Effect of Forced, Hot-Air Treatment of Papaya Fruit on Fruit Quality and Incidence of Postharvest Diseases

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ABSTRACT

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Forced, hot-air (48.5 C for 3–4 hr) treatment of papaya fruit (*Carica papaya*), a recently developed quarantine treatment for fruit flies, did not significantly reduce incidences of postharvest diseases when compared with fungicide or hot-water treatments. However, when hot-air treatment was combined with thiabendazole (TBZ) (4 g a.i./L) application or hot-water immersion (49 C for 20 min), the incidence of most postharvest diseases was reduced. Although disease incidences were not significantly affected by the sequence of hot-air or hot-water application, degreening (lack of surface ripening), along with pitting and scalding symptoms significantly ($P < 0.01$) increased when hot-water preceded hot-air treatment, but these symptoms did not occur when hot-air preceded hot-water treatment. The hot-air treatment was associated with an increase in the incidence of internal lumpiness (hardened lumps of flesh in ripe fruit) when compared with untreated fruit.

Additional keywords: *Botryodiplodia theobromae, Colletotrichum gloeosporioides, Mycosphaerella* sp., *Phomopsis* sp.

Before shipment to U.S. mainland markets, Hawaii-grown papaya fruit (*Carica papaya* L.) are subjected to quarantine treatment to disinfest the fruit of three fruit fly (Diptera: Tephritidae) species: Mediterranean fruit fly (*Ceratitis capitata* (Wiedemann)), melon fruit fly (*Dacus cucurbitae* Coquillett), and oriental fruit fly (*D. dorsalis* Hendel). Currently, a majority of the fruit receive a two-stage, hot-water treatment (13), which has been associated with occasional fruit damage (4,10) and with failure to destroy eggs and larvae of fruit flies in fruit with defective blossom ends (26).

A forced, hot-air treatment for papaya fruit was developed by Armstrong et al (4) and was subsequently approved by USDA-APHIS for meeting quarantine regulations. The treatment consisted of sequentially exposing fruit to forced air (airflow, 0.37 m^3/sec) at 43, 45, 46.5, and 49 C until fruit center (seed cavity) temperatures reached 41, 44, 46.5, and 47.2 C, respectively. The treatment required approximately 7 hr, after which fruit were submerged in tap water until fruit center temperatures were less than 30 C. Although fruit quality was not affected, a thiabendazole (TBZ, 2-(4-thiazolyl)-benzimidazole) application was required to control postharvest decay (4). Because the 7-hr treatment time was too lengthy for the commercial treatment of large quantities of fruit (31 million kg in 1988) (14), an alternative treatment was developed that used a single air temperature of 48.5 C for 3.25–3.75 hr until fruit center temperatures reached 47.2 C (J. W. Armstrong et al, *unpublished*). The effect of this treatment on postharvest diseases and fruit quality was unknown.

This study was initiated to evaluate the effect of the single-temperature, hot-air treatment on incidences of postharvest diseases and on fruit quality. Combinations of the hot-air treatment with fungicide or hot-water treatments were also examined.

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MATERIALS AND METHODS

Fruit. Freshly harvested papaya fruit (cv. Kapoho Solo) at the colorbreak (blush of yellow color) to one-fourth ripeness stage, were collected from field bins at a packinghouse in the Puna district on the island of Hawaii. Fruit were washed, then air-dried in the lab (22–23 C) before treatment the next day.

Treatments. Thiabendazole (Decco salt No. 19, Pennwalt Corp., Monrovia, CA) was applied by spraying 1 ml of an aqueous suspension (4 g a.i., 50 μl of Tween 20 per liter) onto each fruit with a plastic bottle sprayer, then spread over the fruit surface by hand. Fruit exposed to the hot-air treatment were held at 22–23 C for 16–18 hr to recover from heat treatment stress (4,7), then treated with TBZ. All fruit requiring TBZ application were treated on the same day.

With the hot-water treatments, approximately 20 medium-sized fruit were immersed in 60–62 L of water at 49 C for 20 min (single dip, SD) (1), or 42 C for 30 min followed by 49 C for 20 min (double dip) (13), in stainless steel tanks (61 × 52 × 35 cm). The heat-treated fruit were then submerged in tanks filled with fresh, unrecirculated, chlorinated tap water (22–23 C) for 20 min. Fruit in hot-air/hot-water combination treatments were also cooled in tap water immediately following the hot-water treatment. Hot-water temperatures were maintained with a circulating water heater (PolyTemp, PolyScience Corp., Niles, IL).

For the hot-air treatment, approximately 22 or 23 fruit were placed in plastic treatment bins (58 × 38 × 20 cm) with open lattice bottoms and arranged with blossom ends up. The bins of fruit were placed on a tablelike stand in a hot-air chamber that consisted of a walk-in environmental room. A plywood box that formed a wind tunnel was centered over the bins and was equipped with an exhaust fan that circulated heated, humidified (40–60% relative humidity) air through the bins. The air in the room was heated and recirculated by a heat pump (Forma Scientific, Marietta, OH). Equipment for the hot-air treatments was described in detail by Armstrong et al (4).

The single-temperature, hot-air treatment exposed fruit to the forced air (airflow, 0.37 m^3/sec) at 48.5 C for 3–4 hr until fruit center temperatures were 47.2 C. After the heating phase, fruit were submerged in plastic containers filled with running tap water (22–23 C) until fruit temperatures were less than 30 C, a period of about 1 hr. Air (or water) and fruit center temperatures were measured with thermistors that were placed outside of treatment bins or inserted through the blossom end into the seed cavity of one fruit in each bin, and recorded on an Omnidata Polycorder (Omnidata International, Logan, UT).

All fruit were packed in fiberboard cartons, stored at 10 C for 7 days to simulate shipping and storage conditions, and then held at 22–23 C until ripe (5–7 days). Ripened fruit were evaluated for postharvest diseases based on visible signs and symptoms.

Evaluation. Diseases with external symptoms included stem-end and surface rots caused by *Colletotrichum gloeosporioides* (Penz.) Penz. & Sacc. in Penz., *Phomopsis* sp., *Botryodiplodia theobromae* Pat., *Mycosphaerella* sp., *Rhizopus stolonifer* (Ehr.:Fr.) Vuill., *Phytophthora palmivora* (Butl.) Butl., *Stemphylium lycopersici* (Enjoji) Yamamoto, *Guignardia* sp., *Fusarium solani* (Mart.) Sacc., and *Cercospora* sp. Diseases with internal symptoms included fungal and bacterial decay caused by *Cladosporium* sp., *Penicillium* sp., *Fusarium* spp., *Erwinia herbicola* (Loehnis) Dye, and *Enterobacter cloacae* (Jordan) Hormaeche and Edwards.

When visual observations were insufficient for disease diagnosis, decayed or damaged tissue was aseptically excised, surface-sterilized in 0.5% sodium hypochlorite for 30–60 sec, and plated on water agar. Samples of microbial growth were transferred to 10% V8 juice agar (10% Campbell's V8 juice, 0.02% $CaCO_3$, and 2% agar) or potato-dextrose agar (PDA, Difco, supplemented with 0.5% agar), and identified according to descriptions of papaya diseases (2,18,19) and taxonomic references (5,24).

Fruit were also examined for skin-scalding (brownish sheen), pitting ('pinhole'-like depressions on surface), degreening (blotchy coloring on ripe fruit), internal lumpiness (hardened lumps of flesh), and 'hard shell' (4,7–9, 15). Degreening, which was present in both mild and severe forms of injury, was selected as the external indicator of physiological injury, and internal lumpiness was used as the internal indicator.

Experiments. During June to August of 1988, the first of two experiments was conducted in which individual sets of approximately 35 papaya fruit were either treated with TBZ, or exposed to hot-air treatment, or exposed to hot-air treatment and treated with TBZ the next day, or not treated with either TBZ or hot air (untreated control). The experiment was arranged in a randomized complete block design and repeated six times.

A second experiment was conducted from August to September. Sets of 20 fruit were immersed in a single hot-water dip, or a double hot-water dip, or exposed to hot-air treatment, or immersed in single hot-water dip and exposed to hot air 1–1.5 hr later, or exposed to hot air and then immersed in a single hot-water dip, or not exposed to either hot water or hot air (untreated control). The experiment was arranged in a randomized complete block design and repeated four times.

Data analysis. Incidences of the most commonly occurring diseases were analyzed individually or as part of total disease (diseases with external or internal symptoms on the same fruit combined). Data on incidences of *C. gloeosporioides* in the first experiment, and of *Phomopsis* sp. in the second experiment, were not analyzed because the incidences in control fruit were too low (1.19 and 2%, respectively). Incidences of disease or physiological injury, expressed as percentages, were transformed by the arcsine-square root method (22), calculated in degrees, and analyzed using SAS statistical software for personal computers (21).

The analysis of variance (ANOVA) procedure for balanced design was used to analyze for main and interactive effects of fungicide, air, and block in the first experiment, and the general linear models (GLM) procedure for unbalanced design was used to analyze for main and interactive effects of water, air, and block in the second experiment. Data in the second experiment were analyzed as separate data sets, single dip (SD)+hot-air and hot-air+SD, in which data for all treatments except hot-air+SD or SD+hot-air were included, respectively. Differences among treatments and blocks were evaluated by the two-way ANOVA procedure, and comparisons of means of transformed data were based on orthogonal contrasts (22), or the Student-Newman-Keuls test at $P = 0.05$ level of significance.

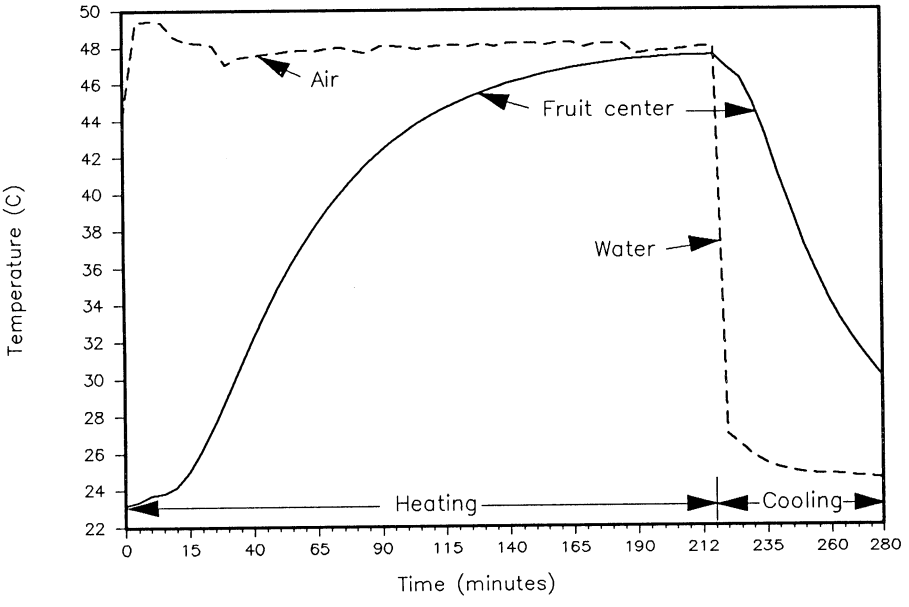

Fig. 1. Temperature profile of fruit center, air (heat phase), and water (hydrocooling phase) during the forced, hot-air treatment of papaya fruit.

RESULTS

In the forced, hot-air treatment with air temperature set at 48.5 C, the heating phase required approximately 3.5 hr to reach fruit center temperatures of 47.2 C. The hydrocooling phase (tap water at 22–23 C) required approximately 1 hr to lower fruit center temperatures to less than 30 C (Fig. 1).

In the first experiment, the hot-air treatment alone did not significantly reduce incidences of most postharvest diseases (Tables 1 and 2). Although incidence of *Phomopsis* sp. was reduced by 62% of the untreated control, incidences of *B. theobromae* and *Mycosphaerella* sp. were not affected. Incidence of total disease was also not affected by the hot-air treatment, according to orthogonal contrasts of selected treatments. The TBZ treatment alone afforded the best control of postharvest diseases by significantly ($P < 0.01$) reducing the incidences of diseases caused by *Phomopsis* sp. (95%), *B. theobromae* (97%), and *Mycosphaerella* sp. (100%). Reduction in incidence of total disease (96%) was also significant ($P < 0.001$), according to orthogonal contrasts. When TBZ application was combined with the hot-air treatment, disease incidences were not significantly different from the TBZ treatment alone except for incidence of total disease. The hot-air+TBZ combination treatment reduced incidence of total disease by 80%, whereas TBZ treatment alone reduced incidence by 96%. These treatments were significantly ($P < 0.01$) different, according to orthogonal contrasts. The greater effect of TBZ alone as compared with hot-air+TBZ, accounted for the significant ($P < 0.01$) interaction between the fungicide and hot-air treatments (Table 2).

In the second experiment, hot-air

Table 1. Effect of forced, hot-air and thiabendazole (TBZ) treatments on incidences of postharvest pathogens and disorders of papaya (*Carica papaya*) fruit[v]

Treatment[w]	Total disease (%)[x,y]	*Phomopsis* sp. (%)	*Botryodiplodia theobromae* (%)	*Mycosphaerella* sp. (%)	Fruit degreening (%)	Lumpy fruit (%)
Untreated	40.4	7.7 a[z]	22.9 a	5.0 a	1.7 a	0.4
TBZ	1.6	0.4 b	0.8 b	0.0 b	1.6 a	0.0
Hot air	34.4	2.9 b	22.5 a	5.8 a	1.7 a	6.5
Hot air+TBZ	8.0	0.4 b	1.8 b	0.8 b	1.5 a	11.5

[v] Papaya fruit were collected from field bins at a packinghouse on the island of Hawaii from June to August 1988.
[w] Treatments consisted of untreated control, TBZ applied at 4 g a.i./L, forced, hot air (48.5 C for 3–4 hr), and hot air followed by TBZ application the next day.
[x] Percentages represent means of seven tests, each consisting of 30–40 fruit for each treatment.
[y] Data for total disease (all diseases on same fruit combined), individual diseases caused by *Phomopsis* sp., *B. theobromae*, and *Mycosphaerella* sp., degreening, and lumpiness, were transformed to arcsine of square root of proportion of incidence and analyzed by analysis of variance (ANOVA) at $P = 0.05$.
[z] Values in columns followed by the same letter are not significantly different according to the Student-Newman-Keuls test of transformed data at $P = 0.05$.

Table 2. Factorial analysis of variance of thiabendazole fungicide application (4 g a.i./L), forced, hot-air treatment (48.5 C, 3–4 hr), and block on incidence[a] of postharvest pathogens and disorders of papaya (*Carica papaya*) fruit

		F value					
Source	df	Total disease	*Phomopsis* sp.	*Botryodiplodia theobromae*	*Mycosphaerella* sp.	Fruit degreening	Lumpy fruit
Fungicide (F)	1	468.55***[b]	18.68**	301.92***	24.87**	0.26	4.17
Air (A)	1	2.27	3.42	0.02	1.29	3.43	60.21***
Block (B)	6	7.22*	0.85	5.06*	2.79	5,202.43***	8.93**
F × A	1	20.26**	3.19	0.69	0.07	2.84	6.32*
F × B	6	1.69	1.22	0.88	2.65	3.35	1.16
A × B	6	9.09**	1.58	4.79*	0.30	0.95	9.82**

[a] Data were transformed to arcsine of square root of proportion of incidence.
[b] *F* value for main effect or interaction significant at $P = 0.05$ (*), $P = 0.01$ (**), or $P = 0.001$ (***).

Table 3. Effect of forced, hot-air and hot-water immersion treatments on incidences of postharvest pathogens and disorders of papaya (*Carica papaya*) fruit[v]

Treatment[w]	Total disease (%)[x,y]	*Colletotrichum gloeosporioides* (%)	*Botryodiplodia theobromae* (%)	*Mycosphaerella* sp. (%)	Fruit degreening (%)	Lumpy fruit (%)
Untreated	64.0 a[z]	17.0 a	36.0 a	16.0 a	2	0 a
Single dip (SD)	21.2 b	12.1 a	5.1 b	2.0 b	0	0 a
Double dip	20.0 b	8.0 a	2.0 b	4.0 b	1	0 a
Hot air	50.0 a	2.0 a	29.0 a	16.0 a	1	2 a
SD + hot air	16.0 b	7.0 a	4.0 b	3.0 b	28	2 a
Hot air + SD	12.0 b	3.0 a	1.0 b	3.0 b	0	5 a

[v] Papaya fruit were collected from field bins at a packinghouse on the island of Hawaii from August to September 1988.
[w] Treatments consisted of untreated control, single hot-water dip (SD) (20 min at 49 C and 20 min hydrocool), double hot-water dip (30 min at 42 C, then 20 min at 49 C and 20 min hydrocool), hot air (48.5 C for 3–4 hr), single dip 1–1.5 hr before hot air, and hot air followed by single dip.
[x] Percentages represent means of five tests, each consisting of 20 fruit for each treatment.
[y] Data for total disease (all diseases on same fruit combined), individual diseases caused by *C. gloeosporioides*, *B. theobromae*, and *Mycosphaerella* sp., degreening and lumpiness, were transformed to arcsine of square root of proportion of incidence and analyzed by analysis of variance (ANOVA) at $P = 0.05$.
[z] Values in columns followed by the same letter are not significantly different according to the Student-Newman-Keuls test of transformed data at $P = 0.05$.

treatment alone did not significantly reduce the incidences of the individual postharvest diseases, or total disease (Table 3). In contrast, the hot-water treatments reduced incidences of total disease by 67–81%, and diseases caused by *B. theobromae* by 86–97% and *Mycosphaerella* sp. by 75–88% of the untreated control (Table 3). In combined treatments, disease incidences were not affected by the sequence of the hot-water or hot-air treatments (Table 3). Both sequences of treatments reduced incidences of total disease, *B. theobromae*, and *Mycosphaerella* sp., when compared with hot air alone or the untreated control.

When analyzed over all treatments, hot-air treatment significantly ($P < 0.05$) affected the incidences of total disease and *C. gloeosporioides*, whereas hot-water treatment significantly ($P < 0.001$) affected the incidences of total disease and other pathogens (Table 4). Because there was no significant interaction between the hot-water and hot-air treatments, the reduction in incidences of total disease and pathogens were due to individual or additive effects of hot water and/or hot air, not interactive effects (Table 4).

The following external fungal pathogens occurred in 3% or less (mean of all tests) in any single treatment in an experiment: *R. stolonifer*, *P. palmivora*, *S. lycopersici*, *Guignardia* sp., *F. solani*, and *Cercospora* sp. The *Alternaria* fruit spot (2) pathogen, *Alternaria alternata* (Fr.: Fr.) Keissler, was not observed.

Internal diseases caused by *Cladosporium* sp. and *Fusarium* spp. were observed in less than 2% (mean of all tests) in any single treatment in an experiment, and *Penicillium* sp. was not observed. The internal yellowing bacterium, *E. cloacae*, and the purple stain bacterium, *E. herbicola*, occurred in 2% or less and 4% or less (mean of all tests), respectively, in any single treatment in an experiment.

Fruit quality, in the form of physiological injury, was affected by the different treatments. However, hard shell (4,15), regarded as the most severe form of internal heat injury, was not detected in our studies.

Degreening symptoms were not increased by fungicide or hot-air treatments in the first experiment (Tables 1 and 2), or by individual applications of hot water or hot air in the second experiment (Table 3). However, when hot-water immersion was followed by hot-air treatment, degreening, along with pitting and scalding symptoms increased significantly ($P < 0.01$), according to orthogonal contrasts performed on the separate data sets, SD+hot-air and hot-air+SD. Hot-water, hot-air, and interaction between these factors were significant ($P < 0.05$) when single dip preceded hot-air treatment, but not when the treatment sequence was reversed (Table 4).

Internal lumpiness, which was not related to degreening symptoms, was significantly ($P < 0.01$) increased by hot-air treatment in both experiments (Tables 2 and 4). In the first experiment, orthogonal contrasts indicated that the incidence of lumpy fruit was affected significantly ($P < 0.05$) more by the hot-air+TBZ combination treatment than by the hot-air treatment alone, which led to a significant interaction between fungicide and hot air (Table 2). In the second experiment, the increase in lumpy fruit in hot-air and hot-air/hot-water treatments was not significant in the Student-Newman-Keuls test (Table 3), yet in the ANOVA, the effect of hot-air treatment was significant ($P < 0.01$) (Table 4). In contrast, the hot-water treatments did not appear to cause the lumpy fruit condition when applied individually, or in combination with hot-air treatment (Tables 3 and 4). The hot-water and hot-air interaction was not significant (Table 4).

DISCUSSION

Quarantine heat treatments for fruit fly in papaya fruit differ in their ability to control postharvest diseases. The efficacy of such treatments and the potential for fruit damage appear to be affected by the method of heat treatment and its application.

Hot-water treatments in our studies effectively controlled most postharvest diseases of papaya fruit and was consistent with previous reports (2,10–12), except for the lack of significant control of anthracnose caused by *C. gloeosporioides*. A single immersion in hot water (49 C) for 20 min was developed to control *C. gloeosporioides* or *Gleosporium* sp. on papaya fruit (1). However, in our studies, the single-dip treatment applied either alone or in combination with the hot-air treatment, was not effective. Previously, inconsistencies in the effectiveness of hot-water treatment on incidences of *C. gloeosporioides* on papaya fruit were associated with level of disease (11,12). The hot-water treatment was effective when disease incidence was low (8–16%), but not when it was higher than 24%. In our tests, the average incidence among untreated fruit was 17%, but significant ($P < 0.01$) differences among the individual blocks or fruit lots, especially in blocks 2 and 4 (data not shown), contributed to variation among the data. Limitations of the hot-water immersion treatment were also reported for *P. palmivora* infections in papaya during periods of high disease incidence (3).

Table 4. Factorial analysis of variance of hot-water treatment, forced, hot-air treatment, and block on incidence[a] of postharvest pathogens and disorders of papaya (*Carica papaya*) fruit

		F value					
Source	df	Total disease	*Colletotrichum gloeosporioides*	*Botryodiplodia theobromae*	*Mycosphaerella* sp.	Fruit degreening	Lumpy fruit
Analyzed as sorted data set, single dip + hot air[b]							
Water (W)	1	150.73***[c]	0.00	150.07***	53.48***	8.63*	0.02
Air (A)	1	8.42*	7.70*	1.73	0.00	9.87*	15.54**
Block (B)	4	15.90***	7.08**	8.54**	6.03*	2.41	8.87**
W × A	1	2.37	4.95	2.55	0.01	12.60**	0.02
W × B	4	3.28	1.92	12.56**	2.46	1.11	1.69
A × B	4	4.01*	1.99	1.97	1.06	2.32	7.70**
Analyzed as sorted data set, hot air + single dip[d]							
Water (W)	1	145.11***	0.85	176.03***	34.59***	3.81	4.39
Air (A)	1	13.68**	14.26**	5.45*	0.01	1.37	26.77***
Block (B)	4	19.31***	7.00**	11.09**	3.23	3.89*	12.29**
W × A	1	0.07	1.87	0.34	0.00	0.15	4.39
W × B	4	0.66	2.17	10.13**	1.93	1.58	1.69
A × B	4	1.57	2.43	0.58	0.78	0.51	13.91***

[a] Data were transformed to arcsine of square root of proportion of incidence.
[b] Single hot-water dip (49 C for 20 min, hydrocool for 20 min), followed 1–1.5 hr later by forced, hot-air treatment. Other treatments in data set included single dip, double dip (42 C for 30 min, followed by 49 C for 20 min, hydrocool for 20 min), hot air (48.5 C for 3–4 hr), and untreated control.
[c] *F* value for main effect or interaction significant at $P = 0.05$ (*), $P = 0.01$ (**), or $P = 0.001$ (***).
[d] Hot-air treatment followed by single dip. Other treatments in data set included single dip, double dip, hot air, and untreated control.

The hot-air treatment was inconsistent with respect to controlling postharvest diseases in our experiments. In the first experiment, total disease or diseases caused by individual pathogens were not affected by the hot-air treatment. In contrast, hot-air treatment had a significant effect on some pathogens in the second experiment that appeared additive with the effect of hot water. This additive effect appears to warrant further investigation. Previously, hot-air treatment of mango fruit (48 C for 2.5 hr) was reported to reduce severity of postharvest diseases (17).

The greater effectiveness of hot water alone compared with hot air alone for control of postharvest diseases of papaya may be due to the greater efficiency of water as a sterilizing agent and as a heat transfer medium. Water assists the denaturation of proteins during heat coagulation (6) and has a greater thermal capacity (enthalpy) than air.

In hot-water/hot-air combination treatments reported here, the sequence of the treatments was critical to surface heat injury. Fruit immersed in hot water before hot-air exposure were injured with degreening and other symptoms, whereas those exposed to the reverse order were not. These observations were contrary to findings with peaches by Kerbel et al (16), where less surface browning injury resulted when fruit were hot-water treated before hot-air treatment at the same temperatures (40–43 C), than when fruit were hot-water treated only. In our tests with papayas, when hot-water immersion preceded hot-air treatment, the 1.5-hr interval between the two treatments may not have been sufficient for the fruit to adapt to the subsequent heat treatment (25), or to thoroughly dry off (which resulted in residual moisture on the fruit surface). In vapor heat studies (15), papaya fruit that were heated for 16 hr at 43.3 C incurred heat injury when the air was fully saturated with water vapor, but not when relative humidity was reduced to 60%.

The hot-air treatment in our studies also significantly affected incidence of lumpy fruit, which varied among fruit lots in both experiments. Inconsistent occurrences of internal lumpiness may be caused by environmental factors or differences in fruit ripeness that affected the physiology of the fruit. Paull and Chen (20) found that the sensitivity of papaya fruit to heat injury was directly related to fruit ripeness and date (season) of harvest, with one-fourth ripe fruit and winter-harvested fruit being the most sensitive. Because our studies were conducted during the summer months, the tendency for hot-air treated fruit to have more internal lumpiness than control fruit indicates that this condition could become more severe during the winter season.

Although not part of quarantine treatment procedures, storage conditions after treatment affected fruit quality in our tests. Degreening symptoms, which occurred in all treatments of the first experiment, and only in block 3 (data not shown), may have been due to chilling injury of colorbreak ripeness fruit during the 1 wk of storage in the refrigerated (10 C) chamber. Uneven ripening and degreening symptoms may occur when papaya fruit are refrigerated below 10 C (7,9), and green fruit are more sensitive to injury than ripe fruit (7,23).

In conclusion, hot-air treatment alone did not control most postharvest diseases of papaya fruit, whereas hot-water treatment or TBZ application afforded good postharvest disease control. When hot-water treatment was combined with the hot-air treatment, incidences of diseases were significantly reduced by additive effects of hot-water and hot-air treatments. This combination treatment merits further investigation, especially when future reduction in the availability of fungicides for disease control is considered. Although disease incidences were not affected by the sequence of the hot-water/hot-air treatments, physiological damage in the form of degreening symptoms resulted when hot-water preceded hot-air treatment. We also observed that hot-air treatment may increase the incidence of lumpiness in papayas, at least with certain fruit lots. Thus, modifications in the hot-air quarantine treatment may be needed to reduce the potential for fruit injury.

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