Toxicity of the Surfactant Nacconol to Four Decay-Causing Fungi of Fresh-Market Tomatoes

M. W. HOY, Postgraduate Research Plant Pathologist, and J. M. OGAWA, Professor, University of California, Davis 95616

ABSTRACT

Hoy, M. W., and Ogawa, J. M. 1984. Toxicity of the surfactant Nacconol to four decay-causing fungi of fresh-market tomatoes. Plant Disease 68:699-703.

The anionic surfactant Nacconol 90F (active ingredient sodium dodecylbenzene sulfonate) was fungistatic in vitro to mycelia and spores of four decay-causing fungi of California tomato fruit. ED_{50} values for *Botrytis cinerea, Geotrichum candidum, Phytophthora parasitica*, and *Rhizopus stolonifer* were 23, 20, 75, and 27 μg a.i./ml for mycelial growth and 116, 60, 60, and 136 μg a.i./ml for spore germination, respectively. Nacconol was fungicidal to *B. cinerea* spores at concentrations greater than 5,000 μg a.i./ml. In laboratory tests, *B. cinerea* decay of injured fresh-market tomatoes was significantly reduced by treating the fruit in Nacconol solutions of 200, 2,000, or 5,000 μg a.i./ml for 3 min at 38 C followed by a fresh-water rinse. However, Nacconol amendments of 200, 2,000, or 5,000 μg/ml did not enhance the ability of 100- or 400-μg/ml chlorine solutions to reduce the incidence of *B. cinerea* decay. When the water rinse was omitted, Nacconol amendments significantly improved the efficacy of 100-μg/ml but not 400-μg/ml chlorine treatments. Phytotoxicity symptoms developed when fruit were not rinsed after treatment with 2,000 or 5,000 μg/ml Nacconol alone or in combination with chlorine.

Additional key words: postharvest decays

Surface-active agents, or surfactants, are compounds that reduce the surface tension of solutions (10). Certain surfactants have fungistatic properties (3,7) and have been used to control plant diseases including apple scab (4) and apple powdery mildew (6). Postharvest pear decays can be reduced significantly by adding surfactants to postharvest disinfectant chlorine washes (14).

Commercial postharvest treatments to control decay on fresh-market tomatoes (*Lycopersicon esculentum* Mill.) in California usually involve dipping mature-green fruit in heated (38–42 C), chlorinated water, followed by a fresh-water or chlorinated water spray rinse and application of a wax containing 2.5% ortho-phenylphenol. California tomato packers have sought to improve control of decay by adding the anionic surfactant Nacconol 90F to chlorinated water in dump tanks. A similar surfactant, Santomerse F85, which contains the active ingredient of Nacconol, has been reported to reduce postharvest decays caused by *Erwinia carotovora* (Jones) Bergey et al, *Aerobacter cloacae* (Jordan) Bergey et al, and *Alternaria tenuis* Nees when added to chlorine for field-washing tomato fruit (12). This paper reports on the fungitoxicity of Nacconol to four common decay-causing fungi on California fresh-market tomatoes: *Botrytis cinerea* Pers. ex Fr., *Geotrichum candidum* Link ex Pers., *Phytophthora parasitica* Dastur, and *Rhizopus stolonifer* (Ehrenb. ex Fr.) Lind. Additionally, we report some results on the efficacy of Nacconol amendments to heated chlorine washes for postharvest decay control on fresh-market tomatoes.

To simplify information, trade names of products have been used. No endorsement of named products is intended, nor is criticism implied of similar products not mentioned.

Accepted for publication 17 February 1984.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

MATERIALS AND METHODS

Effects of Nacconol on mycelial growth of fungi. The isolates of *B. cinerea, G. candidum, P. parasitica,* and *R. stolonifer* used in this study were obtained from infected tomato fruit. In vitro toxicity of Nacconol 90F (active ingredient sodium dodecylbenzene sulfonate) to mycelial growth of *B. cinerea, G. candidum*, and *R. stolonifer* was determined on Difco potato-dextrose agar (PDA) amended with 4–128 μg a.i./ml Nacconol. Toxicity of Nacconol to mycelial growth of *P. parasitica* was evaluated on V-8 agar (V-8A) (200 ml of V-8 juice, 2 g of $CaCO_3$, 800 ml of distilled water, and 17 g of agar) amended with 50–175 μg a.i./ml Nacconol. Preliminary experiments determined that autoclaving had no effect on the activity of Nacconol; therefore, proprietary Nacconol 90F (Stepan Chemical Company, Northfield, IL 60093) was added to PDA or V-8A before autoclaving. All plates contained 25 ml of V-8A or PDA. Four-millimeter disks of 2-day-old *B. cinerea* or *G. candidum* colonies or 1-day-old nonsporulating *R. stolonifer* colonies on PDA or 3-day-old *P. parasitica* colonies on V-8A were transferred to Nacconol-amended media. Controls consisted of disks transferred to PDA or V-8A without Nacconol. Plates were incubated at the optimum temperature for mycelial growth of each fungal isolate: *B. cinerea* at 21 C, *G. candidum* and *P. parasitica* at 30 C, and *R. stolonifer* at 27 C. Colony diameters for six replicates of each concentration were measured when growth of controls reached the edges of the plates.

Growth inhibition was calculated as percent inhibition of mycelial growth relative to fungicide-free controls. Linear regression equations fitted to data by using logarithms for each concentration, and probits for percentage growth inhibition (2) were used to determine ED_{50} values (concentration resulting in 50% growth inhibition). Dosage-response curves were plotted on logarithmic-probability paper (15).

Fungistatic effects of Nacconol on spore germination of fungi. On the basis of preliminary trials, the fungistatic effects of Nacconol on spore germination were tested on PDA amended with 100–125 μg a.i./ml for *B. cinerea*, 50–75 μg a.i./ml for *G. candidum*, and 100–185 μg a.i./ml for *R. stolonifer*. Fungistatic effects on *P. parasitica* zoospores were tested on 50–70 μg a.i./ml of Nacconol-amended Difco cornmeal agar (CMA). Spores were obtained from 9-day-old *B. cinerea*, 11-day-old *G. candidum*, and 4-day-old *R. stolonifer* cultures on PDA, and spore suspensions in sterile distilled water (SDW) were adjusted to 5×10^4 conidia per milliliter. Suspensions of *P. parasitica* zoospores, which were produced by a previously described method (13), were adjusted to 4×10^4 zoospores per milliliter. The suspension was agitated briefly to induce encystment of zoospores. Six replicate 50-μl drops of spore or zoospore suspensions were then pipetted onto Nacconol-amended media. Controls consisted of spores added to PDA or CMA without Nacconol. Plates of *B. cinerea, P. parasitica, R. stolonifer,* and *G. candidum* were incubated for 24 hr at 21, 24, 27, and 30 C, respectively. Fifty spores were examined for each replicate and were considered germinated when the length of the germ tube equaled or exceeded the length of the spore. ED_{50} values were calculated and dosage-response curves were plotted as described

for mycelial growth inhibition.

To determine the effect of Nacconol on germ tube elongation of *B. cinerea* spores, germ tubes were measured 6, 8, and 12 hr after ungerminated spores were placed on 0, 10, or 100 μg a.i./ml of Nacconol-amended PDA. To determine the effect of Nacconol on germ tube elongation of previously germinated *B. cinerea* spores, sterile cellophane squares (18 mm^2) were placed on Nacconol-free PDA. *B. cinerea* spores were allowed to germinate for 6 hr at 21 C on the cellophane squares. The cellophane squares with germinating spores were then transferred to 0, 10, or 100 μg a.i./ml of Nacconol-amended PDA, incubated at 21 C, and further germ tube elongation was measured 6 hr after the transfer. Spore suspensions for germ tube elongation studies were prepared as described for fungistatic effects on spore germination. Germ tube measurements were recorded for 30 spores per concentration. Analysis of variance was used to test the main effects and the interaction of Nacconol concentration and time. The Bonferroni pairwise comparison procedure, which ensures that all pairs are compared at the same level of significance (5), was used to separate means.

Fungicidal effect of Nacconol on *B. cinerea* spores. In several trials to determine the fungicidal effects of Nacconol on *B. cinerea* spores, equal volumes of suspensions of 9-day-old *B. cinerea* spores ($4.5–5 \times 10^5$ spores per milliliter) and Nacconol were combined in sterile test tubes. In different experiments, final Nacconol concentrations were 750–1,000, 1,000–5,000, or 1,000–10,000 μg a.i./ml. Controls consisted of spore suspensions combined with SDW. After incubation for 20 hr at 21 C, the Nacconol/spore suspensions were centrifuged for 90 sec at 3,000 rpm. The spore pellet was rinsed five times by discarding the supernatant, resuspending the pellet in 10 ml of SDW, and centrifuging for 90 sec. Finally, 50-μl drops of each suspension (average concentration 5×10^4 spores per milliliter) were pipetted onto Nacconol-free PDA and the number of germinated spores was determined after 12 hr.

Effects of Nacconol and chlorine treatments on *B. cinerea* on green tomato fruit. Nacconol and Nacconol-amended chlorine treatments were evaluated for control of *B. cinerea* on healthy, mature-green tomato fruit (cultivar Royal Flush) harvested from a field plot in Davis. Fruit were washed in tap water and allowed to dry and equilibrate to room temperature (24–25 C) overnight. Three 2-mm-deep wounds were made near the stem end of each fruit with the tip of a 2-mm-wide finishing nail and inoculated with 25-μl drops of *B. cinerea* spore suspension (1×10^5 spores per milliliter). Spores were obtained from 9- to 14-day-old cultures on PDA maintained under continuous fluorescent light. Inoculated fruit were incubated for 3 hr before treatment with 0, 200, 2,000, or 5,000 μg a.i./ml of Nacconol; 0, 100, or 400 μg/ml of available chlorine; or combinations of Nacconol and chlorine. All chlorine solutions were prepared from 5.25% sodium hypochlorite and remained stable throughout the treatment period. Concentrations of available chlorine were determined by titration with sodium thiosulfate (1). Nacconol solutions without chlorine ranged from pH 6.6 to 6.9. Solutions containing chlorine or both Nacconol and chlorine were adjusted with HCl to pH 7.5. Controls consisted of inoculated fruit treated with water. All treatment solutions were heated to 38 C as described previously (9). Fruit were immersed and agitated for 3 min in treatment solutions, rinsed in tap water, and incubated at 20 C. In some experiments, the tap water rinse was omitted to determine if the efficacy of treatment solutions could be improved. Disease was evaluated after 7–9 days at 20 C. The data were analyzed statistically by the procedures described for the effects of Nacconol on germ tube elongation.

The efficacy of Nacconol, chlorine, and Nacconol plus chlorine treatments was evaluated further for decay control at a commercial fresh-market tomato packing shed with two dump tanks in King City, CA. Fruit (cultivar Castlemart) were treated for a total of about 3 min in the two dump tanks. Both dump tanks contained chlorinated water amended with either 0, 200, or 2,000 μg a.i./ml of Nacconol. Available chlorine concentrations averaged 98 μg/ml in the first dump tank and 190 μg/ml in the second dump tank. The average pulp temperature of fruit arriving at the shed was 16 C and temperatures in the first and second dump tank averaged 28 and 23 C, respectively. All fruit except controls were sprayed with 100 μg/ml of available chlorine as they came out of the second dump tank. Control fruit were washed in the dump tanks before addition of chlorine or Nacconol. Control and treated fruit were then coated with a wax containing 2.5% ortho-phenylphenol and packed in 25-lb boxes. Ten boxes of fruit per treatment were incubated at 20 C and observed for symptom development for 11 days.

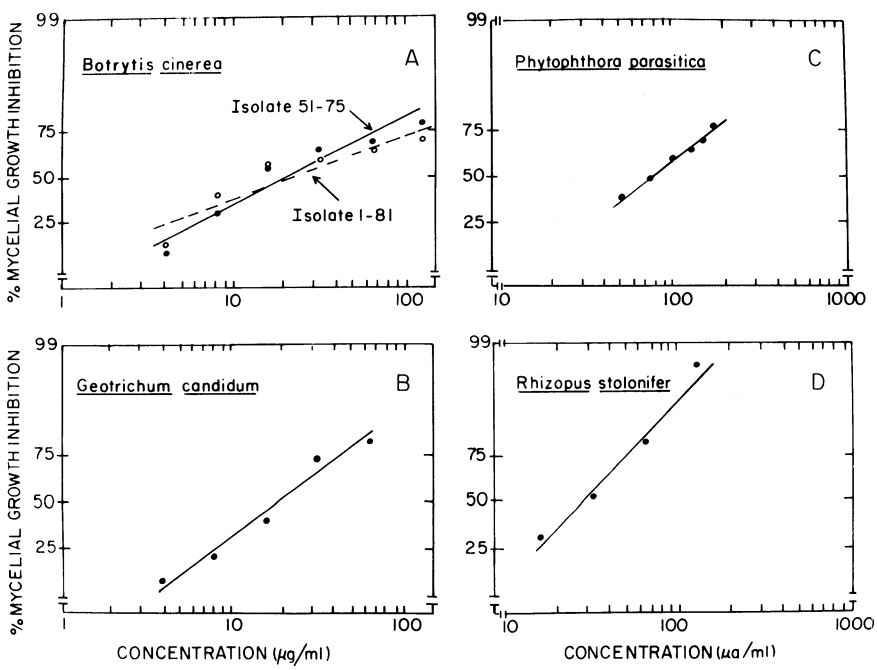

Fig. 1. Dosage-response curves for in vitro toxicity of Nacconol 90F to mycelial growth of **(A)** *Botrytis cinerea* isolates 1-81 and 51-75, **(B)** *Geotrichum candidum*, **(C)** *Phytophthora parasitica*, and **(D)** *Rhizopus stolonifer* 3, 7, 3, or 2 days, respectively, after mycelial disks from Nacconol-free media were placed on Nacconol-amended media. Correlation coefficients were significantly positive at $P \leq 0.01$.

RESULTS

Effects of Nacconol on mycelial growth of fungi. Nacconol was fungistatic to mycelial growth of *B. cinerea, G. candidum, P. parasitica,* and *R. stolonifer*. The ED_{50} values were 23 and 21 μg/ml for *B. cinerea* isolates 1-81 and 51-75, respectively (Fig. 1A), and 20, 75, and 27 μg/ml for *G. candidum, P. parasitica,* and *R. stolonifer*, respectively (Fig. 1B–D). Mycelial growth of *B. cinerea, G. candidum, P. parasitica,* and *R. stolonifer* was reduced by more than 90% at concentrations of 500, 100, 175, and 64 μg/ml, respectively. Nacconol strongly affected mycelial growth of *R. stolonifer* (Fig. 2). Concentrations of Nacconol greater than 16 μg/ml caused restricted, patchy growth of *R. stolonifer* on the agar surface and increased production of aerial mycelium originating on the mycelial plug. At 128 μg/ml, only aerial

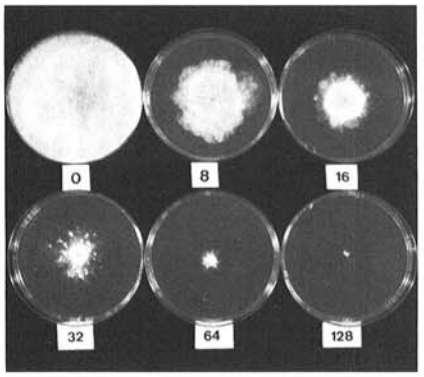

Fig. 2. Fungistatic effect of Nacconol 90F on mycelial growth of *Rhizopus stolonifer* 2 days after 4-mm disks from nonsporulating *R. stolonifer* colonies were placed on potato-dextrose agar amended with 0, 8, 16, 32, 64, and 128 μg a.i./ml of Nacconol.

mycelium was produced. Sporangium production on aerial mycelium was limited or absent.

Fungistatic effect of Nacconol on spore germination of fungi. Nacconol was fungistatic to spore germination of *B. cinerea, G. candidum, P. parasitica,* and *R. stolonifer*. Higher concentrations of Nacconol were required for inhibition of spore germination than for inhibition of mycelial growth except for *P. parasitica,* for which the concentrations required were similar. Spore germination ED_{50} values were 116 and 105 μg/ml for isolates 1-81 and 51-75 of *B. cinerea,* respectively (Fig. 3A), and 60, 60, and 136 μg/ml for *G. candidum, P. parasitica,* and *R. stolonifer*, respectively (Fig. 3B–D).

Both Nacconol concentration and the length of time spores were exposed to Nacconol had a significant effect ($P = 0.01$) on germ tube elongation (Fig. 4). The interaction between Nacconol concentration and length of exposure was also significant ($P = 0.01$), indicating that the response to Nacconol depended on the length of exposure. Twelve hours after *B. cinerea* (isolate 1-81) spores were placed on 10 or 100 μg/ml of Nacconol-amended PDA, germ tubes averaged 64 or 34 μm in length, respectively, whereas germ tubes of spores not treated with Nacconol were significantly longer, averaging 117 μm (Fig. 4). Similarly, when *B. cinerea* spores with an average germ tube length of 34 μm after 6 hr on Nacconol-free PDA were transferred to Nacconol-amended PDA, further elongation of germ tubes was significantly suppressed ($P = 0.05$). Six hours after the transfer, germ tubes of isolate 1-81 averaged 114 μm in length on Nacconol-free PDA, whereas spores transferred to 10 or 100 μg/ml of Nacconol-amended PDA averaged 82 or 54 μm, respectively. Germ tubes of Nacconol-treated spores were shortened, thickened, swollen, distorted, and tended to curl around the spore.

Fungicidal effect of Nacconol on *B. cinerea* spores. The fungicidal effects of Nacconol at concentrations of 500–5,000 μg/ml varied in five trials with different concentration ranges. A 20-hr treatment with 1,000-μg/ml Nacconol solutions prevented germination of as few as 7.7% of spores in one trial or as many as 69.7% in another trial. For all trials, a mean of 98.6% of spores germinated when treated with SDW. Nacconol was consistently fungicidal to spores of *B. cinerea* when concentrations exceeded 5,000 μg/ml. ED_{50} values for the fungicidal activity of Nacconol were not calculated because the variability among trials prevented selection of a concentration range within which fungicidal effects increased gradually and consistently.

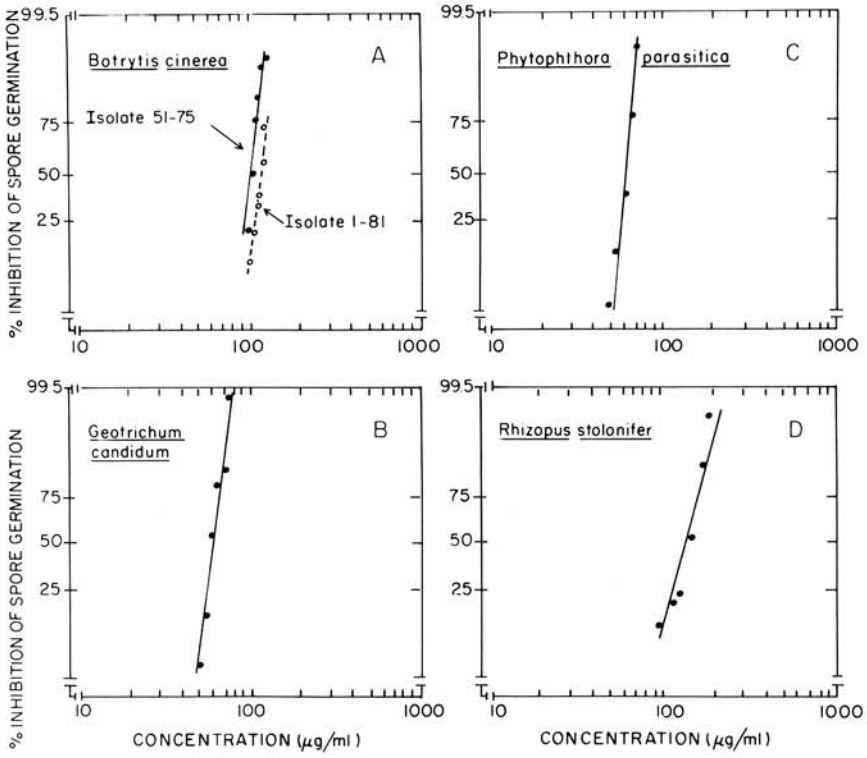

Fig. 3. Dosage-response curves for in vitro fungistatic effects of Nacconol 90F on spore germination of **(A)** *Botrytis cinerea* isolates 1-81 and 51-75, **(B)** *Geotrichum candidum,* **(C)** *Phytophthora parasitica*, and **(D)** *Rhizopus stolonifer,* 24 hr after ungerminated spores were placed on Nacconol-amended media. Correlation coefficients were significantly positive at $P \leq 0.01$.

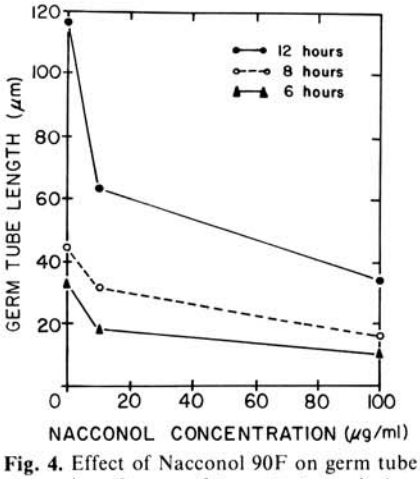

Fig. 4. Effect of Nacconol 90F on germ tube elongation of spores of *Botrytis cinerea* isolate 1-81. Ungerminated spores were placed on Nacconol-amended potato-dextrose agar and germ tubes were measured 6, 8, and 12 hr later. Each value represents the mean of 30 germ tube measurements. Mean differences that exceed 19.5 are significant ($P = 0.05$) according to the Bonferroni comparison procedure.

Nacconol and chlorine treatments on green tomato fruit. In the laboratory, decay caused by *B. cinerea* was significantly ($P = 0.01$) reduced when inoculated green tomato fruit were washed in Nacconol solutions of 200, 2,000, or 5,000 μg/ml and rinsed in tap water (Fig. 5). A significant ($P = 0.01$) interaction between Nacconol and chlorine indicated that the response to Nacconol depended on the concentration of chlorine. Treatment with 200, 2,000, and 5,000 μg/ml of Nacconol alone resulted in significant decay reductions that averaged 38.8, 64.4, and 71.5%, respectively. There was no significant difference between 2,000- and 5,000-μg/ml Nacconol treatments, although 2,000 and 5,000 μg/ml were significantly more effective than 200 μg/ml of Nacconol. When fruit were treated with 100 or 400 μg/ml of chlorine alone, the incidence of decay was significantly reduced by 56.7 or 60.7%, respectively. Addition of 200 μg/ml of Nacconol to 100- or 400-μg/ml chlorine solutions further reduced the incidence of decay by 7.8 or 14.5%, respectively (Fig. 5), but the reductions were not statistically significant. Although the lowest incidence of decay was observed for fruit treated in chlorine solutions amended with 5,000 μg/ml of Nacconol, the differences among 200-,

2,000-, and 5,000-μg/ml Nacconol-amended and Nacconol-free chlorine solutions were not significant (Fig. 5). When fruit were not rinsed after treatment, the efficacy of 100-μg/ml chlorine solutions was significantly ($P = 0.05$) improved by addition of 200, 2,000, or 5,000 μg/ml of Nacconol. Nacconol amendments to 400-μg/ml chlorine solutions made no significant difference in the incidence of decay, however, even when the final rinse was omitted. At concentrations of 2,000 or 5,000 μg/ml of Nacconol alone or Nacconol in combination with 100 or 400 μg/ml of chlorine, green fruit that were not rinsed after treatment developed phytotoxicity symptoms characterized by irregular brown spots 2–8 mm in diameter (Fig. 6A,B). Although the spots were superficial discolorations and fruit tissue beneath the spots appeared green and healthy, damaged fruit were considered not marketable. Treatment with 200 μg/ml of Nacconol alone or with chlorine did not cause phytotoxicity symptoms whether or not the fruit received a final rinse.

In a trial at a packing shed in King City, CA, the overall incidence of postharvest decay was low, resulting in little difference among treatments. Decay caused by *B. cinerea* and total decay, including Geotrichum, Phytophthora, and Rhizopus rots, were slightly but not significantly reduced when chlorine solutions in the two dump tanks were amended with 200 μg/ml of Nacconol. An increase in the concentration of Nacconol from 200 to 2,000 μg/ml made no significant difference in the incidence of *B. cinerea* or other decays but caused excessive foaming in the dump tanks. Fruit treated with Nacconol and chlorine appeared to dry more quickly than fruit washed only in chlorine.

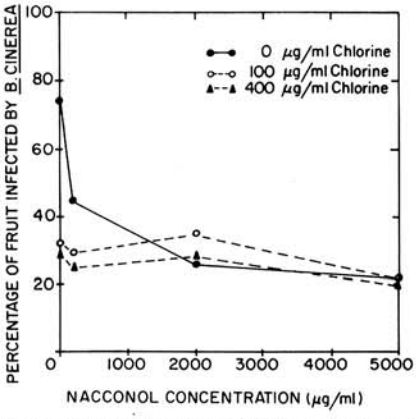

Fig. 5. Effect of Nacconol 90F amendments of 0, 200, 2,000, or 5,000 μg/ml to chlorine washes of 0, 100, or 400 μg/ml at 38 C on the incidence of *Botrytis cinerea* decay on green tomato fruit. Each data point is an arc sine $\sqrt{\text{percentage}}$ transformation and represents the mean of three experiments, each with four replicates of 12 fruits each. Mean differences that exceed 19.8 are significant ($P = 0.05$) according to the Bonferroni procedure.

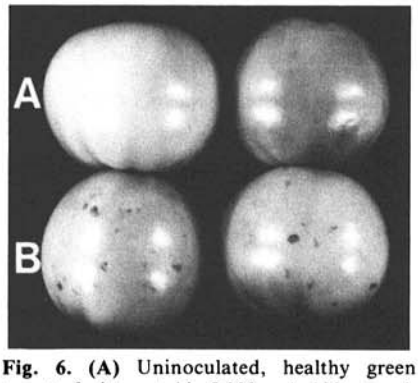

Fig. 6. (A) Uninoculated, healthy green tomato fruit treated in 5,000 μg/ml Nacconol, then rinsed in water, and **(B)** phytotoxicity symptoms on green tomato fruit treated in 5,000 μg/ml Nacconol but not rinsed in water.

DISCUSSION

Nacconol has been used for several years in California fresh-market tomato packinghouse dump tanks without evidence that it contributes to control of postharvest decay. Nacconol is fungistatic in vitro to mycelial growth and spore germination of four important decay-causing fungi on tomatoes in California, *B. cinerea*, *G. candidum*, *P. parasitica*, and *R. stolonifer*. ED_{50} values for mycelial growth and spore germination are similar to those reported for other anionic surfactants (7) and fatty acids (11). Considerably higher (5,000–10,000 μg/ml) Nacconol concentrations were required for fungicidal activity against *B. cinerea* spores, indicating that the fungistatic, not fungicidal, properties of Nacconol were responsible for the decay control achieved when injured tomato fruit inoculated with *B. cinerea* were treated in 200, 2,000, or 5,000 μg/ml of Nacconol (Fig. 5). Additionally, surfactant properties of Nacconol may have increased its penetration into wounds (8) and into the cuticle by dissolving oils and waxes on fruit surfaces.

Although *B. cinerea* decay was controlled by Nacconol solutions, Nacconol solutions alone were no more effective for decay control than were chlorine solutions alone (Fig. 5). Nacconol amendments to chlorine solutions did not significantly enhance decay control unless the chlorine concentration was low (100 μg/ml) and the treatment solution was not removed by a final rinse. At 400 μg/ml of chlorine, the fungistatic effects of Nacconol apparently were not sufficient to enhance the activity of the chlorine, whether or not treated fruit were rinsed. The surfactant must remain on fruit surfaces in order to exert the maximum fungistatic effect. In practice, however, a final rinse is necessary to remove debris as fruit leave the dump tank. A final rinse may also be needed to prevent phytotoxicity at high concentrations of Nacconol. Phytotoxicity could occur if 2,000 μg a.i./ml, the maximum concentration of Nacconol registered for postharvest washes (Parts 170–199 of Code of Federal Regulations 21, revised as of 1 April 1982), were used without a rinse. However, because 2,000 μg/ml of Nacconol caused excessive foaming in the dump tanks and was not significantly more effective than 200 μg/ml, use of such high concentrations is undesirable.

Tomato packers are likely to continue to use Nacconol, in part because it has been suggested that Nacconol-amended chlorine solutions facilitate fruit drying. The data presented here indicate that Nacconol amendments to chlorine in packing shed dump tanks contribute little to decay control. However, the non-significant reductions in decay that occurred in three laboratory experiments using 200-μg/ml Nacconol amendments averaged 7.8 and 14.5% for 100 and 400 μg/ml of chlorine, respectively. Large variations between treatment and experiment replicates caused these reductions to be nonsignificant in the laboratory, but for large tomato packing sheds, these reductions nevertheless might be economically significant. Further packing shed tests are needed to determine whether Nacconol could significantly enhance the activity of chlorine under commercial conditions when the incidence of decay is high.

ACKNOWLEDGMENTS

This research was supported by a grant from the California Fresh Market Tomato Advisory Board. We gratefully acknowledge the assistance of M. Miller, Senior Statistician, Statistics Laboratory, University of California, Davis, and of B. T. Manji, E. A. Bose, and R. L. Meyer.

LITERATURE CITED

1. American Public Health Association. 1976. Standard Methods for Examination of Water and Wastewater. 14th ed. Washington, DC.
2. Bliss, C. J. 1935. The calculation of the dosage-mortality curve. Ann. Appl. Biol. 22:134-167.
3. Brown, J. F., and Sisler, H. D. 1960. Mechanisms of fungitoxic action of *N*-dodecylguanidine acetate. Phytopathology 50:830-839.
4. Burchill, R. T., and Swait, A. A. J. 1977. Eradication of the perithecial stage of apple scab with surfactants. Ann. Appl. Biol. 87:229-231.
5. Chew, V. 1977. Comparisons among treatment means in an analysis of variance. U.S. Dep. Agric. Res. Serv. ARS/H/6. 64 pp.
6. Clifford, D. R., and Hislop, E. C. 1975. Surfactants for the control of apple mildew. Pestic. Sci. 6:409-418.
7. Forsyth, F. R. 1964. Surfactants as fungicides. Can. J. Bot. 42:1335-1347.
8. Furmidge, C. G. L. 1959. Physico-chemical studies on agricultural sprays. II. The phytotoxicity of surface-active agents on leaves of apple and plum trees. J. Sci. Food Agric. 10:274-282.
9. Ogawa, J. M., and Hoy, M. W. 1982. Pre- and postharvest diseases of fresh market tomatoes. Pages 59-73 in: 1981-1982 Annual Report. Calif. Fresh Market Tomato Adv. Board, Bakersfield. 194 pp.
10. Parr, J. F., and Norman, A. G. 1965. Considerations in the use of surfactants in plant systems: A review. Bot. Gaz. 126:86-96.
11. Puritch, G. S., Tan, W. C., and Hopkins, J. C. 1981. Effect of the fatty acid salts on the growth of *Botrytis cinerea*. Can. J. Bot. 59:491-494.
12. Segall, R. H. 1968. Reducing postharvest decay of tomatoes by adding a chlorine source and the surfactant Santomerse F85 to water in field

washers. Proc. Fla. State Hortic. Soc. 81:212-214.
13. Sonoda, R. M., Ogawa, J. M., Lyons, T., and Hanson, J. A. 1970. Correlation between immobilization of zoospores by fungicides and the control of Phytophthora root and crown rot of transplanted tomatoes. Phytopathology 60:783-787.
14. Spotts, R. A., and Peters, B. B. 1982. Use of surfactants with chlorine to improve pear decay control. Plant Dis. 66:725-727.
15. Wilcoxson, F., and McCallan, S. E. A. 1939. Theoretical principles underlying laboratory toxicity tests of fungicides. Contrib. Boyce Thompson Inst. 10:329-338.