Arachidonic Acid Induces Local but not Systemic Synthesis of Salicylic Acid and Confers Systemic Resistance in Potato Plants to *Phytophthora infestans* and *Alternaria solani*

J.-L. Coquoz, A. J. Buchala, Ph. Meuwly, and J.-P. Métraux

Institut de Biologie végétale, 3 rue Albert-Gockel, Université de Fribourg, CH-1700 Fribourg, Switzerland.

We thank H.-R. Hohl (Institute of Plant Biology, University of Zürich) and M. C. Wybrecht (CIBA, Basle, Switzerland) who provided the pathogens; E. Mösinger (Sandoz, Basle, Switzerland) for the tomato PR1 antibody; G. Collet (Swiss Federal Agronomy Station, Changins) for supplying the potato cultivars; and L. Sticher and M. Schneider for critically reading the manuscript. The support of the Swiss National Science Foundation (Grant NF 31-34098.92) is gratefully acknowledged.

Accepted for publication 2 May 1995.

ABSTRACT

Coquoz, J.-L., Buchala, A. J., Meuwly, Ph., and Métraux, J.-P. 1995. Arachidonic acid induces local but not systemic synthesis of salicylic acid and confers systemic resistance in potato plants to *Phytophthora infestans* and *Alternaria solani*. Phytopathology 85:1219-1224.

When the lower leaves of young potato plants (cv. Bintje) were treated with an aqueous suspension of the polyunsaturated fatty acid arachidonic acid (AA), a threefold increase in the amount of free salicylic acid (SA) in the treated leaves was observed 3 h after treatment. The highest level of free SA was measured 6 h after treatment, reaching 3.21 g/g fresh weight compared to 0.15 g/g fresh weight in controls. The levels of conjugated SA in the treated leaves increased up to 51 g/g fresh weight 3 days after treatment compared to 0.83 g/g fresh weight in controls. Four days after treatment with AA the upper leaves became resistant to the potato late blight pathogen, *Phytophthora infestans*, but no accumulation of SA was observed. This systemic protection was observed in plants treated with AA at concentrations as low as 10 ppm and is unlikely to have arisen from AA translocated in the plant. In addition, treatment with AA protected potato plants against the early blight pathogen, *Alternaria solani*, indicating a broader spectrum of protection. Local but not systemic accumulation of a pathogenesis-related (PR)1-like protein was observed 24 h after treatment with AA. Application of SA on potato leaves did not confer resistance against *P. infestans*, but a local accumulation of a PR1-like protein was observed 24 h after treatment. Younger leaves of healthy susceptible and resistant potato plants contained higher levels of SA than older leaves, and this gradient correlated with their natural resistance to *P. infestans*. In general, cultivars showing field resistance contained higher amounts of conjugated SA than did susceptible cultivars.

Additional keywords: local protection, pathogenesis-related protein, *Solanum tuberosum*, systemic acquired resistance.

Infection of potato tubers with an avirulent race of the late blight pathogen, *Phytophthora infestans* (Mont.) de Bary, induces a hypersensitive response (34). A similar response is obtained after treatment of potato tuber slices with arachidonic acid (AA), a major fatty acid in the mycelial cell membrane and spores of the pathogen. AA presumably acts as a natural fungal elicitor, because synthesis of sesquiterpene phytoalexins takes place and tuber slices can no longer be colonized by compatible races of *P. infestans* (2). Potato leaves also respond to local inoculation with virulent races of *P. infestans* by resisting a second challenge-inoculation (systemic) with a virulent race of *P. infestans* (30), but unlike tubers, no sesquiterpenoid phytoalexins are produced (25). Resistance has been obtained when potato leaves have been treated with a cell-wall preparation from *P. infestans* (8) known to contain AA and with the related fatty acid eicosapentaenoic acid (2). More recently, a detailed study has shown that when the lower leaves of potato plants are treated with the fatty acids AA or eicosapentaenoic acid, the upper leaves resist infection by *P. infestans* (6).

In cucumber (17), tobacco (26), *Arabidopsis* (31,32), tomato (13, 16) and potato (30), inoculation of the lower leaves with necrotizing pathogens can induce resistance against pathogens in the upper noninfected leaves, a phenomenon called systemic acquired resistance (SAR). SAR is associated with the production of salicylic acid (SA) (19,20,38), which is thought to be an indispensable component along the signaling pathway leading to SAR (11). Our present aim is to determine if SAR in potato induced by treatment with AA operates via endogenous accumulation of SA.

Corresponding author: J.-P. Métraux
E-mail address: jean-pierre.metraux@unifr.ch

© 1995 The American Phytopathological Society

MATERIALS AND METHODS

Plant material. Potato (*Solanum tuberosum* L.) plants were propagated in vitro from clones obtained from the Swiss Federal Agronomy Station (RAC, Changins, Nyon, Switzerland). The following cultivars, which have no known R genes, were studied: Aula, Bintje, Désirée, Hermes, Panda, Matilda, Sirtema, and Urgenta. Stem segments (1 cm) were initially cultivated on MS agar placed in a growth chamber under a 16/8 h day/night period. The temperature was 19 and 15°C during the day and night periods, respectively. Plants were transferred after 10 days to pots containing garden soil and grown for 3 to 4 weeks in a greenhouse (temperature approximately 25 and 18°C during day and night, respectively). In winter, normal daylight was supplemented with a high-pressure sodium lamp (Philips T 400 W, Philips Lighting, Tumhout, Belgium) and a high-pressure mercury lamp (Philips HPL-N 400 W) to give a minimum irradiation of 70 μmol s^{-1} m^{-2} for 9 h.

Treatment, pathogen, and infection of plants. The lower surface of leaf 3 was sprayed with a sonicated suspension of AA (1,500 ppm) as described by Cohen et al. (6). Control plants were sprayed with deionized water. Lower concentrations (1 and 10 ppm) of AA were applied by injecting (approximately 500 μl) leaf 3 at

three sites, thus filling the leaf with AA suspension. The numbering of leaves was from bottom to top, and by convention, leaf 5 was the first to show developed leaflets. Control plants were injected with deionized water. SA was applied as an aqueous solution (2 mM, pH 6.5) by spraying leaf 3.

P. infestans (isolate 191, race 1,2,3; mating-type A_1) was cultivated in the dark at 18°C and 100% relative humidity (RH) on V8 agar supplemented with 0.005% sitosterol and 0.005% cholesterol (29). *Alternaria solani* Sorauer (Ciba-Geigy, Saint-Aubin, Switzerland) was cultivated on potato carrot agar in the dark at 22°C and 100% RH. A period of 48 h under continuous light was necessary to induce sporulation.

For inoculation with *P. infestans*, potato leaves were sprayed with a suspension (5×10^4 ml^{-1}) of sporangia in a potassium citrate solution (0.01%) 4 days after treatment with AA, unless stated otherwise in the text. Inoculated plants were placed in a moist chamber (100% RH) at 18°C in the dark for 25 h and kept in the same chamber with a normal light regime at 18°C and approximately 80% RH. For inoculation with *A. solani*, potato leaves were sprayed with a suspension (5×10^4 ml^{-1}) of spores in distilled water 4 days after treatment. Inoculated plants were kept for 3 days in a moist chamber (100% RH) at 22°C with reduced light and then transferred to a greenhouse as described above. The extent of the symptoms was determined by measuring the area of the necrotic lesions 7 days after inoculation. Protection against the pathogen was calculated as follows: percent protection = $100 \times (1 - x/y)$, where x and y are the sizes of lesions in treated and control plants, respectively.

Fungal germination test in vitro and fungal growth test. Freshly produced sporangia of *P. infestans* were suspended (1.7×10^4 ml^{-1}) in potassium citrate solutions (0.01%) containing various concentrations (0 to 2 mM) of SA. The suspension was kept for 3 h at 4°C to induce the release of zoospores and then placed in a chamber at 18°C in the dark. The percentages of empty sporangia after release of zoospores and of sporangia germinating with a germ tube were recorded 24 h later.

The effect of SA on growth of *P. infestans* was tested in vitro on solid culture medium. Different concentrations of SA were incorporated in V8 medium in petri dishes. These plates were inoculated with a 5-mm agar plug containing the mycelium and incubated at 18°C in the dark. Colony diameter was measured 3 and 7 days after inoculation.

Quantification of SA. Free and conjugated SA were quantified essentially as described by Meuwly and Métraux (21) with the following modifications. Three hundred nanograms of the internal standard *ortho*-anisic (*o*-anisic) acid was added per sample. Prior to high-pressure liquid chromatography analysis, samples were resuspended in 400 µl (for free SA) and 800 µl (for conjugated SA) of the starting mobile phase composed of KH_2PO_4 (10 mM, pH 3.5) and 17.5% (vol/vol) acetonitrile. Samples (40 µl for free SA and 30 µl for conjugated SA) were applied on a modified version of the deactivated reversed-phase LC-ABZ column (LC-SAL column, Supelco, Buchs, Switzerland) used previously (21). The column was maintained at 35°C and equilibrated with a mixture composed of KH_2PO_4 (10 mM, pH 3.5) and 17.5% (vol/vol) acetonitrile. Elution was programmed at 2 ml min^{-1} beginning with 17.5% acetonitrile for 1 min, raised successively to 19% in 2 min, 20% in 7 min, 75% in 5.5 min, and maintained isocratic for 5.5 min before washing and reequilibrating periods at 100 and 17.5%, respectively. Conditions for fluorescence detection were optimized for each compound as described previously (21). Under these conditions, *o*-anisic acid and SA eluted at 7.5 and 18.0 min, respectively. Experiments were repeated at least three times and gave the same trends, although the absolute values varied from

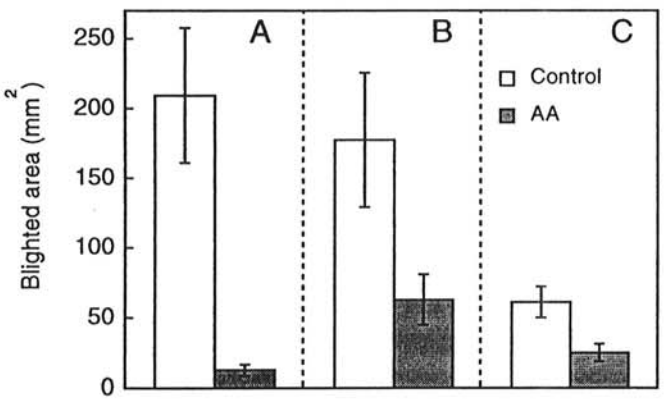

Fig. 1. Systemic protection of potato plants (cv. Bintje) against **A and B,** *Phytophthora infestans* and **C,** *Alternaria solani* by foliar spray application of arachidonic acid (AA) (1,500 ppm, **A and C**) or injection (10 ppm, **B**). Challenge-inoculations were performed 4 (**A and C**) and 6 days (**B**) after treatment with AA. Blighted areas (in square millimeters) were measured 7 days after challenge-inoculation. Means (±SE) of five replicates.

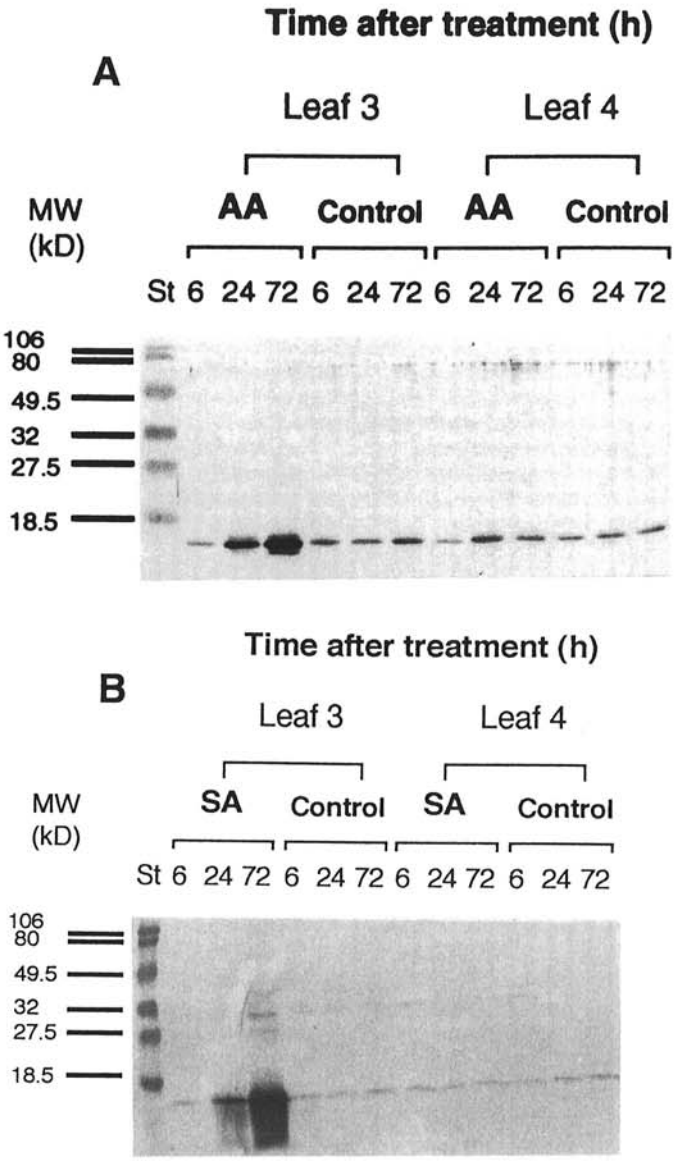

Fig. 2. Time course of accumulation of pathogenesis-related (PR)1-like protein in leaves 3 and 4 of potato plants (cv. Bintje) after spraying leaf 3 with **A,** 1,500 ppm of arachidonic acid (AA) or **B,** 2 mM salicylic acid (SA). Acid-soluble proteins from leaf 3 (treated) and leaf 4 (untreated) were collected 6, 24, and 72 h after treatment and separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis. PR1-like proteins were detected by immunoblotting using an antiserum against the major PR1 protein of tomato (P14) (9).

experiment to experiment. The results shown are those for a typical series of plants, in which three individual plants were analyzed to obtain mean values.

Pathogenesis-related (PR)1 protein measurement. Sodium dodecyl sulfate-polyacrylamide gel electrophoresis was carried out as described by Christ and Mösinger (5) with the acid-soluble proteins derived from material collected 6, 24, and 72 h after spraying leaf 3 with AA (1,500 ppm). Western immunoblotting was performed using an antiserum against the major basic PR1 protein of tomato (P14) (10).

RESULTS

Systemic protection. Spraying leaf 3 of 3-week-old potato plants (cv. Bintje) with 1,500 ppm of AA conferred high resistance (about 90%) to *P. infestans* 4 days later in the immediate upper leaf 4 (Fig. 1A). Injecting 10 ppm of AA in leaf 3 protected leaf 4 against *P. infestans* 6 days after treatment (Fig. 1B). Necrotic areas at the three injection sites were observed 5 days after application of AA. Protection (about 60%) of leaf 4 against the early blight pathogen, *A. solani*, also was observed after spraying leaf 3 with 1,500 ppm of AA (Fig. 1C).

Local protection resulting from spraying leaves with 1,500 ppm of AA was not assessed due to the fact that the leaf rapidly withered, produced necrotic lesions, and abscission occurred about 3 days later. However, at 1 ppm, the highest concentration at which there were no necrotic lesions, no local protection was observed (data not shown). The accumulation of a PR1-like protein (14 kDa) was induced locally in potato leaves 24 h after spraying with 1,500 ppm of AA (Fig. 2). No such accumulation beyond the constitutive level was detected in the immediate upper leaf (leaf 4).

The role of salicylic acid in SAR. The level of free SA in leaves sprayed with AA increased as soon as 3 h after treatment, reaching a maximum value 6 h after treatment with AA (Fig. 3A), whereas increases in conjugated SA were only observed after 24 h (Fig. 3B). This accumulation of the conjugated form was preceded by a drastic decrease of free SA. Surprisingly, no significant increase (Table 1) in SA in either the free or the conjugated form was found in leaf 4 that expressed SAR (Fig. 1A). Similarly, injecting lower concentrations of AA (10 ppm) into leaf 3 resulted in an increase, although less pronounced, in conjugated SA that was evident 7 days after treatment (Table 2), whereas no changes compared to the controls were detected in leaf 4. Nevertheless, leaf 4 expressed resistance against *P. infestans* (Fig. 1B).

Previously, a primary infection of potato leaves with *P. infestans* induced SAR after 5 days against a subsequent infection with the same pathogen (6,30). Increases in free and conjugated SA, lower than those produced after treatment with AA, were detected only in the lower leaf 7 days after primary infection with *P. infestans* (Table 3); no significant effect was observed in the upper leaves. No difference in the levels of SA in leaf 4 after challenge-inoculation with *P. infestans* was observed in plants elicited with AA (10 ppm) on leaf 3. Levels were 0.64 ± 0.11 and 0.40 ± 0.10 g/g fresh weight of free SA and 1.10 ± 0.12 and 0.92 ± 0.11 g/g fresh weight of conjugated SA in control and challenge-inoculated leaves, respectively, 7 days after challenge-inoculation.

The older leaves of potato plants (cv. Bintje) are more susceptible to *P. infestans* (6), and protection induced by a primary infection with *P. infestans* is stronger in young, developing leaves than in fully expanded, older leaves (30). We confirmed these findings and extended them to show the existence of an endogenous gradient of SA (i.e., the younger the leaf, the higher the level of SA) in eight potato cultivars (Fig. 4). High amounts of SA were found in cultivars with high field resistance (28), whereas low amounts occurred in susceptible cultivars. The SA content was more variable for the moderately resistant cultivars (Fig. 4).

Figure 5 compares the SA content in leaves of a susceptible (Bintje) and a resistant cultivar (Matilda) and shows that such a

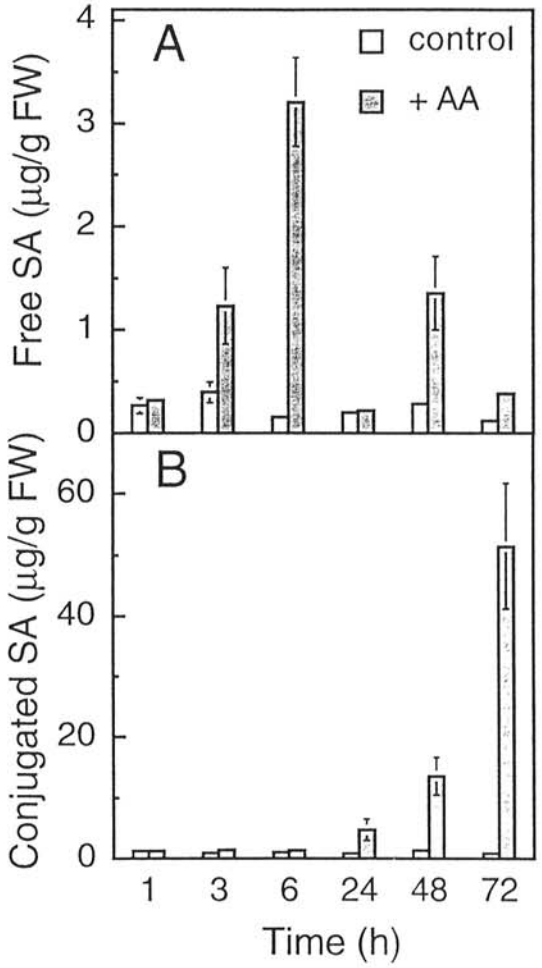

Fig. 3. Levels of **A,** free and **B,** conjugated salicylic acid (SA) in control (open columns) and arachidonic acid (AA)-treated (shaded columns) potato plants (cv. Bintje). Leaf 3 was sprayed with water (control) or 1,500 ppm of AA. SA was measured in the treated leaf. Means (±SE) of three replicates.

TABLE 1. Levels of free and conjugated salicylic acid (SA) (µg/g FW) in leaf 4 of potato plants after spraying leaf 3 with water (control) or 1,500 ppm of arachidonic acid (AA)[a]

Treatment	Time after AA treatment (h)					
SA	1	3	6	24	48	72
AA						
Free	0.42 ± 0.03	0.40 ± 0.07	0.22 ± 0.11	0.13 ± 0.03	0.53 ± 0.21	0.22 ± 0.10
Conjugated	1.02 ± 0.33	1.66 ± 0.43	1.05 ± 0.05	0.71 ± 0.10	1.83 ± 0.39	0.92 ± 0.02
Control						
Free	0.48 ± 0.01	0.45 ± 0.05	0.23 ± 0.07	0.18 ± 0.01	0.54 ± 0.23	0.11 ± 0.02
Conjugated	1.37 ± 0.38	1.34 ± 0.03	1.42 ± 0.21	1.02 ± 0.23	1.48 ± 0.01	1.29 ± 0.16

[a] Means (±SE) of three replicates.

correlation also holds for resistance to infection under optimized laboratory conditions. Such resistance is probably not due to a direct effect of SA on germination or growth of the pathogen since SA only partially inhibited the release of zoospores of the fungus in vitro at 2 mM, whereas the germination of sporangia was not significantly altered in the concentration range of 0 to 2 mM (data not shown).

Foliar application of SA (2 mM) on leaf 3 of susceptible potato plants (cv. Bintje) did not induce SAR against *P. infestans* in leaf 4 (data not shown) but led to the accumulation of a PR1-like protein in treated leaves 24 h after treatment (Fig. 2). Mycelial growth in vitro of *P. infestans* was not affected when the culture medium was amended with SA. Colony diameters were 98.4 and 100.5% of the control when grown in medium containing 1 and 10 mM of SA, respectively).

DISCUSSION

SAR typically is expressed against a wide range of pathogens, independently of the microorganism or chemical inducer used for the primary induction (5,18). In potato, treatment with AA leads to systemic protection not only against *P. infestans* (6) (Fig. 1A and B), an oomycete fungus, but also against the causal agent of early blight, the ascomycete *A. solani* (Fig. 1C), indicating that protection also can operate against organisms other than oomycete fungi. We were not able to separate the induction of SAR from the local necrotic side effects induced by AA, not even when using low amounts of the elicitor. Necrotizing lesions or necrosis-inducing treatments seem to be a common feature in all reported cases of SAR (15,18,22,30).

It is important to show whether AA acts as a true elicitor of SAR or whether AA is mobile in the plant. No acropetal transport of a radiolabeled compound within the plant was detected after injecting radiolabeled AA into leaf 3 (97% of the radiolabel remained in the zone infiltrated). Also, radiolabeled AA released from spores of *P. infestans* inoculated onto potato leaves has limited systemic movement within inoculation sites during the early phases of infection (24). Thus, applied as a foliar spray, it is highly unlikely that AA (6) or one of its metabolites moves from the spraying site. From these results, we infer that systemic resistance, in all likelihood, relies on the induction of host plant defense mechanisms. The dependence on necrotizing treatments, the putative broad spectrum of resistance, and the likely dependence on host plant defense mechanisms all suggest that potato exhibits SAR.

TABLE 2. Levels of free and conjugated salicylic acid (SA) (μg/g FW) in leaves 3 and 4 of potato plants after injecting leaf 3 with water (control) or 10 ppm of arachidonic acid (AA)[a]

Leaf	Treatment SA	Time after AA treatment 6 h	4 days	7 days
3	AA			
	Free	1.37 ± 0.19	0.76 ± 0.22	0.51 ± 0.04
	Conjugated	5.16 ± 0.70	6.66 ± 0.68	8.56 ± 0.74
	Control			
	Free	1.37 ± 0.41	0.47 ± 0.08	0.56 ± 0.19
	Conjugated	4.04 ± 0.24	3.36 ± 0.75	4.07 ± 0.86
4	AA			
	Free	0.32 ± 0.1	0.46 ± 0.08	0.29 ± 0.04
	Conjugated	3.34 ± 0.76	1.99 ± 0.08	1.68 ± 0.31
	Control			
	Free	0.75 ± 0.1	0.35 ± 0.13	0.21 ± 0.03
	Conjugated	3.78 ± 0.13	3.83 ± 0.37	2.63 ± 0.17

[a] Means (±SE) of three replicates.

TABLE 3. Levels of free and conjugated salicylic acid (SA) (μg/g FW) in leaves 3 and 4 of potato plants after spraying leaf 3 with water (control) or with a sporangia suspension of *Phytophthora infestans*[a]

Leaf	Treatment SA	Time after infection (day) 1	3	7
3	*P. infestans*			
	Free	0.10 ± 0.01	0.26 ± 0.04	6.91 ± 3.03
	Conjugate	1.73 ± 0.24	2.26 ± 0.36	9.65 ± 1.17
	Control			
	Free	0.12 ± 0.02	0.11 ± 0.01	0.08 ± 0.01
	Conjugate	1.39 ± 0.21	1.76 ± 0.12	1.75 ± 0.26
4	*P. infestans*			
	Free	0.17 ± 0.01	0.16 ± 0.04	0.17 ± 0.02
	Conjugate	2.62 ± 0.53	2.67 ± 0.44	2.74 ± 0.19
	Control			
	Free	0.12 ± 0.03	0.15 ± 0.08	0.15 ± 0.04
	Conjugate	2.61 ± 0.48	2.73 ± 0.29	2.76 ± 0.76

[a] Means (±SE) of three replicates.

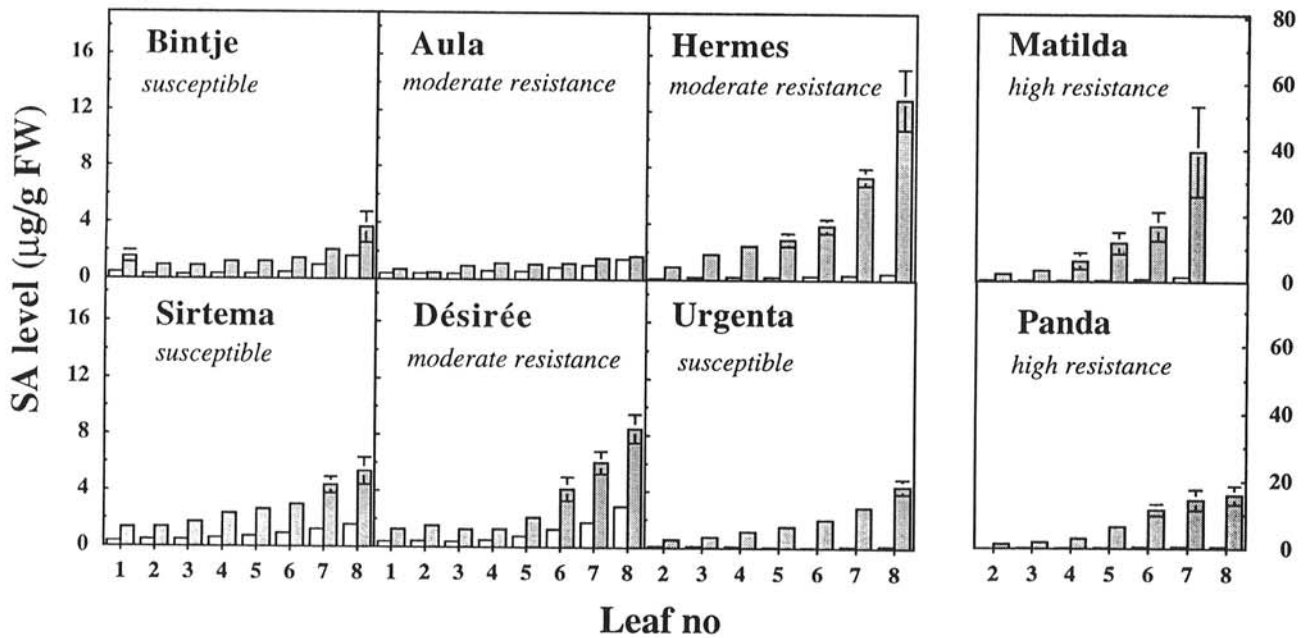

Fig. 4. Endogenous levels of free (open columns) and conjugated (shaded columns) salicylic acid (SA) in leaves 1–8 or 2–8 of 4-wk-old potato plants from various potato cultivars. Means (±SE) of three replicates. Field resistance to *Phytophthora infestans* is indicated.

It has been proposed that SA is a signal molecule necessary for SAR in various plant-pathogen interactions (4,19,20,23,38). In all of the current cases, SA increased both locally and systemically before the appearance of resistance. On the other hand, Vernooij et al. (36) were able to show that in tobacco SA is not the primary translocated signal responsible for inducing SAR. Our data concerning treatment with AA or infection with *P. infestans* do not show any evidence for increases of SA in the upper leaf before (Table 1) or after challenge-inoculation.

In tomato and tobacco, SAR correlates with systemic accumulation of PR1 protein (5,10,37). The slight increase in PR1 protein observed in leaves treated with AA might result from an increase in SA concentration (Fig. 2), since it could be shown that treating the leaves with SA also led to the accumulation of PR1 protein. A similar effect was reported for potato leaves treated with acetylsalicylic acid (aspirin), where PR1 protein was identified on the basis of its molecular mass (12). Due to the fact that local resistance could not be tested, the role of PR1 protein in such a phenomenon remains elusive. However, overexpression of PR1a, a closely related PR protein from tobacco, provides protection of tobacco against oomycetes (1).

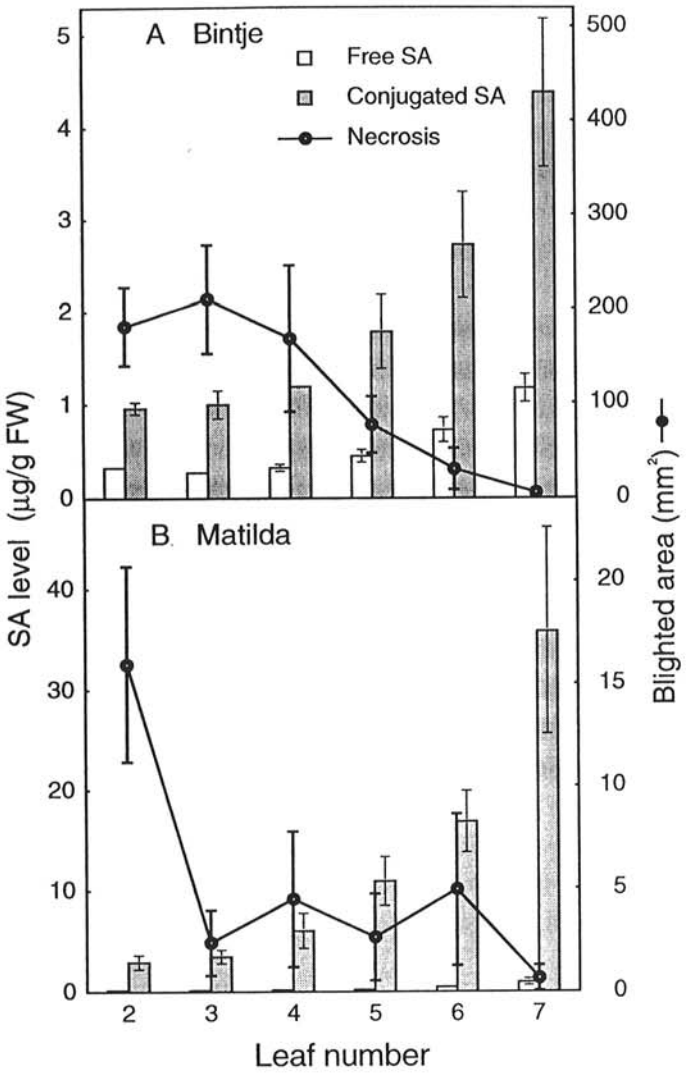

Fig. 5. Levels of free (open columns) and conjugated (shaded columns) salicylic acid (SA) in leaves of healthy potato plants: **A,** cv. Bintje; **B,** cv. Matilda, and necrotic lesions caused by a sporangia suspension of *Phytophthora infestans* (5×10^4 ml^{-1}) applied to leaves. Leaves 2–7 from 4-wk-old potato plants were analyzed for SA. Blighted areas (in square millimeters) were measured 7 days after challenge-inoculation. Means (±SE) of three replicates.

At the systemic level, no changes were found in the levels of PR1 and in SA content. It seems unlikely that PR1 protein is involved in the protection of upper leaves. Thus, potato does not compare to other plant species, such as cucumber (17,22) and tobacco (19,38), in which a primary infection leads to systemic accumulation of SA and PR proteins. Perhaps movement of preexisting SA between intracellular compartments would allow the induction of resistance mechanisms but would escape our present analysis.

It cannot be excluded that SA is involved in the induction of resistance at the local level as suggested by the correlation between the endogenous levels of SA and field resistance to *P. infestans* among various potato cultivars (28). A similar correlation also was observed for the interaction between rice and *Magnaporthe grisea* (27). The potato cultivars examined here have no known major R genes, so any resistance is likely to be at a polygenic level (33). The high level of conjugated inactive SA in the more resistant potato cultivars could represent a pool rapidly converted into free active SA after pathogen infection. Such a role for conjugated SA has already been proposed in tobacco (14). Free SA might inactivate catalase, leading to an increase in active oxygen species that could participate in the induction of resistance responses, as suggested recently for tobacco (4). However, it should be noted that neither foliar treatment of the potato plants with SA nor treatment of tomato plants with aspirin (5) gave rise to SAR. Again one may speculate that the SA could not attain the appropriate cell compartment to trigger SAR. The correlation between SA levels and resistance to *P. infestans* will be the focus of future studies using transgenic potato plants in which the endogenous levels of SA are experimentally decreased by overexpression of salicylate hydroxylase (11).

It has been shown that AA elicits the activity of a lipoxygenase (LOX) in potato callus culture (35) and in potato tubers (3). This could provide a possible link with the synthesis of jasmonic acid via LOX-induced lipid peroxidation products. Jasmonic acid and its octadecanoid precursors have been proposed to participate in a lipid-based intracellular signaling pathway in response to wounding and pathogen attack (9). Recently, Cohen et al. (7) showed that treatment of potato and tomato plants with jasmonic acid led to both local and systemic protection against *P. infestans*. A possible connection between AA and jasmonate signaling in potato plants also will be investigated.

LITERATURE CITED

1. Alexander, D., Goodman, R. M., Gut-Rella, M., Glascock, C., Weymann, K., Friedrich, L., Maddox, D., Ahl-Goy, P., Luntz, T., Ward, E., and Ryals, J. 1993. Increased tolerance to two oomycete pathogens in transgenic tobacco expressing pathogenesis-related protein 1a. Proc. Natl. Acad. Sci. USA 90:7327-7331.
2. Bostock, R. M., Laine, R. A., and Kuć, J. 1981. Eicosapentaenoic and arachidonic acids from *Phytophthora infestans* elicit fungitoxic sesquiterpenes in the potato. Science 212:67-69.
3. Bostock, R. M., Yamamoto, H., Choi, D., Ricker, K. E., and Ward, B. L. 1992. Rapid stimulation of 5-lipoxygenase activity in potato by the fungal elicitor arachidonic acid. Plant Physiol. 100:1448-1456.
4. Chen, Z., Silva, H., and Klessig, D. F. 1993. Active oxygen species in the induction of plant systemic acquired resistance by salicylic acid. Science 272:1883-1886.
5. Christ, U., and Mösinger, E. 1989. Pathogenesis-related proteins in tomato: I. Induction by *Phytophthora infestans* and other biotic inducers and correlations with resistance. Physiol. Mol. Plant Pathol. 35:53-65.
6. Cohen, Y., Gisi, U., and Mösinger, E. 1991. Systemic resistance of potato plants against *Phytophthora infestans* induced by unsaturated fatty acids. Physiol. Mol. Plant Pathol. 38:255-273.
7. Cohen, Y., Gisi, U., and Niderman, T. 1993. Local and systemic protection against *Phytophthora infestans* induced in potato and tomato plants by jasmonic acid and jasmonic acid methyl ester. Phytopathology 83: 1054-1062.
8. Doke, N., Ramirez, A. V., and Tomiyama, K. 1987. Systemic induction of resistance in potato plants against *Phytophthora infestans* by local treatment with hyphal wall components of the fungus. J. Phytopathol. 119:

232-239.
9. Farmer, E. E., and Ryan, C. A. 1992. Octadecanoid precursors of jasmonic acid activate the synthesis of wound-inducible proteinase inhibitors. Plant Cell 4:129-134.
10. Fischer, W., Christ, U., Baumgartner, M., Erismann, K. H., and Mösinger, E. 1989. Pathogenesis-related proteins in tomato: II. Biochemical and immunological characterization. Physiol. Mol. Plant Pathol. 35:67-83.
11. Gaffney, T., Friedrich, L., Vernooij, B., Negrotto, D., Nye, G., Uknes, S., Ward, E., Kessmann, H., and Ryals, J., 1993. Requirement of salicylic acid for the induction of systemic acquired resistance. Science 271:754-756.
12. Hammond-Kosack, K. E., Atkinson, H. J., and Bowles, D. J. 1989. Systemic accumulation of novel proteins in the apoplast of the leaves of potato plants following root invasion by the cyst-nematode *Globodera rostochiensis*. Physiol. Mol. Plant Pathol. 35:495-506.
13. Heller, W. E., and Gessler, C. 1986. Induced systemic resistance in tomato plants against *Phytophthora infestans*. J. Phytopathol. 116:323-329.
14. Henning, J., Malamy, J., Grynkiewicz, G., and Klessig, D. F. 1993. Interconversion of the salicylic acid signal and its glucoside in tobacco. Plant J. 4:593-600.
15. Irving, H. R., and Kuć, J. A. 1990. Local and systemic induction of peroxidase, chitinase and resistance in cucumber plants by KH_2PO_4. Physiol. Mol. Plant Pathol. 37:355-366.
16. Kovats, K., Binder, A., and Hohl, H. R. 1991. Cytology of induced resistance of tomato to *Phytophthora infestans*. Planta 183:491-496.
17. Kuć, J., Shockley, G., and Kearney, K. 1975. Protection of cucumber against *Colletotrichum lagenarium* by *Colletotrichum lagenarium*. Physiol. Plant Pathol. 7:195-199.
18. Madamanchi, N. R., and Kuć, J. 1991. Induced systemic resistance in plants. Pages 422-432 in: The Fungal Spore and Disease Initiation in Plants and Animals. G. T. Cole and H. C. Hoch, eds. Plenum Press, New York.
19. Malamy, J., Carr, J. P., Klessig, D. F., and Raskin, I. 1990. Salicylic acid: A likely endogenous signal in the resistance response of tobacco to viral infection. Science 250:1002-1004.
20. Métraux, J.-P., Signer, H., Ryals, J., Ward, E., Benz-Wyss, M., Gaudin, J., Raschdorf, K., Schmid, E., Blum, W., and Inverardi, B. 1990. Increase in salicylic acid at the onset of systemic acquired resistance in cucumber. Science 250:1004-1006.
21. Meuwly, P., and Métraux, J.-P. 1993. *Ortho*-anisic acid as internal standard for the simultaneous quantitation of salicylic acid and its putative precursors in cucumber leaves. Anal. Biochem. 214:500-505.
22. Meuwly, P., Sticher, L., Mölders, W., Summermatter, K., Coquoz, J.-L., Buchala, A. J., and Métraux, J.-P. 1993. Salicylic acid and systemic acquired resistance in cucumber and potato. Pages 54-64 in: Plant Signals in Interactions with other Organisms. J. Schulz and I. Raskin, eds. American Society of Plant Physiologists, Rockville, MD.
23. Rasmussen, J. B., Hammerschmidt, R., and Zook, M. N. 1991. Systemic induction of salicylic acid accumulation in cucumber after inoculation with *Pseudomonas syringae*. Plant Physiol. 97:1342-1347.
24. Ricker, K. E., and Bostock, R. M. 1992. Evidence for release of the elicitor arachidonic acid and its metabolites from sporangia of *Phytophthora infestans* during infection of potato. Physiol. Mol. Plant Pathol. 41:61-72.
25. Rohwer, F., Fritzemeier, K. H., Scheel, D., and Hahlbrock, K. 1987. Biochemical reactions of different tissues of potato (*Solanum tuberosum*) to zoospores or elicitors from *Phytophthora infestans*. Planta 170:556-561.
26. Ross, A. F. 1961. Systemic acquired resistance induced by localized virus infection in plants. Virology 14:329-339.
27. Silverman, P., Seskar, M., Kanter, D., Schweizer, P., Métraux, J.-P., and Raskin, I. 1995. Salicylic acid in rice: Biosynthesis, conjugation, and role in disease resistance. Plant Physiol. 108:633-639.
28. Stegemann, E., and Schnick, D. 1985. Index 1985 of European potato varieties. Mitt. Biol. Bundesanst. Land Forstwirtsch. Berl.-Dahlem 227:1-128.
29. Stössel, P., and Hohl, H. R. 1980. Sterols in *Phytophthora infestans* and their role in the parasitic interactions with *Solanum tuberosum*. Ber. Schweiz. Bot. Ges. 90:118-128.
30. Strömberg, A., and Brishammar, S. 1991. Induction of systemic resistance in potato (*Solanum tuberosum* L.) plants to late blight by local treatment with *Phytophthora infestans* (Mont.) de Bary, *Phytophthora cryptogea* Pethyb. & Laff., or dipotassium phosphate. Potato Res. 34:219-225.
31. Summermatter, K., Meuwly, P., Mölders, W., and Métraux, J.-P. 1994. Salicylic acid levels in *Arabidopsis thaliana* after treatments with *Pseudomonas syringae* or synthetic inducers. Acta Hortic. 381:367-370.
32. Uknes, S., Mauch-Mani, B., Moyer, M., Potter, S., Williams, S., Dincher, S., Chandler, D., Slusarenko, A., Ward, E., and Ryals, J. 1992. Acquired resistance in Arabidopsis. Plant Cell 4:645-656.
33. Umaerus, V., Umaerus, M., Erjefält, L., and Nilsson, B. A. 1983. Control of *Phytophthora* by host resistance: Problems and progress. Pages 315-326 in: *Phytophthora* Its Biology, Taxonomy, Ecology, and Pathology. D. C. Erwin, S. Bartnicki-Garcia, and P. H. Tsao, eds. The American Phytopathological Society, St. Paul, MN.
34. Varns, J. L., Kuć, J., and Williams, E. B. 1971. Terpenoid accumulation as a biochemical response of the potato tuber to *Phytophthora infestans*. Phytopathology 61:174-177.
35. Vaughn, S. F., and Lulai, E. C. 1992. Further evidence that lipoxygenase activity is required for arachidonic acid-elicited hypersensitivity in potato callus cultures. Plant Sci. 84:91-98.
36. Vernooij, B., Friedrich, L., Morse, A., Reist, R., Kolditz-Jawhar, R., Ward, E., Uknes, S., Kessmann, H., and Ryals, J. 1994. Salicylic acid is not the translocated signal responsible for inducing systemic acquired resistance but is required in signal transduction. Plant Cell 6:959-965.
37. Ward, E. R., Uknes, S. J., Williams, S. C., Dincher, S. S., Wiederhold, D. L., Alexander, D. C., Ahl-Goy, P., Métraux, J.-P., and Ryals, J. A. 1991. Coordinate gene activity in response to agents that induce systemic acquired resistance. Plant Cell 3:1085:1094.
38. Yalpani, N., Silverman, P., Wilson, T. M. A., Kleier, D. A., and Raskin, I. 1991. Salicylic acid is a systemic signal and an inducer of pathogenesis-related proteins in virus-infected tobacco. Plant Cell 3:809-818.