DOI: 10.1111/j.1570-7458.2006.00503.x

MINI REVIEW

The molecular bases of plant resistance and defense responses to aphid feeding: current status

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Accepted: 25 September 2006

Key words: elicitation, plant arthropod defense signaling, allelochemicals, pathways, antioxidants, plant structural defense, homeostasis

Abstract

Plant genes participating in the recognition of aphid herbivory in concert with plant genes involved in defense against herbivores mediate plant resistance to aphids. Several such genes involved in plant disease and nematode resistance have been characterized in detail, but their existence has only recently begun to be determined for arthropod resistance. Hundreds of different genes are typically involved and the disruption of plant cell wall tissues during aphid feeding has been shown to induce defense responses in *Arabidopsis*, *Triticum*, *Sorghum*, and *Nicotiana* species. *Mi-1.2*, a tomato gene for resistance to the potato aphid, *Macrosiphum euphorbiae* (Thomas), is a member of the nucleotide-binding site and leucine-rich region Class II family of disease, nematode, and arthropod resistance genes. Recent studies into the differential expression of *Pto*- and *Pti1*-like kinase genes in wheat plants resistant to the Russian wheat aphid, *Diuraphis noxia* (Mordvilko), provide evidence of the involvement of the *Pto* class of resistance genes in arthropod resistance. An analysis of available data suggests that aphid feeding may trigger multiple signaling pathways in plants. Early signaling includes gene-for-gene recognition and defense signaling in aphid-resistant plants, and recognition of aphid-inflicted cell damage in both resistant and susceptible plants. Furthermore, signaling is mediated by several compounds, including jasmonic acid, salicylic acid, ethylene, abscisic acid, giberellic acid, nitric oxide, and auxin. These signals lead to the development of direct chemical defenses against aphids and general stress-related responses that are well characterized for a number of abiotic and biotic stresses. In spite of major plant taxonomic differences, similarities exist in the types of plant genes expressed in response to feeding by different species of aphids. However, numerous differences in plant signaling and defense responses unique to specific aphid–plant interactions have been identified and warrant further investigation.

Introduction

Aphids (Order Homoptera) are major insect pests of world agriculture, damaging crops by removing photo assimilates and vectoring numerous devastating plant viruses. Many pest aphid species, along with several hundred other insect pests, are resistant to insecticides (Devonshire & Field, 1991). Insect-related crop damage and insecticide resistance have led to the development and cultivation of many aphid-resistant crop varieties (Painter, 1951; Panda & Khush, 1995; Smith, 2005). As the development of aphid-resistant plants has progressed, so has research on the genetics of aphid–plant interactions. Yet only recently have the molecular bases of plant–aphid interactions begun to be understood. Several types of plant resistance genes and plant defense response genes have been identified, and results acquired to date indicate that aphids activate plant defense-signaling pathways dependent on both salicylate and jasmonate signaling molecules (reviewed in Kaloshian, 2004). The origin of the compounds eliciting these signals is poorly understood. There is evidence that molecules eliciting these reactions may be directly synthesized by aphids or may be products of aphid endosymbiotic bacteria (Urbanska et al., 1998; Miles, 1999; Forslund et al., 2000).

Aphids are the largest group of insect phloem feeders. During feeding, aphid salivary stylets penetrate plant

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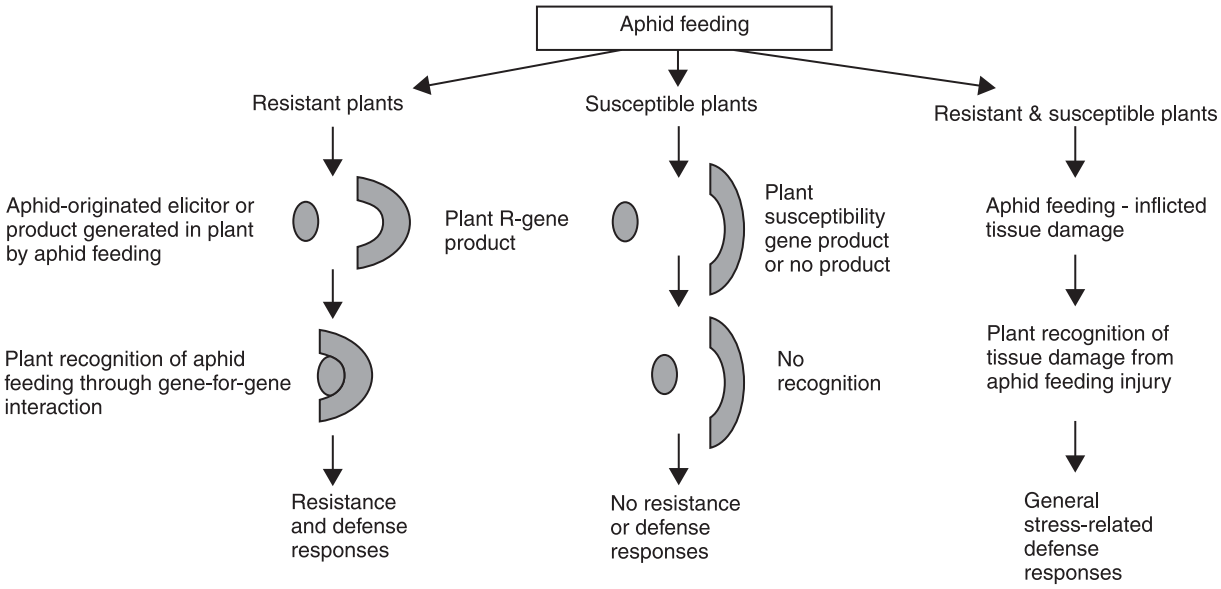

Figure 1 Model of recognition of aphid feeding by resistant and susceptible plants.

tissues to feed on photo assimilates translocating in the phloem sieve elements (Pollard, 1972). While chewing insects cause extensive plant tissue damage, the prolonged interactions of aphid stylets with plant cells result in plant responses to aphids and other phloem-feeding insects that differ from those of chewing insects (Fidantsef et al., 1999; Stout et al., 1999; Walling, 2000). Aphid probing may be influenced by changes in the chemical contents of the sieve element sap or physiological changes induced by aphid saliva (Prado & Tjallingii, 1997; Hays et al., 1999; Telang et al., 1999; Ponder et al., 2001). During feeding, aphids secrete rapidly gelling sheath saliva and watery, digestive saliva. Sheath saliva is composed primarily of proteins, phospholipids, and conjugated carbohydrates. Watery digestive saliva is a more complex mixture of enzymes and other components capable of eliciting plant defense signals (Hori, 1976; Baumann & Baumann, 1995; Urbanska et al., 1998; Miles, 1999).

We hypothesize that two different processes are involved in elicitation of plant response to aphid feeding. One process involves a gene-for-gene recognition of aphid-derived elicitors by plant resistance genes followed by the activation of aphid-specific resistance and defense responses. The second process involves plant recognition of aphid-inflicted plant tissue damage which leads to changes in plant chemistry, followed by the production of plant signaling molecules that trigger a general stress response, similar to the basal plant defense to phytopathogens. While general or basal plant defense responses are involved in signaling in both aphid-resistant and aphid-susceptible plants, gene-for-gene interactions are specific for aphid-resistant plants only (Figure 1).

The intricate interactions between aphids and plants comprise an excellent model system with which to study the coadaptations of plants and herbivorous arthropods described in previous reviews of Stotz et al. (1999), Kessler & Baldwin (2002), and Kaloshian (2004). This mini review will discuss the relationships between different classes of plant sequences and in some cases plant genes putatively involved in aphid resistance and plant defense responses.

Aphid resistance genes in plants

Several crop plant resistance (R) genes and R gene homologues are associated with plant resistance to aphids. Single R genes inherited as a dominant trait control aphid resistance in forages, fruit, and vegetables (reviewed in Smith, 2005). In cereal crops, genes from barley, *Hordeum vulgare* L., rye, *Secale cereale* L., or wheat, *Triticum aestivum* L., confer resistance to the Russian wheat aphid, *Diuraphis noxia* (Mordvilko) (Smith et al., 1999) and the greenbug, *Schizaphis graminum* (Rondani) (Teetes et al., 1999). Results with wheat (Liu et al., 2001, 2002, 2005) and barley (Nieto-Lopez & Blake, 1994; Moharramipour et al., 1997) indicate that aphid resistance R gene loci are located on Triticeae homoeologous groups 1 and 7.

One arthropod resistance gene has been cloned. The *Mi-1.2* gene from wild tomato, *Lycopersicon peruvianum* (L.) P. Mill., confers resistance to the potato aphid, *Macrosiphum euphorbiae* (Thomas) (Kaloshian et al., 1997; Rossi et al., 1998), and to three species of the root knot nematode

genus, *Meloidogyne* (Milligan et al., 1998). *Mi-1.2* is a member of the nucleotide-binding site and leucine-rich region (NBS-LRR) Class II family of disease and nematode resistance genes (Rossi et al., 1998; Martin et al., 2003). The LRR region of *Mi-1.2* functions to signal localized cell death and programmed cell death (Hwang et al., 2000; Wang et al., 2001). A model for *Mi-1.2* interaction with elicitors of aphid or nematode origin was recently proposed by Kaloshian (2004) which suggests a gene-for-gene interaction between aphid or nematode and plant that is similar to plant–disease interactions. The *Vat* (virus aphid transmission) gene from melon, *Cucumis melo* L., controls resistance to the cotton aphid, *Aphis gossypii* Glover (Klingler et al., 1998), and to transmission of some non-persistent viruses vectored by *A. gossypii* (Pitrat & Lecoq, 1980). *Vat* putatively encodes a cytoplasmic protein with NBS-LRR characteristics (Brotman et al., 2002) but has not yet been proven to confer *A. gossypii* resistance.

Boyko et al. (2006) reported that a *Pto* [*Pseudomonas syringae* pv. (tomato)]-like serine/theronine kinase gene and a *Pti1(Pto interactor)1*-like kinase gene are both up-regulated in infested *D. noxia*-resistant wheat plants. Deduced amino acid sequences of both genes have a signature of a functional activation domain, the most important part of any serine/threonine kinase, making the *Pto*-like serine/theronine kinase gene a good candidate for the *D. noxia* resistance gene in wheat (Boyko et al., 2006).

Zhu-Salzman et al. (2004) identified an LRR-containing glycoprotein sequence that is differentially expressed in leaves of sorghum, *Sorghum bicolor* (L.), infested by *S. graminum*. LRR-containing glycoproteins are extracellular, membrane-anchored compounds that in some cases recognize specific tomato leaf mold pathogen *Cladosporium fulvum* (*Cf*)-encoded avirulence gene products. Results of Rooney et al. (2005) indicate that Cf-2 and its Avr2 protein trigger a hypersensitive (resistance) response that also requires an extracellular tomato cysteine protease Rcr3. The binding of Avr2 with and resulting Rcr3 inhibition is proposed as the event that enables the Cf-2 protein to activate a resistance response. A sequence similar to the *Xa1* gene encoding the protein that confers resistance to bacterial blight by recognizing a pathogen elicitor was also found by Park et al. (2005) to be up-regulated by *S. graminum* feeding on sorghum.

Several NBS-LRR sequences have also been cloned and mapped to the vicinity of genetic loci associated with resistance to the cereal cyst nematode, *Heterodera avenae*, and the corn leaf aphid, *Rhopalosiphum maidis* (Fitch), in barley (Lagudah et al., 1997; Seah et al., 1998; Ogbonnaya et al., 2001). Wheat plants containing a *D. noxia* resistance gene contain LZ (leucine zipper) – LRR-NBS sequences (Botha et al., 2003; Lacock & Botha, 2003; Lacock et al., 2003; van Niekerk & Botha, 2003). Swanepoel et al. (2003) found close linkage between an LZ-LRR-NBS sequence and a *D. noxia* resistance gene. Finally, a locus controlling the resistance of *Medicago truncatula* Gaert. (barrel medic) to *Acyrthosiphon kondoi* Shinji, the blue alfalfa aphid, has been mapped to a chromosome region flanked by resistance gene analogs predicted to encode the coiled-coil (CC)-NBS-LRR subfamily of resistance proteins (Klingler et al., 2005).

The cloning and identification of aphid resistance genes and resistance-gene candidates support the contention that aphid–plant interactions occur on a gene-for-gene basis. The variety of identified aphid-induced plant sequences suggests that more than one mechanism is involved in the recognition of aphid feeding by resistant plants and that these mechanisms may lead to specific plant differences in early defense signaling and defense response gene pathways.

Plant defense responses to aphid feeding

Changes in plant metabolism and gene expression induced by arthropod feeding are proving to be multifaceted and include those associated with both the general plant defense responses and specific aphid resistance gene–aphid interactions described above (Walling, 2000; Moran & Thompson, 2001). cDNA micro or macroarrays of plant sequences are providing opportunities to evaluate plant gene expression patterns on a genomic scale in response to aphid feeding, and cDNA arrays have identified a number of plant sequences involved in plant response to aphids, including those involved in signaling, protein synthesis, modification and degradation, maintenance of cell structure and homeostasis, and secondary metabolism. For each of these metabolic functions, examples of plant species-specific differences in plant gene expression in response to aphid feeding are described in Table 1. These responses include feeding of *D. noxia*, *S. graminum*, *Myzus nicotianae* Blackman, and *Myzus persicae* on foliage of *Arabidopsis*, celery, *Apium graveolens* L., cereal, or tobacco plants (Moran & Thompson, 2001; Moran et al., 2002; Voelckel et al., 2004; Zhu-Salzman et al., 2004; Divol et al., 2005; Park et al., 2005; Boyko et al., 2006).

Botha et al. (2006) have also developed expression profiles of *D. noxia* resistant wheat plants to identify numerous transcripts associated with the response of plants to *D. noxia* feeding. There transcripts are similar, though not identical to many of those identified by Boyko et al. (2006) and encode proteins functioning in direct plant defense and signaling, oxidative burst, cell wall degradation, cell maintenance, photosynthesis, and energy production.

Specific plant reactions may also be accompanied by general (basal) plant defense responses (and gene expression

Table 1 Plant sequences expressed after aphid herbivory, as determined by suppression subtractive hybridization, microarray hybridization, or macroarray hybridization

	Plant–aphid interaction[1,2]					
Putative cell function	*Apium graviolens* – *Myzus persicae*[3]	*Arabidopsis thaliana* – *M. persicae*[4]	*Nicotana attenuata* – *Myzus nicotianae*[5]	*Sorghum bicolor* – *Schizaphis graminum*[6]	*S. bicolor* – *S. graminum*[7]	*Triticum aestivum* – *Diuraphis noxia*[8]
Signaling	Ethylene-responsive elements Giberellin synthesis-related protein Auxin-regulated protein Calmodulin Protein kinase Transmembrane protein *TIP1.3*	1-aminocyclopropane-1-carboxylate (ACC) oxidase Calmodulin-related lipoxygenase	Coenzyme A reductase Lipoxygenase *WRKY*	LRR-containing glycoprotein Aldehyde oxidase Nitrite reductase Serine carboxypeptidase	*Xa1*-like protein Harpin-induced protein Phosphatidic acid phosphatase Phosphoinositide kinase GTP-binding protein Adenosylhomocysteinase ARF GTPase activation domain-containing protein GTP-binding protein Ankyrin Acid cluster protein 33 Inorganic pyrophosphotase GDSL-motif lipase/hydrolase Acyl-CoA binding protein Voltage-gated Ca^{2+} channel ϒ2 subunit Phytosulfokine receptor Ras GTPase activation protein binding protein WRKY Phospholipase Aci-reductone dioxygenase-like Stearoyl-acyl-carrier protein desaturase	*Pto*-like kinase *Pti1* protein kinase Sterol Δ-7 reductase 12-OPDA reductase Transketolase Calmodulin-binding protein Heat shock protein 90 AAA-metalloprotease
Pathogenesis-related (PR) proteins	Cytokinin-binding *PR10* β-1,3-glucanase *BGL2* Snapkin/*GAST* Defensin *AMP1* Dehydrin Cysteine proteinase	Pr1 β-1,3-glucanase Defensin Hevein-like protein		*PR10* β-1,3-glucanase Chitinases Thaumatin-like protein Abiotic-stress response proteins	β-1,3-glucanase β-1,4-glucanase S-like RNase Cystein proteinase	
ROS production Allelochemic production		α-dioxygenase ACC oxidase Phenylalanine ammonia lyase	Oxalate oxidase α-dioxygenase Lipoxygenase 3-hydroxy-3-methyl glutaryl coenzyme A reductase Trypsin inhibitor	Aldehyde oxidase Serine carboxypeptidase Serine carboxypeptidase Flavanone 3-hydrolase methyltransferase Wound protease Bowman-Birk protease Subtilisin protease Cysteine protease inhibitors	NOD26-like protein Cystein proteinase Cytochrome P450 monooxygenase β-Glycosidase Aci-reductone like dioxygenase protein Thionin-like protein Cystein proteinase inhibitor Polyphenol oxidase Wilms' tumor-related protein	Sterol Δ-7 reductase Transketolase 12-OPDA reductase Cytochrome P450 monooxygenase Monoterpene synthase β-Glycosidase Epoxide hydrolase Transketolase

Carbohydrate metabolism	PS I reaction center PS I antenna proteins Chlorophyll a/b binding protein PS II 10 kDa & LS1 proteins Phytochrome association *PAP2* Ribulose carboxylase Rubisco Chlorophyllase Carbonic anhydrase Glycolate oxidase Mannitol transporters Mannitol dehydrogenase Sucrose synthase Sorbitol dehydrogenase Glyceraldehyde-3-phosphate dehydrogenase β-galactosidase Cytochromes c1 & b6 Thiamine biosynthesis enzymes	Monosaccharide symporter	Mg protoporphyrin IX chelatase Thiosephosphate isomerase Rubisco	Chlorophyll a/b binding protein O_2 evolving enhancer protein	Bundle sheath cell specific protein PS I reaction center subunit 2 PS I chain D precursor Chlorophyll a/b binding protein Cytochromes b6/f complex PS II 10K protein Ferredoxin Mannose 6-phosphate reductase Ribosomal protein chloroplast-like 29 kDa ribonucleoprotein chloroplast precursor SecA-type chloroplast protein transport factor Peroxisomal membrane protein NADP-specific isocitrate dehydrogenase Citrate synthase Enolase Soluble starch synthase Adenine nucleotide translocator Aspartate aminotransferase RING-H2 finger protein	PS I antenna & assembly proteins Chlorophyll a/b binding protein PS II chlorophyll a binding protein psbB PS II O_2-evolving complex protein 1 PS II protein D1 Ribosomal protein S12 Serine/glycine hydroxymethyl-transferase NADH-dependent glutamate synthase β-amylase ATP/ADP carrier protein
Amino acid & protein synthesis	Histone H3.3 Translation factors Transcription factors Homeobox leucine zipper Dead box RNA helicase S-adenosyl methionine synthetases 40- & 60D ribosomal proteins PolyA-binding protein cyclophylin	Anthranilate synthase	18S rRNA ITS 26S & 18S Ssu pseudogene 60D ribosomal protein	LRR-containing glycoprotein Aldehyde oxidase Nitrite reductase Serine carboxypeptidase	RNA-polymerase subunit Small nuclear ribonucleoprotein Histone H2A Ribosomal protein Transcription factor 70 CCR4-NOT transcription complex subunit 7 Heat shock protein 70	T-complex protein RNA-binding protein Transcription elongation factor Asparaginyl-tRNA synthase Protein disulfide isomerase Heat shock protein 90
Plant self-defense	Mannitol transporters Catalase Metallothioneins Peroxidases Chalcone synthase Phytochelatin synthetase Glutathione peroxidase	Glutathione S-transferases Cu/Zn-superoxide dismutase Fe-superoxide dismutase Peroxidase Chalcone synthase Phenylalanine ammonia lyase	Metallothionein-like protein 3-hydroxy-3-methyl glutaryl coenzyme A reductase	Flavanone 3-hydrolase Lactoylglutathione lyase Catalase-3 isozyme	Phytochelatin synthase-like Cystein-rich protein Cytochrome P450 monooxygenase Peroxidase Catalase Glutathione S-transferase Quinone oxidoreductase Cystein proteinase inhibitor Polyphenol oxidase	Aldose reductase NADH dependent glutamate synthase ATP/ADP carrier protein Protein disulfide isomerase Cytochrome P450 β-Glycosidase ABC transporters T-complex protein AAA-metalloprotease

Table 1 Continued

Putative cell function	Plant–aphid interaction[1,2]					
	Apium graviolens – *Myzus persicae*[3]	*Arabidopsis thaliana* – *M. persicae*[4]	*Nicotana attenuata* – *Myzus nicotianae*[5]	*Sorghum bicolor* – *Schizaphis graminum*[6]	*S. bicolor* – *S. graminum*[7]	*Triticum aestivum* – *Diuraphis noxia*[8]
Structural	Actin depolymerising factor Tubulins Lectin Cellulose synthase Expansin Pectin acetylesterase Pectinesterase Xyloglucan endotransglycosylase ADP-ribosylation factor Microtubule-associated protein	Tyrosine decarboxylase Endo-transglycosylase	Xyloglucan endotransglycosylase		Tubulin Suppressor of actine1 2-Dehydro-3-deoxyphosphooctonate aldolase Caffeic acid O-methyltransferase d-TDP glucose Cellulose synthase Proline-rich protein Peroxisomal membrane protein ADP-ribosylation factor Δ1 pyrroline-5-carboxylate dehydrogenase Glycosyl transferase	Actin Vacuolar proton-ATPase ABC transporters Exocyst complex protein Sec10
N metabolism	Glutamate synthetase Nitrate transporter			Nitrate reductase	Aspartate aminotransferase	NADH dependent glutamate synthase
Homeostasis	Copper factor Metallothioneins Peptide transporter Aquaporins Major intrinsic protein Phytochelatin synthetase Ascorbate oxidase Peroxidases		Metallothionein-like protein	ATP-dependent transmembrane transporter	ATP-dependent transmembrane transporter Adenine nucleotide translocator Adenine phosphoribosyltransferase Polyphenol oxidase	Protein disulfide isomerase Vacuolar proton-ATPase ABC transporters Exocyst complex protein Sec10 Cytochrome P450 monooxygenase
Protein & amino acid degradation	Ubiquitin-conjugating enzyme Cysteine proteinase		Ubiquitin-carrier protein	Ubiquitin-specific protease-like protein	RING-H2 finger protein Legumain-like protease	Ubiquitin fusion degradation protein AAA-metalloprotease FtsH

[1]Underlined sequences are down-regulated; non-underlined sequences are up-regulated.
[2]All interactions are compatible, with the exception of those described by Park et al. (2005) and Boyko et al. (2006).
[3]Divol et al. (2005), susceptible infested plants compared to susceptible un-infested plants.
[4]Moran & Thompson (2001); Moran et al. (2002), susceptible infested plants compared to susceptible uninfested plants.
[5]Voelckel et al. (2004), infested plants compared to uninfested plants.
[6]Zhu-Salzman et al. (2004), susceptible infested plants compared to susceptible uninfested plants.
[7]Park et al. (2005), resistant infested plants compared to susceptible infested plants.
[8]Boyko et al. (2006), resistant infested plants compared to susceptible infested plants.

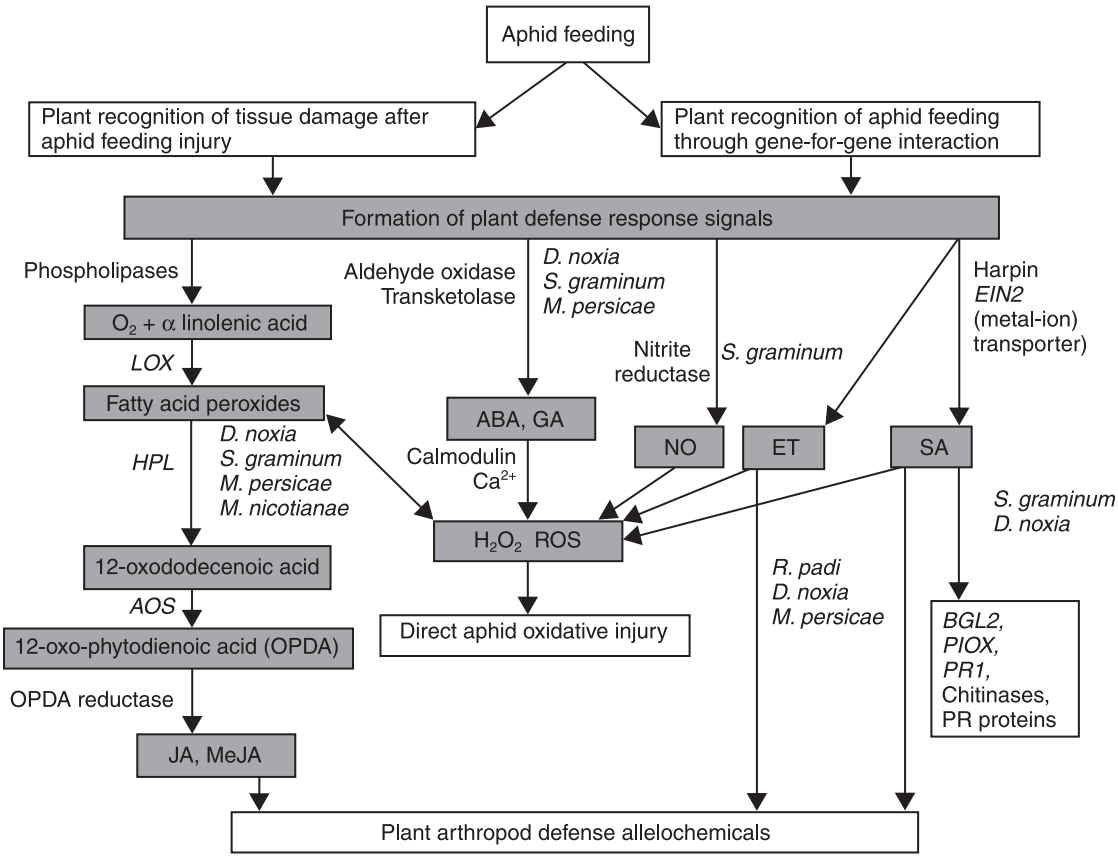

Figure 2 Representative plant signaling pathways involved in aphid resistance and aphid defense response signaling. Arrows indicate pathway activation. Sequence names are those up-regulated by aphid feeding. Aphid species names indicate those activating a pathway response.

patterns) that are common among many plant–aphid interactions. For example, *D. noxia* feeding on wheat foliage and *M. persicae* feeding on *Arabidopsis* plants each induce increased expression of calmodulin binding proteins involved in plant defense signaling. In addition, *D. noxia* feeding on wheat and *M. nicotianae* feeding on tobacco also induce greatly increased expression of glutamate synthase, an enzyme produced and deployed in response to cellular stress. Finally, tobacco, as well as aphid-resistant wheat and sorghum plants all respond to aphid attack by increased production of chlorophyll or photosystem component proteins, presumably as a means of overcoming chlorophyll losses related to aphid feeding (Voelckel et al., 2004; Salzman et al., 2005; Boyko et al., 2006). However, aphid-susceptible sorghum plants actually down-regulate some chlorophyll component proteins after aphid infestation (Zhu-Salzman et al., 2004). The fact that genes encoding these proteins are involved in the response of three different plants to three different aphid species suggests that these are common plant responses to aphid feeding.

Signaling pathways involved in plant responses to aphid feeding

Plant responses to aphid feeding are rapid. *M. persicae* feeding induces resistance responses in foliage of apple (*Malus*) within as little as 2 h, which persist as long as 48 h (Kfoury & Masonie, 1995; Sauge et al., 2002). Results of Klingler et al. (2005) indicate that resistance to the blue alfalfa aphid, *A. kondoi*, in barrel medic, *M. truncatula*, involves an inducible, systemic plant reaction that results in significantly reduced *A. kondoi* growth rates. Plant reactions to aphid feeding may include the activation of general defense response genes, and if aphid resistance traits are present, specific aphid resistance genes, followed by the redirection of normal cell maintenance genes toward plant defense. During response gene activation, plants produce different types of elicitors (activators) that initiate the expression of genes in different defense signaling pathways (Figure 2).

The recognition of aphid feeding probes by plant receptors and ensuing plant defense responses are followed by the transmission of defense response signal cascades that involve various signaling molecules. Plant signaling pathways driven by jasmonic acid, salicylic acid, ethylene, abscisic acid, giberellic acid, reactive oxygen species (ROS), and nitric oxide induce the production of plant defenses in response to attack by numerous species of arthropods, including aphids.

Salicylic acid promotes the development of systemic acquired resistance, a broad-range resistance against pathogens and some aphid species, and is crucial for localized plant tissue hypersensitive (HR) responses (Alvarez, 2000; Walling, 2000; Aviv et al., 2002; Brodersen et al., 2002). Salicylic acid-dependent cascades use salicylic acid and its methyl conjugate to stimulate the expression of defense response genes, including pathogenesis-related (PR) proteins or PR genes with apoplastic localization. Experiments conducted by Moran & Thompson (2001), Moran et al. (2002), Zhu-Salzman et al. (2004), and Divol et al. (2005) demonstrate that in interactions of *M. persicae* feeding on aphid-susceptible *Arabidopsis* and celery, and in interactions of *S. graminum* feeding on an aphid-susceptible sorghum cultivar, major increases occur in the expression of genes associated with the salicylic acid defense signaling pathway, including PR genes such as β-1,3-glucanase, a hevein-like protein, and chitinases (Table 1). However, PR genes are not up-regulated in interactions between *D. noxia* on an aphid-resistant wheat genotype (Boyko et al., 2006) and are down-regulated in the interaction of *S. graminum* on an aphid-resistant sorghum genotype (Park et al., 2005). The combined effects of jasmonic acid and ethylene also control the regulation of chitinase and glucanase PR genes in some pathogen–plant interactions (Pieterse & van Loon, 1999). Although chitinase and glucanase PR genes are highly up-regulated by aphid feeding in both resistant and susceptible plants (van der Westhuizen et al., 1998b; Krishnaveni et al., 1999; Forslund et al., 2000), their regulation is yet to be identified to be under control by jasmonic acid or ethylene.

The octadecanoid pathway leading to jasmonic acid biosynthesis has been studied extensively in relation to the wound-induced systemic induction of proteinase inhibitors and resistance to insect herbivores. Jasmonic acid induces the accumulation of hydrogen peroxide in response to wounding in different plant species, a reaction that may compliment other plant defenses against both herbivores and pathogens (Orozco-Cardenas & Ryan, 1999). Genes putatively involved in jasmonic acid synthesis and jasmonic acid-mediated defense responses [i.e., 12 oxophytodienoate 10,11-reductase, lipoxygenase *(LOX)*, and cytochrome P450] are strongly induced by feeding of *M. nicotianae* Blackman on leaves of *Nicotiana attenuata* Torr. Ex Wats (Voelckel et al., 2004) and *D. noxia* and *S. graminum* on aphid-resistant plants (Park et al., 2005; Boyko et al., 2006) (see Table 1). In addition, Ellis et al. (2002) determined that *M. persicae* population development is greatly reduced on an *Arabidopsis* mutant overexpressing jasmonic acid and ethylene.

Lipoxygenases are located in the cell cytoplasm and function in cell membrane lipid degradation and the production of plant defense response signaling molecules such as jasmonic acid. Transcripts encoding *LOX* genes are strongly induced by feeding of *M. euphorbiae* on tomato foliage (Fidantsef et al., 1999), *M. persicae* feeding on *Arabidopsis* leaves (Moran & Thompson, 2001), and feeding of *M. nicotianae* on leaves of *N. attenuata* (Voelckel et al., 2004). In some plants, linolenic acid released by damaged cell membrane lipids is converted enzymatically to jasmonic acid (Creelman & Mullet, 1997), although this is yet to be demonstrated as a result of aphid–plant interactions.

Methyl jasmonic acid-induced accumulation of ferulic acid and phenolic polymers ultimately leads to cell wall strengthening and increased arthropod resistance in barley and maize, *Zea mays* L. (Bergvinson et al., 1994; Lee et al., 1997). Mewis et al. (2005) demonstrated that *Arabidopsis* jasmonic acid and other oxylipins play important roles in plant defense against feeding by *M. persicae* and the cabbage aphid, *Brevicoryne brassicae* (L.).

This growing body of plant gene expression data supports the established role of salicylic acid in the response of plants to aphid herbivory. However, salicylic acid response increased in the four gene expression studies with aphid-susceptible celery, *Arabidopsis*, and sorghum, while salicylic acid up-regulation was not noted in the study of Park et al. (2005) conducted with aphid-resistant sorghum. Results of related studies in Table 1 also demonstrate the roles of jasmonic acid-regulated pathways in plant defense responses to aphids. Precursors of jasmonic acid appear to function in the protection of aphid-resistant sorghum and wheat plant tissues against aphid herbivory, but are also involved in the defense response of aphid-susceptible *Arabidopsis* and *Nicotiana.*

Relatively little research has been conducted on the involvement of ethylene in the induced defense response of plants to aphids. Yet some experiments have demonstrated that aphid feeding significantly increases ethylene production in the foliage of aphid-resistant barley cultivars compared to susceptible cultivars. Argandona et al. (2001) observed this reaction in barley fed on by both *S. graminum* and *Rhopalosiphum padi*, while Miller et al. (1994) noted the same reaction in barley fed on by *D. noxia*. The expression of genes encoding proteins involved in ethylene production or ethylene signaling (ACC oxidase, sterol

Δ-7 reductase, and ethylene-responsive elements) is up-regulated in aphid-susceptible celery and *Arabidopsis* infested with *M. persicae* (Moran et al., 2002; Divol et al., 2005) and in aphid-resistant wheat infested with *D. noxia* (Boyko et al., 2006) (Table 1). Ethylene is also involved in hypersensitive cell death (Wingler et al., 2005). The case for the involvement of ethylene and jasmonic acid signaling is strengthened by the fact that proteins involved in synthesis of ethylene (ACC oxidase and ACC synthase) have been identified in the phloem sap of pumpkin, *Cucurbita maxima* L., and melon, *C. melo* L. Similarly, precursors of jasmonic acid (*LOX* and *AOS*) have been identified in the phloem of tomato, *Lycopersicon esculentum* (see review by Kehr, 2006). Gene-for-gene interactions between aphids and these plants are well known, and as discussed previously, *Mi-1.2* in *L. peruvianum* confers resistance to *M. euphorbiae* and *Vat* in *C. melo* controls resistance to *A. gossypii*.

Jasmonic acid and ethylene frequently act synergistically, inducing defense responses in plants that are distinct from, and often antagonized by those induced by salicylic acid (Reymond & Farmer, 1998; Bostock, 1999; Pieterse & van Loon, 1999; Walling, 2000; Stotz et al., 2002). Resistance to *M. persicae* in *Arabidopsis* plants has been shown to develop with increased ethylene levels and the expression of several genes essential for ethylene signaling (Dong et al., 2004), including the signal transducer *EIN2*, a bifunctional transducer of ethylene and jasmonic acid signal transduction similar to a disease-response related family of metal-ion transporters which provides a molecular basis for synergy between the two pathways (Alonso et al., 1999). Jasmonic acid–ethylene synergism has also been observed in the induction of foliar defense responses in squash, *Cucurbita moschata* Duchesne, to feeding by the silver leaf whitefly, *Bemisia argentifolii* Bellows and Perring (van de Ven et al., 2000). In contrast, Dong et al. (2004) have shown that the harpin protein, which activates ethylene signaling in *Arabidopsis* and leads to *M. persicae* resistance, does not elicit the involvement of salicylic acid or jasmonic acid in *M. persicae* resistance.

Salzman et al. (2005) used microarray analysis to compare the induced gene responses of sorghum foliage treated with salicylic acid, methyl jasmonate (MeJA), and an ethylene precursor. Results from these experiments demonstrate that jasmonic acid synthesis is induced by both salicylic acid and MeJA, and that salicylic acid also promotes increased jasmonic acid production. Transcriptional cross talk between salicylic acid and jasmonic acid pathways in sorghum also suggests that a subset of genes coregulated by salicylic acid and jasmonic acid may comprise a unique plant signaling pathway tuned to activation by arthropod feeding episodes. The *NPR1* gene (non-expressor of pathogenesis related) and the *WRKY70* transcription factor gene modulate jasmonic acid and signal interactions in *Arabidopsis* plants infected by pathogens (Spoel et al., 2003; Li et al., 2004). Voelckel et al. (2004) demonstrated that *WRKY2* is up-regulated in *M. nicotianae*-infested *N. attenuata* plants but signal modulation roles of *WRKY2* are not known. As indicated by the review of Kaloshian (2004), cross talk between different signaling pathways may allow plants to choose an optimum defense strategy, depending on the type of herbivore feeding stimuli signaling the attack.

The involvement of the growth regulators abscisic acid and giberellic acid in plant responses to aphid feeding is poorly documented. However, abscisic acid is known to be involved in plant response to biotic stresses (van de Ven et al., 2000; Audenaert et al., 2002), and may operate upstream of the octadecanoid pathway, possibly by affecting the release of a jasmonic acid precursor (reviewed in Bostock, 1999). Gibberellic acid plays a role in plant defense response signaling by regulating β-1,3-glucanase release from aleurone cells (Jones, 1971).

Sequences putatively involved in biosynthesis of or activated by abscisic acid or giberellic acid signals (transketolase, aldehyde oxidase, and giberellin among others) are up-regulated in aphid-infested leaf tissues of celery (aphid susceptible) (Divol et al., 2005) and sorghum and wheat (aphid resistant) (Park et al., 2005; Boyko et al., 2006). Conversely, Voelckel et al. (2004) found that a sequence encoding 3-hydroxy-3-methyl glutaryl coenzyme A reductase, which is involved in abscisic acid and giberellic acid biosynthesis, is down-regulated in *N. attenuata* infested by *M. nicotianae*. Park et al. (2005) identified several highly up-regulated genes under abscisic acid control in *S. graminum*-resistant sorghum plants to be involved in cell wall strengthening.

Reactive oxygen species are elicitors of defense signaling pathways with known involvement in the elicitation of plant response to aphid attack (Martin-de Ilarduya et al., 2003; Divol et al., 2005; Boyko et al., 2006). The involvement of ROS in pathogen resistance is well known (Heil & Bostock, 2002), and these compounds may also have direct adverse affects on arthropod midgut tissues. Oligogalacturonides released from plant cell wall polysaccharides by pectinase and polygalacturonase aphid salivary enzymes activate the degradation of linolenic acid, which together with systemin, oligogalacturonic acid, and chitosan, trigger the synthesis of hydrogen peroxide (reviewed in Gatehouse, 2002) and other ROS (Orozco-Cardenas & Ryan, 1999). Linolenic acid degradation also stimulates several different signal pathways to produce defensive allelochemicals (Karban & Baldwin, 1997). Interestingly, tissues of an aphid-resistant apple cultivar have been shown to

up-regulate production of pectin methyl esterase in response to feeding by the rosy apple aphid, *Dysaphis plantaginea* (Passerini) (Qubbaj et al., 2005).

Plants of barley, wheat, and oat, *Avena sativa* (L.), exhibit altered and different peroxide activation patterns in response to feeding by *D. noxia*, the bird cherry oat aphid, *R. padi*, or *S. graminum* (Forslund et al., 2000; Argandona et al., 2001; Ni et al., 2001). Genes involved in oxidative signal transduction through control of cellular hydrogen peroxide concentration, such as peroxidase, catalase, NADH-dependent glutamate synthase, and a mitochondrial adenosine triphosphate/adenosine diphosphate (ATP/ADP) carrier protein, are up-regulated in aphid-infested resistant wheat plants (Boyko et al., 2006) and in *M. persicae*-susceptible celery plants (Divol et al., 2005) (Table 1). However, in aphid-resistant sorghum leaves and aphid-susceptible *Arabidopsis* foliage, several hydrogen peroxide concentration-modulating genes are down-regulated by aphid feeding (Moran & Thompson, 2001; Moran et al., 2002; Park et al., 2005). Calmodulin, involved in Ca^{2+}-mediated defense and hypersensitive cell death (Kawano, 2003) is also involved in plant defense reactions to *M. persicae* and *D. noxia* (Table 1). Calmodulin and peroxidase proteins have been identified in the phloem sap of rape seed, *Brassica napus*, as well as melon and pumpkin (see review of Kehr, 2006).

Sugars also function as messengers in plant signal transduction after aphid infestation, and plant defense responses induced by aphid feeding also stimulate the increased production of intercellular chitinases and β-1,3-glucanases involved in the plant cell wall oligosaccharide release (Botha et al., 1998; van der Westhuizen et al., 1998a,b; Fidantsef et al., 1999; Argandona et al., 2001; Chaman et al., 2001).

In *Arabidopsis*, *M. persicae* feeding induces the expression of *STP4*, a monosaccharide symporter that interacts with invertases to increase carbohydrate import and metabolism, and contributes to the creation of nutrient sinks at aphid-feeding sites (Moran & Thompson, 2001; Moran et al., 2002). Monosaccharide transporters are also induced with other carbohydrate production and metabolism genes during *M. persicae* feeding on foliage of aphid-susceptible celery (Divol et al., 2005) (Table 1). *Diuraphis noxia* feeding on aphid-resistant wheat foliage also alters the expression of genes associated with sugar metabolism and transport (Boyko et al., 2006). These processes are likely to be up-regulated due to phloem sap removal during aphid feeding.

The expression of several genes, including putative aldose reductases, ABC transporters, and mitochondrial ATP/ADP carriers, as well as those potentially involved in oxidation/reduction regulation is altered in both aphid-resistant and aphid-susceptible plants exposed to aphid feeding (Moran et al., 2002; Zhu-Salzman et al., 2004; Divol et al., 2005; Boyko et al., 2006) (Table 1), suggesting that aphid feeding alters the plant redox state and further stimulates defense response signaling. Other up-regulated sequences include those related to production of plant hormones involved in stress signaling, as well as other general stress response compounds such as nitric oxide, heat shock proteins, and metalloproteases (Table 1).

Allelochemical products of plant defense responses

Plant allelochemicals (Whittaker, 1970) once termed secondary plant metabolites are 'non-nutritional chemicals produced by an individual of one species that affect the growth, health, behavior, or population biology of another species' (Seigler, 1998). Allelochemical allomones may occur in plants as volatile herbivore deterrents and repellents, or as non-volatile inhibitors of feeding and oviposition (Tuomi, 1992). Some plants have been shown to respond to aphid feeding damage by producing volatiles that repel or deter aphid settling, such as those derived during the synthesis of MeJA and MeSA (Hardie et al., 1994; Pettersson et al., 1994; Birkett et al., 2000; Vancanneyt et al., 2001). Sequences putatively coding proteins that participate in the synthesis of jasmonic acid (*LOX, 12-ODPA*) and salicylic acid (*WRKY* transcription factors, PR proteins) are up-regulated in several species of plants infested with aphids (Table 1), supporting the concept that unique combinations of plant volatiles are produced in response to attack by different aphid species.

The induction of the various signaling pathways described above also leads to the production of several different types of non-volatile defensive allelochemicals in response to aphid herbivory (Table 1). Cytochrome P450 mono-oxygenases have been identified to be highly up-regulated in aphid resistant varieties of wheat and sorghum (Boyko et al., 2006; Park et al., 2005). However, the functions of mono-oxygenases in aphid defense are difficult to determine, because they serve multiple functions, which include the synthesis of jasmonic acid, salicylic acid, and defense compounds, as well as the detoxification of signaling molecules and exogenous compounds such as aphid salivary enzymes.

Several biochemical and physiological studies have previously demonstrated the involvement of phenols in the reaction of cereal crop plant tissues to aphid damage. Feeding by the grain aphid, *Sitobion avenae* (F.), on aphid-resistant wheat cultivars causes the production of increased levels of phenylalanine ammonia lyase (PAL) and tyrosine ammonia lyase (TAL), key enzymes involved in phenol synthesis (Ciepiela, 1989). Resistance to *R. padi* in wheat is associated with phenol content (Leszczynski,

1985) and has both constitutive and induced components. *Rhopalosiphum padi* feeding on resistant cultivars induces significantly greater amounts of several cell wall-bound phenolic acids, including salicylic, syringic, sinapic, and vanillic acid (Havlickova et al., 1996, 1998). The role of phenols at the molecular level remains unclear. Among the limited number of studies included in Table 1, aphid-susceptible *Arabidopsis* plants down-regulate production of PAL in response to *M. persicae* feeding (Moran & Thompson, 2001) and aphid-resistant sorghum plants down-regulate the production of PPOs in response to *S. graminum* feeding (Park et al., 2005) (Table 1).

There is some evidence to support the role of plant proteinase inhibitors in plant resistance to aphid herbivory. The concentration of chymotrypsin inhibitors increases twofold in the leaves of barley cultivars resistant to *R. padi* following infestation, and when fed to *R. padi* in an artificial diet, the survival of this aphid is greatly decreased (Casaretto & Corcuera, 1998). Similarly, Rhabé et al. (2003a,b) determined that a cysteine proteinase inhibitor from oilseed rape, *Brassica napus* L., and a Bowman-Birk trypsin/chymotrypsin inhibitor from pea, *Pisum sativum* L., are toxic to *M. persicae* and the pea aphid, *Acyrthosiphon pisum* (Harris), respectively.

In regard to proteinase inhibitor gene expression, a gene encoding a cysteine protinesase inhibitor is induced by *S. graminum* feeding in plants of aphid-resistant or aphid-susceptible sorghum genotypes when compared to uninfested plants, but the expression of the inhibitor is down-regulated when infested resistant plants are compared to infested susceptible plants (Zhu-Salzman et al., 2004; Park et al., 2005) (Table 1). A Bowman-Birk type trypsin inhibitor is also highly expressed in tissues of aphid-susceptible sorghum plants fed on by *S. graminum* (Zhu-Salzman et al., 2004). Trypsin proteinase inhibitor expression is also up-regulated in *N. attenuata* foliage infested by *M. nicotianae* (Voelckel et al., 2004). However, in this case, because aphids prefer this site, proteinase inhibitors are viewed to be un-regulated by transcriptional control. Thus, to date there is little evidence to support the concept that the up-regulation of cysteine or trypsin proteinase inhibitors function in plant resistance to aphid herbivory.

The up-regulation of several antimicrobial inhibitor proteins in aphid-susceptible sorghum plants infested by *S. graminum* suggests that these compounds may also be directly involved in plant defense against the symbionts harbored in the gut of pest aphids (Park et al., 2005). The up-regulation of plant allelochemical production is also accompanied by the activation of sequences involved with cellular transport and exocytosis in aphid-resistant wheat plants (Boyko et al., 2006). The synthesis of PR proteins produced within some plants in response to pathogen invasion is up-regulated by aphid feeding on leaves of *Arabidopsis*, celery, and sorghum plants, but is unaffected by aphid feeding in *N. attenuata* and wheat (Table 1).

Plant homeostatic gene responses

Many of the previously described chemical defenses deployed by plants against aphid invasion may also damage plant tissues directly. To avoid this potentially self-inflicted damage, plants respond by producing elevated transcription of many 'housekeeping' or 'civilian' sequences (Karban & Baldwin, 1997) involved in photosynthesis, photorespiration, protein synthesis, antioxidant production, detoxification, and maintenance of cell homeostasis. Some of these gene responses may serve as a form of self-defense to protect the plant from autotoxicity, and others may be involved in addressing the changing source-sink relationships in plants affected by the removal of phloem during aphid feeding.

Photosynthesis results in the production of saccharides, a major source of cell energy and a key source of cellular structural elements. Photosynthesis or photorespiration genes are up-regulated in both aphid susceptible and aphid resistant plant tissues. *Myzus persicae* feeding on leaves of celery, *D. noxia* feeding on wheat foliage, and *M. nicotianae* feeding on *N. attenuata* foliage each promote the up-regulation of such genes, and some photosynthesis genes are down-regulated after feeding by *M. nicotianae* or *S. graminum* (Table 1). This occurrence may reflect the reallocation of plant metabolites from normal growth processes to defensive functions after the elicitation of induced plant responses by aphid feeding. Aphid feeding on celery and wheat foliage leads to the up-regulated transcription of a number of sequences involved in protein syntheses, while such sequences are only mildly affected or unaffected in *Arabidopsis* and sorghum. *Myzus nicotianae* feeding actually down-regulates the synthesis of major ribosomal components in *N. attenuata* (Table 1).

The maintenance of cellular structures and cellular homeostasis are also very important metabolic activities required by plants in order for them to survive aphid-inflicted stresses. The leaves of aphid-infested celery, sorghum, and wheat plants up-regulate a number of sequences participating in cell wall and cell membrane strengthening, as well as redox homeostasis and detoxification (Table 1). Sequences coding proteins involved in protection against and detoxification of ROS and other toxins are up-regulated in celery and wheat, but are down-regulated in *N. attenuata* (Table 1). As pointed out in the review of Thompson & Goggin (2005) plants must find a balance between producing ROS for defense and producing ROS detoxifying enzymes to help stabilize plant tissue damage due to oxidative degradation.

Enzymes involved in ROS scavenging, such as peroxidases, are also prerequisites for plant cell wall building. Barley, oat, and wheat plants produce elevated levels of peroxide in response to feeding by *S. graminum*, *D. noxia*, and *R. padi* on barley, oat, sorghum, and wheat leaves (Forslund et al., 2000; Argandona et al., 2001; Ni et al., 2001; Park et al., 2005). Again, this trend is limited, as aphid-infested *Arabidopsis* plants down-regulate genes involved in the production of cell wall components. Similarly, aphid-infested sorghum plants down-regulate production of an ATP-dependent transmembrane transporter involved in the maintenance of transmembrane electrical potentials. Sequences related to nitrogen assimilation and recycling are up-regulated in celery, sorghum, and wheat. Finally, some sequences involved in the degradation of damaged proteins are up-regulated in celery and wheat, but similar proteins are down-regulated in both *N. attenuata* and sorghum (Table 1).

Conclusions and future directions

Molecular genetic and genomic technologies are now providing exciting new avenues of research in plant–aphid gene-for-gene interactions. These applications are beginning to provide in-depth information about a vast array of plant molecular responses to aphids and other arthropod herbivores. The sequencing of the *Arabidopsis* and rice genomes has begun to provide the first real insights into the structure, function, and location of plant arthropod resistance genes. In addition, commercial oligonucleotide microarrays containing several thousand expressed sequences now allow rapid screening of putative plant resistance-related cDNAs. Arrays for *Arabidopsis*, soybean, *Glycine* spp., barley, tomato and wheat are in use to provide genome-wide representations of plant genes involved in defense responses to arthropod attack.

As additional plant genomes are sequenced, existing and new information about resistance gene synteny will be used to make foresighted decisions in crop plant breeding. The development of future arthropod-resistant crop cultivars should rely on knowledge about the sequences of resistance genes from different resistance sources. In this way, cultivars with resistance genes of diverse sequence and function can be released and deployed to sustain resistance and help delay the development of virulent, resistance-breaking aphid biotypes. The cloning and sequencing of pathogen and arthropod resistance genes and their analogs in many crop plants suggests that current and future plant resistance researchers should increasingly utilize these genetic resources to provide in silico information about the location and function of candidate resistance genes. As a more complete knowledge of crop plant genomes develops, genomic microarrays will provide valuable information about the identity of resistance genes and the gene products mediating their function.

Elicitor-induced responses do play a role in plant resistance to aphids, as discussed and described in this review. However, many gaps remain to be filled in the level and extent of knowledge about elicitor-induced plant resistance to arthropods. Additional research at both the molecular and organismal level is critical to better understand how different species of plants integrate elicitor signals generated as part of defense responses against both arthropods and diseases. The wide variety of specific gene products in both resistant and susceptible plants attacked by arthropods indicates that there are few general plant elicitors of arthropod resistance across the plant kingdom. This is not surprising, given the tremendous variation in the differing degrees of arthropod-host specificity that occur between different arthropod orders and the lack of convergence of evolution of plant CC-NBS-LRR resistance gene homologues in dicot and cereal genomes (Pan et al., 2000).

Conversely, knowledge of how plants recognize the different signals generated by aphid feeding and the elicitors these signals produce is rapidly increasing and a remarkably unified picture is emerging to define the molecular basis of the gene-for-gene interactions between plants and their aphid herbivores. In spite of major plant taxonomic differences, general aphid-induced plant gene expression similarities exist in the different types of plant genes expressed in response to feeding by different species of aphids. Such classes of genes include those involved in jasmonate, abscisic acid, giberellic acid, ethylene, and brassinosteroid synthesis; genes related to calmodulin signaling, ROS, and allelochemical production; as well as genes controlling cell protection, maintenance, and homeostasis.

It is tempting to draw inferences across plant and aphid taxa from the results of the gene expression studies described in Table 1 as to common plant genes involved in aphid resistance. For example, vacuolar H^+-ATPase subunit-like proteins involved in both defense response signaling and plant growth responses are highly up-regulated in both aphid-resistant apple and wheat plants (Qubbaj et al., 2005; Boyko et al., 2006). Similarly, cytochrome P450 monoxygenase genes involved in allelochemical production, chlorophyll a/b binding protein genes involved in carbohydrate metabolism, and cellulose synthase genes presumably involved in structural plant defense are also highly up-regulated in both aphid-resistant sorghum and wheat plants (Boyko et al., 2006; Park et al., 2005). These similarities do illustrate some of the common factors involved in plant–aphid interactions and will aid in the development of a general theory of the molecular bases of plant–aphid interactions.

Yet for as many similarities, there are numerous differences in plant signaling and defense responses that are unique to specific aphid–plant interactions. β-glucosidase sequences highly up-regulated in aphid-resistant wheat plants are down-regulated in aphid-resistant sorghum plants (Park et al., 2005; Boyko et al., 2006) and an ADP-ribosylation factor up-regulated in aphid-resistant apple plants is down-regulated in aphid-resistant sorghum (Qubbaj et al., 2005; Park et al., 2005). Such interactions demonstrate the necessity to study particular aphid-plant systems rather than relying on information obtained using a general model or limited numbers of plant–aphid interactions. Flexible, evolving models of the key genomic processes identified to regulate aphid–plant interactions will be necessary in order to ensure a better understanding of the metabolism of plants induced for defense against aphid attack.

Acknowledgements

This work was funded by grants from the U.S. National Science Foundation, the Kansas Wheat Commission and the Kansas Agricultural Experiment Station. We thank three anonymous reviewers for helpful comments that greatly improved the manuscript.

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