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Lipid-derived signals that discriminate wound- and pathogen-responsive isoprenoid pathways in plants: Methyl jasmonate and the fungal elicitor arachidonic acid induce different 3-hydroxy-3-methylglutaryl-coenzyme A reductase genes and antimicrobial isoprenoids in *Solanum tuberosum* L.

(lipoxygenase/oxylipins/sesquiterpenes/steroid glycoalkaloids)

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ABSTRACT **Induction of 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGR; EC 1.1.1.34) is essential for the synthesis of steroid derivatives and sesquiterpenoid phytoalexins in solanaceous plants following mechanical injury or pathogen infection. Gene-specific probes corresponding to different HMGR genes (*hmg1* and *hmg2*) were used to study HMGR expression in potato tissue following treatment with methyl jasmonate, a lipoxygenase product of linolenic acid, or arachidonic acid, an elicitor present in the lipids of the potato late blight fungus *Phytophthora infestans*. Treatment of potato discs (2.2 cm in diameter) with low concentrations (0.45–45 nmol per disc surface) of methyl jasmonate nearly doubled the wound-induced accumulation of *hmg1* transcripts and steroid-glycoalkaloid (SGA) accumulation, reduced the abundance of *hmg2* transcripts, and did not induce phytoalexins. High concentrations of methyl jasmonate (2–4.5 μmol per disc surface) suppressed *hmg1* mRNA and SGA accumulation but did not affect *hmg2* mRNA abundance or induce phytoalexins. In contrast, arachidonate treatment strongly suppressed *hmg1* and strongly induced *hmg2* mRNA in a concentration-dependent manner. There was a corresponding suppression of SGA accumulation and an induction of sesquiterpene phytoalexin accumulation by this elicitor. Lipoxygenase inhibitors reduced the wound-induced accumulation of *hmg1* transcripts and suppressed SGA levels, effects that were overcome by exogenous methyl jasmonate (45 nmol per disc surface). The results (*i*) suggest that methyl jasmonate can function as a signal for *hmg1* expression and SGA induction following wounding and (*ii*) indicate that the arachidonate- and jasmonate-response pathways are distinct in relation to HMGR gene expression and isoprenoid product accumulation. The results also are consistent with placement of the HMGR activities encoded by *hmg1* and *hmg2* within discrete steroid and sesquiterpenoid biosynthetic channels.**

Many cellular responses in eukaryotic cells appear to be under the control of lipid-based signaling pathways. In mammalian cells, lipoxygenase-mediated oxygenation of arachidonic acid and related polyunsaturated fatty acids leads to leukotrienes and a variety of products with potent physiological activities (1). In higher plants, a class of oxygenated fatty acid metabolites (oxylipins; ref. 2) that have been shown to effect responses in part through transcriptional activation of genes are the jasmonates (2–4). Jasmonic acid (JA) and its methyl ester (MJ) are lipoxygenase products of linolenic acid that are present in most if not all plant species (5–7). Low concentrations of JA and/or MJ occur in many plant organs and can increase dramatically following wounding (8). JA and MJ induce proteinase inhibitors (3, 9), soybean vegetative storage proteins (4, 10), including lipoxygenase(s) (11), anthocyanins, and the expression of a number of wound-inducible genes (12). MJ appears to participate in the induction of alkaloid phytoalexins in cell suspension cultures treated with a fungal elicitor (13, 14) and also modulates the expression of some wound-inducible genes in soybean hypocotyls (8). It has been suggested that jasmonates may function as part of a signal-transduction system involved in regulation of plant defense responses to insects and pathogens (9).

However, it is also apparent that plants can perceive and respond to signals generated during wound and pathological stresses differently and that many of the biochemical barriers that are formed during wound healing are different from those generated during the expression of resistance to potential pathogens (15). For example, in solanaceous plants there is a rapid redirection of isoprenoid biosynthetic pathways from antimicrobial steroid derivatives toward sesquiterpenoid phytoalexins when wounded tissues are exposed to elicitors or isolates of pathogens that induce a hypersensitive response (15, 16). The changes in the levels of these compounds are correlated with changes in the activities of 3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMGR; refs. 17 and 18) and of subsequent enzymes in the pathways leading to their synthesis (19, 20). HMGR catalyzes the first step specific to isoprenoid biosynthesis by converting hydroxymethylglutaryl-coenzyme A to mevalonic acid (21). These changes have been shown to coincide with the induction or suppression of the levels of specific mRNAs corresponding to different HMGR genes (22). The cellular signals that control the expression of these and other genes that encode enzymes involved in wound- and pathogen-responsive isoprenoid pathways are poorly understood. Arachidonic acid (AA), a fatty acid present in the lipids of plant-pathogenic Oomycete fungi, is a potent elicitor of sesquiterpenoid phytoalexins (23) and suppressor of steroid-glycoalkaloids (SGAs) (18), whose activity appears to involve lipoxygenase (24, 25), possibly through the generation of a specific oxygenated metabolite of AA (26).

In this study, gene-specific probes from cloned potato HMGR cDNAs were used to investigate the differential responses of HMGR genes to MJ and AA and the relationship

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Abbreviations: AA, arachidonic acid; ASA, acetylsalicylic acid; HMGR, 3-hydroxy-3-methylglutaryl-coenzyme A reductase; JA, jasmonic acid; MJ, methyl jasmonate; SA, salicylic acid; SGA, steroid-glycoalkaloid; SHAM, salicylhydroxamic acid; nPG, *n*-propyl gallate.

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of their expression to SGA and sesquiterpenoid phytoalexin accumulation. Our results indicate that the plant discriminates the fatty acid signals—one an endogenous wound hormone and the other a fungal elicitor—and manifests this ability at the level of expression of different members of the HMGR gene family and at the level of biosynthesis of different isoprenoid products.

MATERIALS AND METHODS

Chemicals and Plant Materials. Certified seed-grade potatoes (*Solanum tuberosum* L. cv. Kennebec) were stored at 4°C until 24 hr before use. Discs (22 mm × 5 mm) were prepared and AA (Sigma) or MJ (a mixture containing 90.6% 1*R*,2*R* and 8.1% 1*R*,2*S*; Bedoukian Research, Danbury, CT) was applied to the upper surfaces (23, 24). MJ was suspended in water by brief sonication and diluted to the appropriate concentration with water. Because of MJ's volatility and the ability of airborne MJ to induce responses in plants (27), all Petri dishes containing potato discs treated with MJ were sealed separately with parafilm. Controls were similarly sealed. All experiments were conducted at 20°C.

The following compounds also were tested for their effect on plant responses or for their impact on MJ activity: salicylic acid (SA), acetylsalicylic acid (ASA), *p*-aminosalicylic acid, isonicotinic acid, salicylhydroxamic acid (SHAM), and *n*-propyl gallate (nPG). Discs were treated immediately by immersion in a solution of the test compound for 30 min with shaking. The lipoxygenase inhibitors SHAM and nPG (Sigma) were prepared at 5 mM in 50 mM Mes buffer, pH 5.8 (SHAM), or water adjusted to pH 5.8 with NaOH (nPG). SA and ASA (Sigma) were prepared in water (10 mM, pH 6.8). Five to 50 nmol of aqueous isonicotinic acid (Ciba–Geigy) was applied to the upper surface of each disc 24 hr prior to inoculation with *Phytophthora infestans*. An agar disc (3 mm in diameter) from the margin of a 2-week-old culture of *P. infestans* race 1234 was placed on each potato disc 24 hr after treatment of the potato discs with chemicals. The lipoxygenase inhibitors were applied as described above prior to AA or MJ treatment.

Preparation of Probes. DNA probes were prepared from potato HMGR cDNAs as described (22). A probe corresponding to a region that is highly conserved among HMGR genes was prepared by *Sca* I/*Nco* I digestion of the *hmg3* cDNA insert. Gene-specific probes were prepared by PCR using oligonucleotide primers based on the determined cDNA sequences (5′ region of *hmg1* and 3′ regions of *hmg2* and *hmg3*). Each cDNA was amplified in 100 μl under mineral oil with 10 mM Tris·HCl (pH 8.3 at 25°C), 50 mM KCl, 1.5 mM $MgCl_2$, 0.01% gelatin, 20 μg of bovine serum albumin, 0.2 mM each dNTP, 1 μM each specific primer, and 2.5 units of *Taq* polymerase (Perkin–Elmer/Cetus). The PCR products were separated in 1.0% low-gelling-temperature agarose (FMC) in 90 mM Tris/90 mM boric acid/2 mM EDTA. Target fragments were excised from gels and purified with the Gene Clean kit (Bio 101). Radioactive probes prepared with [α-^{32}P]CTP were prepared from gel-purified DNA fragments by the random priming method (United States Biochemical).

RNA Isolation and Gel Blot Analyses. Total RNA was isolated from the top millimeter (≈0.3 g of tissue fresh weight per disc) obtained from several potato discs 24 hr after treatment and analyzed as described (22). RNA samples (20 μg per lane) were fractionated in 1.0% agarose gels containing formaldehyde, and the separated RNAs were transferred to Nytran membranes (Amersham). Hybridization of cDNA probes to RNA blots routinely was carried out at a criterion of 50% formamide/1 M Na^+ at 42°C and washed at the same stringency by standard procedures. The amount of labeled probe hybridized to each RNA sample was estimated with a two-dimensional radioisotope-imaging system (AMBIS Systems).

Analysis of SGAs and Sesquiterpene Phytoalexins. SGAs were extracted from potato discs by a modification of the procedure of Allen and Kuć (28). Each sample was obtained from 3 g of tissue from the top 1 mm of tuber discs 96 hr after treatment. Samples were homogenized in 10 ml of chloroform/acetic acid/methanol (50:5:45, by volume) and further processed (29). Total SGAs were determined by a spectrophotometric assay (30). The absorbance of the samples at 600 nm was measured and values for SGA were calculated based on a standard curve of α-solanine (Sigma).

The sesquiterpenoid phytoalexins rishitin and lubimin were quantified by gas/liquid chromatography (31). Values reported for SGAs and sesquiterpenoids were derived from three (SGAs) or nine (sesquiterpenoids) samples from three separate experiments.

RESULTS

Differential Expression of HMGR Genes in Potato Discs After MJ Treatment. In a previous study (22), we determined that the maximum transcript levels corresponding to different HMGR genes were detected 24 hr after wounding or treatment with the fungal elicitor AA. Therefore, HMGR mRNA levels were monitored at this time point in the experiments described here. RNA samples that were hybridized with a conserved-region HMGR probe yielded a single transcript size of ≈2.5 kb that was strongly induced following treatment of potato discs with high concentrations of MJ (2.2 or 4.5 μmol per disc surface; Fig. 1). The high level of total HMGR mRNA represented by these transcripts was more than the sum of the signals corresponding to transcripts for *hmg1* and *hmg2* (Fig. 1) and for *hmg3*, which was also monitored in this study (data not shown). The HMGR DNA probes have similar size and specific activities as the conserved-region probe. These results suggest the presence of a fourth HMGR gene that is strongly induced by high MJ concentrations.

The wound-induced transcript levels of *hmg1* were enhanced by treatment of the tissue with a low concentration of

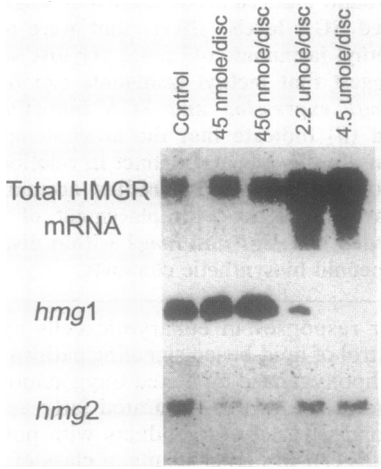

FIG. 1. Differential expression of HMGR genes following treatment of potato discs with MJ. The upper surfaces of discs were treated with various concentrations of MJ and total RNA was extracted from the top millimeter of the discs 24 hr after treatment. RNA samples on gel blots were hybridized with a conserved-region HMGR DNA probe (total HMGR mRNA), an *hmg1*-specific DNA probe, or an *hmg2*-specific DNA probe as indicated. Concentrations of MJ applied per disc surface are indicated above each lane. The increases due to MJ treatment in the *hmg1* mRNA abundance were 79% (450 nmol per disc) and 14% (45 nmol per disc) above that for the untreated control.

MJ (450 pmol per disc surface; Fig. 2). The increase in *hmg1* mRNA following MJ treatments was concentration dependent, and high concentrations of MJ (≥2.2 μmol per disc surface) completely abolished *hmg1* mRNA accumulation (Fig. 1). The level of *hmg1* mRNA almost doubled following treatment of the discs with 450 pmol to 45 nmol of MJ, and enhancement was detected with as low as 45 pmol per disc surface. In contrast to the levels of total HMGR transcripts, the abundance of *hmg1* mRNA corresponded closely with SGA accumulation (Fig. 2). SGA levels were enhanced optimally with 4 nmol of MJ per disc surface, with a significant increase even with 4.5 pmol. The expression patterns for *hmg2* and *hmg3* mRNA levels appeared to be unrelated to SGA accumulation following treatment of the discs with MJ (Figs. 1 and 2), although the *hmg2* mRNA levels were partially suppressed by the optimal concentrations of MJ. The *hmg3* mRNA abundance was strongly enhanced by high concentrations of MJ which suppressed SGA accumulation (data not shown).

Effects of Lipoxygenase Inhibitors on HMGR Gene Expression and SGA Accumulation. Because of the effect of exogenous MJ on *hmg1* mRNA and SGA accumulation, we tested the hypothesis that endogenous jasmonates are responsible for wound induction of these responses. SHAM and nPG were used to inhibit lipoxygenase activity (24, 25), which is required for the biosynthesis of JA in plants (2, 32). The inhibitors were somewhat different in their effects on HMGR gene expression. SHAM did not significantly affect the wound induction of total HMGR or *hmg2* mRNA, whereas nPG generally suppressed total HMGR and *hmg2* transcript levels. Wound-induced *hmg1* mRNA levels were reduced to 38% and 15% of the control levels by SHAM and nPG treatment, respectively, with corresponding reductions in the accumulation of SGA (Figs. 3 and 4). The reduction of *hmg1* mRNA and SGA accumulation by these inhibitors was completely overcome by treatment of the tissue with MJ at 45 nmol per disc surface (Fig. 4). In contrast, and unlike earlier studies wherein SHAM partially suppressed AA-induced sesquiterpenoid phytoalexin accumulation and other responses (24, 25), this inhibitor did not suppress the AA effect on the mRNA levels of *hmg2* (Fig. 3) and *hmg3* (data not shown). These results show again that levels of *hmg1* mRNA are closely correlated with SGA accumulation and that the levels of *hmg2* and *hmg3* mRNAs are unrelated to SGA levels in potato.

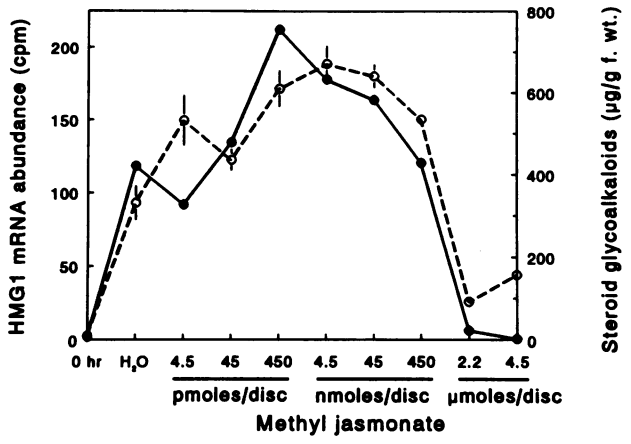

FIG. 2. Effects of various MJ concentrations on *hmg1* mRNA and SGA accumulation. *hmg1* mRNA (●) was determined 24 hr after treatment of the discs (representative data from two experiments) and SGA accumulation (○) was determined 96 hr after treatment. Values for SGA are means ± SD from three samples. f. wt., Fresh weight.

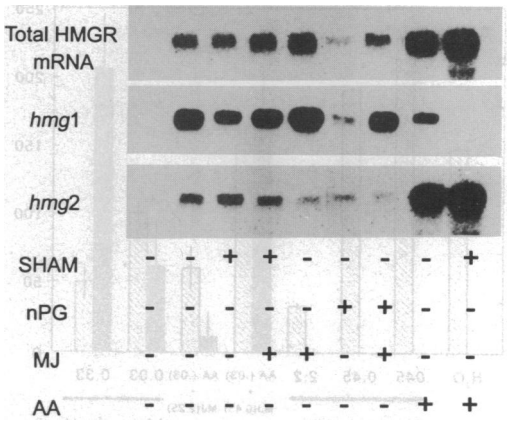

FIG. 3. RNA gel blot of wound-induced HMGR gene expression in the presence or absence of the lipoxygenase inhibitors SHAM and nPG. The results are presented for the effects on total HMGR, *hmg1*, and *hmg2* gene expression of SHAM (5 mM), nPG (5 mM), MJ (45 nmol per disc surface), and AA (0.17 μmol per disc surface) applied in various combinations as indicated below each lane. RNA was extracted from tuber discs 24 hr after treatments. DNA probes were those described in Fig. 1. The first lane contained RNA from discs extracted immediately after preparation (0 hr).

Enhancement of AA Induction of Sesquiterpenoid Phytoalexin Accumulation by MJ. Treatment of potato discs with various concentrations of MJ did not induce sesquiterpenoid phytoalexin accumulation (Fig. 5*A*), unlike an optimal concentration AA, which inhibited SGA accumulation and induced sesquiterpenoid phytoalexins, consistent with previous studies (Fig. 5*A*; refs. 16 and 23). When AA and MJ were applied together at certain concentrations, however, both SGA and sesquiterpenoid phytoalexins accumulated in the tissue. MJ at 450 nmol per disc surface enhanced the AA-induced accumulation of sesquiterpenoid phytoalexins almost 3-fold (Fig. 5*A*). No other concentration of MJ tested enhanced the AA-induced phytoalexin accumulation (Fig. 5*A* and data not shown), and a high concentration of MJ (2.2 μmol per disc surface) inhibited the AA-induced accumulation of phytoalexins. The concentrations of AA used for these experiments were selected on the basis of the concentration–response curve for AA-induced phytoalexin accumulation (23).

When RNA samples from these treatments were hybridized with the different HMGR cDNA probes, a high level of *hmg1* mRNA was detected at a concentration of AA that was

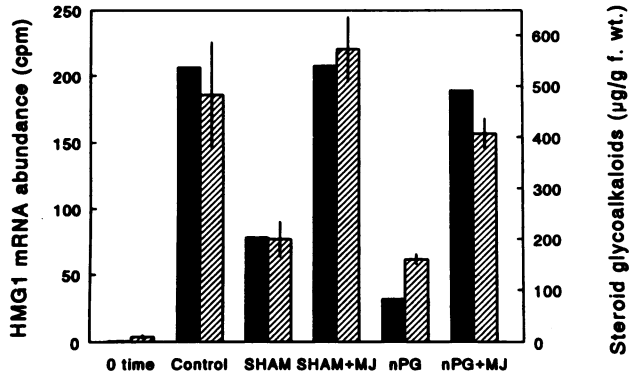

FIG. 4. Effects of lipoxygenase inhibitors (SHAM or nPG at 5 mM) in the presence or absence of MJ (45 nmol per disc surface) on the accumulation of *hmg1* mRNA (solid bars) and SGA (hatched bars). *hmg1* mRNA was determined 24 hr after treatment of the discs (representative data from two experiments), and SGA accumulation was determined 96 hr after treatment. SGA values are the means ± SD from 3 experiments.

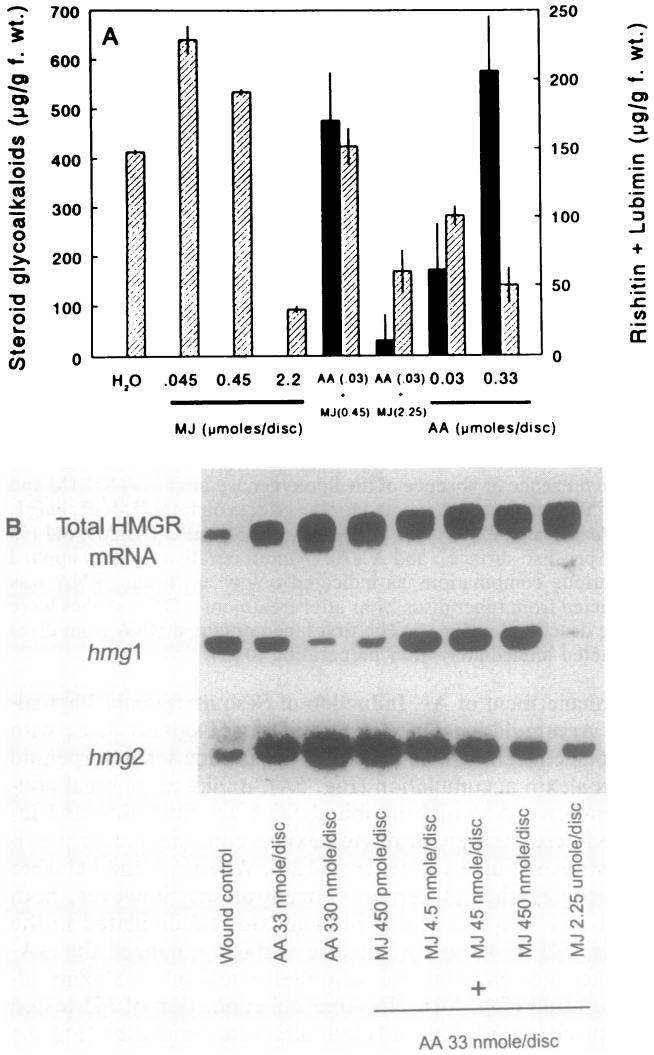

FIG. 5. (*A*) Effects of AA, MJ, or treatment with both compounds on the accumulation of sesquiterpenoid phytoalexins (rishitin and lubimin; solid bars) and SGA (hatched bars) in potato discs 96 hr after treatment. Means and SDs are indicated. (*B*) RNA gel blot illustrating the interaction between AA and MJ on wound-induced HMGR mRNA accumulation. RNA was extracted from tuber discs 24 hr after treatments. DNA probes were those described in Fig. 1. Concentrations of AA and MJ are indicated below each lane.

otherwise inhibitory when applied without MJ, and transcripts for *hmg2* were detected at or near their elicitor-induced levels (Fig. 5*B*). MJ at 450 pmol per disc surface enhanced the AA effect on both *hmg1* and *hmg2* (i.e., MJ enhanced the suppression of *hmg1* and induction of *hmg2* mRNA levels by the elicitor). However, we did not detect a corresponding effect on phytoalexin accumulation at these concentrations.

Specificity of the AA and MJ Effects on HMGR Gene Expression. To further examine the specificity of the response of HMGR genes, several compounds known to induce disease resistance and to activate defense-related genes in plants were tested for their effects on potato *hmg1* expression. SA and ASA induce pathogenesis-related proteins in plants, and SA appears to function as a signal for systemic acquired resistance in some species (33). SA and its derivatives were tested for elicitor activity at 1, 5, and 10 mM, concentrations that induce resistance in other plant species (34, 35). In potato discs, both SA and ASA at 10 mM inhibited wound-induction of *hmg1* expression by ≈95% and suppressed SGA accumulation by >70% relative to the control. SA and ASA did not affect the expression of *hmg2* and *hmg3* and did not induce sesquiterpenoid phytoalexins at any concentration tested.

Effects of Treatments on Tuber Resistance to *P. infestans*. Pretreatment of the potato discs with solutions containing SA, ASA, or *p*-aminosalicylic acid up to 10 mM or MJ at 40 nmol to 4 μmol per disc surface did not protect the tissue from infection and colonization by a compatible race of *P. infestans* (data not shown). Isonicotinic acid (35) was also tested, at 5–50 nmol per disc surface, and did not induce resistance to *P. infestans*. AA at 0.17 μmol per disc surface protected tissues from colonization as previously reported (24).

DISCUSSION

Our results indicate that the effects of MJ and AA on HMGR gene expression and isoprenoid biosynthesis are clearly distinct. AA induces symptoms and biochemical responses similar to those observed during the hypersensitive response to an incompatible race of *P. infestans*. In contrast, MJ appears to function as a signal to evoke or enhance certain wound-responsive programs in potato and, at the optimal concentrations, does not induce the browning and other symptoms characteristic of elicitor-treated tissues.

Although SA, ASA, and high concentrations of MJ suppressed *hmg1* mRNA and SGA levels similarly to AA treatment, these compounds did not induce *hmg2*, sesquiterpenoid phytoalexins, or resistance to *P. infestans*. These results are consistent with earlier studies which indicate that the phytoalexin response in potato is tightly controlled and that the plant perceives specific elements within the structure of AA, resulting in the biochemical responses that are also observed during the hypersensitive response to incompatible races of *P. infestans* (23, 24, 36). These data also indicate that suppression of *hmg1* mRNA alone is insufficient to result in rechanneling of the pathway to phytoalexins and, in fact, may be unnecessary since high levels of *hmg1* mRNA, phytoalexins, and SGA accumulate when tissues are treated simultaneously with certain concentrations of MJ and AA. These results also strengthen the case that the HMGR activity encoded by *hmg2* may be deployed within a channel for the biosynthesis of the sesquiterpenoid phytoalexins.

These results and those of an earlier study (22) are consistent with a model that functionally compartmentalizes the individual HMGR isoforms in the plant and suggest a high degree of control over the biosynthesis of specific pathway products at the level of differentially regulated HMGR genes. The close correspondence between *hmg1* mRNA levels and SGA accumulation, as well as the parallel responses to different MJ concentrations and other chemicals, strongly suggests that *hmg1* encodes a reductase that provides mevalonate within a biosynthetic channel leading to sterols and SGA.

JA and MJ are potent inducers of proteinase inhibitors in tomato and potato (3, 27). Because proteinase inhibitors also are induced by several other types of compounds, such as oligogalacturonides, the peptide systemin, and the fungal elicitor chitosan, a model was suggested that JA is an intermediate involved in the activation of defense responses to insects and pathogens (9). The notion that jasmonates are involved in plant defense against pathogen attack was also extended by Gundlach *et al.* (14), who demonstrated that JA and MJ accumulated in plant cell suspension cultures following treatment with a fungal elicitor. Furthermore, MJ alone could function as an elicitor and induced phenylalanine ammonia lyase and phenolic alkaloid accumulation. However, in these systems, the tissues treated with MJ were not subsequently challenged with a pathogen to determine whether, indeed, there was an enhanced level of resistance.

A role for jasmonates as general inducers of resistance to plant pathogens is not borne out by our studies, since for the

potato responses examined, MJ alone affected only wound-inducible *hmg1* gene expression and SGA accumulation and did not induce phytoalexin accumulation or resistance to *P. infestans*. However, it is possible that jasmonates at certain concentrations can interact with microbial or other signals produced during pathogenesis to optimize cellular responses associated with defense, as suggested by the AA/MJ interaction in our study (Fig. 5). The "aging" effect in which a period of physiological conditioning of wounded tissues optimizes their responses to elicitors and other agents is well documented for potato (36) and other species (37). Perhaps jasmonates function as endogenous signals that participate in this physiological conditioning or sensitization of injured plant tissue.

We attempted to determine jasmonate levels in potato discs under our experimental conditions but found that they were below the threshold of our detection method (≈10 pmol). Hence, we were unable to resolve any treatment effects on JA or MJ levels, and alternative methods are needed for these analyses. Nonetheless, the ability of exogenous MJ to overcome the inhibition caused by lipoxygenase inhibitors supports a role for endogenous jasmonates in the responses examined. A similar approach was used to implicate the involvement of jasmonates in the regulation of soybean vegetative storage protein genes (38) and proteinase inhibitors in tomato (39). The availability of plant mutants that are blocked in their synthesis of, or are altered in their sensitivity to, jasmonates should be useful for establishing the role of these oxylipins in wound- and defense-related responses (40).

ASA, SA, and related hydroxybenzoic acids have been shown to inhibit wound responses, including induction of proteinase inhibitors in tomato plants (41). Subsequently, it was reported that SA was increased systemically after inoculation of tobacco and cucumber plants with various pathogens and induced systemic resistance, perhaps, in part, through its elicitation of pathogenesis-related proteins (33, 35). Wounding does not induce most of the pathogenesis-related proteins in tobacco and does not induce systemic resistance (33, 35). In the present study, SA and ASA inhibited wound-inducible *hmg1* gene expression and SGA accumulation. Hence, there are similarities in the effects of the hydroxybenzoates on tobacco and potato with the effects of AA on potato; specifically, SA and its analogs suppress certain wound responses. However, unlike AA, SA does not induce phytoalexin accumulation or resistance to *P. infestans* in potato, a result that indicates that the signal-transduction pathways connecting AA or SA treatment and the responses in this species are different.

In conclusion, our results indicate that two stress-responsive isoprenoid pathways are regulated in part by different lipid-derived signals that can be present in certain plant/pathogen interactions. Although the present study emphasizes regulation at the level of HMGR expression, our results raise the possibility of regulatory control by MJ and AA of other steps in the pathway, such as squalene synthetase and sesquiterpene cyclase (19, 20). Our studies are consistent with the hypothesis that MJ is an endogenous signal that participates in the regulation of wound-response programs and further demonstrate that the consequences of wounding and certain specific elicitor treatments are different at the level of gene expression associated with secondary metabolism. Collectively, these results and those of other studies reveal that plants discriminate wound and pathogen signals and indicate that the responses observed during hypersensitivity expression in potato are not simply an enhancement of wound responses, but rather an induction of different cellular programs. Isolation of genomic sequences for the various potato HMGRs and analysis of their 5′ and 3′ flanking sequences may reveal elements that confer responsiveness to AA or MJ. Such studies will be informative for elucidating the network of factors that regulate wound- and elicitor-response programs in plants.

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