

Review
Postharvest heat treatments

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Abstract

Postharvest heat treatments are being used for disinfestation and disinfection of an increasing variety of crops, including flowers, fruits and vegetables. This review focuses on the effects of heat on the commodity and its postharvest quality. The effects of a postharvest heat treatment on aspects of ripening and development of thermotolerance is discussed. Ethylene production, respiration, softening, color change and taste components such as soluble solids, acidity and volatiles are dealt with. Heat treatment induction of thermotolerance, both to high and low temperatures, so as to prevent heat injury and chilling injury is discussed, as well as the possible mechanisms of action of this response. Heat damage manifestations from unsuccessful treatments are described. © 1998 Elsevier Science B.V. All rights reserved.

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1. Introduction

During the past few years there has been increasing interest in the use of heat treatments postharvest to control insect pests, prevent fungal rots and affect the ripening or response to temperature extremes of the commodity. Part of this interest is because there is a growing demand to decrease the postharvest use of chemicals against pathogens and insects. Heat treatment substitutes a non-damaging physical treatment for chemical

prevention. A number of previous reviews have dealt with specialized aspects of heat treatments (Couey, 1989; Paull, 1990; Barkai-Golan and Phillips, 1991; Klein and Lurie, 1991, 1992a; Coates and Johnson, 1993; Paull, 1994; Paull and McDonald, 1994). This review will focus on the responses of the commodity to heat treatment.

There are three methods in use to heat commodities; hot water, vapor heat and hot air. Hot water was originally used for fungal control, but has been extended to disinfestation of insects.

Vapor heat was developed specifically for insect control, and hot air has been used for both fungal and insect control and to study the response of commodities to high temperature. The last two methods (vapor heat and hot air) have subdivisions in that sometimes the air is relatively static, and sometimes air flow is quite high; additionally, hot air can have humidity control or not. All of these permutations may affect the response of the commodity to the heat treatment and affect the length of time of exposure needed to achieve a desired effect.

2. Heat treatments

2.1. Hot water dips and sprays

Hot water dips are effective for fungal pathogen control, because fungal spores and latent infections are either on the surface or in the first few cell layers under the peel of the fruit or vegetable. Postharvest dips to control decay are often applied for only a few minutes, at temperatures higher than heat treatments designed to kill insect pests located at the interior of a commodity, because only the surface of the commodity requires heating. Many fruits and vegetables tolerate exposure to water temperatures of 50–60°C for up to 10 min, but shorter exposure at these temperatures can control many postharvest plant pathogens (Barkai-Golan and Phillips, 1991). In contrast, hot water dips for fruit require 90 min exposure to 46°C.

Fungicide effectiveness can be enhanced by applying the fungicide in a hot water bath, thus allowing more effective fungal control with a reduction in chemicals. This has been particularly effective on citrus with the fungicides thiabendazole and imazalil (McDonald et al., 1991; Wild, 1993; Schirra and Mulas, 1995a,b). In addition, generally recognized as safe (GRAS) compounds have been applied in hot water to improve the efficiency of their antifungal action. Heated solutions (45°C) of sulfur dioxide, ethanol or sodium carbonate have been used to control green mold (*Penicillium digitatum*) on citrus fruits (Smilanick et al., 1995, 1997). These compounds were as

effective as an imazalil dip at 25°C in controlling artificial inoculations of the fungus (Smilanick et al., 1995).

A recent extension of the hot water treatment has been the development of a hot water spray machine (Fallik et al., 1996a). This is a technique designed to be part of a sorting line, whereby the commodity is moved by means of brush rollers through a pressurized spray of hot water. By varying the speed of the brushes and the number of nozzles spraying the water, the commodity can be exposed to high temperatures for 10–60 s. The water is recycled, but because of the temperatures used (50–70°C), organisms which are washed off the product into the water do not survive. This machine is in use both to clean and to reduce pathogen presence on a number of fruits and vegetables, such as mangos (Prusky et al., 1997) and peppers (Fallik et al., 1996b). Packers receive a premium for using this machine on these commodities as well as on corn and melons in Israel.

Hot water dips have been used for disinfecting insects as well (Couey, 1989). Since hot water is a more efficient heat transfer medium than hot air (Shellie and Mangan, 1994a), when properly circulated through a load of fruit a uniform temperature profile is established in the bath. For disinfestation a longer treatment is necessary than for fungal control because the total fruit and not just the surface has to be brought to the proper temperature. Procedures have been developed to disinfest a number of subtropical and tropical fruits from various species of fruit fly (Paull, 1994). The times of immersion can be 1 h or more and temperatures are below 50°C, in contrast to many antifungal treatments which are for minutes at temperatures above 50°C.

2.2. Vapor heat

Vapor heat is a method of heating fruit with air saturated with water vapor at temperatures of 40–50°C to kill insect eggs and larvae as a quarantine treatment before fresh market shipment (Animal and Plant Health Inspection Service, 1985). Heat transfer is by condensation of water vapor on the cooler fruit surface. This procedure was first used to kill Mediterranean (*Ceratitidis*

capitata Wiedemann) and Mexican (*Anastrepha ludens* Loew) fruit fly (Hawkins, 1932; Baker, 1952) in a chamber without forced air. However, once ethylene dibromide and methyl bromide came into use as inexpensive chemical fumigants, vapor heat was abandoned. With the ban on use of ethylene dibromide in 1984, and the imminent removal of methyl bromide from use in 2010, vapor heat has again come into use (Gaffney et al., 1990). However, in modern facilities the vapor heat includes forced air which circulates through the pallets and heats the commodity more quickly than vapor heat without forced air. Commercial facilities operate in many countries, mainly for use on subtropical fruits, particularly mango and papaya (Paull, 1994). In addition, studies have been conducted for using vapor or moist forced air to disinfest many fruits and vegetables from various insect pests (Shellie and Mangan, 1993; Shellie et al., 1993; Shellie and Mangan, 1994b).

The treatment consists of a period of warming (approach time) which can be faster or slower depending on a commodity's sensitivity to high temperatures. Then there is a holding period when the interior temperature of the produce reaches the desired temperature for the length of time required to kill the insect. The last part is the cooling down period which can be air cooling (slow) or hydrocooling (fast). Thus, there are a number of components of the treatment which can be manipulated to find the best combination for elimination of the insect pest without damaging the commodity.

2.3. Hot air

Hot air can be applied by placing fruit or vegetables in a heated chamber with a ventilating fan, or by applying forced hot air where the speed of air circulation is precisely controlled. Hot air, whether forced or not, heats more slowly than hot water immersion or forced vapor heat, although forced hot air will heat produce faster than a regular heating chamber. The hot air chamber has been utilized to study physiological changes in fruits and vegetables in response to heat (Klein and Lurie, 1991, 1992a).

Forced hot air, however, has been used to develop quarantine procedures (Gaffney and Armstrong, 1990). One reason is that the high humidity in vapor heat can sometimes damage the fruit being treated, while the slower heating time and lower humidity of forced hot air can cause less damage. A high temperature forced air quarantine treatment to kill Mediterranean fruit fly, melon fly and oriental fruit fly on papayas has been developed (Armstrong et al., 1989; Hansen et al., 1990). This procedure may require rapid cooling after the heat treatment to prevent fruit injury, as may the forced hot air treatment for citrus (Sharp and Gould, 1994; Sharp and McGuire, 1996). Recently, the heat treatment for papaya has been modified so that hydrocooling is not necessary (Armstrong et al., 1995). This method is under investigation for other commodities such as persimmons (Dentener et al., 1996).

Exposure to high temperature forced or static air can also decrease fungal infections. Heating without forced air can reduce decay caused by *Botrytis cinerea* and *Penicillium expansum* in apple fruit (Fallik et al., 1996c; Klein et al., 1997b) and *Botrytis cinerea* in tomatoes (Fallik et al., 1993). The treatments used in these cases are long term heating, from 12 to 96 h at temperatures ranging from 38 to 46°C, and are unlikely to become a commercially attractive treatment. However, the potential to have in hot air treatment a means of beneficially affecting commodity physiology and at the same time preventing both insect and fungal invasion justifies further development of these treatments.

3. Commodity responses

3.1. Flowers

Not being eaten, flowers are not subject to the same restrictions concerning chemical residues as other commodities. On the other hand, with increasing international trade flowers are subjected to the same international restrictions on the import of insects as other commodities. Current postharvest approaches are hand removal, insecticidal dips or fumigation, biological control agents

and temperature treatments. A recent review discusses the advantages and disadvantages of these treatments (Hansen and Hara, 1994).

Both hot water dips and forced vapor heat have been used to kill insects on flowers. Hot water treatments for flowers or cut foliage can either be for hours at temperatures of 42°C or below, or for minutes at temperatures of 46°C or above (Animal and Plant Health Inspection Service, 1992). Aphids in red ginger were destroyed after 5 min in a 47°C water bath without plant injury (Hansen et al., 1991). Hara et al. (1993) achieved quarantine security against magnolia white scale (*Pseudaulacaspis cockerelli* Cooley) on bird of paradise flowers by immersion in 49°C water for 10 min. Flower vase life was reduced by one or two days, depending on the stage of flower opening at the time of treatment. Hot water has been investigated against a number of insects in flowers, foliage and roots and all were controlled by 49°C for 5–12 min (Hara et al., 1997). Some flowers showed greater susceptibility to heat injury at different seasons, and preconditioning in 39°C hot air for 2–4 h before the hot water treatment eliminated this seasonal phytotoxicity (Hara et al., 1997).

Hot water dips can also be used to control fungal pathogens. Hot water treatments at temperatures around 40°C for several hours have been used to treat bulbs, seeds and other planting material against diseases (Gratwick and Southey, 1985). Dipping roses for 30 s at 50°C was effective in preventing *Botrytis cinerea* development (Elad and Volpin, 1991). Botrytis can also cause stem blight on propagated cuttings, and intervals of hot forced air under a greenhouse bench reduced the incidence of the disease (Hausbeck et al., 1996a,b).

Forced vapor heat can also be used successfully to control insects on tropical cut flowers and foliage, although in many cases the vase life is reduced by the length of time required for full insect mortality (Hansen et al., 1992). The same strictures apply for flowers as for other commodities; to find a time–temperature treatment which will destroy the insect or fungal pest without harming the plant tissue. Unlike fruits and vegetables, there have been no reports of heat treatments that improve vase life.

3.2. Fruit ripening

Ripening of most climacteric fruit is characterized by softening of flesh, an increase in the sugar:acid ratio, enhanced color development, and increases in respiratory activity and ethylene production. Exposing fruit to high temperatures attenuates some of these processes while enhancing others. This anomalous situation results in heated fruit being more advanced in some ripening characteristics than non-heated fruit while maintaining their quality longer during shelf life at 20°C.

The inhibition of ripening by heat may be mediated by its effect on the ripening hormone, ethylene. Hot air treatment of 35–40°C inhibits ethylene synthesis within hours in both apples and tomatoes (Biggs et al., 1988; Klein, 1989). Elevated temperatures of 35–38°C can cause endogenous ACC to accumulate in apple and tomato tissue concomitantly with the decrease in ethylene (Yu et al., 1980; Atta Aly, 1992), though raising the temperature higher or holding the fruit longer at the raised temperature will cause the disappearance of ACC as well (Klein, 1989; Atta Aly, 1992). A rapid loss of ACC oxidase activity occurs in many fruit exposed for a few hours to hot water immersion at 42–46°C (Chan, 1986a,b; Dunlap et al., 1990; Paull and Chen, 1990), due primarily to decrease in ACC oxidase mRNA and cessation of enzyme synthesis (Lurie et al., 1996b). ACC synthase is also heat labile (Biggs et al., 1988), but most studies indicate that it is less heat sensitive than ACC oxidase (Klein, 1989; Atta Aly, 1992). The inhibition of ethylene formation is reversed when the fruits are removed from heat (Field, 1984; Biggs et al., 1988; Dunlap et al., 1990; Paull and Chen, 1990; Chan, 1991), and often the level of ethylene rises to higher levels than in non-heated fruits (Klein and Lurie, 1990; Lurie and Klein, 1992b). This recovery requires protein synthesis (Biggs et al., 1988), and studies showed that both mRNA and protein of ACC oxidase accumulate during recovery from a 38°C hot air treatment (Lurie et al., 1996b).

During the heating period, not only is endogenous ethylene production inhibited, but fruits will not respond to exogenous ethylene (Seymour et

al., 1987; Yang et al., 1990). This indicates either a loss or inactivation of ethylene receptors, or the inability to transfer the signal to the subsequent series of events leading to ripening. No information is available on the response of ethylene receptors to heat, but it has been shown that the expression of tomato ripening genes is inhibited by high temperature (Picton and Grierson, 1988). Specific mRNAs associated with ripening processes were found to disappear during a 38°C hot air treatment of tomatoes and reappear during recovery from heat (Lurie et al., 1996b). These included ACC oxidase, polygalacturonase and lycopene synthase.

Fruits subjected to extended hot air treatments of 38 or 40°C often soften more slowly than non-heated fruits, although disinfestation procedures for mangos and papaya of hot forced air for 4 h at 50°C led to faster softening after the treatment (Shellie and Mangan, 1994a). A number of researchers have described the effect of continuous storage in hot air on fruit firmness. Plums (Tsuji et al., 1984), pears (Maxie et al., 1974), avocados (Eaks, 1978), and tomatoes (Biggs et al., 1988) softened more slowly when held continuously at temperatures between 30 and 40°C than at 20°C. The rate of softening increased when heated fruit were returned to 20°C, but it was still less than that of non-heated fruit. Even after 6 months of storage at 0°C and subsequent shelf life for 7 days at 20°C, apples that had been held at 38°C for 3 or 4 days pre-storage were 10 N firmer than non-heated fruit (Porritt and Lidster, 1978; Klein and Lurie, 1990; Klein et al., 1990; Sams et al., 1993; Conway et al., 1994). The texture of hot air treated apples after storage was different quantitatively and qualitatively from non-heated fruit. Conway et al. (1994) using compression tests found the heated apples to be tougher, while Lurie and Nussinovich (1996), using Instron compression and shearing measurements, found heated apples to be crisper than non-heated.

Cell wall studies of apple fruit found less soluble pectin and more insoluble pectin after exposure to 38°C air for 4 days than in fruit that had not been heated, an indication of inhibition of uronic acid degradation (Klein et al., 1990; Ben-Shalom et al., 1993, 1996). In addition, in these

heated apples less calcium was present in the water soluble pectin and more was bound to the cell wall (Lurie and Klein, 1992a). It was thought that this was the result of the activity of pectin esterase creating more sites for calcium binding, but a study of heated and non-heated fruits showed a similar degree of esterification in both (Klein et al., 1995). During the heating period arabinose and galactose content decreased with no accompanying decrease in uronic acids (Ben-Shalom et al., 1993). It is possible that loss of neutral sugar side chains during the heat treatment may lead to closer packing of the pectin strands and in turn hinder enzymic cleavage during and after storage, resulting in firmer fruit.

The decrease in the rate of softening may be due to inhibition of the synthesis of cell wall hydrolytic enzymes such as polygalacturonase (Chan et al., 1981; Yoshida et al., 1984; Lazan et al., 1989) and α - and β -galactosidase (Sozzi et al., 1996). In tomato mRNA for polygalacturonase was absent in fruit during a heat treatment of 1–3 days at 38°C and appeared after the fruit was removed from heat (Lurie et al., 1996b). Depending on the length of treatment, heated tomato fruits may recover and soften to the same extent as non-heated fruits (Lurie and Klein, 1992b), or remain firmer than non-heated fruits (Mitcham and McDonald, 1992). In the former study tomatoes were held for 3 days at 38°C and in the latter for 4 days at 40°C.

Flavor characteristics of fruits can be affected by a heat treatment. Titratable acidity declines in apples held for 3 or 4 days at 38°C while soluble solids concentration is not affected by the treatment (Liu, 1978; Porritt and Lidster, 1978; Klein and Lurie, 1990). The same was found after hot forced air treatment of nectarines at 41–46°C for 1–2 days for insect disinfestation (Lay-Yee and Rose, 1994), and hot water immersion for 15 min at 35, 45 or 55°C of strawberries for decay control (Garcia et al., 1995a). In tomatoes hot air heated at 38°C for 2–3 days (Lurie and Klein, 1991, 1992b; Lurie and Sabehat, 1997) and grapefruit held in forced hot air of 43.5°C for 4.5 h (Miller and McDonald, 1992), neither titratable acidity nor soluble solids content was affected by heat. However, the same fruits in other studies showed

reduction in titratable acidity (D'hallewin et al., 1994; Garcia et al., 1995b; Shellie and Mangan, 1996). The disparate results may be due to cultivar differences or differences in the heat treatment.

In some commodities, sugar content is favorably affected by heat treatment. For example, 3 h of 45°C water before cool storage of muskmelons prevented the loss in sucrose which occurred in non-heated fruit during storage (Lingle et al., 1987). Squash sucrose content can also be raised by holding them at 30°C in air before storage (Bycroft et al., 1997). These heat treated squash were perceived as sweeter by a taste panel. Heated tomatoes were not distinguished from non-heated by a taste panel, but heated Golden Delicious apples (4 days at 38°C) were perceived as crisper, sweeter and overall more acceptable than non-heated fruit (Klein et al., 1997a). In the latter fruit the sweetness was due more to decrease in acidity than increase in sugar content.

Volatiles production can also be affected by a heat treatment of hot water immersion at 42°C for 60 min or hot air for 2 days at 38°C (McDonald et al., 1996). Volatiles production in apples is enhanced during a 38°C hot air treatment, is inhibited immediately following the treatment, and recovers afterwards (Fallik et al., 1997). The profile of the volatiles is also changed, with some being enhanced more than others by the heat. In tomatoes as well the highest volatiles levels in ripe fruit were from fruit heated at the mature green stage and then stored at 13°C before ripening (McDonald et al., 1996).

Heat treatment leads to an accelerated rate of degreening in apples (Liu, 1978; Klein et al., 1990). Chlorophyll content in apple peel, plantain peel and tomato pericarp decreased during a hot air treatment of 35–40°C (Seymour et al., 1987; Lurie and Klein, 1990, 1991). Hot water immersion at 45°C for 30–60 min can also lead to yellowing of cucumbers (Chan and Linse, 1989), as does forced vapor heat for 30 min at 45°C for zucchini (Jacobi et al., 1996) (zucchini are known as courgettes in some countries). Color changes in papaya skin or flesh were not affected by hot water immersion at 42°C for 30 min followed by 49°C for 90 min (Paull and Chen, 1990) and the

same hot air treatment which stimulated degreening of plantains failed to degreen bananas (Seymour et al., 1987). Hot water dips at 43–55°C for up to 10 min delayed yellowing of broccoli (Forney, 1995; Tian et al., 1996, 1997). The difference in responses of different commodities may be an indication of whether new enzymes must be synthesized to effect the color changes or not. In the case of apples chlorophyll degradation reveals the yellow of the underlying carotenoids already present, while other fruits may require synthesis of carotenoids. For example, it has been found that hot air at 38°C or higher inhibits lycopene synthesis in tomatoes (Cheng et al. 1988). The inhibition of lycopene is due to the inhibition of transcription of mRNA for lycopene synthase, a key enzyme in the pathway, and this recovers after removal from heat (Lurie et al., 1996b). In bananas the inhibition of degreening during the heat treatment appears to be due to the absence of the chlorophyll oxidase enzyme resulting in the retention of chlorophyll in the peel (Blackbourn et al., 1989). It is not known if this inhibition is at the level of gene expression.

Respiration rate is enhanced initially for the first day or two by high temperatures of 35–40°C (Lurie and Klein, 1990, 1991), but at longer times at high temperatures the rate decreases (Cheng et al., 1988; Inaba and Chachin, 1989; Lurie and Klein, 1991). With increasing time at 35°C a greater proportion of the respiration is from the cyanide insensitive pathway (Inaba and Chachin, 1989). When fruits return to ambient temperature often the respiration is lower than non-heated fruits (Klein and Lurie, 1990). A heat treatment, depending on temperature and length of exposure, can decrease or increase the climacteric respiration peak as well as advancing or delaying it after treatment (Eaks, 1978; Klein and Lurie, 1990). The response of a particular fruit or vegetable will result from a combination of factors: preharvest environmental conditions, the physiological age of the commodity, the time and temperature of exposure, whether the commodity is removed from heat to storage or to ripening temperature, whether the heat treatment causes damage.

3.2.1. *Thermotolerance*

The mechanism by which a heat treatment causes changes in fruit ripening, such as inhibition of ethylene synthesis, and cell wall degrading enzymes, may be tied to changes in gene expression and protein synthesis. During a high temperature treatment the mRNA of fruit ripening genes disappear and those of heat shock proteins (HSP) accumulate (Picton and Grierson, 1988; Lurie et al., 1996b). An immediate response of high temperature, generally temperatures above 35°C, is disassociation of polyribosomes and then a reassociation of some ribosomes into polyribosomes which preferentially translate the mRNA of HSP (Ferguson et al., 1994). This response both down-regulates normal protein synthesis even without degradation of the mRNAs and upregulates HSP synthesis. The synthesis of HSP is part of the response of all organisms to a heat stress, from man to bacteria (Lindquist, 1986). Studies with many organisms have demonstrated that exposure to elevated, sublethal temperatures induces thermotolerance, which protects them from a second exposure to a normally lethal temperature. Development of thermotolerance has been associated with synthesis of HSP, and loss of thermotolerance with the disappearance of HSP (Vierling, 1991). In addition, the development of thermotolerance is dependent on protein synthesis; pepper discs treated with protein synthesis inhibitors before temperature treatments did not develop thermotolerance (Liu et al., 1996).

Therefore, heat exposure severity will moderate the thermotolerance response. Development of thermotolerance is dependent on exposure temperature. The exposure temperature must be enough to initiate the synthesis of HSP, yet not too hot so that transcription and translation of HSP are inhibited. Temperatures of 35–40°C have been found to be effective, depending upon the commodity. At 42°C or higher, HSP synthesis is attenuated and commodities are more likely to suffer heat damage (Ferguson et al., 1994).

The propensity of a medium heat stress to protect against a higher heat stress has been used to develop treatments to prevent commodity damage while killing fungal pathogens or insect pests. A two-stage hot water treatment, 30 min at 42°C

followed by immersion in 49°C water, was developed for papaya disinfestation (Couey and Hayes, 1986). Induction of heat tolerance in papaya has been reported by Paull and coworkers (Paull et al., 1986; Paull and Chen, 1990). Holding the fruit at 38–42°C for 1 h reduces damage after hot water immersion at 49°C for 70 min. In a similar way, conditioning in 37 or 39°C air before a 46° or 47°C hot water disinfestation reduces treatment damage on avocados and mangos (Joyce and Shorter, 1994; Jacobi et al., 1995a,b). Similar benefits of prior temperature conditioning have been found for avocados (Woolf and Lay-Yee, 1997) and cucumbers (Chan and Linse, 1989).

3.3. *Tolerance to chilling injury*

The correlation between HSP and thermotolerance has been established in many organisms, but only recently has it been found that a heat stress can condition plants to low temperature. Saltveit and coworkers found that prior high temperature exposure of a number of hours at 38–42°C hot air affected chilling sensitivity of tomato discs (Saltveit, 1991), mung bean hypocotyls (Collins et al., 1993) and cucumber cotyledons and seeds (Lafuente et al., 1991; Jennings and Saltveit, 1994). When a heat treatment of 2–3 days in 38°C air was applied to tomato fruit their sensitivity to low temperature was reduced and they could be stored for up to a month at 2°C without developing chilling injury (Lurie and Klein, 1991; Sabehat et al., 1996; Lurie and Sabehat, 1997). This resistance to low temperature injury was found to be contingent on the presence of HSP (Lafuente et al., 1991; Sabehat et al., 1996). In a study with avocado discs, maximal HSP production was found after 4 h at 38°C and heating provided a significant degree of protection from chilling injury (Florissen et al., 1996). This response has been found in numerous other commodities including avocado (Woolf et al., 1995), citrus (Wild, 1993; Rodov et al., 1995; Schirra and Mulas, 1995a), cucumber (McCollum et al., 1995), mango (McCollum et al., 1993), pepper (Mencarelli et al., 1993), persimmons (Burmeister et al., 1997; Lay-Yee et al., 1997; Woolf et al., 1997) and zucchini (Wang, 1994). However, the response may in

some cases be cultivar specific. For example, Whitaker (1994) found no benefit in heating 'Rutgers' tomato fruit.

Other examples of the effectiveness of a heat exposure in reducing chilling sensitivity are the use of a 12–18 h 38°C hot air treatment together with cold quarantine to disinfect avocado from fruit fly without having the fruit develop chilling injury (Sanxter et al., 1994; Nishijima et al., 1995). When the heat treatment was given as a fungicidal hot water dip, rots were also controlled (Jessup, 1991).

The reduction of sensitivity to chilling injury of fruits may not be due solely to the presence of HSP. Chilling injury has long been thought to begin with membrane damage (Lyons, 1973), and a heat treatment of 35–40°C may cause membrane alterations. High temperature (35–40°C) increases membrane leakage (Inaba and Chachin, 1988; Lurie and Klein, 1990, 1991), but after removal from heat stress the tissue recovers and leakage returns to levels found in tissue held at 20°C (Lurie and Klein, 1990). Using membrane leakage as a measure of chilling injury Saltveit (1991) found that conditioning tomato fruit discs at 37°C for 4 h reduced leakage when discs were stored at chilling temperature. An examination of the lipid composition of apple plasma membrane showed that after a 4 day heating in 38°C air and 0°C cold storage for 4 months there were more phospholipids and greater fatty acid unsaturation in heated than in non-heated fruits (Lurie et al., 1995). Whitaker et al. (1997) looking at total apple lipids also found greater fatty acid unsaturation though not greater phospholipid content in fruit heated by the same method. This would indicate more fluid membranes in fruit after exposure to conditioning temperatures, and correspond with lower indiscriminate leakage from conditioned fruit and vegetable tissue. These changes in lipid composition were found also after storage at 2°C in tomatoes treated prestorage for 2 days at 38°C with hot air, or immersed in 46 and 48°C water for 2–3 min, an indication that even short exposure to heat can instigate processes leading to tissue adaptation to low temperature (Lurie et al., 1997).

Apples are normally thought to be fruits which are insensitive to low temperature, but superficial scald is a physiological storage disorder which is a form of chilling injury (Bramlage and Meir, 1990). It is an oxidative process causing peel browning, and has been correlated with the oxidation of α -farnesene, a component of the apple wax (Huelin and Coggiola, 1970). A heat treatment of 3–4 days at 38°C of apples before storage, controls this disorder during the first months of 0°C storage by inhibiting the accumulation of α -farnesene and consequently decreasing the oxidation products (Lurie et al., 1990). The inhibitory effect allows for 3–4 months of air storage without scald developing (Lurie et al., 1990; Combrink et al., 1994). Part of the overall reduction in α -farnesene may be due as well to a thinner wax layer and changes in structure of the wax surface after the heat treatment (Roy et al., 1994; Lurie et al., 1996a).

3.4. Heat damage

Although this review has been focusing on the positive response of commodities to a heat treatment, there is always a danger of tissue damage. This is one reason why there are such a multitude of treatments, to find a time–temperature regime which will produce the desired effect (disinfestation, fungal control) without damaging the commodity. Damage can be both external and internal. External is generally peel browning (Kerbel et al., 1987; Klein and Lurie, 1992b; Shellie et al., 1993; Lay-Yee and Rose, 1994; Woolf and Laing, 1996), pitting (Miller et al., 1988; Jacobi and Gowanlock, 1995), or yellowing of green vegetables such as zucchini (Jacobi et al., 1996) or cucumber (Chan and Linse, 1989). Tissue damage caused by heat will also result in increased decay development (Jacobi and Wong, 1992; Jacobi et al., 1993; Lay-Yee and Rose, 1994). Internal damage can evidence itself in mango and papaya as poor color development, abnormal softening, the lack of starch breakdown and the development of internal cavities (An and Paull, 1990; Jacobi and Wong, 1992; Mitcham and McDonald, 1993; Paull, 1995). In addition, the fruit can soften quickly or show abnormal softening where some

areas of the flesh remain hard while others soften (Paull and Chen, 1990). Internal damage on other fruits can include flesh darkening on lychee and nectarines (Jacobi et al., 1993; Lay-Yee and Rose, 1994). If the produce is stored after the heat treatment at low temperature the heat damage can be confused with chilling injury which has similar symptoms.

4. Conclusions

There has been intensive research during the past few years on heat treatment of commodities, particularly for insect eradication. However, the work has been for the most part empirical, that is, to try a matrix of time–temperature treatments and find the combination which will kill the insect pest with minimal damage to the commodity. What is missing is modeling work to predict the commodity response, similarly to what has been done previously for insects. This kind of research direction might advance the field faster than it is now progressing. In addition, a better understanding of the molecular and biochemical processes occurring in the fruit and vegetable tissue during and following the heat treatment should aid in developing successful treatments. It is tempting to envision an integrated treatment which would control both insect and fungal pests and beneficially affect commodity quality in addition to being economically attractive. Thus far, only a few such treatments have been developed.

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