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Review

Fatty acid signaling in *Arabidopsis*

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Abstract. Many organisms use fatty acid derivatives as biological regulators. In plants, for example, fatty acid-derived signals have established roles in the regulation of developmental and defense gene expression. Growing numbers of these compounds, mostly derived from fatty acid hydroperoxides, are being characterized. The model plant *Arabidopsis thaliana* is serving a vital role in the discovery of fatty acid-derived signal molecules and the genetic analysis of their synthesis and action. The *Arabidopsis* genome sequencing project, the availability of large numbers of mutants in fatty acid biosynthesis and signal transduction, as well as excellent pathosystems, make this plant a tremendously useful model for research in fatty acid signaling. This review summarizes recent progress in understanding fatty acid signaling in *A. thaliana* and highlights areas of research where progress is rapid. Particular attention is paid to the growing literature on the jasmonate family of regulators and their role in defense against insects and microbial pathogens.

Key words: *Arabidopsis* – Fatty acids – Jasmonic acid – Oxo-phytodienoic acid – Signal transduction

Introduction

It is likely that most (and perhaps all) organisms use fatty acids and their derivatives as signal molecules to control processes as diverse as reproduction, social behavior, sleep, metabolism and defenses. In plants, fatty acid signals have crucial roles in defense and development, and their study has implications in basic biology as well as in agriculture. Fatty acid signaling is being studied in dozens of species of higher plants (Farmer 1994; Creelman and Mullet 1997; Mueller 1997; Wasternack and Parthier 1997). One species, however, stands out as having the potential to rapidly yield large amounts of new information in this area: *Arabidopsis thaliana*. The extensive use of mutants for studies of fatty acid metabolism (Ohlrogge and Browse 1995) and the rapidly progressing genome project (http://genome-www.stanford.edu/arabidopsis/) are but two factors that are helping to make *A. thaliana* an indispensable tool in our efforts to understand signal pathways in the development and defense of plants.

Jasmonic acid (JA), a 12-carbon fatty acid cyclopentanone (and/or its precursors), is essential for the completion of the *A. thaliana* life-cycle. Without it, or in plants unable to perceive JA, this plant does not generate viable pollen (Feys et al. 1994; McConn and Browse 1996). Without the ability to generate JA, *Arabidopsis* is highly susceptible to herbivory (McConn et al. 1997), and it is clear that the jasmonate biosynthetic pathway underlies many inducible defenses against herbivory throughout the plant kingdom (Karban and Baldwin 1997). Despite the vital roles of jasmonates in plant physiology we are still unsure of which jasmonates are biological regulators in vivo and whether all plants employ the same jasmonate signal. Additionally, our knowledge of the molecular machinery used to transmit jasmonate signals is only beginning to emerge.

Although much of the review is devoted to signaling via JA and other members of the jasmonate family, *A. thaliana* is no less suited to other areas of fatty acid signaling. Antimicrobial aldehyde production and the possibility that epidermal cells produce fatty acid signals necessary for development are two areas where advances are expected. Several important areas related to fatty acid signaling in *Arabidopsis* are not, however, covered in the review. For example, phospholipase function and

Abbreviations: dnOPDA = dinor-oxo-phytodienoic acid; OPC = oxo (pentenyl) cyclopentane; OPDA = oxo-phytodienoic acid; JA = jasmonic acid; LOX = lipoxygenase; MeJA = methyl jasmonate; 16:0 = hexadecanoic acid; 16:3 = 7Z, 10Z,13Z-hexadecatrienoic acid; 18:2 = 9Z,12Z-octadecadienoic acid (linoleic acid); 18:3 = 9Z,12Z,15Z-octadecatrienoic acid (linolenic acid)

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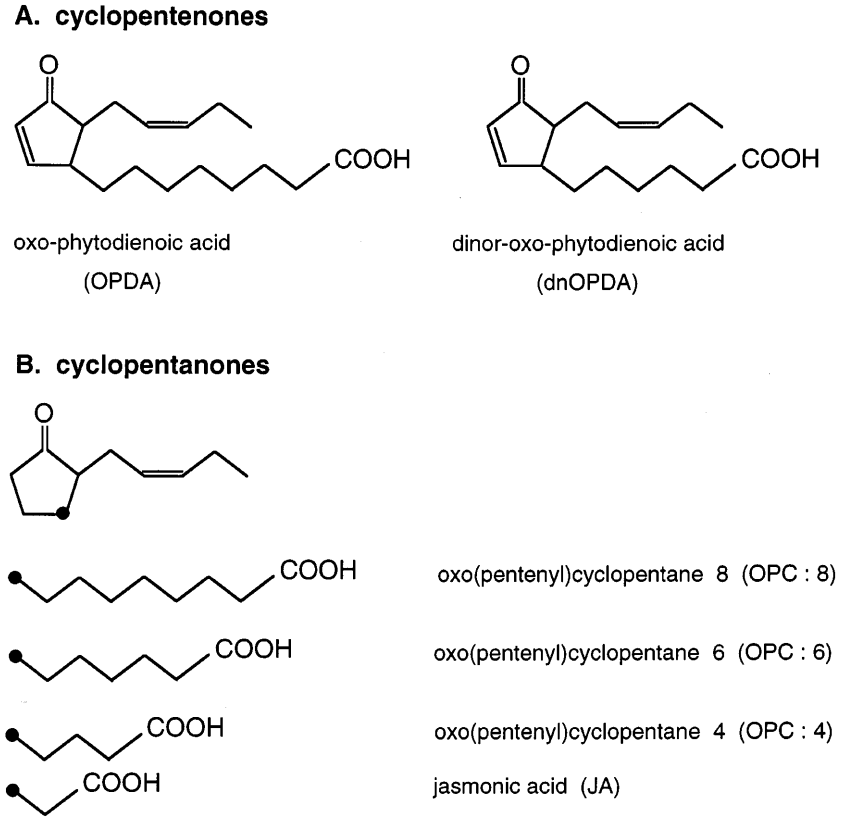

Fig. 1A,B. Jasmonate family signals in *Arabidopsis*. The jasmonate family can be divided into: **A** cyclopentenones (OPDA and dnOPDA); **B** cyclopentanones of the OPC series leading to JA. Both OPDA and dnOPDA are powerful inducers in their own right, and their biological activities in vivo may, in at least some cases, be greater than that of JA

phosphatidyl inositol signaling are not discussed, although progress in these areas is now rapid. Throughout the review we have chosen to refer to mutants in lowercase italics [e.g. *lox* a lipoxygenase mutant], wild-type genes in upper-case italics (e.g. *LOX*) and proteins in simple upper case (e.g. LOX). Many key papers involving research in fatty-acid signaling in species other than *A. thaliana* have, for brevity, not been cited even when they preceeded discoveries in this plant.

The jasmonate signal complex

The jasmonate family is defined here as biologically active cyclopentenones and cyclopentanones of related structure and related origin from the octadecanoid and hexadecanoid biosynthetic pathways. The six members of the jasmonate family that have been detected in *Arabidopsis* are shown in Fig. 1. The molecules are either cyclopentenones [oxo-phytodienoic acid (OPDA), and dinor-oxo-phytodienoic acid (dnOPDA)] or cyclopentanones [the oxo(pentenyl)cyclopentanealkanoic acid (OPC) series including JA itself]. At the time of writing this review, we are still unsure of how many jasmonate family members are signal molecules in vivo in any plant and of their relative contribution to the control of gene expression. The cyclopentenones are expected to be powerful in vivo signals in their own right, based on studies in other plants cited in Stelmach et al. (1998). The recent discovery of dnOPDA (Weber et al. 1997) extends this idea. The term 'jasmonate signal complex' (Farmer 1997) has been used to indicate the potential complexity of jasmonate signaling in plants where single or multiple members derived from the octadecanoid and hexadecanoid pathways may contribute to signal generation in vivo.

A global analysis of oxylipins in *Arabidopsis* reveals a new signal pathway from hexadecenoic acid

Estimates of the levels of JA and OPDA in healthy and wounded *Arabidopsis* leaf tissue have been published and steep rises in these compounds are detected after tissue injury (Laudert et al. 1996; Stelmach et al. 1998). Recently, a method for global analysis of oxylipin levels in *A. thaliana* leaves was developed (Weber et al. 1997). This method allows the simultaneous extraction of many oxylipins and gives 'oxylipin signatures' for any plant tissue. Oxylipins were extracted, separated by gas chromatography and analyzed as a total ion profile by mass spectrometry. Selective ion monitoring was used to display and quantitate peaks for members of the jasmonate family. Results of such an analysis are shown in Fig. 2. Extraction of the data for ion 83 revealed JA and its immediate precursors. A new compound was discovered when the total ion profile was scanned for a fragment of mass 96 which indicates the presence of a cyclopentenone such as that in the 18-carbon compound OPDA. The new 16-carbon compound, which was found to be homologous to OPDA, was named dnOPDA and could be synthesized from hexadecatrienoic acid

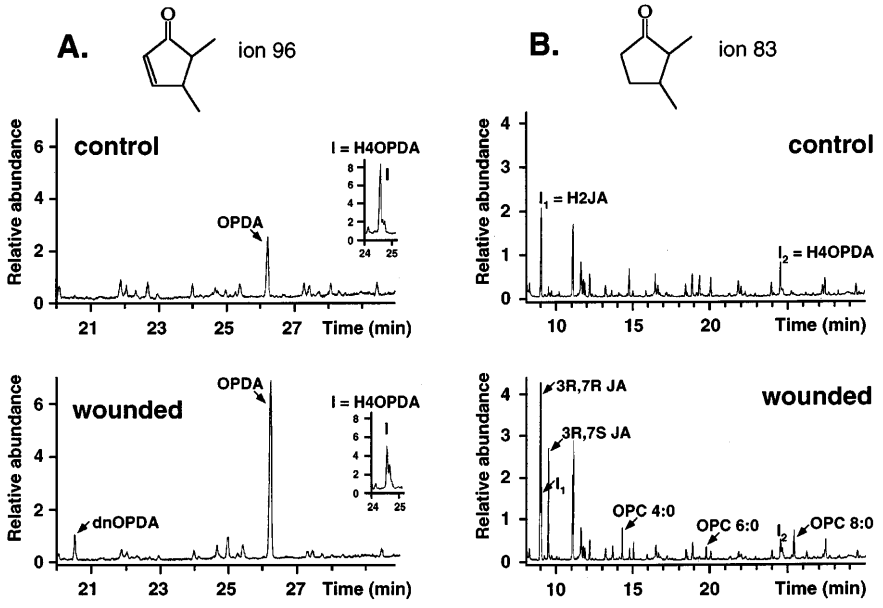

Fig. 2A,B. Oxylipin signatures from *Arabidopsis* leaves. Extracts from healthy leaves or leaves wounded 90 min prior to extraction were analysed by gas chromatography/mass spectrometry. Selective ion monitoring revealed: **A** molecules fragmenting to ions of m/z = 96, from the cyclopentenone ring of OPDA and dnOPDA; **B** ions of m/z = 83 typical of JA and the OPC series. The two internal standards used, dihydro-JA (H2JA) and tetrahydroOPDA (H4OPDA) are indicated as I_1 and I_2, respectively

(16:3) in vitro. Since 16:3 is found in monogalactosyl diacylglycerol, it is possible that a galactolipase releases 16:3 prior to synthesis of dnOPDA. Employment of the *A. thaliana* mutant *fad5* (see review by Ohlrogge and Browse 1995), which lacks the ability to desaturate hexadecanoic acid (16:0), showed that the presence of 16:3 in vivo was a prerequisite for dnOPDA biosynthesis. Thus *Arabidopsis* has at least two pathways to the synthesis of jasmonate family members, the established octadecanoid pathway from linolenic acid (18:3) and the newly discovered hexadecanoid pathway from 16:3 (Fig. 3). It is not yet known whether dnOPDA is reduced by OPDA reductase. It is also possible that a pathway from linoleic acid (18:2) to dihydrojasmonates acid exists in plants (Gundlach and Zenk 1998).

The discovery of a parallel pathway to the synthesis of a jasmonate family member led to the question of what happens to jasmonate signal levels when one branch of the pathway is disrupted by mutation? The *fad5* mutant of *A. thaliana* provided a tool for a preliminary approach to this question. Plants deficient in 16:0 desaturation failed to accumulate dnOPDA and, remarkably, showed reduced levels of OPDA in leaves of uninjured plants (Weber et al. 1997). Levels of OPDA in the wounded leaves of the *fad5* mutant are, however, similar to those of wild-type plants. These results suggest that (i) jasmonate levels are regulated differently in wounded and unwounded *Arabidopsis* and (ii) the relative levels of jasmonates are tightly controlled in uninjured plants and compensate to accommodate the absence of a jasmonate family member (in this case dnOPDA). The first conclusion is consistent with previous research on a cosuppressed lipoxygenase gene, *LOX2*, which is involved in wound-induced JA synthesis; in the absence of LOX2, plants still have basal levels of JA (Bell et al. 1995). In our laboratory, global measurements of jasmonate family members are currently being extended and may shed light on new regulatory functions for jasmonates, for example in the control of flux into prokaryotic and eukaryotic pathways to galactolipid synthesis (Weber et al. 1997). It will now be interesting to apply the oxylipin-signature technique to plants infected with various pathogens or subjected to abiotic stresses such as drought and temperature extremes.

Genes involved in jasmonate metabolism

The enzymology of JA synthesis is increasingly clear (Mueller 1997). Several genes involved in JA biosynthesis have been cloned and their regulation has been studied. The picture which is emerging from these studies is that some of these genes are wound-inducible, for example *FAD7* (Nishiuchi et al. 1997), *LOX2* (Bell et al. 1995), and *CYP74* (allene oxide synthase, Laudert et al. 1996) and often JA-inducible: *FAD7* in root tissues (Nishiuchi et al. 1997), *LOX1* (Melan et al. 1993) and *LOX2* (Bell et al. 1995). Thus the jasmonate biosynthetic pathway is, at least in part, wound-regulated. As well as providing information on the regulation of jasmonate biosynthesis the cloning of genes involved in jasmonate biosynthesis provides valuable tools which could be used to establish which jasmonate family members contribute to biological activity in vivo. The recent cloning of a cDNA for 10,11-oxo-phytodienoic acid reductase (*OPR1*, Schaller and Weiler 1997) may now provide a vital tool to resolve the question of which jasmonate family members are active signals for defense gene expression (Gundlach and Zenk 1998; Stelmach et al. 1998), since altered expression of *OPR1* could disrupt the octadecanoid and hexadecanoid pathways at the cyclopentenone (OPDA and dnOPDA) levels. Treatment of plants with exogenous jasmonate family members causes an increase in LOX2 transcripts (Bell and Mullet 1993) and increases the activity of enzymes involved in jasmonate metabolism (Weber et al. 1997). Jasmonates can thus be regarded as metabolic regula-

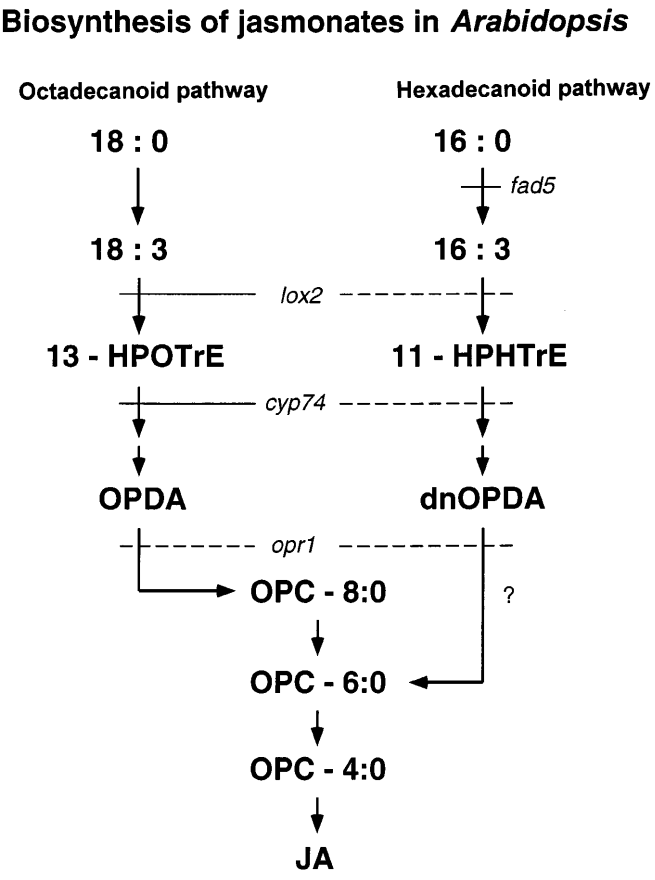

Fig. 3. The biosynthesis of jasmonates in wounded *Arabidopsis* leaves. Mutations affecting the biosynthesis of jasmonates are indicated in *italics*. For each mutant a *solid horizontal line* indicates an established lesion and a *dashed horizontal line* indicates a theoretical lesion. 13-HPOTrE and 11-HPHTrE are the 13- and 11-hydroperoxides of 18:3 and 16:3, respectively. It is not yet clear whether conversion of dnOPDA to OPC-6:0 occurs. *18:0*, octadecanoic acid

tors, although whether they really alter metabolic flux through the jasmonate pathways remains to be tested.

One recently isolated wound-inducible gene (*ACO*) is homologous to acyl-CoA oxidase (Titarenko et al. 1997). The authors speculate that ACO action in the plant response to wounding may include a role in JA catabolism, thus temporally limiting the JA-mediated wound response. The enzyme ACO might be involved in metabolising OPDA to JA, and it would thus be interesting to test whether ACO is OPDA-inducible. Furthermore, the discovery of an inducible component of the β-oxidation machinery could have an impact on our understanding of this process in plants. It will clearly be of interest to follow developments regarding this gene.

Signaling components

The fact that characterized mutants in jasmonate signaling exist in *Arabidopsis* means that we are on the verge of understanding at least some of the cellular machinery involved in jasmonate signal transduction. The mutants characterized so far *jar*1 (Staswick et al. 1992), *coi*1 (Feys et al. 1994), *jin*1 and *jin*4 (Berger et al. 1996), were isolated by their reduced sensitivity to methyl jasmonate (MeJA; and coronatine, a bacterial toxin, in the case of *coi*1). All of these mutations segregate as recessive Mendelian traits. The mutations *jar*1 and *jin*4 may be allelic (Berger et al. 1996). Among the mutants, *coi*1 appears to affect more MeJA-activated responses than do the other mutations. Vegetative storage protein (VSP) does not accumulate in *coi*1 flowers although its levels are high in flowers from wild-type plants as well as in *jin*1 and *jin*4 (Benedetti et al. 1995; Berger et al. 1996). Vegetative storage protein is not MeJA-inducible in the leaves of *coi*1, *jin*1 and *jin*4 plants and MeJA-induction of *LOX*1 gene expression was also inhibited in *coi*1. The *coi*1 plants are, additionally, male-sterile (which is not the case for the mutants *jar*1, *jin*1 and *jin*4) and show no anthocyanin accumulation in response to MeJA. If not alleles, *jin*4 and *jar*1 at least share the characteristic that their root growth is less resistant to MeJA than that of *coi*1 and *jin*1. These data on phenotype and gene expression in *A. thaliana* mutants indicate that the mutants affect different levels in the JA signal transduction chain(s). Berger et al. (1996) speculate that *coi*1 might be upstream of *jar*1, *jin*1 and *jin*4. It may be difficult at this stage to position mutants relative to one another in a simple and universal model; however, work on protein phosphorylation during JA signaling is helping to build an image of this process.

Protein phosphorylation is involved in signal transduction controlling wound-inducible gene expression in tobacco (Seo et al. 1995) as well as in *A. thaliana* (Rojo et al. 1998). Protein kinase and protein phosphatase inhibitors, as well as auxin, were used to inhibit or modulate the expression of several wound-inducible genes, including *VSP*, *JR*1, *JR*2, *JR*3, and a choline kinase homologue, *CK* (Titarenko et al. 1997; Rojo et al. 1998). These studies led to a model for the role of protein kinases and phosphatases (probably of the PP2A type) in jasmonate-dependent and jasmonate-independent gene expression. The model implies that protein phosphorylation is involved in or downstream of the JA perception necessary for gene activation (Rojo et al. 1998). This work represents a step forward in our understanding of jasmonate signaling, and it will be interesting to see whether any of the jasmonate-insensitive mutations are in genes encoding protein phosphatases or protein kinases. No receptors are yet reported for jasmonates in *Arabidopsis* or other plants and their subcellular location is unknown.

Defense against herbivores

Genetic analyses have shown that jasmonate production is essential for defense against the herbivore, *Bradysia*, a fungal gnat that is commonly found in *Arabidopsis* cultures (McConn et al. 1997). The *Arabidopsis fad3-2fad7-2fad8* triple mutant lacks the ability to synthesize the jasmonate precursor 18:3 and also has massively

reduced levels of 16:3. This mutant is rapidly attacked by the larvae of *Bradysia impatiens* (McConn et al. 1997). Treatment of the triple mutant with MeJA restored its defenses to *Bradysia*. Exactly how MeJA works in this process is unknown, and it is important to note that it is not clear which genes play direct roles in defense against *A. thaliana*'s herbivores. Existing data lead to a speculative model for the role of jasmonates in the defense of *Arabidopsis* against herbivores (Fig. 4). In the model the glucosinolate system is thought to play a central role. Like other members of the Brassicaceae, *Arabidopsis* uses the production of glucosinolates (Halkier and Du 1997) as a potentially important part of its arsenal against herbivory. Glucosinolates are degraded into highly toxic products (such as isothiocyanates and nitriles) via the enzyme myrosinase, for which at least three genes have been identified in *A. thaliana* (Xue et al. 1995). If and how these genes are regulated in stress and tissue damage is not yet clear; however, a JA-inducible sulphotransferase potentially involved in glucosinolate synthesis has been identified (Lacomme and Roby 1996). Moreover, parts of the myrosinase-glucosinolate system are jasmonate-inducible in other Brassicaceae (Doughty et al. 1995; Tiapalensuu et al. 1997). The exact role of JA (and other jasmonates) in defense against *Arabidopsis* herbivores remains to be established and defense genes activated by pathways dependent or independent of jasmonates need to be identified. The genome sequencing project is now helping to build up a better picture of defense-related genes in *Arabidopsis* and this is illustrated by the recent discovery of genes potentially involved in cyanogenesis (The EU Arabidopsis genome project: Bevan et al. 1998). Whether these genes are regulated by fatty acid-derived signals such as jasmonates is unknown but cyanogenesis is included in Fig. 4 for completeness.

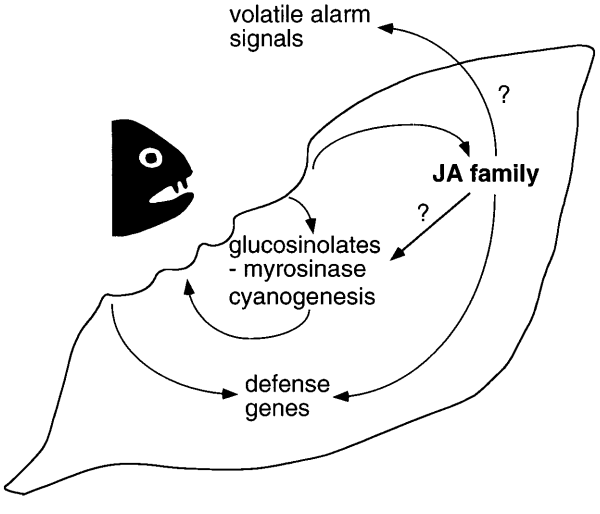

Fig. 4. Speculative model for defenses against herbivory in *Arabidopsis*. Defense genes are activated by members of the jasmonic acid family (*JA family*) or by independent pathways (Titarenko et al. 1997). The glucosinolate/myrosinase system plays important defense roles which may depend to some extent on the production of jasmonates. Note that other genes encoding proven herbivore defenses are yet to be defined in *Arabidopsis*

Herbivore defenses in many plants involve the release of volatile alarm signals from the attacked plant. Recently, a fatty acid-derived elicitor of alarm-signal production was isolated from the regurgitant of an insect herbivore; this molecule, volicitin [*N*-(17-hydroxylinolenoyl)-L-glutamine], activated alarm-signal production in maize (Alborn et al. 1997). Reports of whether or not volicitin (or a related signal) is active in *A. thaliana* are not yet available. If found to be active in *A. thaliana*, these elicitors, which potentially might interact with the jasmonate pathway (Farmer 1997), would be excellent tools for the study of complex signaling between *Arabidopsis* and herbivores. At this point it is worth noting that the great potential of *A. thaliana* and its close relatives as ecological models in the area of herbivore defense is being realised (e.g. Mauricio and Rauscher 1997; http://www.arabis.net/Arabis/resArbis.htm) and, in the future, it will be interesting to see signaling/defense mutants incorporated into these studies. For such experiments the growing number of mutants in epicuticular wax formation (e.g. Aarts et al. 1995; Jenks et al. 1996) are likely to provide excellent materials for studies of the role of cutin quality and quantity in plant-insect interactions.

Fatty acid derivatives in pathogenesis – new models for *Arabidopsis*

What roles do fatty acid derivatives such as JA play in pathogenesis? Several JA-inducible genes which are induced during pathogenesis are known and will help provide answers to this question. Defensins are small antifungal proteins found in the animal and plant kingdoms (Broekaert et al. 1997). In *Arabidopsis*, systemic expression of the defensin gene *PDF*1.2 is induced during infection by the fungus *Alternaria brassicicola* (Penninckx et al. 1996). The *PDF*1.2 gene is up-regulated by JA but not by salicylic acid (SA) and, importantly, after infection with *A. brassicicola* JA accumulates in both infected and non-infected leaves. Furthermore, the lesion-mimic mutant *acd2* displays elevated JA levels as well as increased *PDF*1.2 gene expression (Penninckx et al. 1996). A direct role of the PDF1.2 protein in pathogenesis is not yet confirmed but the protein provides a valuable marker. Other genes which will probably be used to unravel the roles of fatty acids in defense against pathogens include the pathogen- and JA-inducible thionin *THI*2.1 (Epple et al. 1995). Still more roles for fatty acids might be discovered in the regulation of defense gene expression during plant-microbe interaction. Salicylic acid-independent systemic pathogenesis-related gene expression occurs in *A. thaliana* when treated with biocontrol bacteria (Pieterse et al. 1996), and it is not yet known what types of signal molecules regulate these responses.

Bowling et al. (1997) place JA and SA pathways in parallel and propose that the definition of systemic acquired resistance (SAR) in *Arabidopsis* should include both pathways. This is consistent with the systemic increase of both JA and PDF1.2 gene expression

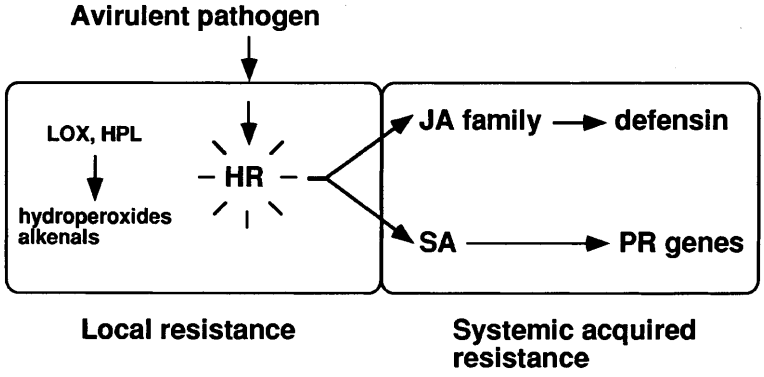

Fig. 5. Potential roles of fatty acids in pathogenesis in *Arabidopsis*. The model is based in part on those of Penninckx et al. (1996) and Bowling et al. (1997). Lipoxygenase (*LOX*) and hydroperoxide lyase (*HPL*) may contribute to the death of pathogen and plant cells during the hypersensitive response (*HR*) by generating toxic fatty acid hydroperoxides and alkenals. Jasmonic acid (*JA*) and salicylic acid (*SA*) up-regulate defense gene expression in distal tissues and contribute to systemic acquired resistance. *PR*, pathogenesis-related

observed by Penninckx et al. (1996). Figure 5 shows a model which provides a framework for further tests of the potential roles of jasmonates in pathogenesis. We have added LOX and hydroperoxide lyase to the model of Bowling et al. (1997) due to their likely in-vivo roles in the production of antimicrobial aldehydes. It has been suggested that these alkenals and aldehydes, (together, perhaps, with their parent fatty acid hydroperoxides) may actually kill cells during hypersensitive cell death in plants (Farmer 1994). Fatty acid hydroperoxides themselves are also toxic to plant cells (data not shown) and might contribute to plant and/or pathogen cell death.

The generation of antimicrobial alkenals and alkanals in infected plant tissues is well known but has not yet received adequate attention in terms of molecular biology. Hexanal and 2E-hexenal are antibiotics thought to play roles in defense against numerous pests (reviewed in Farmer 1994). *Arabidopsis* provides an ideal system in which to investigate the potential roles of these compounds. Hydroperoxide lyases (HPLs) are the enzymes which metabolize fatty acid hydroperoxides to oxoacids and aldehydes. Hydroperoxide lyase activity was recently detected in *A. thaliana* (Avdiushko et al. 1995; Zhuang et al. 1996) and this enzyme(s) may be the source of the volatile C6 aldehydes hexanal and 2E-hexenal. This detailed study has shown that chloroplast fatty acids are likely to be the principle substrates for C6 aldehyde production in *Arabidopsis*. Hydroperoxide lyase from *A. thaliana* is reported to be JA- and wound-inducible (Avdiushko et al. 1995; Rojo et al. 1998). Fatty acid-biosynthesis mutants are also being used to study the production of volatile alkenals and alkanals in *A. thaliana*. Three mutants which have been used are *fad2*, and *fad3*, extrachloroplast desaturases that desaturate octadecanoic acid (18:1) and 18:2, respectively and *fad7*, a chloroplast ω-3 desaturase mutant which blocks conversion of hexadecadienoic acid (16:2) and 18:2 to 16:3 and 18:3, respectively. All three mutants produce altered levels of C6 aldehydes. The mutants *fad2* and *fad3*, (which have reduced levels of extraplastidic 18:2 and 18:3) were found to produce elevated levels of 2E-hexenal, whereas *fad7* produced elevated quantities of hexanal (Zhuang et al. 1996). Although the molecular basis for these effects of the mutations are unclear, it would be worthwhile to test whether these mutants have altered defenses to pathogens and insects.

In summary, studies on the potential roles of fatty acid-derived signals in pathogenesis have now provided good, testable models, and evidence for the roles of jasmonates in defense against pathogens is increasing. If the early literature on antimicrobial aldehydes (reviewed in Farmer 1994) is to be believed, genetic tests of their roles will be fruitful. These tests could include examining the susceptibility of antisense hydroperoxide lyase plants to a battery of different microbial pathogens (and insects). The *Arabidopsis* genome project provides sequence information on regulatory regions of genes potentially involved in fatty acid signal and toxin generation. Careful analysis of existing sequence data will accelerate the design of experiments to identify the role of gene function in signal transduction and defense. For genes such as *HPL* these analyses should be helpful since little information is available for their regulation.

Very-long-chain fatty acids and cuticle formation

Just as C16 and C18 fatty acids can be metabolized to signal compounds in *A. thaliana*, longer-chain fatty acids could serve as substrates for the in-vivo synthesis of biologically active molecules. A potential source of such molecules is the epidermis – the site of cuticle biosynthesis. The formation of cuticle requires the biosynthesis of very-long-chain fatty acids (VLCFAs) by elongation of C18 and C20 fatty acids such as stearic and eicosanoic acids. Healthy vegetative tissues of *Arabidopsis* contain very low levels of C20 and C22 fatty acids but the levels of these compounds can be increased by the ectopic expression of the seed fatty acid elongation gene (*FAE1*) in vegetative tissues (Millar and Kunst 1997). These plants have a dramatically altered phenotype raising the possibility that VLCFAs are metabolized to signals or toxins in these tissues. Interestingly, this possibility provides a parallel with on-going work on fatty acid elongation in vegetative tissues. The fatty acid elongation enzymes which function naturally in vegetative tissues may differ from those in seed tissues. In the case of vegetative tissues it appears that the early steps of stearic acid elongation in epidermal cells may be indispensable for plant growth. No mutants in the elongation of stearic acid in epidermal cells have been reported (Negruk et al. 1996), and this raises the possibility that essential developmental signals derived

from VLCFAs might exist. Together with the results on FAE1 over-expression, these observations make the search for such putative signals attractive although the existance of VLCFA-derived regulators of development remains an open question.

Conclusion

Answers to the following crucial questions related to fatty acid signaling are likely to involve work with *Arabidopsis thaliana*: What is the signaling machinery for jasmonate perception and signal transduction? Which genes participate directly in herbivore defenses in *Arabidopsis*? How general are the roles of fatty acid-derived signals such as jasmonates in pathogenesis? In addition, the growing numbers of *Arabidopsis* mutants in fatty acid metabolism and signaling make this plant especially attractive for ecological studies. The genome sequencing project will surely impact this field of investigation in *Arabidopsis*.

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References

Aarts MGM, Keijzer CJ, Stiekema WJ, Pereira A (1995) Molecular characterization of the CER1 gene of *Arabidopsis* involved in epicuticular wax biosynthesis and pollen fertility. Plant Cell 7: 2115–2127

Alborn HT, Turlings TCJ, Jones TH, Stenhagen G, Loughrin JH, Tumlinson JH (1997) An elicitor of plant volatiles from beet armyworm secretion. Science 276: 945–949

Avdiushko S, Croft KPC, Brown GC, Jackson DM, Hamilton-Kemp TR, Hildebrand D (1995) Effect of volatile methyl jasmonate on the oxylipin pathway in tobacco, cucumber, and *Arabidopsis*. Plant Physiol 109: 1227–1230

Bell E, Mullet JE (1993) Characterization of an *Arabidopsis* lipoxygenase gene responsive to methyl jasmonate and wounding. Plant Physiol 103: 1133–1137

Bell E, Creelman RA, Mullet JE (1995) A chloroplast lipoxygenase is required for wound-induced jasmonic acid accumulation in *Arabidopsis*. Proc Natl Acad Sci USA 92: 8675–8679

Benedetti CE, Xie D, Turner JG (1995) *COI1*-dependent expression of an *Arabidopsis* vegetative storage protein in flowers and siliques and in response to coronatine or methyl jasmonate. Plant Physiol 109: 567–572

Berger S, Bell E, Mullet JE (1996) Two methyl jasmonate-insensitive mutants show altered expression of *AtVsp* in response to methyl jasmonate and wounding. Plant Physiol 111: 525–531

Bevan M, Bancroft I, Bent E, Love K, Goodman H, Dean C, Bergkamp R, Dirkse W, Van Staveren M, Stiekema W, Drost L, Ridley P, Hudson SA, Patel K, Murphy G, Piffanelli P, Wedler H, Wedler A, Wambutt R, Weitzenegger T, Pohl TM, Terryn N, Gielen J, Villarroel R, De Clerck R, Van Montagu M, Lecharny A, Auborg S, Gy I, Kreis M, Lao N, Kavanagh T, Hempel S, Kotter P, Entian KD, Rieger M, Schaeffer M, Funk B, Mueller-Auer S, Silvey M, James R, Montfort A, Pons A, Puigdomenech P, Douka A, Vouketalou E, Milioni D, Hatzopoulos P, Piravandi E, Obermaier B, Hilbert H, Dürsterhöft A, Moores T, Jones JDG, Eneva T, Palme K, Benes V, Rechman S, Ansorge W, Cooke R, Berger C, Delseny M, Voet M, Volckaert G, Mewes HW, Klosterman S, Schueller C, Chalwatzis N (1998) Analysis of 1.9 Mb of contiguous sequence from chromosome 4 of *Arabidopsis thaliana*. The EU Arabidopsis Genome Project. Nature 391: 485–488

Bowling SA, Clarke JD, Liu Y, Klessig DF, Dong X (1997) The *cpr5* mutant of *Arabidopsis* expresses both NPR1-dependent and NPR1-independent resistance. Plant Cell 9: 1573–1584

Broekaert WF, Cammue BPA, De Bolle MFC, Thevissen K, De Samblanx GW, Osborn RW (1997) Antimicrobial peptides from plants. Crit Rev Plant Sci 16: 297–323

Creelman RA, Mullet JE (1997) Biosynthesis and action of jasmonates in plants. Annu Rev Plant Physiol Plant Mol Biol 48: 355–381

Doughty KJ, Kiddle GA, Pye BJ, Wallsgrove RM, Pickett JA (1995) Selective induction of glucosinolates in oilseed rape leaves by methyl jasmonate. Phytochemistry 38: 347–350

Epple P, Apel K, Bohlmann H (1995) An *Arabidopsis thaliana* thionin gene is inducible via a signal transduction pathway different from that for pathogenesis-related proteins. Plant Physiol 109: 813–820

Farmer EE (1994) Fatty acid signalling in plants and their associated microorganisms. Plant Mol Biol 26: 1423–1437

Farmer EE (1997) New fatty acid-based signals: a lesson from the plant world. Science 276: 912–913

Feys BJF, Benedetti CE, Penfold CN, Turner JG (1994) *Arabidopsis* mutants selected for resistance to the phytotoxin coronatine are male sterile, insensitive to methyl jasmonate, and resistant to a bacterial pathogen. Plant Cell 6: 751–759

Gundlach H, Zenk MH (1998) Biological activity and biosynthesis of pentacyclic oxylipins: the linoleic acid pathway. Phytochemistry 47: 527–537

Halkier BA, Du L (1997) The biosynthesis of glucosinolates. Trends Plant Sci 2: 425–431

Jenks MA, Rashotte AM, Tuttle HA, Feldmann KA (1996) Mutants in *Arabidopsis thaliana* altered in epicuticular wax and leaf morphology. Plant Physiol 110: 377–385

Karban R, Baldwin IT (1997) Induced responses to herbivory. University of Chicago Press, Chicago

Lacomme C, Roby D (1996) Molecular cloning of a sulfotransferase in *Arabidopsis thaliana* and regulation during development and in response to infection with pathogenic bacteria. Plant Mol Biol 30: 995–1008

Laudert D, Pfannschmidt U, Lottspeich F, Holländer-Czytko H, Weiler EW (1996) Cloning, molecular and functional characterization of *Arabidopsis thaliana* allene oxide synthase (CYP 74), the first enzyme of the octadecanoid pathway to jasmonates. Plant Mol Biol 31: 323–335

Mauricio R, Rausher MD (1997) Experimental manipulation of putative selective agents provides evidence for the role of natural enemies in the evolution of plant defense. Evolution 51: 1435–1444

McConn M, Browse J (1996) The critical requirement for linolenic acid is pollen development, not photosynthesis, in an *Arabidopsis* mutant. Plant Cell 8: 403–416

McConn M, Creelman RA, Bell E, Mullet JE, Browse J (1997) Jasmonate is essential for insect defense in *Arabidopsis*. Proc Natl Acad Sci USA 94: 5473–5477

Melan MA, Dong X, Endara ME, Davis KR, Ausubel FM, Peterman TK (1993) An *Arabidopsis thaliana* lipoxygenase gene can be induced by pathogens, abscisic acid, and methyl jasmonate. Plant Physiol 101: 441–450

Millar AA, Kunst L (1997) Very-long-chain fatty acid biosynthesis is controlled through the expression and specificity of the condensing enzyme. Plant J 12: 121–131

Mueller MJ (1997) Enzymes involved in jasmonic acid biosynthesis. Physiol Plant 100: 653–663

Negruk V, Yang P, Subramanian M, McNevin JP, Lemieux B (1996) Molecular cloning and characterization of the *CER2* gene of *Arabidopsis thaliana*. Plant J 9: 137–145

Nishiuchi T, Hamada T, Kodama H, Iba K (1997) Wounding changes the spatial expression pattern of the *Arabidopsis* plastid

ω-3 fatty acid desaturase gene (*FAD7*) through different signal transduction pathways. Plant Cell 9: 1701–1712

Ohlrogge J, Browse J (1995) Lipid biosynthesis. Plant Cell 7: 957–970

Penninckx IAMA, Eggermont K, Terras FRG, Thomma BPHJ, De Samblanx GW, Buchala A, Métraux J-P, Manners JM, Broekaert WF (1996) Pathogen-induced systemic activation of a plant defensin gene in *Arabidopsis* follows a salicylic acid-independent pathway. Plant Cell 8: 2309–2323

Pieterse CMJ, Van Wees SCM, Hoffland E, Van Pelt JA, Van Loon LC (1996) Systemic resistance in *Arabidopsis* induced by biocontrol bacteria is independent of salicylic acid accumulation and pathogenesis-related gene expression. Plant Cell 8: 1225–1237

Rojo E, Titarenko E, Leon J, Berger S, Vancanneyt G, Sanchez-Serrano JJ (1998) Reversible protein phosphorylation regulates jasmonic acid dependent and independent wound signal transduction pathways in *Arabidopsis thaliana*. Plant J 12: 153–165

Schaller F, Weiler EW (1997) Molecular cloning and characterization of 12-oxophytodienoate reductase, an enzyme of the octadecanoid signaling pathway from *Arabidopsis thaliana*. J Biol Chem 272: 28066–28072

Seo S, Okamoto M, Seto H, Ishizuka K, Sano H, Ohashi Y (1995) Tobacco MAP kinase: a possible mediator in wound signal transduction pathways. Science 270: 1988–1992

Note added in proof

The *COI1* gene involved in jasmonate signal transduction has been shown to encode an F-box protein (Xie et al., 1998, Science 280: 1091–1094). Jasmonate is essential for the defense of *Arabidopsis* against *Pythium mastophorum* (Vijayan et al., 1998, Proc. Natl. Acad Sci USA. 95: 7209–7214).

Staswick PE, Su W, Howell SH (1992) Methyl jasmonate inhibition of root growth and induction of a leaf protein are decreased in an *Arabidopsis thaliana* mutant. Proc Natl Acad Sci USA 89: 6837–6840

Stelmach BA, Muller A, Hennig P, Laudert D, Andert L, Weiler EW (1998) Quantitation of the octadecanoid 12-oxo-phytodienoic acid, a signalling compound in plant mechanotransduction. Phytochemistry 47: 539–546

Tiapalensuu J, Falk A, Ek B, Rask L (1997) Myrosinase-binding proteins are derived from a large wound-inducible and repetitive transcript. Eur J Biochem 243: 605–611

Titarenko E, Rojo E, Leon J, Sanchez-Serrano JJ (1997) Jasmonic acid-dependent and -independent signaling pathways control wound-induced gene activation in *Arabidopsis thaliana*. Plant Physiol 115: 817–826

Wasternack C, Parthier B (1997) Jasmonate-signalled plant gene expression. Trends Plant Sci 2: 302–307

Weber H, Vick BA, Farmer EE (1997) Dinor-oxo-phytodienoic acid: a new hexadecanoid signal in the jasmonate family. Proc Natl Acad Sci USA 94: 10473–10478

Xue J, Jorgensen M, Pihlgren U, Rask L (1995) The myrosinase gene family in *Arabidopsis thaliana*: gene organization, expression and evolution. Plant Mol Biol 27: 911–922

Zhuang H, Hamilton-Kemp TR, Andersen RA, Hildebrand DF (1996) The impact of alteration of polyunsaturated fatty acid levels on C_6-aldehyde formation of *Arabidopsis thaliana* leaves. Plant Physiol 111: 805–812