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Quantity and Potential Biological Activity of Caffeic Acid in Sweet Potato [*Ipomoea batatas* (L.) Lam.] Storage Root Periderm

HOWARD F. HARRISON,*,† JOSEPH K. PETERSON,† MAURICE E. SNOOK,‡ JANICE R. BOHAC,† AND D. MICHAEL JACKSON†

U.S. Vegetable Laboratory, Agricultural Research Service, U.S. Department of Agriculture, 2875 Savannah Highway, Charleston, South Carolina 29414, and Department of Chemistry, University of Georgia, P.O. Box 5677, Athens, Georgia, 30163

The caffeic acid content of storage root periderm and cortex tissues of genetically diverse sweet potato [*Ipomoea batatas* (L.) Lam.] cultivars and breeding clones was quantified by high-performance liquid chromatography. Periderm caffeic acid content of the clones ranged from 0.008 to 7.97 mg/g dry weight, whereas the highest cortex content was 0.047 mg/g. Clones varied greatly in periderm caffeic acid content in all experiments, but there were also differences between experiments in content averaged for all clones. This indicates that periderm caffeic acid content is subject to genetic and environmental influences. Caffeic acid inhibited the growth of four sweet potato pathogenic fungi and germination of proso millet seeds in bioassays. Inhibitory activity in the bioassays suggests that high periderm caffeic acid levels contribute to the storage root defense chemistry of some sweet potato genotypes.

KEYWORDS: ***Fusarium oxysporum*; *Fusarium solani*; *Lasiodiplodia theobromae*; *Rhizopus stolonifer*; *Panicum milliaceum*; *Cyperus esculentus*; phenolic acid; fungicide; pest resistance; allelopathy**

INTRODUCTION

Caffeic acid is ubiquitous in plants. As an early intermediate of phenylpropanoid metabolism, it is a precursor for structural polyphenols and many biologically active secondary compounds that are important in the defense chemistry of plants (*1*). Many biological activities have been reported for free caffeic acid. In bioassay experiments, it inhibited the growth of plants (*2*−*4*), fungi (*5*−*8*), bacteria (*9*, *10*), and insects (*11*). In several plant species, the levels of caffeic acid and other phenolics appear to be related to pest resistance (*12*−*14*) and allelopathic potential (*2*). Caffeic acid is one of many phenolics considered to be an important part of the general defense mechanism of plants against infection and predation (*15*, *16*).

Sweet potato storage roots are subject to infection from a number of fungal and bacterial pathogens, and differences between cultivars in susceptibility have been reported for several root diseases (*17*). The best-described defense system elicited by fungal infection of sweet potato involves terpene metabolism. Ipomoeamarone is the most abundant of approximately 30 furanoterpenoids that were produced by sweet potato in response to infection by pathogens (*18*−*21*). The furanoterpenoids are toxic to mammals and impart an unpleasant flavor, and sweet potatoes with high levels of these compounds are not suitable for human or livestock consumption (*21*, *22*); thus, breeding sweet potatoes for increased furanoterpenoid production may not be desirable due to their negative effect on quality.

Sweet potato phenolics were first isolated by Rudkin and Nelson (*23*) who found chlorogenic acid and related compounds. Caffeic acid, and the caffeoylquinic acid derivatives, chlorogenic and isochlorogenic acids, accumulate in wounded tissue or in response to infection by the black rot fungus, *Ceratosystis fimbriata* Ell. and Halst (*24*, *25*). Nematode resistant sweet potato clones have been reported to accumulate higher levels of phenolic compounds than nonresistant clones (*26*). Analysis of the outer 3 cm layer of sweet potato storage roots of five cultivars indicated that phenolics comprised up to 0.92% of the fresh weight of this tissue (*27*). The cultivars varied in total phenolic content and in content of each phenolic compound, and most of the phenolic component consisted of chlorogenic acid and other caffeoylquinic acids. Caffeic acid constituted less than 10% of the total phenolic content in four cultivars but was the largest component (36% of total phenolic content) in Jewel. Snook et al. (*14*) found high levels of fatty acid esters of coumaric and ferulic acids in the root and vine latex of sweet potatoes. The levels of the esters in sweet potato leaves were inversely correlated with feeding indices for the sweet potato weevil (*Cylas formicarius*) indicating that they contribute to insect resistance. Stange et al. (*28*) reported that extracts of a combination of periderm and outer cortex tissue of sweet potato

* To whom correspondence should be addressed. Tel: (843)556-7531. Fax: (843)766-7013. E-mail: howardh@awod.com.
† U.S. Department of Agriculture.
‡ University of Georgia.

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inhibited the growth of *Rhizopus stolonifer*. The most inhibitory fractions contained caffeic, chlorogenic, and dicaffeoylquinic acids. Caffeic acid was determined to be a less important inhibitor of fungus growth than the caffeoyl quinic acids. Islam et al. (*29*) recently analyzed the foliar phenolic content of a large collection of sweet potato genotypes and found contents to be from 1.42 to 17.1 g/100 g dry weight. Caffeic acid was a relatively minor component of the total leaf phenolics with contents from 7.33 to 179.65 mg/100 g dry weight.

The sweet potato periderm is considered to be a virtually impenetrable barrier for root pathogens, and most require mechanical or insect feeding injury for infection (*30*). The hydrophobic nature of this tissue provides a physical barrier to microbes, and phenolic compounds found in suberized tissues have antimicrobial properties (*15*). In previous investigations of the allelopathic properties of sweet potato, we found that the periderm tissue contained components that inhibited the growth of weeds, insects, and disease fungi (*31*−*35*). Most of the plant and insect growth inhibition by Regal sweet potato periderm extracts was attributed to several partially characterized resin glycosides (*31*, *33*). The resin glycosides are not as inhibitory to pathogenic fungi in Petri dish bioassays as other components of periderm extracts (*32*; unpublished data). Noda et al. (*36*) characterized the structure of several resin glycosides from the sweet potato cultivar Simon, which is cultivated as a health food in Japan and as a folk medicine in Brazil.

During investigations of root components that are involved in storage root defense, the periderm of some sweet potato clones was found to contain high levels of caffeic acid. The objectives of this study were to determine the caffeic acid content of storage root tissues of sweet potato clones with diverse genetic background. The activity of caffeic acid against pathogenic fungi and weed seed germination was assessed in order to determine if the relatively high levels in some clones contribute defense chemistry of sweet potato roots.

MATERIAL AND METHODS

Plant Material. Marketable-sized sweet potato storage roots utilized for caffeic acid determination were selected from replicated field germplasm evaluation experiments that were grown using standard cultural practices at the Clemson University Edisto Research and Education Center, Blackville, SC (Edisto), in 1999 or at the U.S. Vegetable Laboratory, Charleston, SC (Charleston), in 1997, 1999, and 2000. The experiments from which these roots were obtained were conducted as part of the sweet potato breeding project at the U.S. Vegetable Laboratory, and different varieties were available in the three years included in this report. Freshly dug (not cured) storage roots were washed with a soft brush to remove soil. The periderm was separated from the cortex by gently scraping with a scalpel only from undamaged areas of sweet potato storage roots. Periderm tissue was dried at 55 °C, ground to a fine powder under liquid nitrogen with a mortar and pestle, and stored at −20 °C under nitrogen until extraction. Periderm and cortex tissues were collected from sweet potatoes grown at Charleston in 2000. Cortex tissue was obtained from storage roots following careful removal of periderm and scar tissue. Roots were sliced into disks, and cortex tissue was separated from stele tissue with a knife. Cortex samples were lyophilized, ground with a mill to pass through a 0.55 mm screen, and stored at −20 °C under nitrogen until extraction.

Caffeic Acid Analysis. Ground tissue samples were weighed (approximately 200 mg) into Teflon-lined, screw-capped test tubes, 2.0 mL of methanol containing 0.08 mg of chrysin (recrystallized from amyl alcohol) as an internal standard was added, and the test tubes were ultrasonicated for 20 min in ice-cooled water. The test tubes were centrifuged, and the supernatant was filtered through Nylon-66 filters (0.45 μm) into autoinjector vials. Caffeic acid concentrations were determined by reverse phased high-performance liquid chromatography (HPLC) (model 1050, Hewlett Packard Inc., Atlanta GA) using 20 μL of the solution. A H_2O/MeOH linear gradient from 10 to 90% MeOH in 35 min was used. The column was a 250 mm × 4.6 mm i.d., 5 μm Ultrasphere C18 (Beckman Instruments Inc., Norcross, GA). The flow rate was 1.0 mL/min and detection at 340 nm. Each solvent contained 1% H_3PO_4. Purified caffeic acid was used as an external standard to determine response factor vs chrysin for quantification. The identity of caffeic acid in the sweet potato periderm extract was confirmed by spectroscopic analysis and cochromatography with authentic caffeic acid (Aldrich Chemical Co., Milwaukee, WI).

Fungus Growth Bioassays. The effect of caffeic acid on the growth of four fungi that incite field and postharvest rotting of sweet potatoes: *Fusarium oxysporum* Schlecht. f. sp. *batatas* (Wollenw.); *Fusarium solani* (Sacc.) Mart.; *Lasiodiplodia theobromae* (Pat.) Griffon and Maubl.; and *R. stolonifer* (Ehr. ex Fr.) Lind. was assessed using a previously described bioassay (*32*). Caffeic acid concentrations were 0, 0.31, 0.63, 1.25, 2.5, and 5.0 mg/mL. Authentic caffeic acid (Sigma Co., St. Louis, MO) was dissolved in warm, sterile potato dextrose agar medium (PDA) (BBL Potato Dextrose Agar, Beckton Dickinson Microbiology System, Cockeysville, MD), and 1.5 mL of medium was pipetted into 35 mm × 10 mm Petri dishes and allowed to gelate. Small, equal segments of mycelium taken from cultures grown on PDA plates were transferred to the center of each plate. Fungi were grown in a dark incubator at 25 °C for 60, 60, 40, and 18 h for *F. oxysporum*, *F. solani*, *L. theobromae*, and *R. stolonifer*, respectively, at which time the diameter of the fungal growth was measured with a caliper.

Proso Millet Seed Germination Bioassay. The effect of caffeic acid on proso millet seed germination was determined using a previously described bioassay (*33*). Caffeic acid was dissolved in methanol and added to 10 cm Petri dishes containing two filter papers, and the methanol was evaporated at room temperature. Five milliliters of distilled water and 100 seeds were added to each dish, and the dishes were incubated in the dark at 22 °C for 42.5 h after which they were frozen to halt germination. Seeds with radicles longer than the diameter of the seed were counted as germinated. Test concentrations were 0, 0.25, 0.375, 0.5, 0.75, 1.0, and 1.5 mg/mL.

Yellow Nutsedge Bioassay. A bioassay described in previous reports (*31*) was used to test the effect of caffeic acid on yellow nutsedge growth. Caffeic acid test concentrations were 0, 0.125, 0.25, 0.5, 1.0, and 2.0 mg/mL. Caffeic acid dissolved in methanol was pipetted into 5 mL vials, and the methanol was evaporated in air. Two milliliters of Hoaglunds no. 5 nutrient solution (Sigma Co.) was added to each vial and stirred with a spatula to dissolve the caffeic acid. A small yellow nutsedge shoot with all roots excised with scissors were placed in each vial and supported with a foam collar. The vials were then placed in a growth chamber at 25 °C under constant lighting for 5 days. Nutrient solution was added to tubes daily as necessary to maintain 2 mL volume. After 5 days, yellow nutsedge root lengths and dry weights were measured.

Statistical Analysis. Sweet potatoes used for caffeic acid quantitation were obtained from replicated field cultivar and germplasm evaluation experiments. The 1997 and 1999 experiments at Charleston had four replicates, and the 1999 experiment at Edisto had three replicates. Tissue samples from four replications of the 2000 experiment at Charleston were pooled prior to caffeic acid analysis. Following analysis of variance using a completely randomized design, means were separated by Fisher's protected LSD ($P < 0.05$). Fungus bioassay experiments were arranged in a completely randomized design with five replicates. Dose−response data for the fungi were subjected to analysis of variance in a completely random design where replicates were the concentration means from five repetitions of the experiments and sigmoidal regression lines best fitting the data were determined using the Regression Wizard function of SigmaPlot 2000 (SPSS, Inc.). Proso millet seed germination bioassays were arranged in a completely random design with 10 replicates. Means from four repetitions of the proso millet seed germination bioassay experiments were analyzed in the same manner as the data from the fungus experiments.

RESULTS AND DISCUSSION

Caffeic Acid Contents. Periderm caffeic acid levels for clones that were included in these studies ranged from 0.008 to

Table 1. Caffeic Acid Contents of Storage Root Periderm for 12 Sweet Potato Cultivars and Breeding Clones Grown at Charleston in 1997

clone	periderm caffeic acid content (mg/g dry wt)
Sumor	3.27
Jewel	3.26
SC 1149-19	3.18
Tinian	2.99
W-263	2.73
Regal	2.49
Excel	2.16
Beauregard	2.01
W-241	1.98
W-274	1.58
Centennial	1.46
Sulfur	0.45
$LSD_{0.05}$[a]	1.05
average	2.30

[a] Least significant difference ($P \leq 0.05$) for comparing means within a column.

Table 2. Caffeic Acid Contents of Storage Root Periderm for 24 Sweet Potato Cultivars and Breeding Clones Grown at Charleston and Edisto, SC, in 1999

	periderm caffeic acid contents (mg/g dry wt)	
	Edisto	Charleston
NC-718	7.97	0.85
Hi Dry	6.93	2.01
W-326	6.50	2.90
W-325	5.97	0.63
95-161	5.80	0.83
W-345	5.57	0.03
Tinian	5.40	0.67
SC 1149-19	5.37	0.78
94-127	4.37	0.97
PI 538354	3.73	0.38
96-51	3.70	0.47
95-161	3.60	0.83
W-332	3.60	1.84
PI 399163	2.87	0.13
Sumor	2.67	0.68
94-145	2.34	1.42
97-92	2.23	1.48
96-51	1.93	0.47
Picadito	1.77	1.98
97-95	1.00	0.39
97-82	0.93	0.72
97-95	0.10	0.04
92-294	0.09	0.04
97-82	0.09	0.07
$LSD_{0.05}$[a]	2.04	0.81
average	3.51	0.86

[a] Least significant difference ($P \leq 0.05$) for comparing means within a column.

7.97 mg/g periderm dry weight (**Tables 1−3**). In 1999, caffeic acid contents were determined for a number of clones in an unreplicated screening trial (data not presented). Several clones from that trial did not contain detectable caffeic acid, and the levels of several others exceeded 1.0% of the periderm dry weight. Caffeic acid contents differed between clones in all experiments; however, there was variation between replications within clones as indicated by the relatively large $LSD_{0.05}$ values. Caffeic acid levels also varied between locations and years; levels for clones grown at Edisto in 1999 averaged 4.01 mg/g and were consistently higher than those grown at Charleston in 1999 with average contents of 0.97 mg/g (**Table 2**). For clones present in all experiments, the average caffeic acid levels in the 1997 and 2000 experiments at Charleston (**Tables 1−3**) were generally intermediate between levels observed in the 1999 experiments. The comparison of the two 1999 experiments is most meaningful because the same clones were present at both locations. Caffeic acid levels for most clones were over five times higher at Edisto than at Charleston in 1999; however, one clone, Picadito, was similar at both locations and several other clones with relatively high caffeic acid levels were less than 2-fold greater at Edisto than at Charleston. Average cortex caffeic acid levels at 0.018 mg/g were much lower than average periderm caffeic levels at 1.478 mg/g (**Table 3**), and marked differences between cortex and periderm levels were observed for all clones except two, which had very low periderm caffeic acid contents.

Table 3. Caffeic Acid Content in Storage Root Periderm and Cortex Tissues of Sweet Potato Clones Grown at Charleston in 2000

	caffeic acid content (mg/g dry wt)	
clone	periderm	cortex
Beauregard	0. 657	0.014
Carolina Bunch	2.141	0.016
Excel	0.656	0.012
Jewel	1.699	0.013
PI 399163	0.602	0.021
Regal	0.423	0.014
SC 1149-19	0.653	0.028
Sulfur	0.008	0.003
Sumor	1.135	0.004
Tinian	4.555	0.047
W-274	2.300	0.020
TIS 80/637	0.034	0.004
TIS 9101	2.088	0.037
TIS 70357	3.748	0.024
average	1.478	0.018

The stimulus for caffeic acid accumulation in sweet potato periderm has not been reported. Abiotic stress and attack by herbivores or diseases are known to elicit the accumulation of phenolic compounds in sweet potato and other species (*7*, *12*, *25*, *37*−*39*). The sweet potatoes grown at Edisto were probably subject to greater moisture stress than those grown at Charleston. Rainfall was supplemented with sprinkler irrigation at Charleston, whereas the Edisto experiment was not irrigated, and 1999 was a drought year in the area. Further research is planned to assess the effect of environment on sweet potato storage root composition. There may be differences between clones in response to the stimulus that causes caffeic acid accumulation, because not all clones were markedly different between Edisto and Charleston. Several had relatively high caffeic acid at Charleston, and several were low at both locations. This suggests that some clones have relatively high constitutive caffeic acid levels in the absence of the stimulus. Other clones may not accumulate high levels under any conditions.

Most investigations found chlorogenic acid and other caffeoylquinic acids to be the most abundant phenolics in sweet potato. In contrast are the high levels of *p*-coumarate fatty acid esters reported in latex (*14*). Only Son et al. (*27*) found high levels of caffeic acid in sweet potato, where the concentration for five cultivars ranged from 0.34 to 1.37 mg/g fresh weight in the outer 3 mm of storage roots. This layer probably contained more cortex than periderm tissue, because the periderm thickness of 16 clones evaluated by Schalk et al. (*40*) ranged from 0.104 to 0.298 mm. A similar combination of tissues was analyzed by Stange et al. (*28*) who concluded that a fraction containing caffeic acid was not as important as a fraction containing 3,5-dicaffeoyl quinic acid in the *R. stolonifer* growth inhibition caused by extracts of the tissue. Although the differences in

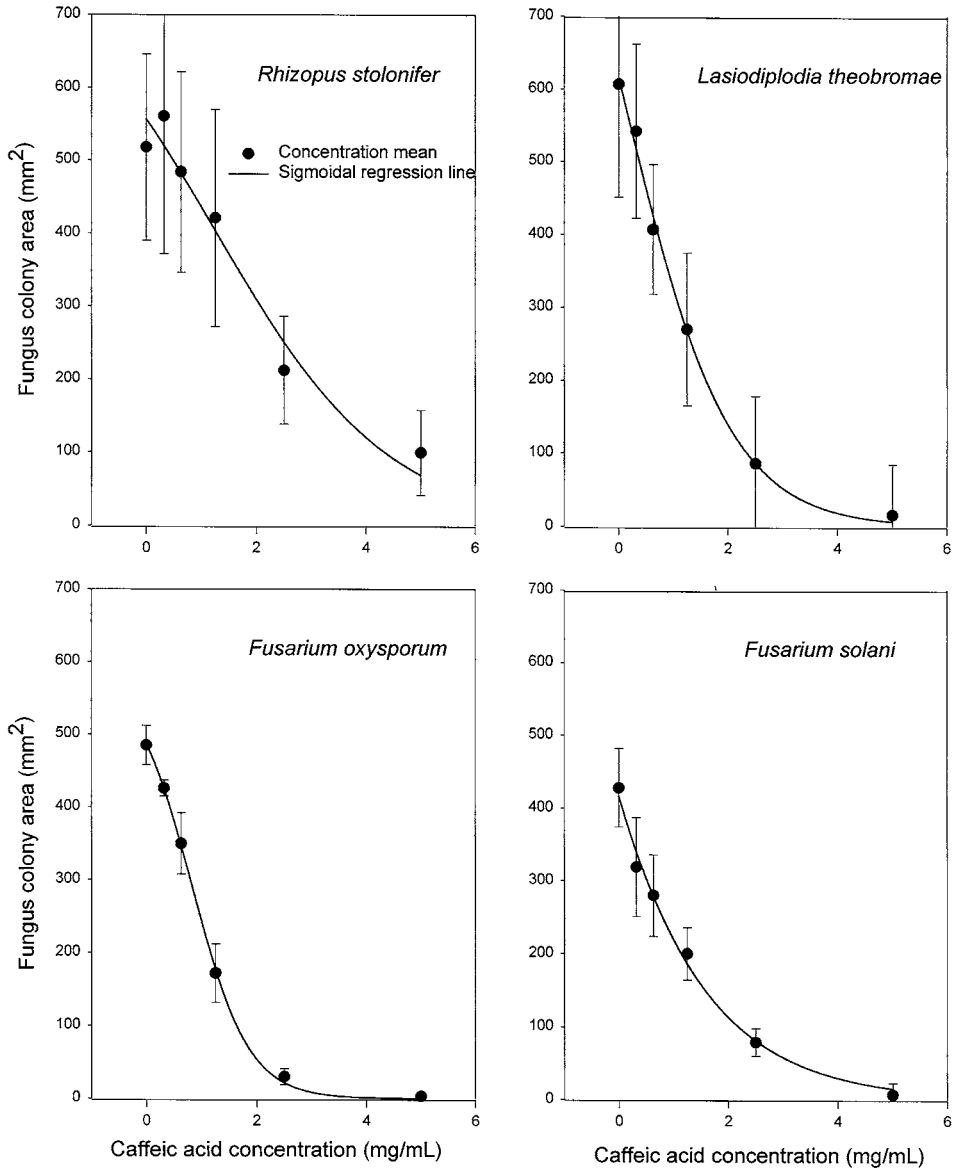

Figure 1. Effect of caffeic acid in PDA medium on the growth of four sweet potato root pathogenic fungi. Vertical bars are standard errors of the mean ($n = 5$). The equation used to derive the sigmoidal regression line was $f = a/(1 + \exp(-(x - x_0)/b))$; $a = 1052.5$, $b = -1.86$, $x_0 = 0.296$, and $r^2 = 0.976$ ($p = 0.002$) for *R. stolonifer*; $a = 1170.1$, $b = -0.947$, $x_0 = 0.100$, and $r^2 = 0.9993$ ($p = 0.0002$) for *L. theobromae*; $a = 585.3$, $b = -0.514$, $x_0 = 0.821$, and $r^2 = 0.999$ ($p = 0.0001$) for *F. oxysporum*; $a = 5897$, $b = -1.48$, $x_0 = -3.80$, and $r^2 = 0.989$ ($p = 0.0006$) for *F. solani*.

periderm thickness (*40*) are not large in comparison to differences in caffeic acid content, the relative thickness contributes to the differences in total root caffeic acid content or in content expressed on a root surface area basis. The difference between the tissues in contents of caffeic acid (**Table 3**) and other phenolics (unpublished data) indicate the necessity to examine tissues separately.

The origin of the relatively high caffeic acid content in sweet potato storage root periderm is not known; however, the discrepancy between caffeic acid levels in the cortex and periderm (**Table 3**) suggests that PCA is formed in the periderm. It is possible that caffeic acid accumulates through de novo synthesis or through enzymatic or nonenzymatic release from more complex phenolic constituents common in suberized tissue. Treatment with ultraviolet-C radiation (*41*) and innoculation with a nonpathogenic strain of the fungus (*42*) induce resistance to *F. oxysporum* root rot in sweet potato. Stevens et al. (*41*) found that the ultraviolet-C radiation treatments that induced resistance in Jewel sweet potato also increased phenylalanine ammonia lyase activity, a rate-controlling step in the phenylpropanoid pathway, activity in the outer layer of sweet potato storage roots. This suggests that the mechanism of induced resistance may involve increased synthesis of phenylpropanoid compounds and supports speculation that caffeic acid accumulation occurs through de novo synthesis. Chlorogenic acid and other caffeoyl quinates also occur in the periderm region (*27*, *28*). Periderm caffeic acid could also arise from these; however, the cinnamoyl ester hydrolase activity needed to cleave the caffeoyl−quinic acid esters has not been reported in sweet potato *(43)*.

Effect of Caffeic Acid on Fungus and Plant Growth. Caffeic acid in PDA medium inhibited the growth of the four pathogenic fungi, all exhibited a strong concentration response, and all fungi except *R. stolonifer* hardly grew on PDA containing 5.0 mg/mL (**Figure 1**). *R. stolonifer* was somewhat less sensitive than *F. oxysporum*, *F. solani*, and *L. theobromea*, which responded similarly to caffeic acid. The lowest concentrations

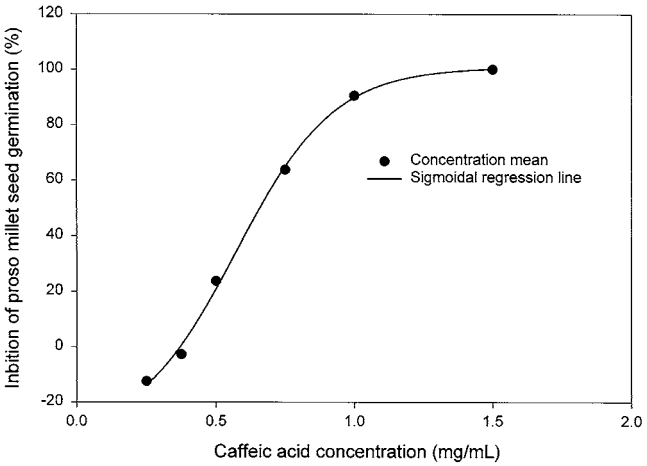

Figure 2. Effect of caffeic acid on the germination of proso millet seed in a Petri dish bioassay. Vertical bars are the standard errors of the mean ($n = 4$). The equation used to derive the standard regression curve was $f = y_0 + a/(1 + \exp(-(x - x_0)/b))$; $a = 133.7$, $b = 0.179$, $x_0 = 0.571$, $y_0 = -32.87$, and $r^2 = 0.995$ ($p = 0.003$).

that inhibited growth in comparison to the control were 0.31, 0.31, 0.63, and 1.25 mg/mL for *F. oxysporum*, *F. solani*, *L. theobromea*, and *R. stolonifer*, respectively. Caffeic acid at up to 2.0 mg/mL in nutrient solution did not reduce yellow nutsedge root growth (data not presented); however, it was inhibitory to millet seed germination at 0.5 mg/mL and higher (**Figure 2**), and inhibition exceeded 90% at 1.0 mg/mL. The lowest concentration, 0.125 mg/mL, stimulated millet germination slightly.

Resin glycosides from the sweet potato periderm are more inhibitory than caffeic acid in the yellow nutsedge and proso millet bioassays (*32*, *33*) and have been reported at higher levels (over 3% of the periderm dry weight) (*34*). They are the most important inhibitors in the allelopathic effect of sweet potato against yellow nutsedge, but they do not inhibit all weed species. Caffeic acid has been reported as an active component in several instances of plant allelopathy (*2*−*4*), and it may affect the growth of weeds in the sweet potato root zone.

These results indicate that caffeic acid may contribute to the protection provided by the sweet potato periderm against root pathogens. The periderm continually sloughs off and is reformed during root growth (*44*). Thus, caffeic acid levels in the root−soil interface region may reach levels inhibitory to the growth of pathogenic fungi and the germination of weed seeds. Linear regression analysis indicated no correlation (data not shown) between caffeic acid levels and insect injury ratings for sweet potato clones that ranged from highly susceptible to highly resistant to soil insects. The thin layer of periderm tissue is probably not a barrier to insect feeding, and cortex caffeic acid levels (**Table 3**) may be too low to influence insect feeding. Many biological activities have been reported for caffeic acid (*2*−*16*), and caffeic acid may have biological functions in sweet potato other than the fungus and weed growth inhibition included in this study. Several other phenolic compounds, including chlorogenic acid other caffeoyl quinic acids, *p*-coumaric acid, scopolin, and scopoletin, have also been found in the periderm (data not reported), usually at much lower levels than the caffeic acid levels found in high caffeic acid genotypes. Further research is needed to fully understand the role of caffeic acid and other phenolics in the complex biochemical defense systems that protect sweet potato roots.

LITERATURE CITED

(1) Buchanan, B. B.; Gruissem, W.; Jones, R. L. *Biochemistry and Molecular Biology of Plants*; American Society of Plant Physiology: Rockville, MD, 2000.

(2) Baghestani, A.; Lemieux, C.; Leroux, G. D.; Baziramkenga, R.; Simard, R. R. Determination of allelochemicals in spring cereal cultivars of different competitiveness. *Weed Sci.* **1999**, *47*, 498−504.

(3) Miller, H. G.; Ikawa, M.; Peirce, L. C. Caffeic acid identified as an inhibitory compound in asparagus root filtrate. *Hortic. Sci.* **1991**, *26*, 1525−1527.

(4) Singh, M.; Tamma, R. V.; Nigg, H. N. HPLC identification of allelopathic compounds from *Lantana camara*. *J. Chem. Ecol.* **1989**, *15*, 81−87.

(5) Bostock, R. M.; Wilcox, S. M.; Wand, G. Adaskaveg. Suppression of *Monilinia fructicola* cutinase production by peach fruit surface phenolic acids. *Physiol. Mol. Plant Pathol.* **1999**, *54*, 37−50.

(6) Kasenberg, T. R.; Traquair, J. A. Effects of phenolics on growth of *Fusarium oxysporum* f.sp. *radicis-lycopersici in vitro*. *Can. J. Bot.* **1987**, *66*, 1174−1177.

(7) Kuc, R. E.; Henze, R. E.; Ullstrup, A. J.; Quackenbush, F. W. Chlorogenic and caffeic acids as fungistatic agents produced by potatoes in response to inoculation with *Helminthosporum carbonium*. *J. Am. Chem. Soc.* **1956**, *78*, 3123−3125.

(8) Ravn, H.; Andary, C.; Kovacs, G.; Molgaard, P. Caffeic acid esters as *in vitro* inhibitors of plant pathogenic bacteria and fungi. *Biochem. Syst. Ecol.* **1989**, *17*, 175−184.

(9) Bowles, B. L.; Miller, A. J. Caffeic acid activity against *Clostridium botulinum* spores. *J. Food Sci.* **1994**, *59*, 905−908.

(10) Reinders, R. D.; Biesterveld, S.; Bijker, P. G. H. Survival of *Escherichia coli* O157:H7 ATCC 43895 in a model apple juice medium with different concentrations of proline and caffeic acid. *Appl. Environ. Microbiol.* **2001**, *67*, 2863−2866.

(11) Summers, C. B.; Felton, G. W. Prooxidant effects of phenolic acids on the generalist herbivore *Helicoverpa zea* (Lepidoptera: Noctuidae): potential mode of action for phenolic compounds in plant anti-herbivore chemistry. *Insect Biochem. Mol. Biol.* **1994**, *24*, 943−953.

(12) Cvikrova, M.; Nedelnik, J.; Eder, J.; Binarova, P. Changes in pattern of phenolic acids induced by culture filtrate of *Fusarium oxysporum* in alfalfa plants differing in susceptibility to the pathogen. *J. Plant Physiol.* **1993**, *142*, 1−5.

(13) Maher, E. A.; Bate, N. J.; Ni, W.; Elkind, Y.; Dixon, R. A.; Lamb, C. J. Increased disease susceptibility of transgenic tobacco plants with suppressed levels of preformed phenylpropanoid products. *Proc. Natl. Acad. Sci. U.S.A.* **1994**, *91*, 7802−7806.

(14) Snook, M. E.; Data, E. S.; Kays, S. J. Characterization and quantitation of hexadecyl, octadecyl and eicosyl esters of *p*-coumaric acid in the vine and root latex of sweetpotato [*Ipomoea batatas* (L.) Lam.]. *J. Agric. Food Chem.* **1994**, *42*, 2589−2595.

(15) Faulds, C. B.; Williamson, G. The role of hydroxycinnamates in the plant cell wall. *J. Sci. Food Agric.* **1999**, *79*, 393−395.

(16) Friend, J. Phenolic substances and plant disease. *Ann. Proc. Phytochem. Soc.* **1985**, *25*, 367−372.

(17) Clark, C. A.; Moyer, J. W. *Compendium of Sweetpotato Diseases*; American Phytopathological Society Press: St. Paul, MN, 1988.

(18) Clark, C. A.; Lawrence, A.; Martin, F. A. Accumulation of furanoterpenoids in sweetpotato tissue following inoculation with different diseases. *Phytopathology* **1981**, *71*, 708−711.

(19) Hyodo, H. I.; Uritani, I.; Akai, S. Production of furanoterpenoids and other compounds in sweet potato tissue in response to infection by various isolates of *Ceratocystis fimbriata*. *Phytopathol. Z.* **1969**, *65*, 332−340.

(20) Uritani, I.; Akazawa, T. Antibiotic effect on *Ceratostomella fimbriata* of ipomeamarone, an abnormal metabolite in black rot of sweetpotato. *Science* **1954**, *121*, 216−217.

(21) Wilson, B. J.; Boyd, M. R.; Harris, T. M.; Yang, D. T. C. A lung edema factor from mouldy sweet potatoes (*Ipomoea batatas*). *Nature* **1971**, *231*, 52−53.
(22) Wilson, B. J. Toxicity of mold-damaged sweetpotatoes. *Nutr. Rev.* **1973**, *31*, 73−78.
(23) Rudkin, G. O.; Nelson, J. M. Chlorogenic acid and respiration in sweetpotato. *J. Am. Soc. Chem*. **1947**, *69*, 1470.
(24) Uritani, I. Abnormal substances produced in fungus contaminated foodstuffs. *J. Assoc. Off. Anal. Chem.* **1967**, *50*, 105−114.
(25) Uritani, I.; Miyano, M. Derivatives of caffeic acid in sweet potato attacked by black rot. *Nature* **1955**, *175*, 812.
(26) Gapasin, R. M.; Valdez, R. V.; Mendoza, E. M. T. Phenolics involved in sweetpotato resistance to *Meloidogyne incognita* and *M. javanica*. *Ann. Trop. Res*. **1988**, *10*, 63−73.
(27) Son, K.-C.; Severson, R. F.; Snook, M. E.; Kays, S. J. Root carbohydrate, organic acid and phenolic chemistry in relation to sweetpotato weevil resistance. *HortSci.* **1991**, *26*, 1305−1308.
(28) Stange, R. R., Jr.; Midland, S. L.; Holmes, G. J.; Sims, J. J.; Mayer, R. T. Constituents from the periderm and outer cortex of *Ipomoea batatas* with activity against *Rhizopus stolonifer*. *Postharvest Biol. Technol.* **2001**, *23*, 85−92.
(29) Islam, M. S.; Yoshimoto, M.; Yahara, S.; Okuno, S.; Ishiguro, K.; Yamakawa, O. Identification and characterization of foliar polyphenolic composition in sweetpotato (*Ipomoea batatas* L.) genotypes. *J. Agric. Food Chem.* **2002**, *50*, 3718−3722.
(30) Jenkins, P. D. Differences in the susceptibility of sweet potatoes (*Ipomoea batatas*) to infection by storage fungi in Bangladesh. *Phytopathol. Z.* **1981**, *102*, 247−256.
(31) Harrison, H. F., Jr.; Peterson, J. K. Sweetpotato components inhibit yellow nutsedge (Cyperus esculentus) growth. *Weed Technol.* **1994**, *8*, 198−171.
(32) Harrison, H. F.; Peterson, J. K.; Clark, C. A.; Snook, M. E. Sweetpotato periderm components inhibit the growth of root rotting fungi. *HortSci.* **2001**, *36*, 927−930.
(33) Peterson, J. K.; Harrison, H. F., Jr. Isolation of a substance from sweetpotato that inhibits seed germination. *J. Chem. Ecol.* **1991**, *17*, 943−951.
(34) Peterson, J. K.; Harrison, J. K.; Muckenfuss, A. E. Sweetpotato [*Ipomoea batatas* (L.)] glycosides: evidence of antibiosis effects in the diamondback moth *Plutella xylostella* L. (Lepidoptera: Plutellidae). *Allelopathy J.* **1998**, *5*, 43−52.
(35) Peterson, J. K.; Harrison, H. F., Jr.; Snook, M. E. Comparison of three parameters for estimation of allelopathic potential in sweetpotato [*Ipomoea batatas* (L.) Lam.] germplasm. *Allelopathy J.* **1999**, *6*, 201−208.
(36) Noda, N.; Yoda, S.; Kawasaki, T.; Miyahara, K. Resin Glycosides. XV. Simonins I−V, Ether soluble resin glycosides (jalapins) from the roots of *Ipomoea batatas* (cv. Simon). *Chem. Pharm. Bull.* **1992**, *40*, 3163−3168.
(37) Lyons, P. C.; Wood, K. V.; Nicholson, R. Caffeoyl ester accumulation in corn leaves inoculated with fungal pathogens. *Phytochemistry* **1990**, *29*, 97−101.
(38) Pomenta, J. V.; Burns, E. E. Factors affecting chlorogenic, quinic and caffeic acid levels in sunflower kernels. *J. Food Sci.* **1971**, *36*, 490−492.
(39) Ramamurthy, M. S.; Biswanath, M.; Thomas P.; Nair, P. M. High-performance liquid chromatography determination of phenolic acids in potato tubers (*Solanum tuberosum*) during wound healing. *J. Agric. Food Chem.* **1992**, *40*, 569−572.
(40) Schalk, J. M.; Peterson, J. K.; Jones, A.; Dukes, P. O.; Walter, W. M., Jr. The anatomy of sweet potato periderm and its relationship to wireworm, *Diabrotica, Systena* resistance. *J. Agric. Entomol.* **1986**, *3*, 350−356.
(41) Stevens, C.; Khan, V. A.; Lu, J. Y.; Wilson, C. L.; Chalutz, E.; Droby, S.; Kabwe, M. K.; Huang, Z.; Adeyeye, O.; Pusey, L. P.; Tang, A. Y. A. Induced resistance of sweetpotato to Fusarium root rot by UV−C hormenesis. *Crop Prot.* **1999**, *18*, 463−470.
(42) Ogawa, K.; Komada, H. Biological control of Fusarium wilt of sweet potato with cross-protection by prior inoculation with nonpathogenic Fusarium oxysporum. *Jpn. Agric. Res. Q.* **1985**, *19*, 20−25.
(43) Kroon, P. A. Hydroxycinnamates in plants and food: current and future perspectives. *J. Sci. Food. Agric.* **1999**, *79*, 355−361.
(44) Steinbauer, C. E.; Kushman, L. J. Sweet potato culture and diseases. Agricultural Handbook 338; U.S. Department of Agriculture: Washington, DC, 1971.

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