Potential for Postharvest Disease in Tomato Fruit Infiltrated with Chlorinated Water

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ABSTRACT

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The incidence of disease associated with the infiltration of tomato fruits with water was reduced but not eliminated by adding 50–1,000 mg of free-chlorine per liter (ppm Cl_2) to the water. Bacterial soft rot and sour rot affected nearly 18% of fruits infiltrated with water containing 1,000 ppm Cl_2 and then stored for 2 wk at 24 C. Addition of chlorine (sodium hypochlorite) to water enhanced the infiltration of submerged fruit. Nearly 40% of fruit treated with 100–1,000 ppm Cl_2 absorbed 0.5 g or more of water compared with just 5% of those similarly treated with water alone. Water uptake increased with chlorine concentration, period of immersion, and depth of immersion. Disease incidence increased as chlorine concentration decreased. Under conditions that simulated commercial handling, water uptake was affected more by the period of immersion, 2–60 min, than by immersion depths of 1–15 cm or concentrations of 0–250 ppm Cl_2. Appropriate chlorination practices should control postharvest fruit rots; however, chlorination increases the potential for infiltration, and infiltrated fruit are likely to become diseased. Therefore, factors responsible for infiltration, such as the depth and period of immersion, must be controlled.

Extensive postharvest disease results when fruits of tomato (*Lycopersicon esculentum* Mill.) become infiltrated with aqueous suspensions of fruit rot pathogens (1–3,6). Absorption of water through stem scar tissues occurs when the pressure of water on the surface of submerged fruit overcomes the hydrophobic nature of the surface of stem scars and internal gas pressures in fruit (3). Pressure imbalances occur as a direct result of immersion (3) or when submerged fruit cool (6).

Strategies to prevent infiltration of fruit in tomato packinghouses have been proposed (15). Water in dump tanks and flumes should be warmed (5 C higher than the highest fruit pulp temperature); immersion periods and depths should be limited to 2 min and 60 cm, respectively. In several progressive packinghouses in Florida, water temperatures were consistently higher than fruit pulp temperatures; fruit were submerged for 1 to occasionally 20 min; immersion depths ranged from about 1 to 15 cm, which represented one to three layers of fruit floating in the water; and chlorine concentrations varied from 35 to >180 mg of free-chlorine per liter (ppm Cl_2) (J. A. Bartz and M. Sherman, *unpublished*). Thus, water temperatures and immersion depths, but not immersion periods, were within those recommended.

Because the water used in packinghouse systems usually is chlorinated, the need to prevent infiltration may be questioned. Recommended concentrations of 100–150 ppm are maintained by adding chlorine gas or some source of hypochlorite ion (9,14). Free-chlorine is the accepted term for unreacted hypochlorous acid and hypochlorite ion (10). In laboratory tests, free-chlorine at 0.2–5 mg/L of water inactivated cells of various gram-negative bacteria within seconds (7). In contrast, spores of *Alternaria alternata* were 10-fold less sensitive than cells of *Escherichia coli* (13). Chlorine levels 20–300 times higher than those used in the laboratory tests are recommended for dump tanks and flumes as an attempt to maintain concentrations over time that will quickly inactivate introduced inocula (9). In addition, such levels of free-chlorine reduce populations of pathogens in shallow wounds on fruit surfaces and thus delay the onset of disease (4).

Hypochlorous acid inactivates microbes on contact (7). It is a strong oxidizing agent and will react with many different types of inorganic and organic species (7,10). The products of these reactions either have reduced or no antimicrobial activity. Therefore, hypochlorous acid has only limited ability to penetrate organic matter (debris, wounds on plants, etc.) and kill microbes that are partially or fully embedded (8). Chlorine dioxide, a chemical that is not as reactive toward many chemical species as free-chlorine (7), has been proposed as an alternative biocide for dump tanks and flumes. It may be more specific for target microbes than chlorine.

The purpose of this work was to assess the probable impact of immersion period and depth on the potential for fruit rots in tomato fruit treated with water, to determine if chlorine dioxide had the same effect on the incidence of fruit rots as hypochlorous acid/hypochlorite ion, and to determine if fruit infiltrated with those solutions are likely to develop fruit rots. A preliminary report was issued (5).

MATERIALS AND METHODS

General methods. Tomato fruit, cultivars Flora-Dade, Florida MH-1, and Hayslip, were hand-harvested from plants in experimental plots or commercial fields. Mature green or "breaker" fruit selected for the tests were free of injury and disease. Bacterial inoculum was prepared from shake cultures of *Erwinia carotovora* subsp. *carotovora* (*E. c.* subsp. *carotovora*) (Florida strain SR38) that grew in nutrient broth. Cells were pelleted from 24-hr suspensions by centrifugation, then resuspended in distilled water. Concentration of these suspensions was estimated from their optical density at 600 nm based on previous tests in which an aqueous suspension with an optical density of 0.3 contained about 2×10^8 colony-forming units (cfu) per milliliter. Concentrated stock suspensions were diluted with distilled water as needed for the tests.

Chlorine solutions (hypochlorite ion/hypochlorous acid) were prepared by diluting standard commercial laundry bleach (5.25% NaOCl) with tap water. Concentrations of free-chlorine in the test solutions were measured with the DPD-test (*N,N*-diethyl-*p*-phenylenediamine) (Hellige Inc., Garden City, NY). Stabilized chlorine dioxide solutions were prepared by dilution from 5% chlorine dioxide (Odocine Plus, Odco Laboratories International Inc., Fort Myers Beach, FL). Concentration of active ingredient, determined by a DPD method outlined by Palin (11), was 4.5%. The pH of various test solutions was measured with a model 701A Orion pH meter (Orion Research Inc., Cambridge,

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MA). Surface tensions of chlorine solutions were measured with a Fisher Surface Tensiomat, model 21 (Fisher Scientific Corp., Pittsburgh, PA).

Tomato fruit, weighed to the nearest 10 mg, were exposed to immersion depths of 1–152 cm. For some treatments, a rubber mat weighted with beakers of water was used to force fruit below the surface of water contained in a sink. The distance from the surface of the water to the top of the fruit represented the immersion depth. In other treatments, fruit submerged in water in a 19-L pressure cooker were exposed to air pressure. Immersion depth was calculated from the pressure applied. After treatment, fruits were toweled dry, reweighed, and stored on trays at 24 C and >95% relative humidity for up to 2 wk. The treated fruit were observed daily for fruit rot. Diseased fruit were removed as needed to prevent secondary spread of disease. Locations of lesions (at wounds or inside fruit) were noted and type of rot—bacterial soft rot, sour rot, Rhizopus rot, or Alternaria rot—was diagnosed on the basis of visual symptoms and lesion pH (1). Data were analyzed with the Statistical Analysis System (SAS Institute, Cary, NC) software package for the personal computer. Appropriate programs (ANOVA, GLM) were used. Fruit weight affected the amount of water absorbed (J. A. Bartz, *unpublished*); therefore, water absorption was analyzed on the basis of percent weight increase of fruit. Analyses of covariance for sample weight were performed to determine if sample size contributed to variation in percent weight increase among treatments. In some tests, *F*-test values were used to assess the relative contribution of the different independent variables to variation in the dependent variable. Disease incidence was expressed as a percentage. The latter values were transformed by the arc sine square root method before analyses were performed. Statistical analyses unique to the different tests are detailed below.

Incidence of disease after storage of fruit infiltrated with solutions of hypochlorite and chlorine dioxide. Samples of cultivar Flora-Dade were submerged in aqueous solutions of chlorine or chlorine dioxide and then exposed to a hydrostatic pressure (152 cm) for a period of time (2 min) that caused some of the fruit to become infiltrated. Concentrations of chlorine represented 1-, 5-, and 10-fold recommended dosages, i.e., 100, 500, and 1,000 ppm Cl_2. The four 10-fruit replicates of each treatment were incubated 13 days. The incidence of decay was analyzed directly and also was divided by the weight increase so that disease among treatment replicates was based on an equivalent amount of water uptake.

Incidence of disease and weight increase of fruit infiltrated with chlorinated water as affected by immersion depth, period of immersion, and solution pH. Fruits of the cultivar Flora-Dade were submerged in 0, 50, or 150 ppm Cl_2 to a depth equivalent to 91 cm for 10 min or 152 cm for 2 min to determine if the rate of infiltration with chlorinated water would affect the incidence of fruit rots. In addition, to determine if the ratio of hypochlorous acid to hypochlorite ion affected water uptake or disease incidence, solution pH was adjusted to 6.8 or 9.6. Solutions of hypochlorite at pH 6.8 were prepared by adding an amount of Decco 311 buffer (PennWalt Inc., Monrovia, CA) equal to the amount of laundry bleach used or, for the water-alone treatment, adding a sufficient amount of 0.1 N NaOH to the amount of buffer used in the 50-ppm Cl_2 treatment. The pH 9.6 represents that of laundry bleach diluted to 150 ppm Cl_2. The pH of the solutions and concentration of free-chlorine did not change during treatment of fruits. The experimental design was factorial with three 10-fruit replicates. Disease incidence was totaled after 12 days of storage. Fruits were added to the respective solutions, inoculum was added (1×10^6 cfu of *E. c.* subsp. *carotovora* per milliliter), and fruits were stirred 3–5 sec, then exposed to one of the different hydrostatic pressure treatments.

Weight increase associated with prolonged submersion of tomato fruit in chlorinated water. Effects of chlorine concentration, period of immersion, and depth of immersion on water absorption were examined in a split-plot factorial test. Chlorine concentrations (100 or 250 ppm Cl_2), periods of immersion (10 or 20 min), and depths of immersion (1, 8, or 15 cm) represent those observed in commercial packinghouses in Florida. A prolonged exposure period, 60 min, was added to simulate fruit that become trapped behind flume diverters or remain in water during equipment breakdowns or lunch breaks. Fruit, cultivar Flora-Dade, were treated simultaneously at three depths in sinks filled with water; therefore, depth was analyzed as a subplot factor. The immersion depths represented one, two, or three layers of floating fruit. The layers were separated by plastic trays. Fifteen fruits, weighed individually, were treated at each combination of depth, time, and concentration. The pH of the solutions was not adjusted. The temperature of the solution, 26 C, was slightly below that of the fruit, 28 C. If fruit cooled the full 2 degrees C, the apparent depth of immersion would increase by about 7 cm. However, the water would warm particularly during the longer exposures. Therefore, the temperature effect would be constant among depth and concentration treatment combinations within each exposure period and might bias only comparisons of time period treatments.

After a 2-wk incubation period, fruit free of apparent disease were dissected and examined for internal lesions. The number of fruit with internal lesions was added to the number discarded with disease to provide total disease incidence. Fruit were not exposed to inoculum in the laboratory as part of the test. Therefore, all disease originated from casual or epiphytic populations of fruit rot pathogens that existed before the test.

In a second test of the period of immersion at depths found in packinghouses, fruit were treated in a factorial design with 0 or 100 ppm Cl_2, immersion depths of 2 or 15 cm, and immersion periods of 2 or 20 min. For weight increase, there were six five-fruit replicates of each treatment. These were combined to provide three 10-fruit replicates for disease incidence. Fruits were randomly selected from a bulk sample of Florida MH-1 and Hayslip fruits, rinsed for 5 sec or less in 5×10^6 cfu of *E. c.* subsp. *carotovora* per milliliter, and allowed to dry before treatment. The pH of the chlorine solutions was reduced to pH 7 by adding 1 N HCl. The temperature of both fruit and water at the time of treatment was about 26 C.

Surface tension measurements. For surface tension measurements, solutions were added to 100-ml beakers. The surface tension at the air-water interface was measured, then mineral oil was added. After the oil-water interface had stabilized, the surface tension at that interface was measured. There were three separate measurements of each interface. Serial 10-fold dilutions of 0.1%, w/v, Tergitol NPX (Fisher Scientific, Fair Lawn, NJ) in distilled water were measured for comparison.

RESULTS

Incidence of disease after storage of fruit infiltrated with solutions of hypochlorite and chlorine dioxide. Fruit rots were reduced but not prevented by adding hypochlorite ion to water before pressure-treating the fruits. The incidence of disease after 13 days was 37, 30, 24, and 18% for treatment with 0, 100, 500, and 1,000 ppm Cl_2, respectively. In contrast, addition of ClO_2 had no effect. Disease incidences ranged from 45 to 58% after treatment with 100 to 1,000 mg/L. Symptoms of phytotoxicity, sunken stem scars, developed on fruits treated with 500 and 1,000 mg/L.

Fruits treated with free-chlorine were free of external symptoms of phytotoxicity. In some fruits treated with the higher concentrations, craters were occasionally observed in the white connective tissue beneath the stem scar. The difference among the means for disease in fruits treated with hypochlorite was significant (*F*-test value = 7.68, $P > F = 0.004$). Adjustment of disease for water uptake improved the coefficient of determination (= R^2 or amount of variation in disease attributable to the treatments)

from 0.32 to 0.66.

Addition of chlorine to the water also increased the amount of water absorbed by the fruit (F-test value = 3.96, $P > F = 0.036$). The mean weight increase for the control fruits was 0.13%, whereas that of fruit in the three chlorine treatments was 0.29%. Only 8% of the fruit treated with water alone absorbed at least 0.5 g, whereas 37% of the fruit treated with chlorine absorbed that amount.

Incidence of disease and weight increase of fruit infiltrated with chlorinated water as affected by immersion depth, period of immersion, and solution pH. Two immersion treatments were used in an effort to achieve the same water uptake but at different rates. Water uptake by submerged fruits was not affected significantly by the treatments (Table 1). In contrast, disease incidence, particularly among fruits treated with water alone, was much higher among fruit infiltrated during the shorter time period. However, if disease was weighted by amount of water absorbed, then differences in incidence attributed to the immersion treatments were eliminated (analysis not shown).

Adding chlorine to the water before applying pressure decreased the incidence of fruit rots as expected; however, disease was observed among fruits treated with 50 or 150 ppm Cl_2 (Table 1). The first symptoms of disease, water-soaked areas adjacent to the stem scar, were observed on the second day after inoculation but only among fruit treated with water alone. By the fourth day, 35 of the total 39 diseased fruits were from the water-alone treatments. Thus, the chlorine treatment also delayed disease onset.

The effect of chlorine concentration on disease incidence was quadratic. The initial 50 ppm Cl_2 had more effect on disease than did the next 100 ppm. Chlorine concentration also affected water uptake but in a linear manner. Uptake increased directly with chlorine concentration. Concentration interacted with immersion treatment in a linear manner with regard to weight increase and in a quadratic manner with regard to disease incidence. In the first interaction, the effect of chlorine concentration on water absorption appeared to be greater among fruit infiltrated over the 10-min compared with the 2-min exposure period. In the second, the difference in incidence between the 0- and 50-ppm Cl_2 treatments was much greater among fruits infiltrated rapidly than among those treated more slowly, primarily because of the relatively high disease in the rapid infiltration, water-alone treatment. This interaction appeared to be caused by differences in water absorbed, because it was eliminated when incidence was weighted by uptake.

Disease incidence was lower among fruit treated at pH 9.6 than among those treated at 6.8. The pH treatment had no effect on water absorption. Weighting incidence by absorption narrowed the difference between the means from 14 to 6%, but the difference remained statistically significant ($P > F = 0.0133$).

Weight increase associated with prolonged submersion of tomato fruit in chlorinated water. Average weight increases for fruit treated in simulation of exposure extremes in packinghouse flumes and dump tanks ranged from 0.04 to 1.66%. The lower value was for fruits submerged to 1 cm in 250 ppm Cl_2 for 10 min, whereas the higher value was for fruits submerged in the same chlorine concentration but to a 15-cm depth for 60 min. Both the period and depth of immersion affected water uptake significantly (Table 2). Both affected uptake in a quadratic manner, because the difference in water uptake attributed to the initial increment (of period or depth) was much larger than that attributed to the subsequent increment.

The concentration of chlorine did not affect water uptake by submerged fruit significantly; however, it interacted with both depth and period of immersion. Both interactions had highly significant linear components, whereas the quadratic interaction of period with concentration also was highly significant and that of depth with concentration was significant at $P = 0.05$. Both interactions resulted from a lower uptake of the 250-compared with the 100-ppm Cl_2 solution at the shortest and shallowest exposures and a higher uptake of that concentration at the longest and deepest exposures, respectively.

The period and depth of immersion also interacted. Both independent variables in this interaction had linear and quadratic effects on water absorption. Differences attributed to the initial increment of depth or time were much greater than those attributed to subsequent increments except for depth at the 10-min exposure period, where the largest difference was associated with the second increment of depth.

Mean disease incidences were 27, 16, and 16% for fruits submerged to depths of 1, 8, and 15 cm, respectively, for the 250-ppm treatments. Means within the 100-ppm treatments were 18, 16, and 16%, respectively.

In the preceding experiment, the design did not include an adequate number of fruit for statistical analysis of

Table 1. Percent weight increase (%WI) and disease incidence (%DI) in tomato fruit as a result of submersion in chlorinated water[a]

	Main effects		Interactions			
			%WI		%DI	
Concentration (ppm)	%WI	%DI	91 cm/10 min	152 cm/2 min	91 cm/10 min	152 cm/2 min
0	0.13[b]	64[b]	0.11	0.14	48	79
50	0.14	29	0.13	0.16	26	32
150	0.18	19	0.19	0.16	16	22
F values						
Linear	14.06****[c]	150.00****	4.78*		9.45***	
Quadratic	0.00	38.17****	0.56		7.88**	
Immersion treatment						
91 cm/10 min	0.13	30				
152 cm/2 min	0.17	44				
F value	1.43	30.01****				
Solution pH						
6.8	0.14	42				
9.6	0.16	32				
F value	0.45	16.42****				

[a] Preweighed fruit were submerged in water with 0–150 mg Cl_2/L at pH 6.8 or 9.6. Cells of *Erwinia carotovora* subsp. *carotovora* were added (10^6 cfu/ml), the mixture was stirred about 4 sec, and 91 or 152 cm hydrostatic pressure was applied for 10 or 2 min, respectively. The fruit were removed, reweighed, and stored at 24 C for 12 days.

[b] Each value is the average of three 10-fruit replicates.

[c] Probability of a greater F-test value: * = 0.05, ** = 0.01, *** = 0.001, and **** = 0.0001.

disease incidence. Therefore, in an additional test, period of immersion in water, with or without a recommended level of chlorine and depths that simulated packinghouse operation were compared. Fruit in this test were not as porous as those used in the previous test. The average weight increase for fruit submerged to 15 cm for 20 min was 0.09% compared with 1.13% in the earlier test. None of the fruit submerged to 1 cm in 100 ppm Cl_2 for 2 min were diseased after 8 days, whereas 50% of fruit submerged to 14 cm in water alone for 20 min developed lesions during that storage interval. As in previous tests, adding chlorine to water before depth treatment of submerged fruit did not prevent the later development of fruit rots if infiltration occurred and did increase the amount of water absorbed (Table 3). Chlorination did not affect the incidence of disease significantly unless the latter was weighted by water absorption, ranked, and then analyzed. In that analysis, addition of chlorine was the only independent test variable to affect disease (F-test value = 7.14 and $P > F = 0.0167$).

The weight increase for the five-fruit samples ranged from 0.00 to 2.67 g. The lowest treatment mean, 0.00 g/sample, was recorded for fruit submerged to 1 cm for 2 min in water alone, whereas the highest, 1.64 g/sample, was for fruit submerged to 15 cm in 100 ppm Cl_2 for 20 min.

Water uptake but not disease incidence was affected by interactions of period of exposure with both depth and addition of chlorine (Table 3). In the first interaction, the effect of depth on water uptake was more pronounced at the longer exposure period. In the second interaction, the effect of depth was more pronounced when chlorine was present.

Effect of chlorination on water surface tension. Addition of chlorine from commercial laundry bleach to water at concentrations of 62.5–1,000 ppm Cl_2 had little effect on the surface tension at the air-water interface. Such additions had a small effect on the surface tension at a mineral oil-water interface. Surface tensions at the air-water interface for 100 ppm Cl_2, 1,000 ppm Cl_2, 0.01% Tergitol NPX, and 0.1% Tergitol NPX were 76, 65, 35, and 35 dynes/cm^2, respectively. At a mineral oil-water interface, the values were 47, 39, 6, and 4 dynes/cm^2, respectively. Values for water alone were 77 and 49 for interfaces with air and oil, respectively.

Table 2. Percent weight increase (%WI) for tomato fruit submersed in chlorinated water[a]

Main effects		Interactions				
		Concentration (ppm)		Depth (cm)[b]		
Period of immersion (min)	%WI	100	250	1	6	15
10	0.09	0.13	0.07	0.05	0.08	0.17
20	0.78	0.86	0.69	0.31	0.89	1.13
60	1.03	0.93	1.14	0.21	1.49	1.41
F values						
Linear	378.2****[c]	20.90***		D-Lin. 45.3***	P-Lin. 79.6****	
Quadratic	173.3****	5.31*		−Quad. 49.0****	−Quad. 14.7***	
Depth (cm)						
1	0.18	0.27	0.11			
6	0.81	0.81	0.83			
15	0.90	0.85	0.95			
F values						
Linear	222.4****	11.92***				
Quadratic	99.4****	0.96				
Concentration (ppm)						
100	0.64					
250	0.65					
F value	0.02					

[a] Fifteen tomato fruits were weighed individually, submerged in 100 or 250 ppm aqueous solution of chlorine for 10, 20, or 60 min, removed, and then reweighed. Depth, the subplot factor, was 1, 6, or 15 cm. Statistics were performed on percent increase in weight.
[b] Error term for depth was replicate × depth.
[c] Probability of a greater F-test value: * = 0.05, ** = 0.01, *** = 0.001, and **** = 0.0001.

Table 3. Percent weight increase (%WI) and disease incidence (%DI) in tomato fruit that were contaminated with *Erwinia carotovora* subsp. *carotovora*, submerged in chlorinated water, and then incubated for 8 days[a]

Main effects			Interactions (wt inc)			
			Depth (cm)		Concentration (ppm)	
Period of immersion (min)	%WI	%DI	1	15	0	100
2	0.01[b]	13	0.01	0.02	0.03	0.05
20	0.10	30	0.06	0.15	0.04	0.11
F value	62.19****[c]	5.18*	15.10***		4.77*	
Concentration (ppm)						
0	0.04	18				
100	0.08	25				
F value	11.46**	1.75				
Depth (cm)						
1	0.03	14				
15	0.08	28				
F value	19.08****	6.82*				

[a] Tomato fruit were rinsed in an aqueous suspension of 5×10^6cfu *E. c.* subsp. *carotovora* per milliliter, allowed to dry, weighed, submerged in water with or without chlorine for indicated time period, reweighed, and then incubated at 24 C.
[b] There were six five-fruit replicates for weight increase. These were combined to provide three 10-fruit replicates for disease.
[c] Probability of a greater F-test value: * = 0.05, ** = 0.01, *** = 0.001, and **** = 0.0001.

DISCUSSION

The risk of postharvest disease associated with absorption of water by submerged tomato fruits in commercial handling is reduced but not eliminated by adding chlorine to the water. Previously, the ability of recommended levels of chlorine to inactivate, on contact, pathogens that accompany fruit entering packinghouse handling systems was questioned (3). A rapid kill would be necessary for those situations where fruits begin water absorption within seconds after immersion (2). For adequate control of decay, pathogens must be inactivated before they become embedded in potential infection courts (8).

High concentrations of free-chlorine, higher than six times recommended levels, did not prevent postharvest rots if fruits became infiltrated with water. Addition of chlorine reduced disease significantly compared with the water-alone treatments; however, chlorination enhanced infiltration. Higher weight increases were recorded for fruits submerged in chlorinated water compared with water alone. Water absorption was a critical factor in disease incidence. When disease was based on equivalent water uptake, then differences attributed to period or depth of exposure were no longer significant.

Most of the fruit rots observed were consistent with inoculation by infiltration. Relatively few lesions were associated with wounds on the fruit surfaces. Many were located adjacent to or beneath the stem scar. In commercial handling, fruits that become inoculated probably do so through infiltration. Fruits with noticeable wounds would be culled, whereas maintenance of free-chlorine in the system would prevent inoculation of many of the small wounds that escaped detection.

In the tests reported here, the quadratic (curvilinear) effect of multiple levels of period and depth of immersion on water uptake was suggestive of a threshold for infiltration. At some combination of depth and period of immersion, water intrusion into stem scar tissues begins. At or near the threshold, incremental increases in depth and/or period would have a larger effect on water uptake than at increments above or below the thresholds. Additional nonlinearity would occur as the water absorbed saturates available intercellular spaces in the core of the fruits (Table 2). Absorption of water above this amount apparently is possible and leads to deep cracks in the shoulder of fruits (15).

Chlorination also appeared to have a nonlinear effect on disease incidence. Use of percent values to rate the effect of chlorine concentration on disease is closed ended and could account for some nonlinearity. However, initial increments of free-chlorine would destroy inoculum suspended in the water as well as exposed inoculum on fruit surfaces and thus would have more effect on disease than additional increments that could act only on the partially and fully embedded pathogens that remain.

Recommendations for dump tank management need provisions for depths and periods of immersion that are below the threshold for infiltration. Both parameters need to be controlled. However, based on *F*-test values (Tables 2 and 3), the period of immersion over the range of 2–60 min appeared to have more influence on water uptake than did depth in the range of one to three layers of fruit.

Theoretically, a slower rate of water uptake would allow hypochlorous acid more time to inactivate pathogens before they became embedded in stem scar tissues. When this possibility was addressed (Table 1), however, the amount of water absorbed rather than the rate of absorption was the critical factor in disease incidence. Moreover, chlorine enhanced infiltration more at longer exposure intervals. Thus, control of the immersion period would be more important than control of depth, particularly if the tank design precludes the stacking of several layers of fruit.

The enhancement of water uptake effect associated with chlorination was not caused by the source of hypochlorite or by hypochlorite per se. The surface tension of the chlorine solutions was similar to that of water alone and differed from that of solutions of the surfactant Tergitol-NPX, which previously was associated with increased infiltration (2). Fruits wetted with chlorinated water feel "greasy," evidence for some effect of hypochlorous acid on natural fruit waxes. Chlorine may erode the hydrophobic constituents on the surface of the stem scar and, as a result, facilitate water intrusion into those tissues. This would be consistent with the observed interaction of chlorination with period of immersion.

Solution pH is important to the efficacy of chlorinated water as a disinfectant (7,9,10,13). At pH 6, nearly 98% of the hypochlorite ion added to water is in the form of hypochlorous acid, whereas at pH 9.6, nearly 99% is in the ion form (7). The acid form is up to 80 times more bactericidal than the ion form. In several different tests on the effect of chlorinated water on viruses and bacteria, decreases in the pH of the solution were associated with increases in efficacy. In one of the tests reported here, however, significantly less disease was observed among fruit treated with chlorinated water at pH 9.6 than at pH 6.8, a result completely opposite that expected. Because the ion form is more stable (less reactive) than the acid form, perhaps at pH 9.6 free-chlorine solutions penetrated more deeply into stem scar tissues than did the pH 6.8 solutions and, as a result, inactivated more pathogens.

Chlorine dioxide from a stabilized commercial product had no effect on postharvest disease when fruit were infiltrated; however, the pH of the solutions was not modified. There is some evidence that stabilized chlorine dioxide must be acidified to release the active ingredient. Directions for acidification (pH 4) are found on some product labels. Thus, stabilized chlorine dioxide has different characteristics from the form generated on site, which appears to be effective over a wide range of pHs (7). The generated form may be more effective against fruit rot pathogens at the pHs found in most dump tanks.

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