Influence of Fungicide and Chemical Salt Dip Treatments on Crater Rot Caused by *Rhizoctonia carotae* in Long-Term Storage

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ABSTRACT

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Nine fungicides and two chemical salts were tested for their ability to reduce mycelial growth of *Rhizoctonia carotae* on V8 agar. Among the fungicides, only sodium orthophenylphenate (SOPP, Dowcide A) and mancozeb (Dithane M-45) completely inhibited growth and were fungicidal. Benomyl, chlorothalonil, and iprodione also inhibited mycelial growth but were fungistatic. The fungicides captan, dicloran, thiabendazole, and thiophanate methyl reduced growth of the fungus by 4–85% of that in unamended agar. Two chemical salts, potassium carbonate (K_2CO_3) and sodium bicarbonate ($NaHCO_3$), at 0.1 M totally inhibited growth of *R. carotae* and were fungicidal. Potassium carbonate was also fungicidal at 10 mM. The fungicides SOPP, benomyl, thiabendazole, and iprodione, the salts K_2CO_3 and $NaHCO_3$, and hot water treatment (58 C), were evaluated alone or in various combinations for disease control on carrot cvs. Danvers 126 and Gold King over 4 yr of storage trials. A dip treatment (1 min) of naturally infected carrots was made before an 18- to 24-wk storage period, or 4 wk after trials were initiated. The most effective treatment (52–70% reduction in disease incidence over 4 yr) was SOPP, used either alone or in combination with 0.1 M K_2CO_3. Treatments applied after 4 wk in storage were as effective as those made after harvest. The addition of K_2CO_3 to the SOPP solution maintained a high optimal pH; the combined cost of the treatment was about 30–40¢ per metric ton.

Additional keywords: *Daucus carota*, storage rot

Large tonnages of carrots (*Daucus carota* L.) frequently are stored after harvest for periods of 4–6 mo at temperatures of 1–2 C and a relative humidity (RH) in excess of 95% to provide a year-round supply for processing needs, such as for soup production. The high humidity is required to reduce losses attributable to shrinkage, which can range from 7 to 16%. One of the few fungal pathogens that causes significant losses under these storage conditions is *Rhizoctonia carotae* Rader, the causal agent of crater rot (14). Although the fungus grows slowly even at its optimal temperature range of 10–21 C, it can cause significant losses on carrots stored over a 3- to 4-mo period because of its ability to grow at 0–1 C (7,14,16). The occurrence of *R. carotae* in fields cropped to carrots in North America is not common, and the pathogen has been reported only from Illinois, New York, and Ohio (14,16–18). The disease occurs in many parts of the United Kingdom and Europe (1,5,7,8), where low temperatures preceding harvest may facilitate infection. For example, in the late 1960s, more than 50% of the carrots in Danish storage facilities were destroyed within 2–3 mo (8). Carrots grown near Napoleon, OH, generally cannot be stored for periods longer than 2 mo without extensive losses caused by crater rot. In one storage facility with a capacity of 1,100 t, losses have ranged from 15 to 50% over a 5-yr period. In 1949, significantly higher losses (around 90%) were reported from storage facilities near Chicago, IL (18).

Control of *R. carotae* on stored carrots is difficult. Jensen (8) reported that the most economical method to prevent the development of crater rot was to eliminate fungal inoculum on wooden crates by steam sterilization. Because carrots in Napoleon are stored in bulk, however, disease control strategies must be targeted to reduce incoming inoculum in or on the carrot roots. Rader (16,17) attempted unsuccessfully to reduce severity of crater rot with fungicide dips. Wells and Merwarth (20) reported that dips in aqueous solutions (in μg a.i. ml^{-1}) of sodium orthophenylphenate (SOPP) (1,000), dicloran (450), or benomyl (500) resulted in a lower incidence of storage rots, of which a low proportion was attributed to *R. carotae*. A hot water (52 C) dip for 10–20 sec was reported to be as effective as fungicide dip treatments (20). Several other chemicals have been evaluated to prevent storage rots on carrots (3,6,9–11), but none have been tested against growth of *R. carotae* or development of crater rot. In a recent report, benomyl or iprodione (500 μg a.i. ml^{-1}) provided control of storage rot caused by *Botrytis cinerea* Pers.:Fr. or *Sclerotinia sclerotiorum* (Lib.) de Bary, but neither chemical was effective against *R. carotae* (5).

The objective of this study was to determine whether practical and economical control of crater rot could be achieved with postharvest applications of fungicides and chemical salts. We have screened selected compounds against mycelial growth of *R. carotae* in vitro and have evaluated the effectiveness of some of the chemicals in preventing disease development in storage trials that were conducted over a 4-yr period

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(1984–1988). Several other parameters, such as the time and frequency of application, hot water treatment, and the use of sodium hypochlorite, were also evaluated for their effectiveness in preventing disease development during long-term storage.

MATERIALS AND METHODS

Effect of fungicides and chemical salts on mycelial growth of *R. carotae*. Five isolates of *R. carotae*, obtained in 1984 from diseased carrots taken from storage facilities in Napoleon, OH, were cultured on V8 agar (200 ml of V8 juice, 800 ml of water, and 15 g of water agar per liter) at room temperature. Plugs (7 mm diameter) were transferred from 2- to 3-wk-old cultures to 100 × 15 mm petri dishes containing V8 agar amended with 50 mg/L each of streptomycin sulfate and penicillin G sulfate (to prevent bacterial contamination) and the appropriate fungicide or chemical salt to be evaluated. The antibiotics and fungicides/chemicals were added directly to warm (50 C) autoclaved medium which was subsequently dispensed into the petri dishes.

The following fungicides (obtained directly from chemical distributors or manufacturers) were tested: sodium orthophenylphenate (SOPP) (Dowcide A, 97%), captan (Orthocide, 50WP), dicloran (Botran, 75WP), thiabendazole (Mertect 340-F, 45.5%), mancozeb (Dithane M-45, 80WP), benomyl (Benlate, 50WP), chlorothalonil (Bravo 500-F, 50%), iprodione (Rovral 50WP), and thiophanate methyl (Topsin M, 70WP). The first four fungicides are registered for postharvest disease control on carrots (4) and were evaluated at the recommended rates (Table 1). Mancozeb, benomyl, and chlorothalonil were tested at rates comparable to those used for foliar disease control (1) and iprodione and thiophanate methyl were evaluated at rates recommended for postharvest treatment of fruits (Table 1). Two chemical salts, potassium carbonate (K_2CO_3) and sodium bicarbonate ($NaHCO_3$), were also tested in this study. Both of these salts have been reported to inhibit germination of sclerotia of *Sclerotium rolfsii* Sacc. (15) and in preliminary studies with artificially inoculated carrot roots reduced infection by *R. carotae*.

Each dish containing a fungicide or chemical salt was inoculated with a plug from each of the isolates; the five plugs were arranged equidistant in the shape of a pentagon approximately 1 cm from the periphery of the dish. Each treatment was replicated four times, and dishes were incubated in the laboratory at 20–24 C. Radial growth from the plug site toward the center of the dish was measured after 8–11 days. Plugs with less than 1 mm of growth were transferred to V8 agar and incubated for 7 days to determine if the inhibition was attributable to fungistatic or fungicidal activity of the compound.

For comparative purposes, two storage pathogens of carrot, *B. cinerea* and *S. sclerotiorum*, were also evaluated for their sensitivity to the fungicides and chemical salts that had been added to V8 agar. A single mycelial plug was placed in the center of each dish, and the extent of growth was rated 3–4 days later.

Effect of fungicides and chemical salts on disease development in storage. Over a 4-yr period (1984–1988), carrot cvs. Gold King or Danvers 126 were mechanically harvested during September–October from commercial fields with a history of producing carrots with poor storage quality. Small, distorted, or injured carrots were discarded. The remaining carrots were divided into lots of approximately 11 kg and placed in a wooden or plastic crate. Each lot was dipped for 1 min in 95 L of the appropriate chemical and stored for a period of 18–24 wk in one of three storage facilities that were maintained at approximately 1 C and >95% RH. For each treatment, there were four single crate replicates, and controls consisted of carrots that were dipped in water or left dry. At the end of each experiment, the carrots were graded (good, acceptable, or unacceptable) based on visual ratings of the percentage of surface area that either had pits (crater rot) or visible surface mycelium (14). Hyphae were examined microscopically for the characteristic branching pattern and presence of clamp connections to confirm the presence of *R. carotae* (14). The grading scale used was as follows: good = no evidence of decay or mycelial growth; acceptable = <10% of the surface area diseased or covered with mycelium; and unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. The carrots in each grade category were then weighed, and final disease severity was expressed as the weight of carrots in each grade/final total weight × 100. In the first year of the storage trials, carrots in each grade category were also counted and weighed. Weight and number were well correlated, as numbers accounted for 96–99% of the variation in weight. Thus, only fresh weights were used in subsequent experiments. The data were transformed by arcsine-square root where appropriate and analyzed with Duncan's multiple range test or by appropriate *t* tests.

Influence of application procedure, time and frequency of application, hot water treatment, and sodium hypochlorite on disease development in storage. The following parameters were evaluated to determine their influence on the development of crater rot: 1) application procedure—a mixture of SOPP + K_2CO_3 was used as a dip (1 min) for the carrots or 5.7 L of the solution was poured over the carrots in each crate with a watering can; 2) time of application—the carrots were treated after harvest or 4 wk after storage was initiated (for the latter, the crates were removed from storage, dipped, and returned to storage within 3 hr); 3) frequency of application—crates were dipped into a solution of SOPP once or twice after harvest or after harvest and again after 4 wk in storage, and finally, once after harvest and again after 16 wk in storage; 4) high temperature—carrots were dipped in water or a solution of SOPP + K_2CO_3 at 58 C and compared with that of water or the solution at 18 C; and 5) bleach

Table 1. Activities of fungicides and chemical salts in V8 agar on mycelial growth of *Rhizoctonia carotae* and of two storage pathogens of carrot

Compound	Rate (μg a.i. ml^{-1})	Inhibition of growth (%) of[w]		
		Rhizoctonia	*Botrytis*	*Sclerotinia*
SOPP[x]	930*[y]	100 (C)	100 (C)	100 (C)
	260	100 (C)	ND	ND
K_2CO_3	0.1*[z]	100 (C)	100 (S)	100 (S)
	0.01[z]	100 (C)	89	100 (S)
SOPP + K_2CO_3	930 + 0.1[z]	100 (C)	ND	ND
$NaHCO_3$	0.1*[z]	100 (C)	100 (S)	100 (S)
	0.01[z]	2–32	28	61
Thiabendazole	2,000	4–70	ND	ND
	1,500*	0–70	98	94
SOPP + thiabendazole	930 + 1,500*	100 (C)	ND	ND
Dicloran	900	44–77	100 (S)	100 (S)
Captan	1,200	85–100	61	100 (S)
Benomyl	2,900*	100 (S)	100 (C)	100 (C)
Mancozeb	2,700	100 (C)	39	100 (C)
Iprodione	3,300*	100 (S)	100 (C)	100 (C)
Chlorothalonil	2,600	94–100	79	82
Thiophanate methyl	3,400	29–58	96	92

[w] Mean value of four replicates for one isolate. Ranges indicate mean values for the five isolates. Extent of inhibition was calculated as follows: (control growth − treatment growth)/control growth × 100. ND = not determined. (C) = Fungicidal activity; (S) = fungistatic activity.
[x] SOPP = sodium orthophenylphenate.
[y] * = Rates of compounds used for all subsequent storage trials.
[z] Rates in M.

—an unbuffered solution containing 0.525% NaOCl was applied as a dip treatment.

RESULTS

Effect of fungicides and chemical salts on mycelial growth of *R. carotae*. Among the fungicides and chemical salts tested for their ability to inhibit mycelial growth of *R. carotae* on V8 agar, SOPP (930 μg a.i. ml^{-1}), mancozeb (2,700 μg a.i. ml^{-1}), 0.1 M and 0.01 M K_2CO_3, and 0.1 M $NaHCO_3$ totally inhibited growth of the fungus and were fungicidal (Table 1). A combination of SOPP with either K_2CO_3 or thiabendazole was similarly inhibitory. Benomyl and iprodione also inhibited growth of the fungus, but when the plugs were transferred to unamended media, growth resumed. The remaining fungicides inhibited mycelial growth of *R. carotae* to different extents. By comparison, *B. cinerea* and *S. sclerotiorum* were totally inhibited by SOPP, dicloran, benomyl, iprodione, K_2CO_3, and $NaHCO_3$, some of which were fungicidal.

Effect of fungicides and chemical salts on disease development in storage. In the 1984–1985 trial conducted over an 18-wk period, crater rot development resulted in 64% of the Danvers 126 that had been dipped in water to be unacceptable for processing (Table 2). By comparison, the most effective treatment (SOPP) significantly reduced the percentage of unacceptable carrots to 31%.

In the 1985–1986 trial, 28% of the carrots in the water control were unacceptable, compared with 35% in the dry treatment. In contrast, only 6% of the carrots treated with SOPP in combination with K_2CO_3 were unacceptable. The other dip treatments provided varying levels of disease control (Table 3).

In the 1986–1987 trial, the most effective treatment again was SOPP, used either alone or in combination with K_2CO_3, applied either as a dip or a drench (Table 4). The percentage of unacceptable carrots was lower in the water controls (42%) than in carrots not dipped in water (57%). When treatments were applied after 4 wk in storage, the results were similar, except that the percentage of unacceptable carrots was higher in the water control (48%) and in the dry control (69%) (Table 4). Dip treatment of carrots in bleach after harvest or after 4 wk of storage resulted in 26–40% lower disease development than in the water control.

The rate of development of crater rot (expressed as the percentage of unacceptable carrots) over an 18-wk period and final disease levels at the end of each of the storage trials during 1985–1986, 1986–1987, and 1987–1988 were different. In all years, little to no disease was detected before 5 wk, after which time disease increased at different rates. During 1987–1988, disease development was most rapid and reached a maximum of 92% by 18 wk.

Influence of frequency of application and hot water treatment on disease development in storage. Treatment of carrots with SOPP at the beginning of the storage trial, twice on the same day, did not improve disease control over a single application (Table 5). With two applications made at different times, at the initiation of the trial and after 4 wk of storage, the percentage of unacceptable carrots was lower compared with two applications made after harvest. Treatment once at the initiation of the trial and again after 16 wk of storage provided a similar level of disease control as two applications made after harvest (Table 5). Use of hot water or a solution of SOPP + K_2CO_3 at 58 C did not enhance their effectiveness over dips conducted at 18 C (Table 6). Disease development in this storage trial (1987–1988) was very high in the water control (92%), but the percentage of unacceptable carrots was significantly reduced to 16–28% by treatment with SOPP + K_2CO_3 at either temperature.

Table 2. Effect of fungicide and chemical salt dip treatments on development of *Rhizoctonia carotae* on stored carrots, 1984–1985[w]

Treatment[x]	Total carrots in each disease category (%)[y]		
	Good	Acceptable	Unacceptable
SOPP[z]	37 a	32 ab	31 a
Thiabendazole	21 a	46 b	33 a
SOPP + thiabendazole	30 a	19 a	51 abc
$NaHCO_3$	23 a	35 ab	42 ab
K_2CO_3	17 a	29 ab	54 bc
Water	11 a	25 a	64 c

[w] Carrot cultivar was Danvers 126; storage period was 18 wk.
[x] Rates used are indicated in Table 1, except for thiabendazole, which was tested at 912 μg a.i. ml^{-1}.
[y] Good = no evidence of decay or mycelial growth, acceptable = <10% of the surface area diseased or covered with mycelium, unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. Values within each column followed by the same letter are not statistically different ($P = 0.05$) according to Duncan's new multiple range test.
[z] SOPP = sodium orthophenylphenate.

Table 3. Effect of fungicide and chemical salt dip treatments on development of *Rhizoctonia carotae* on stored carrots, 1985–1986[w]

Treatment[x]	Total carrots in each disease category (%)[y]		
	Good	Acceptable	Unacceptable
SOPP[z]	71 cd	7 a	22 ab
Thiabendazole	41 ab	13 a	46 bc
SOPP + thiabendazole	58 abcd	9 a	33 ab
K_2CO_3	64 bcd	10 a	26 ab
SOPP + K_2CO_3	85 d	9 a	6 a
Iprodione	66 bcd	9 a	25 ab
Benomyl	35 a	7 a	59 c
Water	61 abcd	11 a	28 ab
None (dry)	50 abc	14 a	35 abc

[w] Carrot cultivar was Danvers 126; storage period was 18 or 23 wk.
[x] Rates used are indicated in Table 1.
[y] Good = no evidence of decay or mycelial growth, acceptable = <10% of the surface area diseased or covered with mycelium, unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. Values within each column followed by the same letter are not statistically different ($P = 0.05$) according to Duncan's new multiple range test.
[z] SOPP = sodium orthophenylphenate.

DISCUSSION

The standard treatment generally used by growers to reduce the incidence of storage rots on carrots in Napoleon, OH, including that attributable to *R. carotae*, is to dip harvested and washed carrots in thiabendazole or thiabendazole + SOPP (Mertect + Dowcide A) and occasionally in a 10% solution of commercial bleach. However, in the results reported here, neither bleach or thiabendazole treatment significantly affected the development of *R. carotae* on carrots in long-term storage. Treatment with a combination of thiabendazole + SOPP was less effective than treatment with SOPP alone. In contrast, SOPP used alone or in combination with K_2CO_3 consistently provided a level of disease control in each of the 4 yr of this study. The addition of 0.1 M K_2CO_3 to SOPP increased the pH of the mixture from 10.3 to 11.6. The activity of SOPP is enhanced at pH 11.6 or higher (19). Generally, the addition of NaOH to SOPP is recommended to achieve the high pH

(21). However, we observed that the use of NaOH frequently resulted in the formation of a precipitate, whereas the use of K_2CO_3 did not (M. D. Ricker and Z. K. Punja, *unpublished data*). In addition, K_2CO_3 and $NaHCO_3$ are toxic to fungi such as *R. carotae* and *S. rolfsii* (15) because of the disassociation of the carbonate (CO_3^{2-}) and bicarbonate (HCO_3^-) ions in alkaline solution (15).

The use of K_2CO_3 and $NaHCO_3$ alone (at 0.1 M) as a solution dip for carrots can effectively reduce the development of other fungal pathogens in short-term storage, such as *Pythium* and *Thielaviopsis* (M. D. Ricker and Z. K. Punja, *unpublished data*), and was partially effective against *R. carotae* in this study. However, some injury may result to the root surface because of the extremely alkaline nature of the salts. For control of crater rot in commercial storage, SOPP + K_2CO_3 should be applied to carrots after soil has been washed from the root surface. Application as a dip provided a similar level of control as a drench in the present study and is easier to use. Treatments can be effectively applied once after harvest. Solution temperatures did not appear important to the effectiveness of SOPP + K_2CO_3. In one experiment (1985–1986), a second treatment applied after 4 wk of storage resulted in lower development of disease, but such an application would be impractical once carrots are already placed in bulk storage. The application of SOPP + K_2CO_3 also has the potential to control other storage pathogens of carrot, such as *B. cinerea* and *S. sclerotiorum*, because both of these compounds completely inhibited growth of these fungi in vitro.

Over several years of this study, less disease developed in the water controls when compared with carrots not dipped in water. A similar reduction after dips in water was noted by Geeson et al (5). Dipping or washing carrots in water before storage also has been shown to influence disease severity attributable to other decay organisms (2,9,10,13,20). The lower disease development attributable to *R. carotae* in this study after water dips could be attributed, in part, to physical removal of some surface mycelium. Infection by *R. carotae* on carrot is preceded by abundant mycelial growth on the root surface (14), and removal of some mycelium may have delayed disease development. Alternatively, the presence of a film of moisture on the root surface may have increased the growth of competing fungi or antagonistic bacteria, which may have reduced the slow-growing *R. carotae*. However, when the incoming inoculum level was high, e.g., in 1987–1988, where the final percentage of unacceptable carrots was 92%, water dips had no effect on reducing disease.

The final percentage of carrots un-

Table 4. Effect of fungicide and chemical salt treatments, applied at two different times, on development of *Rhizoctonia carotae* on carrots, 1986–1987[w]

Treatment[x]	Total carrots in each disease category (%)[y]		
	Good	Acceptable	Unacceptable
Applied at initiation of storage			
SOPP[z]	98 c	2 a	0 a
Thiabendazole	26 a	10 bc	64 c
SOPP + K_2CO_3	98 c	1 a	1 a
SOPP + K_2CO_3, drench	90 c	4 b	6 a
Bleach	56 b	13 c	31 b
Water	43 ab	16 c	42 bc
None (dry)	23 a	19 c	57 bc
Applied after 4 wk of storage			
SOPP	95 c	4 ab	1 a
Thiabendazole	38 ab	16 cd	47 bc
SOPP + K_2CO_3	99 c	1 a	0 a
SOPP + K_2CO_3, drench	88 c	7 b	5 a
Bleach	61 b	10 bc	29 b
Water	30 ab	21 d	48 bc
None (dry)	21 a	9 bc	69 c

[w] Carrot cultivar was Gold King; storage period was 23 wk.
[x] Rates used are indicated in Table 1. All treatments, except where indicated, were applied as a dip.
[y] Good = no evidence of decay or mycelial growth, acceptable = <10% of the surface area diseased or covered with mycelium, unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. Values within each column followed by the same letter are not statistically different ($P = 0.05$) according to Duncan's new multiple range test.
[z] SOPP = sodium orthophenylphenate.

Table 5. Influence of time and frequency of application of sodium orthophenylphenate (SOPP) on development of *Rhizoctonia carotae* on carrots[x]

Time of application[y]	Frequency	Total carrots in each disease category (%)[z]		
		Good	Acceptable	Unacceptable
Initiation of storage	Once	62 ab	11 b	27 a
	Twice	64 ab	8 b	28 a
Initiation of storage and 4 wk later	Once each	92 b	3 a	5 a
Initiation of storage and 16 wk later	Once each	53 a	12 b	35 a

[x] Carrot cultivar was Danvers 126; storage period was 24 wk.
[y] Roots were submerged for 1 min in an aqueous solution of SOPP (930 μg a.i. ml^{-1}) at the times indicated.
[z] Good = no evidence of decay or mycelial growth, acceptable = <10% of the surface area diseased or covered with mycelium, unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. Values within each column followed by the same letter are not statistically different ($P = 0.05$) according to Duncan's new multiple range test.

Table 6. Influence of temperature of dip solution on development of *Rhizoctonia carotae* on carrot[w]

Temperature (C)[x]	Treatment[y]	Total carrots in each disease category (%)[z]		
		Good	Acceptable	Unacceptable
18	SOPP + K_2CO_3	72 a	13 a	16 a
	Water	1 b	4 b	95 b
58	SOPP + K_2CO_3	59 a	13 a	28 a
	Water	4 b	6 b	90 b
	None	2 b	6 b	91 b

[w] Carrot cultivar was Danvers 126; storage period was 19 wk. Data are from 1987–1988.
[x] Temperature of the treatment solution was adjusted by heating the solution just before treatment.
[z] Good = no evidence of decay or mycelial growth, acceptable = <10% of the surface area diseased or covered with mycelium, unacceptable = >10% of the surface area with disease or with one or more craters deeper than 3 mm. Values within each column followed by the same letter are not statistically different ($P = 0.05$) according to Duncan's new multiple range test.

acceptable for processing at the end of the storage trials in the controls in each of the years of this study (1984–1988) was different and ranged from 28 (1985–1986) to 92% (1987–1988). Therefore, a direct comparison of results after treatments from one year to the next cannot be made. This yearly variation in disease severity most likely was attributable to differences in disease incidence in different fields, resulting in variable incoming inoculum (infections) on the harvested carrots. There are no previous reports on the source of inoculum for disease development in storage, because previous investigators have not noted visible mycelium or lesions on harvested carrots (6,16). In 1988 and 1989, during late harvest of carrots (at the end of October), mycelium of *R. carotae* was observed growing on the remnants of petiole tissues at the crown, generally as dense clumps on senescent leaf tissue or within cracks on the root. No visible lesions were observed. We postulate that growth of *R. carotae* from soil onto the carrots occurs late in the season, when temperatures are low, with the fungus first colonizing senescent petiole or leaf tissue at the crown and subsequently developing on the root surface during storage. This is supported by the observation that most initial infections are at or near the crown, with the fungus subsequently spreading in storage by growth of mycelium from root to root (M. D. Ricker and Z. K. Punja, *unpublished data*). Eliminating the incoming inoculum on petiole tissues at the crown is difficult because the carrots are topped but not crowned. Occasionally, sclerotia of *R. carotae* have been observed on naturally infected roots after prolonged storage (M. D. Ricker and Z. K. Punja, *unpublished data*). Sclerotia have been previously observed on artificially inoculated roots after 6 mo of storage, and they form readily in culture (14). The role of sclerotia in initiating disease in nature is not known, and they may serve as the overwintering structures. Sclerotia could form in nature on unharvested carrots left in the soil or on discarded roots in cull piles.

None of the treatments tested in each of the years of this study, including SOPP + K_2CO_3, completely prevented development of *R. carotae* in storage. Mycelium tightly woven into the crown tissue, in soil clumps adhering to roots, or as incipient infections may not have been exposed to the chemicals. The significant reduction in disease, however, has warranted the commercial use of SOPP + K_2CO_3 to treat carrots over the last 3 yr in the Napoleon, OH, area. The only additional equipment needed was a conveyer belt and an 1,800-L tank for dipping the carrots. The additional cost of the chemicals was estimated to be 30–40¢ per metric ton, for a product valued at $50–150 per ton. In 1988, the combination of washing carrots in water followed by dipping in SOPP + K_2CO_3 led to an estimated 95% reduction in losses in commercial storage as compared with untreated carrots.

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