Accumulation of Furanoterpenoids in Sweet Potato Tissue Following Inoculation with Different Pathogens

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ABSTRACT

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Accumulation of 4-ipomeanol and 1,4-ipomeadiol in sweet potato storage roots was positively correlated with accumulation of ipomeamarone and total furanoterpenoids. *Streptomyces ipomoea*, *Monilochaetes infuscans*, and internal cork virus did not induce accumulation of detectable levels of ipomeamarone, 4-ipomeanol, or 1,4-ipomeadiol. *Rhizopus stolonifer* and *Erwinia carotovora* induced accumulation of relatively low concentrations of ipomeamarone (58–2,675 μg/g), 4-ipomeanol (from not detectable [ND] to 112 μg/g), and 1,4-ipomeadiol (ND–16 μg/g). *Plenodomus destruens*, *Diaporthe batatatis*, *Diplodia tubericola*, *Fusarium solani*, and *Ceratocystis fimbriata* induced accumulation of relatively high concentrations of ipomeamarone (63–16,523 μg/g), 4-ipomeanol (5–236 μg/g) and 1,4-ipomeadiol (ND–1,406 μg/g). *Macrophomina phaseoli* and *Sclerotium rolfsii* induced accumulation of relatively high concentrations of ipomeamarone (ND–23,346 μg/g) and 4-ipomeanol (4–227 μg/g) but did not induce accumulation of 1,4-ipomeadiol. Mercuric acetate induced accumulation of low concentrations of total furanoterpenoids, ipomeamarone, 1-ipomeanol, 1,4-ipomeadiol. *Fusarium oxysporum* f. sp. *batatas* did not induce accumulation of furanoterpenoids in sweet potato vines. Concentrations of 4-ipomeanol and 1,4-ipomeadiol were highest in tissue infected with certain isolates of *D. tubericola* and *F. solani*. The ipomeanols accumulate in tissue infected by any of several sweet potato pathogens in conjunction with a general furanoterpenoid accumulation and are not specific degradation products of *E. solani* infection.

Additional key words: Hepatotoxin, *Ipomoea batatas*, lung edema toxin, phytoalexin.

The phytoalexin ipomeamarone has been studied extensively since it was discovered in 1943 (15). The biosynthetic pathway has been elucidated by Uritani and co-workers (1,23,29), and several similar 15-carbon furanoterpenoids occur in decayed sweet potato tissue (4–6,23,34). Based on differential sensitivity to and induction of ipomeamarone by strains of *Ceratocystis fimbriata* Ell. and Halst., it has been suggested that ipomeamarone may play a role in determining host specificity (17,19,20). Certain sweet potato pathogens (internal cork virus, *Streptomyces ipomoea* Person and W. J. Martin) Waks., and Henrici, *Monilochaetes infuscans*Ell. and Halst. ex Harter, and *Meloidogyne incognita* (Kofoid and White) Chitwood do not induce appreciable concentrations of ipomeamarone (less than 100 μg/g); *Rhizopus stolonifer* (Ehr. ex Fr.) Lind. induces low concentrations of ipomeamarone (100–1,000 μg/g); and *Fusarium oxysporum* Schlecht., *Sclerotium rolfsii* Sacc., *Diplodia tubericola* (Ell. and Ev.) Taub., *Macrophomina phaseoli* (Maubl.) Ashby, *Plenodomus destruens* Harter, and *C. fimbriata* induce high concentrations (more than 1,000 μg/g) of ipomeamarone (21).

Sweet potato tissue infected with *Fusarium solani* contains, in addition to the 15-carbon furanoterpenoids, a group of 9-carbon furanoterpenoids: 1-ipomeanol, 4-ipomeanol, 1,4-ipomeadiol, and ipomeanine (3,33). Furthermore, *F. solani* converts the 15-carbon furanoterpenoid, 4-hydroxymyoporone, to form all four 9-carbon furanoterpenoids in vitro but *C. fimbriata* does not (7). Several phytoalexins are metabolized by either the host (16,25) or the pathogen (2,11,13,14,26,31), and concentrations of ipomeanols are somewhat higher during late stages of infection (12). Although ipomeamarone disappears from culture fluid when incubated with *S. rolfsii*, the fate of the ipomeamarone has not been determined (28).

Ipomeamarone and other 15-carbon sweet potato furanoterpenoids have hepatotoxic activity in mice (32); the ipomeanols are more acutely toxic and induce lung edema (33), a symptom similar to that reported in cattle fed moldy sweet potatoes (24). To determine the relative importance of these compounds as toxins, it would be necessary to know which sweet potato pathogens cause their accumulation.

The objective of this study was to determine the ability of sweet potato pathogens to induce accumulation of the ipomeanols in relation to accumulation of ipomeamarone and total furanoterpenoids. Two subordinate questions were whether formation of ipomeanols was a specific response to *F. solani* or a general feature of furanoterpenoid accumulation and whether low ipomeamarone concentrations following inoculation with certain pathogens is due to conversion of ipomeamarone to other furanoterpenoids.

MATERIALS AND METHODS

All pathogens used were initially isolated from infected sweet potatoes. Isolates of *Erwinia carotovora* and deep-rotting isolates of *F. solani* were provided from North Carolina by J. W. Moyer and E. Echandi. The remaining isolates originated in Louisiana. Surface rotting isolates of *F. solani* differ in depth of penetration of the deep-rotting North Carolina isolates in infected sweet potatoes (10).

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Sweet potatoes (*Ipomoea batatas* (L.) Lam. 'Centennial') naturally infected with the scurf pathogen, *M. infuscans*, and of the cultivar Goldrush infected with internal cork virus were used for analysis of tissue. All other tissue was collected from artificially inoculated Centennial sweet potatoes. To reduce the chances of contamination, whole unblemished sweet potatoes were thoroughly rinsed in tap water, peeled, surface-sterilized in 0.5% sodium hypochlorite for 5 min, sliced in half longitudinally, and placed in moist chambers. Agar plugs from actively growing colonies of each of the various sweet potato pathogens were inverted and placed on the center of the slice. Sweet potatoes were incubated for varying periods at 20–24 C depending on the normal rate of development of the particular pathogen. Tissue was collected when the rate of lesion enlargement was maximal. Infected tissue was scraped from each of three slices and pooled for furanoterpenoid analysis. Terminal vine cuttings with petioles and leaves removed were inoculated by dipping in a bud cell suspension containing 10^6 cells per milliliter of *Fusarium oxysporum* f. sp. *batatas* (Wr.) Snyd. & Hans. Inoculated cuttings were planted in a sand-soil (1:1, v/v) mixture and grown for 2 wk in a greenhouse.

Furanoterpenoids were extracted by the method of Catalano et al (9). Up to 25 g of tissue was diced and comminuted in a blender for 3 min in 100 ml of methanol with 3 g of sodium chloride. The extract was filtered through a glass fiber filter (Reeve Angel 934 AH) on a Büchner funnel and the cake was washed three times with a total of 100 ml of methanol. Celite C211 Ea analytical filter aid was added to the filtrate, which was then filtered through Whatman 2-V fluted filter paper. The filtrate was partitioned three times against one-half volume of methylene chloride. The combined methylene chloride fraction was filtered through granular anhydrous sodium sulfate and evaporated to dryness on a rotary evaporator. This crude extract was redissolved in 1 ml of methylene chloride for analysis.

Ipomeamarone was assayed by the gas-liquid chromatographic (GLC) method of Catalano et al (8). An aliquot (10–25 μl) of the crude extract was injected into a Hewlett-Packard 5700-A gas chromatograph, fitted with an injection port glass liner packed with glass wool. The gas chromatograph was equipped with dual columns and flame ionization detectors, and coupled to a Hewlett-Packard 7132A recorder and a Hewlett-Packard 3373 B integrator. The column was a 6-mm-diameter stainless steel coiled tube, 88 cm long, packed with 10% UC-W98 (methyl vinyl silicone) on 177–149 μm (80- to-100-mesh) Gas-Chrom Q. Flow rates were 60 ml of nitrogen carrier gas through each column (30 ml/min air to each flame). The temperatures were 200 C at inlet, 300 C at detector, and 180 C in column oven. After 16 min, the temperature of the oven was increased to 210 C for 8 min before the next injection. The electrometer settings were: range = 1,000, attenuation = 16. Under these conditions, the retention time was 6.5 min. This method detects 85–95% of the total extractable ipomeamarone (8).

Total furanoterpenoids were estimated by an Erlich's reaction method of Hyodo et al (17). An aliquot (10–25 μl) of the crude extract was added to 2 ml of ethanol, after which 1 ml of 10% *p*-dimethylamino benzaldehyde in ethanol and 2 ml of 40% (v/v) aqueous sulfuric acid were successively added. The mixture was incubated at 30 C for 15 min and the optical density at 527 nm was measured. Ipomeamarone was used as a standard and data are thus expressed as ipomeamarone-equivalent. Because the different furanoterpenoids reacted differently with Ehrlich's reagent, the values can only be considered relative measures of total furanoterpenoid content.

Thin-layer chromatography (TLC) was used to separate 4-ipomeanol and 1,4-ipomeadiol on Whatman K5F silica gel plates in a 10% (v/v) methanol in benzene solvent system. R_f values for this system were 1-ipomeanol = 0.67, 4-ipomeanol = 0.65, 1,4-ipomeadiol = 0.42, and ipomeamarone = 0.93. The plates were sprayed with Ehrlich's reagent and heated at 30 C for 15 min, after which the appropriate zones of the plate were scraped and the chromophores were eluted with a total of 5 ml of methanol. The optical density (OD) was measured at 527 nm. Because 1-ipomeanol produces a highly unstable chromophore with Ehrlich's reagent, it could not be assayed. Standard curves for 4-ipomeanol, 1,4-ipomeadiol, and ipomeamarone were made by spotting known amounts of these compounds on TLC plates, spraying with Ehrlich's reagent, heating, eluting, and measuring OD at 527 nm. This procedure detected 60% of the 4-ipomeanol and 1,4-ipomeadiol in spiked samples. Assays for ipomeamarone agreed closely with the GLC assays.

RESULTS

The concentrations of ipomeamarone induced in sweet potato storage roots by the different sweet potato pathogens (Table 1) were similar to those reported by Martin et al (21). In addition, *E. carotovora* induced accumulation of relatively low levels of ipomeamarone, whereas *F. solani* induced relatively high levels of ipomeamarone. Concentrations of ipomeamarone induced by the various pathogens were positively correlated with estimates of total furanoterpenoids ($r = 0.53$). Concentrations of 4-ipomeanol and 1,4-ipomeadiol were positively correlated with both ipomeamarone ($r = 0.54$ and 0.40, respectively) and total furanoterpenoids ($r = 0.39$ and 0.34, respectively). Pathogens such as *S. ipomoea*, *M. infuscans*, and internal cork virus, which did not induce ipomeamarone accumulation, did not induce accumulation of 4-ipomeanol or 1,4-ipomeadiol and induced formation of total furanoterpenoid concentrations only slightly higher than in uninoculated controls.

Quantities of total furanoterpenoid were often estimated to greatly exceed quantities of ipomeamarone. Two factors that may contribute to this relationship are that as many as 12 to 15 spots positive to Ehrlich's reagent were detected on some of the TLC plates and that the degree of reaction with Ehrlich's reagent for a set concentration differed among furanoterpenoid compounds. Ipomeamarone always gave the most intense spot. However, other furanoterpenoids were also present.

Sweet potato disks treated with 50 mM mercuric acetate and incubated for 14 days at 30 C contained 1,657 μg total furanoterpenoid, 1,075 μg ipomeamarone, 3 μg 4-ipomeanol, and 5 μg 1,4-ipomeadiol per gram of tissue.

Sweet potato vines of cultivars Porto Rico 198 (susceptible), Centennial (intermediate), and Goldrush (resistant) inoculated with the wilt pathogen (*F. oxysporum* f. sp. *batatas*) and uninoculated vines of Centennial were assayed for content of total furanoterpenoids and ipomeamarone. Concentrations of total furanoterpenoid were 8, 95, 10, and 11 μg/g for inoculated Centennial, Porto Rico 198, Goldrush, and uninoculated Centennial, respectively. Ipomeamarone was not detected in any of these samples. Vine and foliar tissue have not previously been assayed for furanoterpenoid content, and thus we could not determine whether lack of furanoterpenoid formation in these vines is due to lack of induction by the pathogen or lack of response by the host.

DISCUSSION

Although other workers (7) have found that *F. solani* could and that *C. fimbriata* could not convert 4-hydroxymyoporone to ipomeanols in vitro, in this study *C. fimbriata* and other pathogens induced formation of ipomeanols in vivo. This suggests that the ipomeanols may also form as a normal result of general activation of the furanoterpenoid biosynthetic mechanism of the host or that they form as a result of degradation of ipomeamarone by the sweet potato, as demonstrated for other phytoalexins in other hosts (16,25). This is further confirmed by results of mercuric acetate induction. Clearly, the low levels of ipomeamarone induced by some pathogens cannot be accounted for by the hypothesis that ipomeamarone is degraded to other furanoterpenoids by these pathogens since the same pathogens also induced accumulation of proportionally low levels of the ipomeanols and total furanoterpenoids.

Previous studies have indicated that ipomeanine is transitory and does not accumulate in diseased tissue, and thus it has not been considered an important component of the furanoterpenoid complex (7,32). Measurements of ipomeanol concentrations by

high performance liquid chromatography (HPLC) indicate that 1-ipomeanol and 4-ipomeanol are normally present in equivalent concentrations (9). Although we were unable to quantitatively measure 1-ipomeanol concentrations in this study, intensity of the 1-ipomeanol spots on TLC plates sprayed with Ehrlich's reagent was similar to that of 4-ipomeanol. Seven of our samples were assayed by HPLC and the results confirmed our TLC-Ehrlich's assays and the observation that concentrations of 1-ipomeanol and 4-ipomeanol are relatively equivalent. Concentrations of furanoterpenoids varied considerably among experiments and among isolates of certain pathogens (*P. destruens* and *D. batatatis*). Little is known concerning variation within most sweet potato pathogen species; thus, it cannot be determined at present whether variation in furanoterpenoid accumulation is related to variation in virulence. Variation among experiments may be due to the use of sweet potatoes stored for different lengths of time after harvest.

Imaseki and Uritani (18) showed with radioactive tracers that synthesis of ipomeamarone is initiated in the healthy tissue immediately adjacent to the necrotic tissue and that it is completed in the necrotic tissue. They suggested that an intermediate "migrates" from the healthy to the necrotic tissue. On the basis of ability to induce furanoterpenoid accumulation, the pathogens used in this study can be grouped as noninducers (*S. ipomoea*, *M. infuscans*, and internal cork virus), low-level inducers (*R. stolonifer* and *E. carotovora*), and high-level inducers (*C. fimbriata*, *D. batatatis*, *D. tubericola*, *F. solani*, *M. phaseoli*, *P. destruens*, and *S. rolfsii*). These three groups also differ in the nature of the diseases

TABLE 1. Total furanoterpenoids, ipomeamarone, 4-ipomeanol, and 1,4-ipomeadiol in decayed tissue of cultivar Centennial sweet potato tissue inoculated with different isolates of sweet potato pathogens

Pathogen	Isolate	Samples analyzed (no.)	Necrotic tissue ($\mu g/g$)[a]			
			Total furanoterpenoid[b]	Ipomeamarone[c]	4-Ipomeanol[d]	1,4-Ipomeadiol[d]
None	···	3	25 (8–57)	ND	3 (ND–9)	ND
Streptomyces ipomoea	MS	2	372 (62–682)	ND	2 (ND–4)	ND
Monilochaetes infuscans	field	2	134 (ND–267)	ND	ND	ND
Internal cork virus[e]	LSU	1	1,271	58	5	ND
Erwinia carotovora	E-1	2	2,403 (1,304–3,520)	673 (213–1,133)	<1 (ND–1)	ND
Rhizopus stolonifer	M-22	1	732	58	11	ND
	77-25	2	8,314 (394–16,234)	1,363 (50–2,675)	66 (20–112)	8 (ND–16)
	field	1	400	116	1	ND
Plenodomus destruens	M-9	1	5,667	344	10	ND
	M-13	2	37,947 (25,000–50,893)	11,605 (6,686–16,523)	55 (45–65)	271 (96–446)
	M-14	3	3,482	1,327	33	ND
Diaporthe batatas	M-12	3	42,437 (881–106,429)	2,640 (492–4,141)	13 (6–24)	20 (ND–60)
	M-16	3	26,803 (10,000–52,333)	3,250 (720–7,858)	21 (18–25)	69 (ND–208)
	M-17	1	11,892	3,226	5	135
Diplodia tubericola	78-2	2	52,838 (25,676–80,000)	8,447 (6,915–9,979)	88 (19–156)	ND
	78-9	1	56,250	6,702	156	1,406
	78-13	2	21,116 (12,857–29,375)	4,064 (816–7,311)	26 (16–36)	128 (89–167)
	78-14	1	57,212	7,110	337	481
	78-18	1	3,482	1,327	33	ND
Fusarium solani (deep)	NC-M10	2	59,340 (35,498–83,182)	4,580 (1,808–7,352)	124 (28–219)	272 (170–373)
	NC-N1	2	55,379 (17,007–93,750)	8,002 (7,782–8,232)	196 (156–236)	350 (230–469)
Fusarium solani (surface)	77-29	2	13,117 (10,000–16,234)	1,220 (1,102–1,338)	59 (6–112)	8 (ND–16)
	78-32	1	5,984	1,301	18	133
	78-36	2	38,847 (14,693–55,000)	1,759 (242–3,276)	92 (83–101)	154 (ND–307)
	78-47	2	11,250 (7,500–15,000)	999 (63–1,934)	27 (21-32)	85 (ND-169)
Macrophomina phaseoli	M-10	1	26,250	6,734	30	ND
	78-54	1	67,333	4,270	94	ND
Ceratocystis fimbriata	M-11	2	35,636 (26,271–45,000)	6,724 (4,074–9,373)	24 (23–24)	121 (85–156)
Sclerotium rolfsii	M-8	2	16,490 (533–36,364)	7,069 (ND–15,184)	116 (4–227)	ND
	M-15	2	33,230 (23,404–43,056)	15,364 (7,381–23,346)	15 (3–27)	ND

[a] The top set of numbers are the means of all samples analyzed. Numbers in parentheses indicate the range of concentrations determined for each treatment. ND = none detected.

[b] Measured by reacting crude extract with Ehrlich's reagent and measuring optical density at 527 nm with ipomeamarone as a standard. Data are expressed as ipomeamarone equivalents.

[c] Determined by gas-liquid chromatographic assay of crude extracts.

[d] Determined by separation by thin-layer chromatography, elution, reaction with Ehrlich's reagent, and measuring optical density at 527 nm.

[e] Internal cork-affected tissue was of the sweet potato cultivar Goldrush.

they cause. The noninducers all cause diseases that are very slow to develop and have a sharp demarcation between healthy and infected tissue. The low-level inducers both cause soft rots that develop very rapidly. The high-level inducers all cause dry rots that develop faster than the symptoms caused by the noninducers but slower than the soft rots caused by the low-level inducers. We suggest that the differences in the nature of diseases may relate to the extent of furanoterpenoid accumulation. Thus, noninducers may, as a function of the sharp demarcation between healthy and infected tissue, fail to stimulate formation of Imaseki and Uritani's (18) intermediate, and the low-level inducers may overgrow and kill healthy tissue so rapidly that only small quantities of the intermediate are formed. The high-level inducers may create a situation in which activities in the healthy and infected tissues are in a balance that optimizes furanoterpenoid accumulation.

The toxicity of the various furanoterpenoid constituents of diseased sweet potatoes has not been extensively investigated. Ipomeamarone is more toxic to strains of *C. fimbriata* from other hosts and nonpathogens than to *C. fimbriata* from sweet potato (20,22,27,30). Although their toxicity to mammals has been studied (3,32,33), no studies have been reported on the toxicity of the ipomeanols or other furanoterpenoids to microorganisms. Although several furanoterpenoid constituents of diseased sweet potatoes have been identified (3,5,6,17,23,34), many of the minor or transitory compounds have not been quantified. To properly assess the importance of the observed levels of various sweet potato furanoterpenoids, their relative toxicities to sweet potato pathogens should be determined.

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