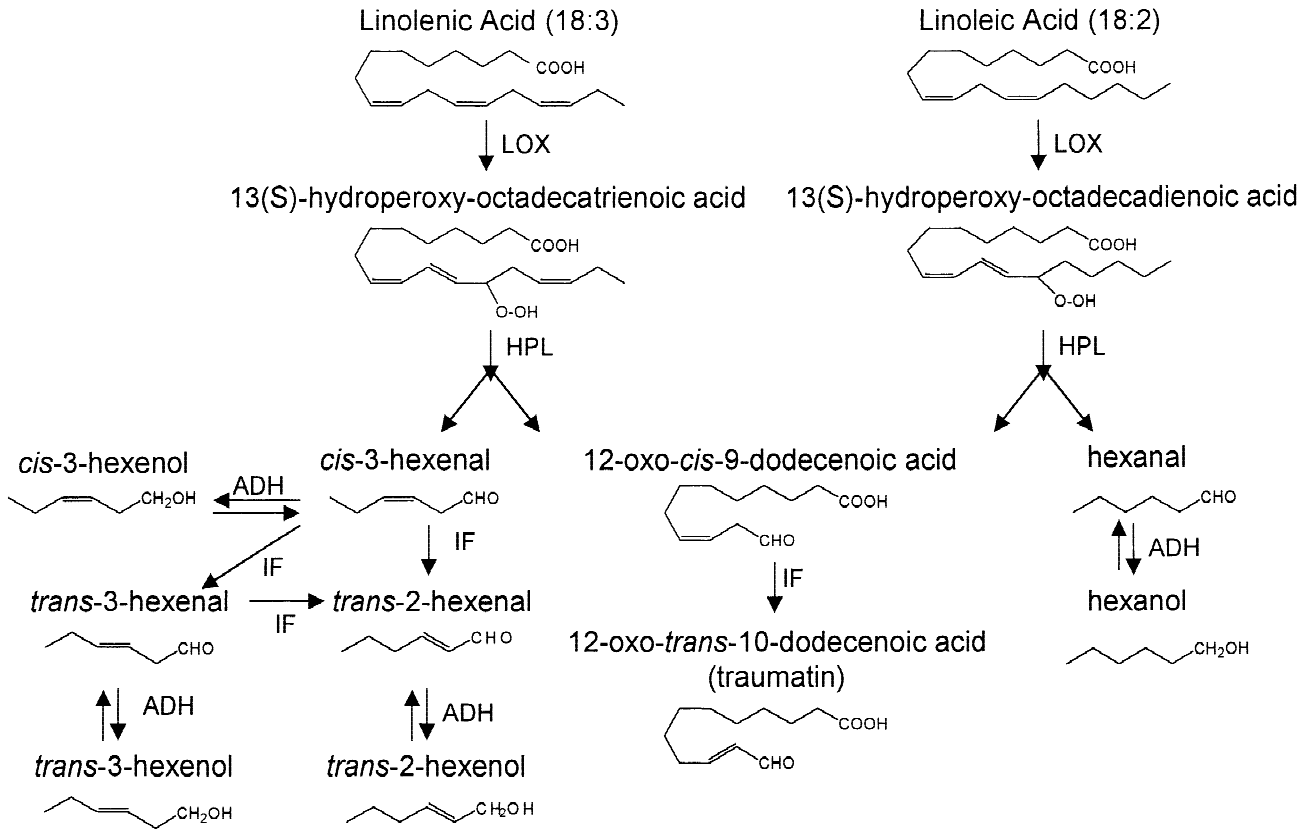

Figure 7. Traumatin and C_6 volatile synthesis. The scheme for C_6 volatile synthesis is presented. Structures for each intermediate, the C_6 volatiles and traumatin are illustrated. LOX (lipoxygenase), HPL (hydroperoxide lyase), ADH (alcohol dehydrogenase), IF (isomerization factors).

The volatile blends released by each herbivore-plant interaction are qualitatively and quantitatively distinct. The most abundant terpenoid volatiles are emitted by many different plant-herbivore interactions (Páre and Tumlinson 1999). It is the molar ratio of the individual volatiles that distinguishes each plant-herbivore interaction (De Moraes and others 1998). These quantitative differences are specific cues that either attract or repel natural enemies from herbivore-infested plants (De Moraes and others 1998). For example, the specialist parasitic wasp *Cardiochiles nigriceps* is preferentially attracted to plants infested by its host, *Heliothis virescens* (boll worm), rather to than plants infested with a nonhost insect, *Helicoverpa zea* (corn ear worm). These observations indicate that the interaction between plants, herbivores, and the herbivore natural enemies is complex and finely tuned.

Volatiles and Phloem-feeding Herbivores

Compared with caterpillars and spider mites, relatively little is known about the volatile blends released by herbivores that do not extensively damage plant tissues. Aphid-infested plants release volatile blends to make them attractive to parasitoid wasps (Du and others 1998) and mediate the density of aphids on plants (Bernasconi and others 1998; Quiroz and others 1997). The corn leaf aphid (*Rhopalosiphum maidis*)-maize interaction releases the most common terpenoids emitted after caterpillar and spider mite infestation: β-ocimene, linalool, 4,8-dimethyl-1,3,7-nonatriene, α-farnesene, β-farnesene, 4,8,12-trimethyl-1,3,7,11-tridecatetraene, and acetylated C_6 volatiles (Figures 6, 7) (Bernasconi and others 1998; Páre and Tumlinson 1999; Quiroz and others 1997).

In addition to terpenoids and C_6 volatiles, MeSA is released after aphid feeding (Bernasconi and others 1998). MeSA may have two roles. MeSA, like SA, activates the SA-dependent defense-signaling pathway and induces *PR* gene expression and SAR (Figure 1) (Shulaev and others 1997). Although controversial, MeSA may also provide an interplant communication that activates defense responses in neighboring plants (Preston and others 1999; Shu-

laev and others 1997). MeSA and the volatile terpenoid β-farnesene are strong aphid repellents (Figures 3, 6) (Hardie and others 1994; Pickett and Griffiths 1980). Bernasconi and others (1998) have speculated that these compounds allow aphids to avoid settling on plants with high aphid densities. These infested plants have a decreased nutritional quality, attract more parasitic wasps, and have higher induced defenses. All would be conditions detrimental for aphid survival.

JA and Volatile Release

The release of terpenoid and C_6 volatiles are strongly influenced by the release of linolenic and linoleic acids from membranes. These C_{18} fatty acids provide substrates for the synthesis of JA, C_6 volatiles (See: *C_6 Volatiles and Herbivore Interactions*) or insect-modified lipid elicitors for volatile production (See: *Elicitors of Volatile Production*). JA treatments of plants induce release of volatiles not emitted by healthy plants. JA releases a complex blend of terpenoid volatiles from *Phaseolus lunatus* (lima beans), maize, and *Gerbera jamesonii,* but the volatiles are not equivalent to those released after herbivore feeding (Dicke and others 1999; Gols and others 1999; Hopke and others 1994). For example, although the carnivorous mite (*Phytoseiulus persimilis*) is attracted to volatiles from JA-treated plants relative to healthy plants, they prefer plants infested with their prey *T. urticae*. These data indicate that the qualitative and quantitative differences in the volatiles released from mite-infested and JA-treated plants provide important cues to natural spider mite enemies. Finally, in the field, JA-treatment of insect-infested tomato plants enhanced plant-herbivore-herbivore enemy interactions. Tomato plants treated with JA and infested with the beet army worm (*Spodoptera exigua*) enhanced the attraction and/or retention of the parasitoid wasp *Hyposoter exiguae* relative to plants not treated with JA (Thaler 1999).

Nevertheless, a dichotomy exists because wounding does not induce the array of volatiles emitted after JA treatment, but wounding induces JA synthesis. Wounding may repress the synthesis of the JA- and herbivore-induced volatiles to prevent herbivore enemies from being attracted to noninfested, but mechanically damaged, plants. This suppression could be due to differences in the balance of the oxylipin intermediates and jasmonates that occur after wounding and exogenous JA treatments. Alternatively, repression may be due to mechanisms that are independent on the products of the octadecanoid pathway. Because JA stimulates activities of some enzymes critical for terpenoid volatile biosynthesis (Bouwmeester and others 1999), it is possible that the volatile-repression mechanisms may influence expression of volatile biosynthesis genes or regulation of enzymatic activities that control or limit volatile production. The insect-derived elicitors for volatile production may antagonize this suppression mechanism.

C_6 Volatiles and Herbivore Interactions

Although large changes in volatile terpenoids occur after herbivore feeding in many plants, changes in C_6 volatiles can also be dramatic and have a strong impact on herbivore-plant interactions. C_6 volatiles are emitted at low levels by healthy plants and are rapidly released in response to insect feeding and mechanical damage. C_6 volatiles, the "green" odors emitted from freshly cut grass, are synthesized from linolenic (18:3) and linoleic (18:2) acids (Blée 1998). With the activity of *LOX,* 13-hydroperoxide lyases (HPLs), alcohol dehydrogenase (ADH), isomerization factors (IF), and acylases, the C_6 volatiles *cis*-3-hexenol, *trans*-2-hexenal, *trans*-2-hexenol, hexanol, and *cis*-3-hexenyl acetate are formed (Hatanaka and others 1987) (Figure 7). Because the levels of 18:3 and 18:2 fatty acids in each organ and plant vary, the balance of the linolenic- and linoleic-derived C_6 volatiles may be distinct and provide important signals in plant-herbivore-herbivore enemy interactions. In some plants, the substrates and enzymes critical for C_6 volatiles are synthesized de novo in response to injury. This is consistent with the fact that wounding and JA enhance phospholipase A_2, HPL, and *LOX* activities, thereby increasing substrates for C_6 volatile production (Avdiushko and others 1995; Narváez-Vásquez and others 1999).

C_6 volatiles influence plant-herbivore and plant-pathogen interaction at several levels (Table 3). First, C_6 volatiles stimulate expression of wound-response genes (Bate and Rothstein 1998). It is not presently known whether C_6 volatiles induce a novel set of genes important in herbivore-plant interactions. Second, C_6 volatiles reduce aphid fecundity, spider mite fecundity, and caterpillar feeding (Avdiushko and others 1997; Hildebrand and others 1993; Kasu and others 1995). Third, C_6 volatiles are used as attractants for the Colorado potato beetle and specialist aphids (Bolter and others 1997; Visser and others 1996). Fourth, C_6 volatiles have antimicrobial and antifungal activity at biologically relevant concentrations. (Andersen and others 1994; Croft and others 1993). Because defense- and wound-signaling pathways cross talk, deterrence of pathogen growth at the wound site might enhance activity of wound-response pathways critical for

Table 3. Impact of C_6 Volatiles on Herbivore-plant Interactions

C_6 Volatile[a]	Impact on Herbivores and Plant Gene Expression	Reference
trans-2-hexenal	Reduces aphid fecundity	Hildebrand and others 1993
	Reduces spider mite fecundity	Kasu and others 1995
	Increases in *AOS, LOX, PAL, VSP, CHS,* and *DFR* RNAs	Bate and Rothstein 1998
	Reduces *M. sexta* feeding	Avdiushko and others 1997
cis-3-hexenol	Reduces aphid fecundity	Hildebrand and others 1993
Hexanol	Reduces aphid fecundity	Hildebrand and others 1993
	Increases in *LOX* RNAs	Bate and Rothstein 1998
Hexanal	Reduces spider mite fecundity	Kasu and others 1995
	Deters *M. sexta* feeding	Avdiushko and others 1997
cis-3-hexenal	Increases in *LOX* RNAs	Bate and Rothstein 1998
trans-3-hexenyl acetate	Attractant for the cabbage aphid	Visser and others 1996
C_6 Mixture	Attractant for Colorado potato beetle	Bolter and others 1997

[a]*Structures for C_6 volatiles are shown in Figure 7.*

wound healing and containment of herbivore damage (See: *Plant Responses to Pathogen Attack*). Finally, C_6 volatiles inhibit pollen tube germination (Hamilton-Kemp and others 1992), which may have a significant impact on the timing and success of fertilization (a fitness cost) in herbivore-infested plants.

Elicitors of Volatile Production

Two insect elicitors of volatile release have been identified: volicitin and β-glucosidase. Volicitin [*N*-(17-hydroxylinolenyl)-L-glutamine] (Figure 3) was identified in regurgitant from the beet armyworm (Turlings and others 1993) and is synthesized by the concerted efforts of the plant and insect (Páre and others 1998). Linolenic acid is supplied by the plant and the glutamine is of insect origin. Application of volicitin to a wound induces the same array of volatiles released by beet armyworm feeding on maize.

β-Glucosidases are the second class of elicitors that mediate volatile release. Treatment of lima beans and *B. oleraceae* (cabbage) with an almond β-glucosidase releases acyclic terpenes that are typically emitted by the red-spotted spider mite and *P. brassicae* larvae, respectively (Hopke and others 1994; Mattiacci and others 1995). The detection of a β-glucosidase in *P. brassicae* regurgitant suggests that an insect-derived β-glucosidase may be an elicitor for volatile production. The recent identification of a squash β-glucosidase–like protein gene (*SLW3*) that is induced by insect feeding (van de Ven and others 2000) suggests that plant-encoded β-glucosidases could potentially play a role in this signaling process.

Regurgitant also contains signaling molecules that enhance expression of wound-response genes (Korth and Dixon 1997a), increase JA biosynthesis (McCloud and Baldwin 1997), and enhance the induced resistance stimulated by insect feeding (Lin and Kogan 1990). It is not presently known whether volicitin, β-glucosidases, or another component of insect regurgitant is responsible for these changes. Relatively little is known about the composition of herbivore regurgitant and less is known about the saliva of chewing insects. The recent identification of a *H. zea* salivary glucose oxidase (Eichenseer and others 1999) suggests that this H_2O_2-generating enzyme could play an important role in ROS production at the site of feeding. ROS are important signals in the plant defense and wound responses (Bolwell and Wojtaszek 1997).

Future Directions

The field of plant-herbivore interactions has entered an exciting period. Herbivore-plant interactions are exceedingly complex. Insects from different feeding guilds induce distinct changes in plant gene expression. Whereas phloem-feeding whiteflies and aphids activate defense-response pathways induced by pathogen attack, chewing and cell-content feeding herbivores activate wound-response pathways. Most studies to date have focused on herbivores that consume foliage. Virtually nothing is known about the signaling pathways activated in response to herbivores that feed on seeds or roots. It will also be interesting to determine whether other phloem-feeding herbivores that cause more extensive damage to foliage induce the pathogen defense-response

pathways like whiteflies or whether there is a balance or shift from defense-signaling pathways to wound-signaling pathways during infestation.

In the field, plants are challenged by multiple herbivores simultaneously, and they must prioritize the signals and determine which pathway to activate. Treatment of plants with SA, BTH, and JA, and analysis of transgenic plants with a suppressed wound-induced MAP kinase or altered levels of PAL, support the idea that SA- and JA-regulated signaling pathways are reciprocally regulated (Felton and others 1999; Fidantsef and others 1999; Peña-Cortés and others 1993; Seo and others 1995). The mechanisms that control the balance of SA- versus JA-mediated responses have a profound impact on plant tolerance to herbivores. Furthermore, the interactions of the JA-defense and JA-wound response pathways are not understood. Although JA-dependent defense response and wound-response genes use both JA and ethylene as cues, the mechanisms to activate these sets of genes appear to be distinct. Many JA-regulated defense-response transcripts accumulate in response to ethylene but are not activated by wounding (or systemin), whereas JA-regulated wound-response transcripts do not accumulate in response to exogenous ethylene treatments (Chao and others 1999; van Wees and others 1999). Because JA-wound response genes induce products that actively antagonize herbivore feeding or decrease the nutritional value of food (PPO, peroxidase, pins, Cys proteases, LOX) (Broadway and Duffey 1986; Felton and others 1992; Johnson and others 1989; Pechan and others 2000), understanding the networks that coordinate the flux between these pathways is important.

Finally, there is substantial evidence that herbivores produce novel elicitors to influence direct and indirect defenses. Two of these elicitors have been identified, and no doubt more will be identified in coming years. Understanding these elicitors and interactions with *R* genes in herbivore-resistant plant interactions is a priority. In addition, labor-intensive experiments to characterize biochemically the constituents of insect oral secretions and the specific defense responses they induce in different plant species should be done. Finally, it is important to remember that many phloem-feeding insect-plant interactions actually represent the interactions of three organisms: the plant, herbivore, and endosymbiont(s). The importance of endosymbionts in production of signals from phloem-feeding insects will be important to discover.

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