Postharvest Temperature Effects on Wound Healing and Surface Rot in Sweetpotato

L. W. Nielsen and J. T. Johnson

Professor and Research Technician, respectively, Department of Plant Pathology, North Carolina State University, Raleigh 27607.

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ABSTRACT

Freshly harvested roots of sweet potato cultivars 'Porto Rico', 'Centennial', 'Nugget', and 'Goldrush' were immersed for 2 h in water baths at five temp from 4.5 to 45 C, or exposed to cold air at 3 C, or to sunlight to obtain differential root temp. The temp-treated roots were artifically wounded, cured at 29 C for 7 days, stored at 13 C for 6 mo. and examined for wound infection by *Fusarium oxysporum*. In general, surface rot developed in more wounds of roots exposed to 4.5 and 45 C, and least developed in wounds of roots exposed to 32 or 38 C. The cultivars responded similarly, but differed in magnitude of response. More wound infections developed in roots of cultivars Centennial and Goldrush than in those of Nugget and Porto Rico. The relative infection was related to the rapidity of wound healing and retardation of the healing processes by the 4.5 and 45 C treatments. Suberin had formed across root wounds of Porto Rico and Nugget exposed to 38 C in 6 days, but it was incomplete across root wounds in Centennial and Goldrush. Temperatures of 4.5 and 45 C retarded wound healing in roots of all cultivars, and the phellogen formed at greater distances below the wound surface. Suberization was retarded more and phellogen formed deeper in the tissues in Goldrush and Centennial roots than in Porto Rico and Nugget roots.

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Sweetpotato (*Ipomoea batatas* [L.] Lam.) surface rot (*Fusarium oxysporum* Schlecht.) developed in more surface wounds when freshly harvested roots were exposed on the soil surface several hours or days prior to curing (8). Solar irradiation and night temp were related to the increased incidence of surface rot, and wound infection of cultivar Goldrush roots was increased by a 2-h exposure to cold water at approximately 43 C. Least surface rot developed in root wounds of four cultivars placed at 29 C within 1 h after harvest.

Temperature plays a significant role in wound healing and storage of sweetpotato roots. Storage temp below 10 C adversely affect the physiology of sweetpotato roots and lead to internal breakdown (1, 3, 4). The duration of root exposure to cold temp to initiate these physiological changes diminishes as the temp approaches 0 C (1). Cured 'Orange Jersey' roots chilled 4 days at 0 C formed suberin and periderm over wounds more slowly than those held at 15.5 C (7), and cured 'Nemagold' roots chilled at 7 and 0 C for 1 to 8 days, developed more Rhizopus soft rot in inoculated wounds than roots held constantly at 13 C. (6). Freshly harvested and noncured roots are more sensitive to cold temperature than cured roots (1, 3, 5). Suberization and phelloderm formation in 'Yellow Jersey' roots proceeds most rapidly at 32 C (2). Rapid wound healing is a significant function of the curing process which results in the control of several storage rots including Fusarium surface rot (4).

Less is known about the effects of high temp on sweetpotato root tissues. Wound healing is retarded at 35 C (2), but surface rot was controlled by curing roots at 36.5 C (4). The cortical tissue of roots exposed to sunlight reached 41.5 C and surface rot developed more extensively in wounds of these roots (8). The evidence indicates that temp in excess of 35-40 C have an adverse effect on the wound healing processes and wound infection.

The objectives of this research were to determine: (i) the role of temp in predisposing freshly harvested sweetpotato roots to infection by *F. oxysporum*, and (ii) the effect of extreme temp on wound healing during the curing process.

MATERIALS AND METHODS.—Roots of sweetpotato cultivars Centennial, Nugget, Goldrush, and Porto Rico were used in all experiments. The sweetpotatoes were planted in June at the Central Crops Research Station, Clayton, N. C. and harvested in late September. Roots were treated within 24 h after harvest.

The roots were exposed to different temp in water baths, cold air, and sunlight. They were immersed 2 h in water baths at 4.5, 13, 32, 38, and 45 C. To simulate natural conditions, other roots were chilled at 3 C until internal tissue reached the desired temp, or exposed to sunlight on black plastic sheeting to obtain high temp. In each case, the temp of three roots at a depth of 3 to 5 mm were measured with thermocouples and samples removed when the average desired temp was reached. The temp obtained in the cold room and from sun irradiation are presented with the data (Table 1).

After exposure, each root was wounded twice by rubbing it on two parallel dowel rods covered with coarse sandpaper (8). Roots exposed to sunlight were

TABLE 1. Development of *Fusarium* surface rot in root wounds following exposure of harvested sweetpotatoes to different temp and 6 mo storage

Sweetpotato cultivar	Percentage of wounds infected in roots treated in –						
	Water baths–2 h exposure			Cold air and sunlight			
				Test 1		Test 2	
	Bath temp (C)	Test 1	Test 2	Temp (C)	Inf.	Temp (C)	Inf.
Porto Rico	4.5	70.8	16.0	4	14.8	4	3.0
	13	46.3	8.0	11	4.2	10	0.5
	32	7.0	4.8	32	5.2	33	4.3
	38	2.0	11.8	40	6.0	38	4.8
	45	10.5	18.5	42	11.5	39	7.3
Goldrush	4.5	80.3	79.1	4	42.6	4	4.0
	13	66.3	43.1	11	38.5	10	0.0
	32	14.5	51.0	32	10.3	33	2.0
	38	5.5	25.0	40	34.8	38	2.5
	45	15.0	31.3	42	54.2	39	2.0
Centennial	4.5	79.8	41.3	4	30.8	4	9.2
	13	73.5	39.3	11	36.8	10	4.5
	32	9.8	36.2	32	22.7	33	3.5
	38	19.8	24.1	40	36.2	38	3.0
	45	42.5	35.6	42	37.5	39	9.5
Nugget	4.5	74.0	16.3	4	54.8	4	34.8
	13	24.3	5.3	11	49.5	10	25.0
	32	1.0	9.0	32	26.6	33	16.0
	38	2.5	16.5	40	6.5	38	20.7
	45	36.5	63.3	42	11.5	39	12.2
LSD, $P = 0.05$[a]		11.7	13.8		14.4		8.4

[a]The mean differences between cultivars, temp and the interaction cv X temp were significant in all tests at $P = 0.01$ except temp in Test 2 of roots exposed to cold air and sunlight where it was $P = 0.05$.

injured on the irradiated side. There were four replications of 25 roots for each treatment. The cultivar roots and heat treatments were randomized and the replications layered in storage crates and separated with waterproof paper. *F. oxysporum* inoculum was that naturally present in adhering soil or on the root surfaces.

The treated and wounded roots were cured at 29 C for seven days then stored at 13 C. They were examined for surface rot in early March, or after ca 6 mo storage. Data are presented as percentage of wounds infected. The tests were performed twice and extended over three harvest seasons.

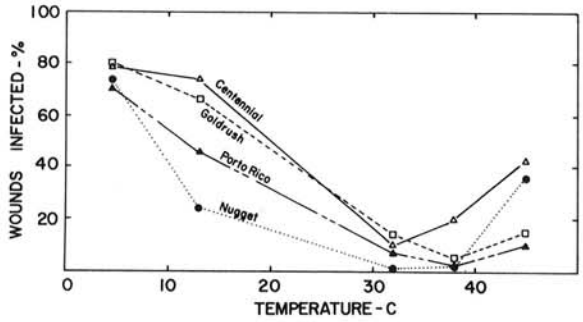

Fig. 1. Development of Fusarium surface rot in root wounds of four sweetpotato cultivars following 2-h exposures of freshly harvested roots in water baths and 6 mo storage. (Figure by Marvin Williams).

Wounds from roots of the four cultivars treated in the water baths at 4.5, 38, and 45 C were excised three and six days after wounding for examination of the wound healing processes. The excised wounds were killed and fixed in formalin-acetic acid-ethyl alcohol solution. Sections were cut with a microtome (Model 900, American Optical, Buffalo), stained in sudan IV (9), mounted in 50% glycerol and examined microscopically for suberin and phellogen formation.

RESULTS.–*Wound infection in temperature treated roots.*–Data from the first water bath test illustrates graphically the relationship between root wound infection and exposure temp for the four cultivars (Fig. 1). Roots of the cultivars responded similarly. More wounds became infected when the roots were exposed to the extreme temp and fewer infections developed in wounds of roots exposed to 32 or 38 C.

Although the cultivar roots responded similarly to the exposure temp they differed in the magnitude of response (Table 1). More wound infections developed in roots of Centennial and Goldrush than in those of Nugget and Porto Rico. This difference between cultivars was evident in the statistical analyses of the four tests and the interaction cultivar X temp. The temp effect was least evident in Test 2 with roots exposed to cold air and sunlight. The maximum temp reached in this test was 39 C which is near the temp that induced minimal predisposition to infection and only Nugget roots developed more infection following the cold treatments.

Influence of exposure temperature on wound healing.—The progress of wound healing in roots of the four cultivars at 29 C was examined in tissue samples taken 3 and 6 days after wounding. Sections were cut from four wounds for each sample. Suberin and phellogen development generally started at the edges of the wounds and progressed toward the center. The extent of suberization was recorded by estimating the percentage of the wound surface that had suberin deposited in cell walls. Early examinations of sections for phellogen development indicated it was forming at variable depths below the wound surface. Consequently, the distance between wound surface and phellogen layer was measured in microns at three positions for each wound; i.e., ca one-quarter of the wound width from either margin and at the center. The three readings were averaged for each wound, and the means of the four wounds for each treatment were used for making comparisons.

The wound healing processes were retarded in roots exposed to the extreme temp (Table 2). The 2-h exposure at 4.5 and 45 C retarded suberization in root tissues of the four cultivars. No suberin had formed after 3 days in the heated roots, but some was deposited in cell walls of chilled roots. The deleterious effect of these temp on suberization was still evident in root wounds after 6 days at 29 C. As the interaction cultivar X temp indicates, cultivar roots responded differently to the temp. Suberization was retarded more in Goldrush roots exposed to 4.5 C than Nugget roots. Suberization proceeded most rapidly in root tissues exposed to 38 C. After 6 days at 29 C, suberin had formed in cells across Nugget and Porto Rico root wounds, while it was incomplete in Centennial and Goldrush roots.

The exposure temp also influenced the depth at which phellogen formed below wound surfaces. In general, phellogen formed deeper in root tissues exposed to the extreme temp than in those exposed to 38 C. Here again, the interaction of cultivar X temp indicated a different response by the cultivar to temp, viz, Nugget vs. Goldrush at 4.5 C.

The rapidity of suberization and the depths of phellogen below the wound surface of roots exposed to the three temp are related to the percentage of root wounds of the four cultivars that developed Fusarium surface rot in storage (see water bath test 2, Table 1). The depth of the phellogen is directly related to infection and the rapidity of suberization is inversely related.

DISCUSSION.—Fusarium surface rot infection data from the two water bath tests were in better agreement than those from exposures to cold air and sunlight. The water-sweetpotato system is a more efficient energy exchanger than the air-sweetpotato system. In the 4.5 C bath, the center of roots weighing 130 and 550 g reached equilibrium with the water in 45 and 100 minutes, respectively. Thus, root tissues exposed 2 h to this and other water bath temp were probably at equilibrium with the bath temp. In contrast, it required 15 to 16 h to obtain the minimum root temp in the cold room. When root tissue 3 to 5 mm deep reached the desired temp, the roots were removed and injured. Root tissues exposed in this manner were at the temp indicated in Table 1 for a relatively short time in comparison with similar root tissues exposed 2 h in the water baths. This may account for the differences in wound infection obtained by exposing roots by the two procedures.

The maximum root temp obtained from exposure to sunlight did not reach the 45 C of the water bath treatments. These exposures were made on 23 September on successive years. Both were cloudless days, but the daily maxima for tests 1 and 2 were 31 and 26 C, respectively, and accounts for the different maxima obtained in the two tests. In comparison with the results from the other tests (Table 1) the 39 C maximum reached with sunlight in Test 2 was below that required to predispose tissues to infection or to retard appreciably wound healing. In North Carolina high root temp from sunlight would occur during late August and early September harvests. Most of these sweetpotatoes go to fresh market and processing and surface rot would not be a problem. Roots for storage are harvested from late September to early November and during the latter weeks of this period roots are often exposed to low temp. Managers of storage facilities concede that late-harvested sweetpotatoes are "poor keepers", particularly when they are not cured at 29 C after harvest.

Sweetpotato cultivars (8, 10) and selections (10)

TABLE 2. Relative suberization and depth of phellogen in root wounds of sweetpotatoes after harvested roots were exposed to water bath temperatures for 2 h and held at 29 C after treatment[a]

Cultivar	Exposure temp (C)	Suberization index[b]		Phellogen distance from wound surface (μm)
		3 days	6 days	
Porto Rico	4.5	3.3	4.3	550
	38	4.5	6.0	245
	45	0.0	2.5	481
Goldrush	4.5	1.0	1.3	775
	38	4.3	5.0	189
	45	0.0	4.3	779
Centennial	4.5	0.5	2.8	503
	38	3.0	4.5	205
	45	0.0	1.5	762
Nugget	4.5	4.3	6.0	196
	38	4.8	6.0	118
	45	0.0	3.3	537
LSD, $P = 0.05$[d]		[c]	2.0	167

[a]Roots from water bath test 2, Table 1.

[b]Indices for percentage of wound surface with suberin, 0, none; 1, 0-10; 2, 10-25; 3, 25-50; 4, 50-75; 5, 75-<100; 6, 100.

[c]Too few data for analysis.

[d]The mean differences between cultivars, temp, and the interaction cv X temp for suberization and depth of phellogen were $P = 0.01$ except for cv X temp in suberization where it was $P = 0.05$.

appear to differ in their susceptibility to Fusarium surface rot. This study shows that cultivars also differ in their rate of wound healing and this is related to infection by *F. oxysporum*.The greater susceptibility to surface rot observed in some cultivars may reflect the inability of their roots to form suberin and phellogen rapidly enough to prevent infection by the pathogen, or their root tissues exposed to low and high temp respond differently to the retardation of the healing processes rather than the tissue being more susceptible to the pathogen.

Slower suberization and the deeper location of phellogen in the tissue following exposure to low or high temp would favor the pathogen. The delayed deposition of suberin in cells would permit tissue penetration and the increased volume of dead tissue due to the deeper phellogen would provide additional substrate for the pathogen to become established in the tissue.

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