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Postharvest Heat Treatment of Fresh Fruits and Vegetables for Decay Control

Postharvest heating to kill or weaken plant pathogens offers a pesticide-free method to control postharvest diseases. Heat treatment of fresh fruits and vegetables for decay control differs from other uses of heat on produce, such as curing to promote wound healing or heating to suppress nematodes, insects, or viruses (2), because these treatments usually require longer heating times than for postharvest decay control. Postharvest heat treatments to control decay often are applied for only 3-5 minutes because the target pathogens are found on the surface or within the few outer cell layers of the produce. To achieve a significant degree of pathogen control, heat is necessary for only the exterior surfaces.

For our discussion, heat treatment is the application of heat at temperatures above 40 C for control of postharvest pathogens. Fruits and vegetables commonly tolerate temperatures of 50-60 C for 5-10 minutes, but shorter exposure at these temperatures controls many postharvest plant pathogens (30). The pathogen can be killed or injured while the host is changed very little. With the trend toward less reliance on chemical control, postharvest use of heat treatment warrants greater study and further development.

The Effect of Heat on Pathogen and Host

The efficacy of heat on the pathogen is usually measured by reduced viability

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of the heated propagules. However, heat effects may be lethal or sublethal (8). The response of a pathogen to heat can be influenced by the moisture content of spores, metabolic activity of the pathogen or its inoculum (17), age of the inoculum (15), and chemical composition (23) and water activity of the treatment medium. Even culture media on which the pathogen grew after heat treatment (8.15) can influence its apparent viability. Many factors that modify the effect of heat on the pathogen may also affect the

Genetic differences among fungi are expressed by considerable variation in sensitivity to high temperature (33) (Fig. 1). For a given species, spore inactivation increases with both temperature and duration of the treatment. Spores of Alternaria tenuis Nees may be inactivated equally by treatment for 2 minutes at 48 C or for 4 minutes at 46 C (9) (Fig. 2).

Water relations before or during exposure to heat can markedly influence transfer of heat and its effect on pathogens. When dehydrated and moist conidia of Penicillium digitatum (Pers.:Fr.) Sacc. were compared, 10% of the dry spores but 90% of the moist spores were killed in 30 minutes at 70 C. Surviving dry conidia infected citrus fruit, but onset of symptoms was delayed 24 hours. Moisture also influences physiological activity, such as spore germination. Germinated fungal spores are markedly more sensitive than nongerminated spores to heat. We have found that at 42 C, water does not affect dormant conidia of A. tenuis but does inactivate many germinated conidia (Fig. 3). The LD₅₀ temperature for sporangiospores of Rhizopus sp. exposed to hot water for 4 minutes was 39 C for germinating spores but 49 C for dormant spores (16).

Heat may control pathogens by pro-

tein denaturation, lipid liberation, destruction of hormones, asphyxiation of tissue, depletion of food reserves, or metabolic injury with or without accumulation of toxic intermediates (6). Some or all of these mechanisms may be involved simultaneously. Ultrastructural changes in heat-treated nongerminated spores of Monilinia fructicola (G. Wint.) Honey illustrated progressive destruction of the mitochondrial cristae. matrix, and outer membranes; disruption of vacuolar membranes; and formation of gaps in the conidial cytoplasm (24). The site most sensitive to heat in dormant conidia of M. fructicola may be in the mitochondria, probably in the inner membrane. Evidence to support this hypothesis is that cytoplasmic protein synthesis or DNA synthesis inhibitors in Penicillium expansum Link did not affect recovery of heat-injured dormant conidia (8). Ultrastructural changes in germinated M. fructicola spores include changes in the nuclei or the cell wall, or both, (7) and indicate that the nucleus may also be injured in germinating spores.

Heat treatments can affect the host by altering ripening (3), fruit color, electrolyte leakage, sugar metabolism, ethylene production, ethanol production, pectic enzyme activity, and susceptibility to pathogens (17,26). The complex structure of the host can greatly influence the rate of heat transfer. Heat transfer from tissue to tissue within the leaf, stem, root. or fruit can vary greatly. These morphological factors of the host contribute to inconsistent results from heat treatment. The colored outer layer (flavedo) of the citrus rind may have little intercellular space and may transfer heat faster than the underlying spongy albedo. A berry, such as grape, may transfer heat faster than tissues of a pome fruit, such as apple.

Location of the pathogen on or within the host also can affect consistency of response (26). Immersion in water at 46-49 C for 4 minutes arrested development of *Phytophthora citrophthora* (R.E. Sm. & E.H. Sm.) Leonian in lemons only if the fungus had not yet penetrated the outer layer of the rind (27).

Some variables that interact with the efficacy of heat treatment result from preharvest cultural and weather conditions. For example, temperature prevailing in citrus groves during the rainy season affects fungus development and determines the efficacy of heat treatment in arresting decay (27). Preharvest environmental factors may influence hot

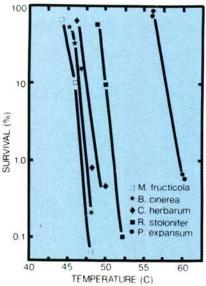


Fig. 1. Survival of spores of Monilinia fructicola, Botrytis cinerea, Cladosporium herbarum, Rhizopus stolonifer, and Penicillium expansum after 4 minutes at the indicated temperatures. (From Sommer et al [33])

water injury to lemons (32). Some understanding of this variation may be reached by examining fruit produced under differing cultural practices or growing temperatures (27). Maturity and the cultivar's inherent heat tolerance, coupled with the preharvest environment, must be considered when developing or applying postharvest heat treatment.

Methods Used in Heat Treatment

Heat is usually delivered to a commodity by air or water. The water content of air greatly influences heat transfer, and heated moist air usually kills pathogens more effectively than dry air at the same temperature (36). Moist heat may be more effective than dry heat because moist spores have higher physiological activity than dry spores but also because moist air transfers heat more efficiently than dry air. When the air is dry, no condensation forms on the target commodity and the rate of heat transfer depends largely on the air passing over the surface of the fruit and the heat conductivity of the commodity. When moisture condenses on fruit, latent heat transfer from the water is significant and heat transfer depends less on air movement. When the air is saturated with water (vapor heat), condensation forms on surfaces that are cooler than the air and heat is transferred rapidly to the surface (17).

Maintaining an effective temperature range depends on heat added to or stored in the treatment medium. Heat transfer to the commodity may be sufficient without adding heat to the water if the commodity-to-water ratio is low and little heat is needed to warm the surface of the product. This is a very simple treatment system. To maintain the water temperature for prolonged treatment or

when the commodity-to-water ratio is high, heat must be added during treatment. A minimum commodity-to-water ratio of 1:10 can result in satisfactory surface heating. Sophisticated equipment is available for heat treatment for insect control (4), but simple hot water tanks may be used successfully for postharvest treatments to control decay (2). Heat input is important when heating with air because the air does not have the heat-holding capacity of water. Thus, the heat required for the commodity should be matched carefully to the heaters. Additional work is needed to develop an air treatment designed specifically for decay control.

For effective heat treatment, temperature is often near the level injurious to the commodity, and temperature must be carefully controlled and measured. Temperature can be measured in the commodity or in the medium (air or water) used to heat it. Temperature readings within the commodity differ according to the depth of the probe within the tissue. Covering the probe with a water barrier may be necessary to avoid undue evaporative cooling of the instrument. Determination of plant tissue temperature also may be influenced by heat moving through the sensor probe, and probed fruits may heat faster than unprobed fruits. The initial temperature and size of the commodity, as well as the position of the fruit within the treatment chamber, can greatly influence product temperature. A standard location for measuring the temperature is often chosen, but using a sample or test product that is truly representative is difficult.

Effects and Limitations of Heat Treatments

Many postharvest treatments with hot water (Table 1) or hot air (Table 2) have

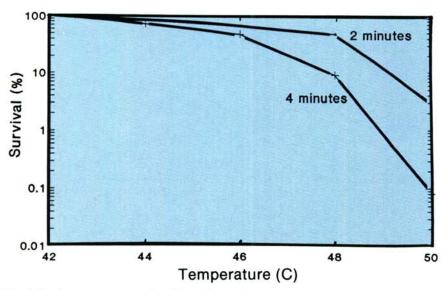


Fig. 2. Heat response curve for *Alternaria tenuis* after 2 or 4 minutes at the indicated temperatures. (From Barkai-Golan [9])

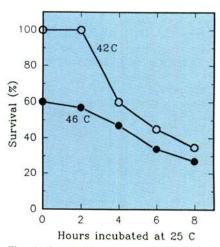


Fig. 3. Sensitivity to heat treatment (2 minutes at 42 or 46 C) in conidia of Alternaria tenuis that were freshly harvested or had been incubated in a 25 C water bath for 2, 4, 6, or 8 hours. (From Barkai-Golan [9])

been reported. Effective water treatments are usually between 46 and 60 C, with exposure times ranging from 30 seconds to 10 minutes. Treatments using air range from 43 to 54 C for 10-60 minutes. Either water or air can be effective, and choosing a method seems to depend on such factors as a need to control desiccation, hydration, or time of application, Tropical fruits, such as mangos and papayas, may be inherently more heat-tolerant than fruits from temperate zones (14). Some treatments providing decay control improve quality of the treated commodity by reducing pesticide residues, reducing physiological disorders, controlling premature softening, or improving host resistance to disease (14). The effect of heat can differ with each pathogen-host combination. For example, pectic enzyme activity of *Rhizopus*-infected apricots appears to continue even after the fruits are canned and sterilized. However, a short exposure to heat can significantly lower pectic enzyme production by heated spores or decrease the natural pectic enzyme activity of heated fruit

Injuries caused by heat treatment include increased water loss, discoloration, increased susceptibility to contaminating microorganisms, and decreased shelf or storage life (17). The deleterious effects are quite inconsistent and also are influenced by many environmental factors. The greatest limitations to using heat are the lack of residual protection against recontamination by pathogens (17) and

injury to the host. Some pathogens may be heat-resistant, but the development of resistance, as has occurred with the use of some fungicides, is not anticipated.

Improving Heat Treatments

Improved use of heat treatments may be possible when the nature of the phytotoxicity caused by the heat is understood. For example, the effect of heat alone needs to be separated from the effect of or interaction with the heating medium. In air or water, both drying and hydration need to be considered as potentially damaging when interacting with heat.

The host may be protected from injury by preconditioning. Lemons that are slightly wilted or held 2-8 days at 15.5 C

Table 1. Hot water treatment of fruits and vegetables for control of postharvest decay

| Commodities | Pathogens | Temper- ature (C) | Time (min) | Possible injuries | Refer- ences |
|---------------|---|-------------------------|---------------|-------------------------|------------------|
| Apple | Gloeosporium sp. | 45 | 10 | Reduced storage life | 17 |
| Bean | Penicillium expansum Pythium butleri Sclerotinia sclerotiorum | 52 | 0.5 | | 38 |
| Cherry | Monilinia fructicola | 52 | 2 | Slight discoloration | 21 |
| Cranberry | Godronia sp. Pestalotia sp. Sporonema sp. | 52 | 2.5 | Physiological breakdown | #### 17 i |
| Grapefruit | Phytophthora citrophthora | 48 | 3 | | 27 |
| Lemon | Penicillium digitatum Phytophthora sp. | 52 | 5–10 | | 19 |
| Litchi | Aspergillus sp. Rhizopus sp. | 52 | 2 | Some browning | 28 |
| Mango | Colletotrichum gloeosporioides | 52 | 5 | No stem rot control | 35 |
| Melon | Fungi | 57-63 | 0.5 | | 21,36 |
| Nectarine | Monilinia fructicola Rhizopus stolonifer | 52 | 2.5 | Reduced storage life | 29,34 |
| Orange | Diplodia sp. Phomopsis sp. Phytophthora sp. | 53 | 5 | Poor degreening | 32 |
| Papaya | Fungi | 48-49 | 20 | | 20 |
| Peach | Monilinia fructicola Rhizopus stolonifer | 52 | 2.5 | Motile skin | 29,30 34 |
| Pear | Mucor piriformis | 47 | 30 | | 25 |
| Pepper (bell) | Erwinia sp. | 53 | 1.5 | Slight spotting | 21 |
| Prune (fresh) | Monilinia fructicola | 52 | 3 | | 22 |

Table 2. Hot air treatment of fruits for control of postharvest decay

| Commodities | Pathogens | Temper- ature (C) | Time (min) | RH (%) | Possible injuries | Refer- ences |
|-------------|---|-------------------------|---------------|-----------|----------------------|-----------------|
| Apple | Gloeosporium sp. Penicillium expansum | 45 | 15 | 100 | Deterioration | 17 |
| Melon | Fungi | 30-90 | 35 | Low | Marked breakdown | 36 |
| Nectarine | Monilinia fructicola Rhizopus stolonifer | 52 | 15 | 90-100 | Slight discoloration | 3 |
| Peach | Monilinia fructicola Rhizopus stolonifer | 54 | 15 | 80 | ••• | 30 |
| Strawberry | Alternaria sp. Botrytis sp. Cladosporium sp. Rhizopus sp. | 43 | 30 | 98 | | 31 |

have improved tolerance of heat treatment (19).

The pathogen can also be preconditioned to increase its sensitivity to heat. Alternaria rot was controlled more effectively in tomatoes heat-treated 8 hours after inoculation than in those treated immediately after inoculation (9). Most spores germinate during the 8 hours of incubation, and the "sporelings," or young hyphal cells, are more sensitive to heat than nongerminated spores.

Moisture control to avoid water condensation during hot air treatment may protect the host. Damage to papayas during hot air treatment for insect control was minimized by reducing condensation or by heating in stages (4).

The increased water loss that often follows hot water treatment can be reduced by applying wax, with or without fungicides (37), after treatment. Produce also can be wrapped with plastic film before or after heat treatment to prevent water loss (3,30,36). We have found that plastic-wrapped nectarine fruit, when heated in air for 15 minutes at 52 C in 90% relative humidity, are protected not only from water loss or gain but also from recontamination and discoloration. Plastic wrap did not interfere with heat exchange but did prevent water from condensing directly on the fruit surface (3) and thereby seemed to improve the fruit quality.

Hot water containing fungicides is more effective than water or fungicide alone for decay control in peaches, plums, and nectarines (39); apples; mangos; citrus fruits (18); litchis; guavas; and melons. In commercial exports of mango from Jamaica to Britain that took 24 days at 13 C, almost all mangos treated with 55 C water for 5 minutes had mild, superficial symptoms of anthracnose; the addition of benomyl (500 mg/L) to the hot water resulted in

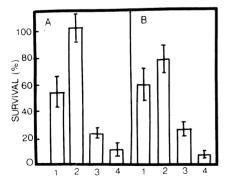


Fig. 4. Survival of conidia after heat treatment and irradiation (1 = irradiation only, 2 = heat only, 3 = irradiation followed by heat, 4 = heat followed by irradiation): (A) Botrytis cinerea subjected to 44 C for 4 minutes or a 75-krad dose, or both, and (B) Penicillium expansum subjected to 56 C for 4 minutes or a 20-krad dose, or both. Vertical lines indicate standard deviations. (From Sommer et al [33])

entirely disease-free fruit. Peaches and nectarines treated for 1.5-2 minutes in water at 52 C decayed when held longer than 3 weeks but not when heated to 46 C in water containing 100 mg/L of benomyl. With the fungicide, a lower temperature was effective and no injury was observed in the stored fruit (29). The mechanism of control with heated fungicide mixes may be related in part to the direct effect of heat or to increased chemical activity, but control may also be improved by increased penetration and deposition of fungicide on the product when the treatment solution is heated (39).

Nonpesticide chemicals can be added to hot water to protect the commodity and improve treatment effectiveness. External discoloration of peaches and nectarines held 18-24 hours at 20 C was rated (1 = no discoloration and 2 = some[one or two spots], 3 = moderate [three to 10 spots], and 4 = severe [more than 10 spots discoloration) after treatment of the fruit for 10 minutes in cool (21 C) water or in hot (52 C) water with or without sucrose. Discoloration ratings were: 1.78 with cool water, 3.54 with hot water and no sucrose, 1.66 with hot water and 171 g/L of sucrose, and 1.62 with hot water and 342 g/L of sucrose. Sucrose has been reported to increase the survival of bacteria and yeasts that have been heated (5,13). Perhaps sucrose stabilizes the proteins of these microorganisms (5) and may similarly protect the fruit surface, or perhaps sugar slows hydration of the surface exposed to the hot water treatment.

Combinations of heat and controlled atmospheres have been tried for several commodities, but without success. An atmosphere of 5% CO₂ and 3% O₂ hastened development of heat-related physiological disorders in apples stored for 19 weeks. When heat-treated peaches stored 6 weeks in a controlled atmosphere were ripened, excessive decay developed (29). Applying 10% CO₂ for 18 hours to Galia melons before a 2-minute dip in hot (52 C) water did not control fungi but did seem to improve flavor over heated fruit not treated with CO₂ (36).

Irradiation combined with heat may reduce the dose of radiation needed for pathogen control (33). The combined effects of heat and irradiation have been explored in various host-pathogen interactions, including oranges with P. digitatum (10); nectarines with M. fructicola (33); mangos with Colletotrichum gloeosporioides (Penz.) Penz. & Sacc. in Penz., *Hendersonia* sp., and stem-end rot fungi (12); papayas with various postharvest pathogens (12); and tomatoes with soft rot bacteria. Heat and irradiation act synergistically to inactivate spores (10,11,33), lessening the time of exposure needed for each. The effect of combined treatment is influenced by the sequence and is generally greater when

heat precedes irradiation than when the sequence is reversed (Fig. 4) (33). The interval between heating and irradiation also affects the synergism. Irradiation should be applied within 24 hours of the hot water treatment (10). Dipping citrus fruit in hot water before irradiation may also reduce irradiation-induced peel injury.

Conclusions

Heat treatment of fresh fruits and vegetables can provide good control of decay but does not as yet provide the same protection of fruit quality that postharvest fungicides do. Induced injury to commodities and lack of residual protection are serious limitations to the use of heat treatment. The use of polymer film wrap during treatment or the addition of nonpesticide chemicals to hot water may increase the effectiveness of heat treatment, but more information is needed for the various commodities. Today, the best candidates for heat treatment are fruits and vegetables that are sold soon after harvest rather than stored. With continued effort, heat treatments may provide new, safe, and effective disease control for many types of fresh produce.

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