Essential Principles of Postharvest Heat Treatments in Fruit Crops

Abstract

In recent years, the field of heat treatment for commodities, particularly in the context of pest control and pathogen reduction, has advanced significantly. Hot water treatments (HWT) have emerged as a promising solution, addressing a range of postharvest challenges, from insect control to preventing fungal development and storage disorders like chilling injury. These treatments involve diverse time and temperature conditions, spanning from extended exposure at temperatures around 35-39°C in hot air to brief bursts at temperatures up to 63°C in hot water. Historically, much of the research in this field has been focused on solving specific problems, with less attention given to understanding how commodities themselves respond to these treatments. However, since the beginning of the 21st century, several research groups have taken an active interest in exploring the molecular responses and changes that occur within commodities subjected to heat treatment. This review centres on examining how fruits and vegetables react at the molecular level to high-temperature postharvest stress. It delves into the genetic (transcriptome), protein (proteome), and metabolic (metabolome) changes that occur in response to various heat treatments. By investigating these responses, researchers aim to not only optimize heat treatment strategies but also gain a deeper understanding of how commodities interact with these processes. In essence, this review provides valuable insights into the molecular dynamics of commodities undergoing heat treatments, shedding light on the intricate biochemical processes that influence their postharvest quality and safety.

Keywords: Heat Treatment, Postharvest, Molecular Responses, Pest Control, Pathogen Reduction

Introduction

In recent years, there has been increasing interest in the use of post-harvest heat treatments to control insect pests, prevent fungal rot, and affect ripening or reactivity. with the extreme temperature of the product. Part of this concern is due to the growing need to reduce the use of post-harvest chemicals against pathogens and insects. Heat treatment replaces harmless physical treatment to prevent chemicals. Several previous reviews have addressed specific aspects of heat treatment (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 product's response to heat treatment. There are a number of methods used to heat the product; hot water, steam heat and hot air, and we will study responses to heat stress in horticultural products. Hot water was originally used to control fungi but has since been expanded to disinfect insects. Steam heat was developed specifically for insect control, and hot air was used to control fungi and insects as well as to study the response of the product to high temperatures. The last two methods (thermal steam and hot air) have subdivisions where sometimes the air is relatively still and sometimes the airflow is quite high; Furthermore, warm air may or may not have humidity control. All of

these permutations can affect the response of the product to heat treatment and affect the contact time required to achieve the desired effect. The majority of published studies describe the physical and physiological responses of fruits or vegetables to heat treatment. In some studies, biochemical data were collected on heat shock protein (HSP) and other stress proteins such as resistant pathogens (RP), antioxidants, etc. However, much of the research is focused on developing an effective treatment for a problem, whether it's an insect pest, a pathogen attack, or a storage disorder.

This review will focus on the molecular responses of fruits and vegetables to high-temperature postharvest stress. It will summarize what has been published in the 21st century and will emphasize the study of 'omics'; metabolism, proteomics and transcription. It will not cover research on the effects of high temperatures on minimally processed products, which is of interest. It also will not treat heat damage, which is also significant (Ghasemnezhad*et al.,* 2008). The knowledge gained in these studies will help develop more precise treatments with less chance of unwanted side effects to the product.

Heat treatments

Hot Soaking and Spraying Hot water soaking are effective in controlling fungal pathogens because fungal spores and potential infections are found on the surface or in the first cell layers under the skin of a fruit or vegetable.Postharvest dipping for rot control is usually applied for only a few minutes, at a higher temperature than heat treatment designed to kill pests inside the product, because only heating is required.Many fruits and vegetables tolerate exposure to water temperatures of 50-60°C for up to 10 minutes, but shorter exposures to these temperatures can control many of the following plant pathogens.In contrast, a hot bath for fruit requires 90 minutes of exposure at 46°C.The fungicidal effect can be improved by applying the fungicide in a warm bath, allowing for more effective fungal control with a reduction in chemicals.Additionally, compounds generally recognized as safe (GRAS) have been used in hot water to improve their antifungal efficacy.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.,* 1997) These compounds were as effective as imazalil immersion solution at 25°C in controlling fungal culture (Smilanick*et al.*, 1995). A recent extension of hot water treatment is the development of water heaters (Fallik*et al.*, 1996a). Hot baths have also been used to disinfect insects (Couey, 1989).Since hot water is a more efficient means of heat transfer than hot air (Shellie and Mangan, 1994a), when properly circulated through a quantity of fruit, a uniform temperature profile is established in the bath.For sterilization, a longer treatment is required than for fungal control because the whole fruit and not just the surface must be brought to the correct temperature.Steam heating is a method of heating fruit with air saturated with steam to a temperature of 40-50°C to kill insect eggs and larvae as a pre-quarantine treatment.Heat transfer takes place because water vapour condenses on the cooler surface of the fruit. This procedure was first used to kill the Mediterranean fruit fly (*Ceratitiscapitata Wiedemann*) and the Mexican fruit fly (*Anastrephaludens* Loew) (Hawkins, 1932; Baker, 1952) in a pulseless air chamber. However, once ethylene dibromide and methyl bromide were used as inexpensive fumigants, the steam heat was eliminated. With the ban on the use of ethylene dibromide in 1984 and the discontinuation of methyl bromide in 2010, steam heat was once again used (Gaffney *et al.*,1990).

However, in modern installations, steam heat consists of forced air that circulates through pallets and heats goods faster than steam heat without forced air. Commercial plants operate in many countries, mainly for the use of subtropical fruits, especially mango and papaya (Paull, 1994). In addition, studies have been conducted on the use of steam or humid forced air to disinfect a variety of fruits and vegetables from various insect pests (Shellie and Mangan, 1993; Shellie *et al.,* associates, 1993; Shellie and Mangan, 1994b). The processing including a warm-up (access time) can be faster or slower depending on the sensitivity of the product to high temperatures. There is then a holding period when the internal temperature of the product reaches the desired temperature for the time it takes to kill the insects. The last part is the cooling stage which can be air-cooled (slow) or water-cooled (fast). Therefore, several treatment components can be manipulated to find the best combination for pest control without damaging the product.

Hot air

Hot air can be provided by placing fruit or vegetables in a fan-heated chamber or by a forced hot air supply where the air circulation rate is precisely controlled.Hot air chambers have been used to study the physiological changes of fruits and vegetables in response to heat (Klein and Lurie, 1991, 1992a).One reason is that the high humidity of steam heat can sometimes damage processed fruit, while the slower heating time and lower humidity of forced hot air can cause less spoilage. A forced air isolation treatment at high temperatures for the control of Mediterranean fruit flies, melon fruit flies and oriental fruit flies on papaya has been developed (Armstrong *et al.,* 1989). This process may require rapid cooling after heat treatment to avoid fruit damage, similar to forced hot air treatment for citrus fruit (Sharp and Gould, 1994; Sharp and McGuire, 1996).Recently, the heat treatment of papaya has been changed to not require water cooling (Armstrong *et al.,* 1995).

Exposure to static or forced air at high temperatures can also reduce fungal infections.Heating without forced air can reduce rot diseases caused by *Botrytis cinerea* and *Penicillium expansum* in apples (Fallik*et al.,*1996b).However, the potential of hot air treatment as a means of beneficially influencing product physiology, while also preventing both insect and fungal entry, demonstrates further development of these pathogens.

Heat stress response

Temperature is one of the most important environmental factors regulating plant growth and development. Plants have evolved signaling pathways to detect changes in environmental temperature and regulate their metabolism and cellular function to prevent heat-related damage.Many features of the heat stress response pathway are conserved in prokaryotes and eukaryotes (kotak*et al.,* 2007). Because plants are stalkless organisms that cannot escape stressful conditions, they invest valuable resources in altering their metabolism to prevent heat damage, in a process called heat stress.HSP accumulation, under the control of heat stress transcription factors (HSFs), plays a central role in the heat stress response and acquired heat tolerance in plants and other organisms.Heat stress affects the stability of many proteins, membranes, RNAs and cellular structures, and alters the efficiency of cellular enzymatic reactions, causing metabolic imbalance (McClung *et al.,* 2010). In fact, ROS is generally a component of heat stress, and increased antioxidation is part of the response to heat stress (Mittler *et al.,* 2012). In response to heat stress, plants reprogram their transcriptional, protein, metabolic and lipid sets (Figure 1).These changes establish a new

steady equilibrium of metabolic processes that can allow the body to function at the new temperature.High temperatures will affect most macromolecules and cause changes such as increased membrane fluidity, partial fusion of DNA and RNA chains, dissociation of protein subunits, and exposure.The specific alteration that may be high temperature stress sensing is unknown and may vary in different plants.However, mild heat stress may not lead to protein swelling and plants exhibit stress response.may also be helpful in responding to other stresses, such as the low temperatures of storage.

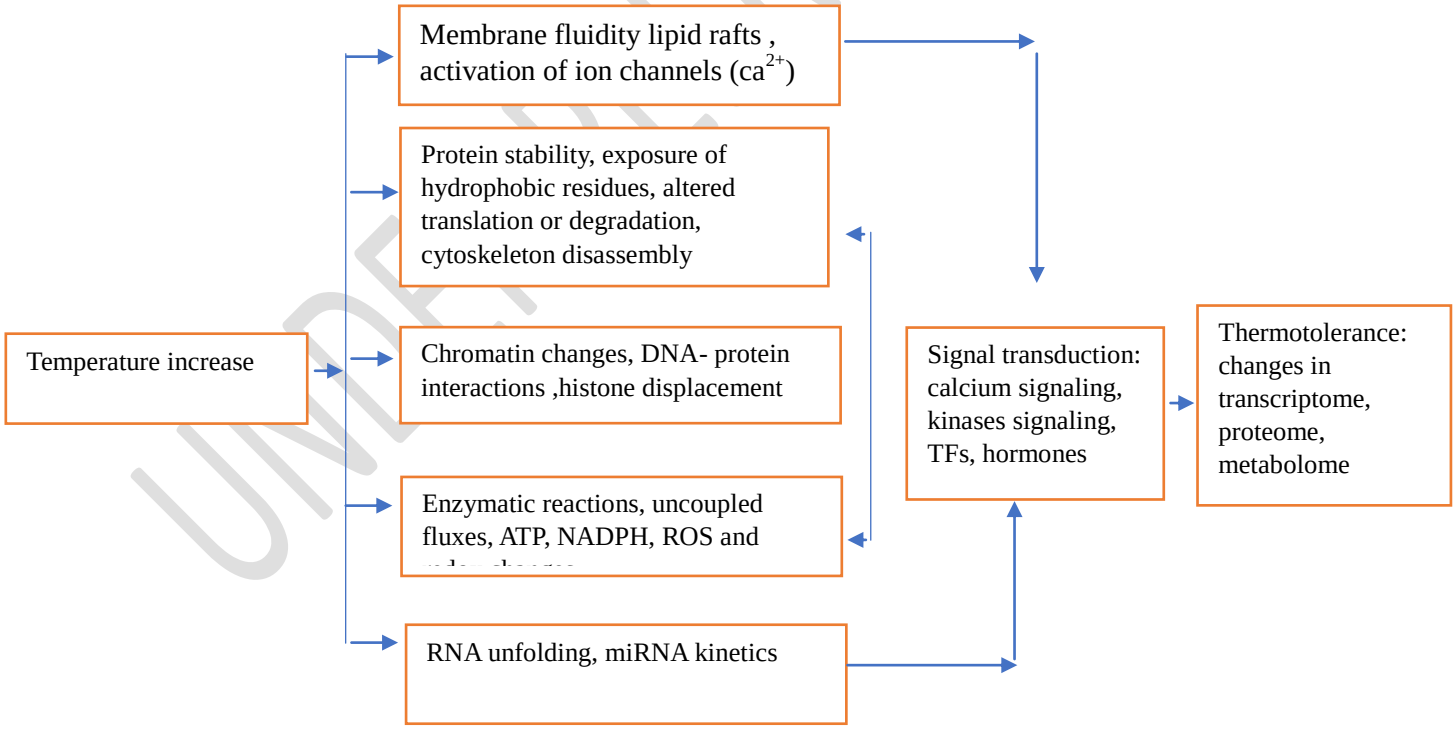

Fig1: A conceptual model of temperature sensing in plants. Temperature changes can cause membrane characteristics to change and a calcium channel to open. The subsequent inward calcium flux will activate signal transduction processes and change plant metabolism to attain thermotolerance. Other temperature-induced modifications include changes in protein stability and the exposure of hydrophobic protein residues. Ubiquitination in the cytosol and ER, histone displacement, which allows HSFs to bind to DNA, accumulation of ROS, and changes in cellular energy levels, as well as the unfolding of RNA species that may alter miRNA.

These pathways will also cause other signal transduction processes can result in thermotolerance (from Mittler *et al.*,8 2012).

Mode of Action

The defence mechanism against pathogens in fruit involves complex interactions, involving a variety of responses, such as the development of mechanical barriers against microorganisms and the production of compounds.Heat treatment induces such protective mechanisms and triggers physiological and pathological responses that enable fresh produce to withstand extreme conditions during storage and reduce the development of rotting disease.Postharvest HWD treatment at 53°C for 3 min significantly reduced the rot rate of pear melon (*Cucumis melo* L).This treatment enhanced the activity of defence-related enzymes such as phenylalanine ammonia lyase (PAL), cinnamate-4-hydroxylase (C4H), 4-coumarate-CoA ligase (4CL), polyphenol oxidase (PPO).This treatment effectively maintains fruit firmness by inhibiting the activities of endo-1,4-ß-d-glucanase (EGase), ß-glucosidase (ß-Glu), polygalacturonase (PG) and pectinesterase (PME), while promoting the accumulation of suberin and callose (Yuan *et al.,* 2013). In melons treated with HWT at 45°C for 10, 15, 20, and 25 min, (Sui *et al.,*2014) found that protein damage and ATP consumption were activated by treatment, improved control of Fusarium root rotConcludedthat the effect of HWT on Fusarium rot may be related to both direct inhibition of fungi and initiation of defence responses in fruit.

Treatment of peaches with HWD at 40°C for 5 and 10 min induced the expression of defence-related genes, including chitinase (CHI), ß-1.3-glucanase (GNS) and PAL, also trigger reactive oxygen species (ROS) accumulation, collapse of mitochondrial membrane potential, and decrease in intracellular ATP in *M.laxa conidia*; then, after 15 minutes and 3, 6, 12, 24 and 48 hours, they were treated by soaking in hot water at 60°C.The effect of heat treatment on several cell wall genes involved in maturation, such as ß-galactosidase (ß-GAL), pectin lyase (PL), PG, and pectin methyl esterase (PME), were investigated.Expression levels of genes involved in PAL and CHI protection, heat stress-related genes such as heat shock proteins 70 and 90 (HSP70, HSP90), and ROS-determining genes were also evaluated by qRT-PCR (Spadoni *et al.,* 2014). Expression levels of cell wall genes (ß-GAL, PL, PG and PME) showed an overall decrease in HWT-exposed fruits compared with controls, whereas PAL, CHI, HSP70 and ROS-scavenging increased their expression levels in heat-treated fruit compared with untreated fruit, and thus inhibited the development of brown rot by 86%.The treatment induces the expression of CpPGIP, which then rapidly induces the expression of CpNPR1, and this sequence regulates the expression of the CpPR1 gene, which can improve the fruit anthracnose resistance and reduce decomposition rate (Linle*et al.,* 1987) at 52°C for 2 min revealed resistance-related proteins such as GNS, type III chitinase, (Yun *et al.,* 2013), the H_2O_2 content decreased and the lignin content increased in the heat-treated pods compared with the control, In addition, flavonoids, which are known to be effective in reducing external stress, were up-regulated in the heat-treated pods.Interpretation of protein and metabolite data suggests that ROS and lignin play important roles in heat treatment-induced pathogen resistance and fruit physiological disorders (Yun *et al.,* 2013;Zhao *et al.,*2014)by inducing gene expression NoxA produces ROS and causes oxidative damage to spores and germs. Heat treatment reduced the viability of *B.cinerea* bacteria and spores, and this effect was at least partly responsible for the control of GrayMold observed in fruits investigated HWRB-induced global gene expression on fruit pods at 55°C for 15-20 s and

investigated genes involved in potentially related mechanisms.The discovery of three major gene clusters that are differentially expressed after HWRB treatment provides a molecular basis for the biochemical and physiological consequences of postharvest HWRB treatment; consequences include resistance to pathogens, improved colour development, and the appearance of lenticel discolouration (Luria *et al.*,2014). Treat with HWD at 40°C for 40 min and yeast antagonists, *Candida guilliermondii*. The combination of the antagonist and the HWT showed better control effects than their separate applications:Application of HWT did not affect the growth of *C.membranaefaciens* on wounds in tomato, while HWT induced a significant increase in the activities of PAL, CHI and GNS on fruit.The mechanism by which HWT enhances the biocontrol effect of antagonistic yeasts may be related to the elicitation of biochemical defence responses in tomato fruit (Zong *et al.*,2010). *Guilliermondii*stimulated a rapid increase in H_2O_2 and higher lignin deposition in cherry tomato fruit, suggesting that the oxidative burst and biosynthesis of lignin may play an important role in the preservation reaction.Furthermore, the reduction in the susceptibility of cherry tomato fruits to pathogens by combined treatment was positively correlated with an increase in the activity of PAL and GNS, both of which are involved in the protective response.(Zhao*et al.*,2009) These treatments controlled the development of caries, possibly by increasing the polyamine content in the fruit and thus contributing to the maintenance of membrane integrity (Silveira *et al.*, 2014) discussed the effectiveness of treatments for HWD at 52°C for 180 s and 56°C for 20 s as an alternative to control postharvest rot.HWD treatment at 56°C significantly increases the concentration of fatty alcohols (fats), esters and aldehydes; therefore, the lower rot rate values observed in fruits treated with HWD at 56°C may be due to increased levels of oxidizing monoterpenes, esters and aldehydes (Strano *et al.*,2014).

Fruit ripening

The ripening of most climate fruits is characterized by tenderness of the flesh, increased TSS:acid ratio, improves colour development and increases respiratory activity and ethylene production.Hot air treatment at 35-40°C inhibits ethylene synthesis for several hours in apples and tomatoes (Biggs *et al.*,1988). High temperatures of 35-38°C can induce endogenous ACC accumulation in apple and tomato tissues with a decrease in ethylene (Yu *et al.*, 1980; Atta Aly, 1992), although it either increases the temperature or Keeping fruit longer at high temperature will also cause ACC to disappear (Klein, 1989; Atta Aly, 1992).Rapid loss of ACC oxidase activity occurs in many fruits when soaked in hot water at 42–46°C for several hours (Chan, 1986a,b; Dunlap *et al.*, 1990; Paull and Chen, 1990), mainly due to a decrease in ACC oxidase mRNA and a cessation of enzyme synthesis (Lurie *et al.*,1991) ACC synthase is also heat-labile (Biggs *et al.*,1988), but most studies indicate that it is less sensitive to heat than ACC oxidase (Klein, 1989; Atta Aly, 1992).Inhibition of ethylene formation was reversed when the fruit was removed from heat (Field, 1984; Biggs *et al.*, 1988), and studies have shown that ACC oxidase mRNA and protein accumulate during recovery after hot air treatment at 38°C (Lurie *et al.*,1995). During heating, not only was the endogenous ethylene production inhibited, but the fruit also did not react with exogenous ethylene (Seymour *et al.*, 1987). There is no information on the response of ethylene receptors to heat, but the expression of tomato ripening genes has been shown to be inhibited by high temperature (Picton and Grierson, 1988).

Specific mRNAs involved in ripening were found to disappear upon 38°C hot air treatment of tomatoes and reappear upon recovery from heat (Lurie *et al.*,1995a)Several

researchers have described the effect of continuous hot air storage on fruit firmness.Even after 6 months of storage at 0°C and a subsequent shelf life of 7 days at 20°C, apples that had been stored at 38°C for 3 or 4 days before storage were still 10 N stiffer than with unpreserved fruit (Porritt and Lidster, 1978; Klein and Lurie, 1990; Klein *et al.*,1994) using compression tests showed that heated apples were firmer, while (Lurie and Russinovich; 1996), using Instron shear and compression force measurements, found that apples were made Crispy hot than unheated apples.Cell wall studies of apples showed less soluble and more insoluble pectin after exposure to 38°C air for 4 days compared with unheated fruit, which indicates inhibits the breakdown of uronic acid (Klein *et al.*,1989). In addition, in these heated apples, there is less calcium in the water-soluble pectin and more calcium is attached to the cell wall (Lurie and Klein, 1992a).

This is thought to be the result of pectin esterase activity producing more calcium binding sites, but a study of heated and unheated fruit showed similar levels of esterification in both two (Klein *et al.*,). It is possible that the loss of the neutral sugar side chain during heat treatment could result in the pectin fibers being more tightly packed and thus hindering enzymatic cleavage during and after storage, resulting in hard fruit.The reduced softening rate may be due to inhibition of synthesis of cell wall hydrolytic enzymes such as polygalacturonase (Chan *et al.*,1981) In tomato, polygalacturonase mRNA was absent from the fruit during 1-3 days of heat treatment at 38°C and appeared after the fruit was removed from heat (Lurie *et al.*,1996b). Depending on the time of treatment, heated tomato fruit may recover and become as soft as untreated fruit (Lurie and Klein, 1992b), or remain firmer than untreated fruit (Mitcham and McDonald, 1992).The flavor properties of fruit can be affected by heat treatment.The reduced titratable acidity in apples was kept for 3 or 4 days at 38°C while soluble solids concentrations were not affected by treatment (Liu, 1978; Porritt and Lidster, 1978; Klein and Lurie, 1990) .strawberries to control powdery mildew (Garcia *et al.*,1995a). In tomatoes, hot air was heated to 38°C for 2-3 days (Lurie and Klein, 1991, 1992b; Lurie and Sabehat, 1997) and grapefruits were kept in forced hot air at 43.5 h (Miller and McDonald, 1992), titratable acidity as well as dissolved solids content were both unaffected by heat.However, similar fruits in other studies have shown a decrease in titratable acidity (D`hallewin*et al.*, 1994; Garcia *et al.*,1995b).

3.3. Tolerance to chilling injury

A correlation between HSP and heat tolerance has been established in many organisms, but it was only recently discovered that heat stress can drive plants to low temperatures.Salveit and coworkers found that several hours of previous exposure to high temperatures of 38–42 °C in hot air affected the chilling sensitivity of tomato discs (Saltveit, 1991), mung bean hypostems (Collins *et al.*, 1993; Lurie and Sabehat, 1997). When tomato fruits were exposed to air at 38°C for 2-3 days, their sensitivity to low temperatures was reduced and they could be stored for up to a month at 2°C without frost damage (Lurie and Klein, 1991).This resistance to low-temperature damage was found to depend on the presence of HSP (Lafuente *et al.*,1991) In a study using avocado slices, maximal HSP production was observed after 4 h at 38°C and heating provided significant protection against chilling injury (Florissen *et al.*, 1995; Schirra and Mulas, 1995), cucumber (McCollum *et al.*,1995). , mango (McCollum *et al.*, 1993) For example, Whitaker (1994) found no benefit from heating Rutgers tomato fruit.Other examples of the effectiveness of heat exposure in reducing chilling sensitivity include a 12-18 h 38 °C hot air treatment with cold quarantine to disinfect

avocados from fruit flies without causing chilling injury to the fruit (Sanxter*et al.,* 1994). Heat treatment such as fungicidal hot trip also controlled rot (Jessup, 1991).The reduced cold sensitivity of fruits is not necessarily due to the presence of HSP alone.Chilling injury has long been thought to result from membrane damage and heat treatment at 35–40°C can cause membrane changes.

High temperature (35–40°C) increases membrane leakage (Inaba and Chachin, 1988; Lurie and Klein, 1990, 1991), but after removal of heat stress the tissue recovers and leakage returns to levels found in tissue maintained at 20°C.Using membrane leakage as a measure of chilling injury (Saltveit*et al.,* 1991), treatment of tomato fruit discs at 37°C for 4 h was found to reduce leakage when the discs were stored at freezing temperatures.An examination of the plasma membrane lipid composition of apples showed that after 4 days of heating at 38 °C in air and 4 months at 0 °C in the cold, heated fruits had more phospholipids and fatty acid unsaturation than unheated fruits. The total lipid content of apples was also found higher fatty acid unsaturation, although not higher phospholipid content, in fruits heated by the same method.This suggests that fruits have more fluid membranes when exposed to conditioning temperatures and leak less indiscriminately from treated fruit and vegetable tissues.These changes in lipid composition were also observed after storage at 2°C in tomatoes that were previously stored at 38°C for 2 days in hot air or immersed for 2-3 min in 46 and 48°C water.brief exposure to heat can initiate processes that lead to tissue adaptation to low temperatures (Lurie *et al.*,1998). Apples are generally considered to be fruits insensitive to low temperatures, but superficial burn is a physiological storage disease that is a type of frost damage (Bramlage and Meir, 1990).Heat treatment of apples for 3-4 days at 38°C before storage controls this disease during the first months of 0°C storage, preventing the accumulation of α-farnesene and thus reducing oxidation products (Lurie *et al.*,1996a). The inhibitory effect allows storage for 3-4 months in the air without burning some of the reduction in total α-farnesene may also be due to a thinner wax layer and changes in wax surface structure after heat treatment (Roy *et al.*,1994).

Heat treatment of postharvest commodities

Harvested raw materials are always exposed to low temperatures to slow down the respiration of the productand slow down ripening and ageing. Many commodities cause frostbite if the temperature is too low or if the cold conditions last too long. In many cases, heat treatment has been foundto delay or prevent the onset of freezing. For many commodities, this has been shownto be associated with the long-term presence of HSPs in the tissue and their protective effects. (Zhang *et al.,* 2005; Yi *et al.,* 2006; Sevillano *et al.,* 2010; He *et al.,* 2012).

HSPs increase duringheat stress and usually disappear rapidly when the plant is returned to ambient temperature. (Sabehat*et al.*,1996) were the first to show that HSPs were not metabolized when the commodity was placed at 2 ^{0}C instead of 20^{0} C after heat stress. Thus, heat tolerance due to heat stress may provide protection against cold stress. Postharvest heat treatments also alternormalprotein synthesis and cell metabolism during heat stress. After the application of heat stress, polyribosomes are rapidly degraded and protein synthesis stops for a while and then resumes with a new set of proteins, including HSPs (Ferguson *et al.,* 1994). As a result of this exchange, normal ripening processes are inhibited, and if the product is then placed at a low temperature, the inhibition lasts for some time. Reheated after

storage, ripening continues. Therefore, post-harvest heat treatments can modulate theripening of goods and in addition, prevent post-harvest service disorders. Studies have been published on the transcriptome, proteome and metabolic changes of several fruits from postharvest heat treatment. The investigated commodities are citrus fruits, mango, peach, potato and tomato.

Citrus

Citrus fruits are susceptible to chilling injury when exposed to temperatures lower than 2–5 ^{0}C. High-temperature treatments using HAT (sala *et al.,* 2000) and HWT(Schirra *et al.,* 1997) can delay the appearance of chilling injury and inhibit fungal infection. (Sala *et al.,* 2000) found a correlation between catalase (CAT) activity and resistance to chilling injury when mandarins were stored at 2 0 C following a 3 min HWT at 53 0C or 3-day HAT at 38 0C hot air. They did not find induction of ascorbate peroxidase (APX), glutathione reductase or superoxide dismutase (SOD) in the heated fruit. This treatment of HAT can also increase polyamine concentration and the increase correlated with lower chilling sensitivity of mandarins (Gonzalez *et al.,* 2000). The most common treatment for citrus is a HWT for 2–3 min at temperatures of 50–53 0C to control fungal development. (Yun *et al.,* 2013) conducted a proteomic and metabolomic study of mandarin peel during storage after a 2 min dip at 52^{0}C. This treatment successfully suppressed *Penicillium italicum* development and reduced chilling injury during storage. Two-dimensional gel electrophoresis (2-DE) found 50 proteins (out of 600 detected spots) that were altered at least 1.5-fold between heated and control fruit during storage ranging from 1 to 39 days after treatment. These proteins fell into the categories of glycolysis and TCA cycle, redox regulation, stress response and protein folding. The stress proteins were higher in the heated fruit and included HSPs and PR proteins, and some remained higher than control for up to 39 days after treatment. The ROS metabolism-related proteins that were found differentially in the 2-DE gels were lower during storage in heat-treated fruit and included a number of oxidoreductases and Cu/Zn SOD. However, a chloroplast APX was induced by the heat treatment and remained high in storage. CAT was not found differentially in this study. Although the ROS enzymes were not higher in the heated fruit, H2O2 decreased in the heat-treated fruit during storage, while it increased in the control fruit.

The metabolomic study identified 62 metabolites which were grouped into alcohols, amino acids, sugars, organic acids and fatty acids. Most sugars and fatty acids increased in the peel after HWT, but the sugar levels returned to control levels during storage. Organic acids were lower immediately after HWT and then recovered to a higher level than control fruit during storage. Amino acids were also decreased by HWT but remained lower than control even during storage. An exception was ornithine which was 2.5 times higher than the control fruit. This amino acid is required for the synthesis of polyamines and alkaloids, which contribute to abiotic stress tolerance in plants (Kalamaki*et al.,* 2009).

Immediately after the HWT, many flavonoids were much higher than in control fruit, including quercitin, hesperetin, naringenin and rutin. Most of these have antioxidant activity and may have contributed to the decrease in H_2O_2 (Savirantа*et al.,* 2011). Some also have antimicrobial properties.36 Most increases in these compounds disappeared during the first few days of storage.

A transcriptomics approachfound many similarities to the proteomic results of (Yun *et al.,*2013). The treatment was a HWB of grapefruit for 20 s at 62 ^{0}C, and the fruit were stored

at 2 ^{0}C for 8 and 14 weeks. There was 80% decrease in chilling injury in the fruit after storage. cDNA libraries were constructed and sequenced and then subtraction analysis was performed. Eleven different cDNAs were evaluated after the HWB and cold storage. These were six stress-responsive, three antioxidant and two lipid modification genes chosen for verification with RNA Northern blots. The HSPs were highly expressed following the HWB as were other stress genes such as dehydrin and glucanase. Lipid modifying genes, fatty acid desaturase, lipid transfer protein and lipase were induced by HWB and decreased in storage. The antioxidant genes of SOD and CAT increased during cold storage. Many of the gene expression patterns showed a priming effect from the HWB; the expression was not higher immediately after the treatment but increased over time during exposure to chilling.

In contrast to the HWT and HWB which are short heat treatments, antioxidants were increased by in oranges by a HAT for 48 h at 37 ^{0}C followed by storage at 5 ^{0}C for 30 and 60 days (Perotti *et al.,* 2011). Proteomics was conducted on both flavedo (peel) and juice sacs, and activity gels were also run for SOD, POD and APX. The activity of all three enzymes increased after HAT and remained higher than control fruit during storage. CAT and glutathione reductase activities did not change due to HAT. From the proteomic 2-DE results of juice sacs, 61 differential protein spots (out of 500 spots) were detected, either after the HAT or after 30 or 60 days of storage. The classes of proteins increased by the HAT were defence, including both HSPs and PR proteins, and metabolism. However, after 60 days at 5 0C, the differential spots found between HAT or control fruit were all higher in the control fruit. Metabolic analysis was made of both flavedo and juice sacs after treatment and after 60 days of storage and 28 compounds were analyzed. Sugars were higher in the HAT at both time points. The main organic acids in the juice, citric and malic, were not affected by the HAT, while they decreased in the flavedo initially and then recovered during storage as occurred in the HWT of (Yun *et al.,* 2013) Glycerol and putrescine increased in the HAT flavedo, while proline was very high in the fruit sacs. Proline has been found to be correlated in many plants with adaptation to cold temperatures. Putrescine, one of whose precursor's ornithine was high in HWT mandarins, was also found by (Gonzalez Aguilar *et al.,*2000) to increase in mandarins during HAT. Putrescine is essential to counteract cold stress in Arabidopsis40 and to allow ABA increases in cold-stressed plants (Cuevas *et al.,* 2009).

Peach

Both HWT and HAT were performed on peach fruits to investigate molecular changes in the fruit. 48 ^{0}C HWT for 10 min followed by storage time was used to study proteomic changes during ripening. 600 protein spots were detected by 2-DE and 35 differentially expressed spots were analyzed. Almost half of the identified proteinswere stress and defence, including antioxidants. As in the grapefruit HWB study (Sapitnitskaya*et al.,* 2006), in many cases the increase in protein content occurred with a delay of one or two days after treatment. Both APX and DHAR protein levels were higher 1 and 3 days aftertreatment, as were the two HSPs.

A series of papers have been published detailing the changes in peach fruit due to three days of HAT 390. This treatment was found to slow the development of internal decay in stored peaches, although it improved the red colour of both skin and flesh. (Lara *et al.,* 2009) observed fruit recoveryfromheat stress and recovery from ripening that was inhibited during HAT, especially softening and ethylene gene expression. D-glucose, D-fructose,

sucrose, sorbitol, malic acid and citric acid were determined using enzymatic kits. Analysis of these metabolites showed that, as in citrus fruits, sugars (glucose, fructose, sorbitol) were higher than in control fruits during and after HAT, while organic acids (citric and malic acid) were lower. Sucrose degradation and glycol and fermentation-related enzyme activity and transcript levels were analyzed by qRT-PCRafter HAT and 3 days later. Enzyme activity and expression levels of invertases (both acid and neutral), pyruvate kinase and pyruvate decarboxylase, ATP-dependent and PPi-dependent phosphofructokinase were higher in HAT fruits than in control fruits 3 days after treatment, although not at the end oftreatment. period hot treatment A 2D-DE study of peach proteins identified 600 spots and temperature significantly affected 57 spots. Forty-four different proteins were identified. 27 per cent of the identified proteins corresponded to HSPs. Some of them appeared during HAT treatment, others increased after treatment. Examination of fruit extracellular proteins during harvest found that proteases, peroxidases, porin and a small HSP were the most abundant proteins.

Glyceraldehyde-3-phosphate dehydrogenase was abundant in HAT fruits andlocalized to the cell wall by immunolocalization. qRT-PCR expression analysis of cell wall modifying genes (not detected in the proteomic study, possibly because the technique extracted only weakly related cell wall proteins) showed lower expression during HAT, consistent with inhibited softening. A transcriptomic and metabolomic study was performed on HAT peaches after five days at 0 0 °C. The aim was to determine which responses and metabolic pathways were activated under both stresses and which were limited to hot or cold.45,46 Differential transcriptional analysis ofpeaches found 127 unigenesdifferentially expressed at 20 °C heated or unheated, 60 unigenes. was induced by HAT and 45 unigenes were repressed (Lauxmann*et al.*, 2012). 20 per cent of the genes are involved in protein conversion, transcription, and RNA metabolism. This group may be responsible for the signal transduction causing the previously reported proteomic changes. Another functional gene group that was overrepresented in the induced genes were those responding to heat, cold or cold stress. A group of genes that were either induced or repressed by HAT was selected for further study and their expression was determined in peaches with 0 0 . 75% of induced HAT genes and 95% of repressed HAT geneswere found to be similarly activatedbycold treatment. Whenpeach co-genes were compared to their Arabidopsis orthologs, 70% of them were cold-responsive. This study concluded thatheat treatment activates many of the same genes as cold stressand that some of them increase the resistance of the fruitto freezing damage. 47 metabolites were identified in the metabolic study. This is quite different from the transcriptional study, wheretranscripts activated by both heat and cold stress converged. The metabolites belonged to the same classesanalyzed in the citrus studies; sugars, organic acids, amino acids, fatty acids andputrescine and urea In the peach study, flavonoids were not analyzed, as in the case of citrus fruits. The content of organic acidsin HAT fruit decreased with harvest level, as well as their behaviour in citrus peel.

Table 1 :**Reviewing the effect of Post-harvest treatment on plants**

Fruit	**Post-harvest Treatment**	**Efficacy**	**Reference**
Apple	Hot water treatment (45 °C, 10 minutes) and inoculation of	Increased thermotolerance of apple genes, affecting fruit	A. Spadoni*et al.*

	Penicillium expansum spore suspension (inoculation-heat treatment, heat treatment-inoculation; 1, 4, 24 hours), storage T= 20 °C)	resistance to blue mold infection	
Peach	Steam blanching (T= 38 °C, t= 3 hours), hot water blanching (T= 48 °C, t= 10 minutes) and cold storage (T= 4 °C)	Steam and hot water blanching can maintain firmness, increase antioxidant activity and slow down the damage. Whereas hot water blanching is more efficient to prevent internal browning of the material	C. Huan *et al.*
Papaya	Hot water (T= 48 °C, t= 20 minutes) followed by soaking in 1% Ca (heat treatment-Ca) solution	Inhibits anthracnose and damage	L. E. Ayón-Reyna
Tomato	Hot water blanching (T= 40 °C, t= 10 minutes)	Slowsdamageand extends shelf-life	P. Boonkorn
Cherry	Hot water (T= 44 °C, t= 114 minutes) and storage (T = 20 °C)	Inhibits blue mold decay by increasing resistance to disease and slowing cell softening	L. Wang*et al.*
Kiwi	Hot water (T= 45 °C, t= 10 minutes), storages (T= 4 °C and 25 °C)	Inhibiting damage by inhibiting the growth of B. cinerea and *P. expansum* spores and increasing the antioxidant activity of catalase and peroxidase enzymes	Chen *et al.*
Lemon	Hot water treatment (non-chemical, kinetin and potash alum (chemical), MAP: polyethylene	Extend shelf life. Hot water treatment is more effective than kinetin and potash alum	Morgado*et al.*

Melon	Hot water dipping (50 °C, 30 minutes), MAP (Oriented polypropylene) and biodegradable film (polylactic acid), cold storage (T= 6 °C)	Decreased fruit respiration rate. Oriented polypropylene MAP packaging is more effective than polylactic acid. The combination of heat treatment with MAP is effective in reducing browning and other quality degradation, as well as increasing beta-carotene levels	Kayesh*et al.*
Sweet orange	Heat treatment (T= 50 °C, t= 0-20 minutes), Cold storage (T= 5 °C)	Firmness and weight loss are maintained at a certain storage time limit, then after that, it decreases	A. Rab *et al.*

Conclusion

In conclusion, the field of heat treatment for commodities, especially in the context of pest control and pathogen reduction, has made significant strides in recent years. However, there is a notable gap in systematic modelling, which could greatly accelerate progress. While empirical approaches involving various time-temperature combinations have yielded valuable insights, a more structured and predictive approach is needed to optimize treatments.Understanding how commodities respond to heat treatments through modelling is crucial. This scientific framework could help identify the most effective and efficient combinations, reducing the trial-and-error nature of current practices. Such modelling holds the potential to revolutionize the field and expedite the development of treatments that not only combat pests and pathogens but also preserve the quality of the commodities.As the demand for fresh produce continues to rise, ensuring its safety becomes paramount. The application of hot water treatments (HWT) has emerged as a promising solution, addressing issues from disinfestation to chilling injury control. However, achieving precise and uniform temperature control during these treatments is essential to prevent conditions that favour pathogen growth.Furthermore, the long-term goal is to replace hazardous synthetic chemicals like fungicides and pesticides with safer, sustainable alternatives. To achieve this, a deeper understanding of the biochemical and molecular processes involved in hot-water-treated produce is required.

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