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Temperature Extremes

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I. INTRODUCTION AND DEFINITIONS

A. Variation in Temperatures

Temperature variations are normal in any natural environment. Changes in the air and plant temperature occur within minutes as clouds obscure the sun and wind patterns change, within hours as the sun's position changes during the day, and within days as weather patterns move through the area and the seasons change. Leaves and exposed tissue suffer the greatest temperature variations, shaded large fruit experiences smaller changes, while underground tissues experience the smallest variations in temperature. To avoid being injured, plant tissue must stay within a narrow physiologically tolerable range of temperatures, which are bracketed by extreme low (i.e., chilling or freezing) and high temperatures. However, in addition to these limits, which directly affect viability, many plants require specific diurnal or seasonal fluctuations of temperatures for normal growth and development—i.e., vernalization for flowering and setting fruit and for the fruit to develop and ripen to high quality.

The kinetic energy content or temperature of a tissue depends on the balance between inputs of heat energy from sunlight and warm air and losses of heat energy due to radiation, conduction, convection, and the evaporation of water. The exact temperature of the tissue depends on its energy content and thermal mass. Conduction of heat energy within a commodity is a relatively slow process, so localized areas of excessive energy inputs or losses can result in localized areas of extreme temperatures.

Temperatures are often expressed in degrees Fahrenheit (F) or Celsius (C), scales that have arbitrary set points and divisions and can lead to erroneous deductions about temperature changes. For example, increasing the temperature from 10 to 30°C does not mean that the thermal energy in the system increased threefold. A temperature scale that is based on physi-

cal and thermodynamic properties is the Kelvin scale. A temperature of 0 K (there are no degrees) is called "absolute zero" (it is about −273°C); it is the temperature at which there is no thermal energy left in the system. A change from 100 to 200 K represents a doubling of the thermal energy in a system. Likewise, when the change from 10 to 30°C is expressed as a change from 283 to 303 K, the real magnitude of the change is seen as only a 7% increase in thermal energy. Although small in relation to the total energy content of the tissue, such small changes in temperature often mean the difference between life and death.

The temperatures at which physiological processes can occur range from −10°C for algae and lichens in polar areas and atop mountains to over 100°C for bacteria in thermal pools and deep sea hydrothermal vents. Living things can exist for a limited time at even higher and lower temperatures, especially when in a dormant state (e.g., seeds, and spores).

The limits for physiological processes in higher, vascular plants are more restricted than for nonvascular plants, while the range for the commercial production and marketing of vegetable crops is even more circumscribed, generally lying between 4 and 36°C (Fig. 1). While it is true that many vascular plants have adapted to exist in areas that experience an extraordinary range of temperatures (i.e., −40 to 40°C), commercially grown vegetable crops have a more limited range of temperatures within which they produce the high-quality commodities demanded by today's consumers. Many fruits and vegetables cannot tolerate the extremes of 0 to 10°C and 30 to 40°C for more than short exposures without an adverse effect on their growth, development, maturation, and ripening.

B. Temperature Quotient

The rate of a chemical reaction increases with increasing temperature. In general, the reaction rate increases between two- and threefold with every 10°C rise in temperature. This concept, first postulated by the Dutch chemist van't Hoff, is expressed in the following equation: $Q_{10} = R_2/R_1 \approx 2$ to 3. In this equation, Q_{10} = the temperature quotient for a 10°C change, R_1 = the rate of the reaction at temperature T_1, and R_2 = the rate of the reaction at temperature T_2 (where $T_2 = T_1 + 10$°C). The more general form of the van't Hoff rule that can be applied to temperature intervals other than 10°C is given in Chapter 2. The Q_{10} equation can be used to calculate either the Q_{10} from known reaction rates at known temperatures or an unknown reaction rate for a given temperature difference from a known Q_{10} and reaction rate.

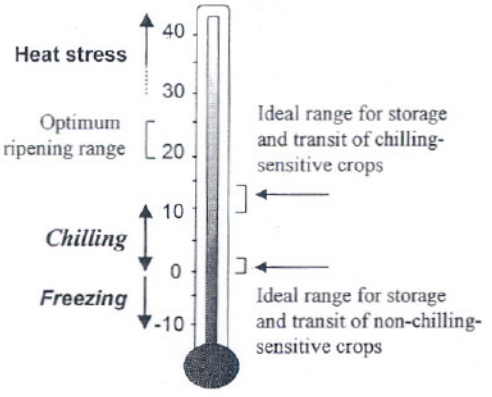

Figure 1 Temperatures that could be encountered by harvested vegetables and the ranges associated with temperature extremes.

The possibility that a calculated reaction rate may not be accurate increases as the difference between the two temperatures increase, since Q_{10} values are not constant but change with temperature. For example, we have found that the Q_{10} for the respiration of tomato (*Lycopersicon esculentum* Mill.) fruit is 5.1 between 0 and 5°C, which is within the range of temperatures causing chilling injury, but it is only 1.6 between 15 and 20°C, the range of temperature conducive to optimal ripening (Saltveit, unpublished).

C. Effect of Temperatures on Plants

Temperature can affect many complex physiological processes, such as respiration, photosynthesis, absorption of minerals and water, transpiration, ripening, growth, development, and senescence. There is not only the physical effect of temperature on the rate of chemical reactions, as expressed by the Q_{10}, but also an effect of critical temperature limits on the tissue—e.g., freezing, chilling, membrane- and protein-phase transitions, heat shock, and thermal denaturation of proteins.

Not all reactions within a given metabolic pathway are influenced by temperatures to the same degree. Complex metabolic pathways (e.g., respiration; the synthesis of compounds such as lignin, ethylene, and lycopene; and the breakdown of starch, chlorophyll, and protein) are usually regulated by one rate-limiting step that integrates a number of signals (e.g., level of substrate or product, hormone concentrations, presence of inhibitors or promoters) to provide an orderly coherence of the many competing and sustaining processes occurring within the cell. Fluctuations of temperature outside of the range in which this amalgam of reactions has evolved to function appropriately can disrupt the normal growth, development, and postharvest behavior of the commodity.

Control of a metabolic pathway may shift from one reaction to another, as each becomes the rate-limiting reaction when the temperature approaches one of the boundaries constituting the "normal" range of temperatures. These shifts, and the inherent differences in the temperature response of the component reactions within a complex series of reactions, usually produce significant changes in the Q_{10} over a range of temperatures from the normally expected values near 2 (Table 1). For example, the Q_{10} for the reduction in

Table 1 Temperature Quotient (Q_{10}) for the Rate of Deterioration of a Number of Harvested Vegetables

	Temperature range (°C)		
Commodity	0 to 10	10 to 20	20 to 30
Asparagus			
Reduced visual appearance	2.7	2.4	1.8
Loss of sugar	5.8	2.7	1.4
Increased fiber weight	10.0	2.0	2.0
Brussels sprouts (visual)	3.8	2.7	1.9
Celery (visual)	4.1	2.3	1.9
Head lettuce (visual)	2.5	2.2	1.9
Peas (visual)	3.3	2.8	2.0
Peas (loss of sugar)	2.7	2.6	1.5
Spinach (visual)	3.3	2.5	1.8
Sweetcorn (loss of sugar)	3.9	3.6	1.5

Source: Data from Hardenburg et al., 1986.

visual quality of asparagus (*Asparagus officinalis* L.) drops from 2.7 to 1.8 as the temperature increases from 0 to 30°C (Lipton, 1990). In contrast, the Q_{10} for loss of sugar and the Q_{10} for increased fiber weight drop from 5.8 to 1.4 and from 10 to 2, respectively, as the temperature increases from 0 to 30°C (Lipton, 1990).

Ethylene-induced increases in phenylalanine-ammonia lyase (PAL) activity (Fig. 2A) and the subsequent development of russet spotting (Fig. 2B) in lettuce (*Lactuca sativa*

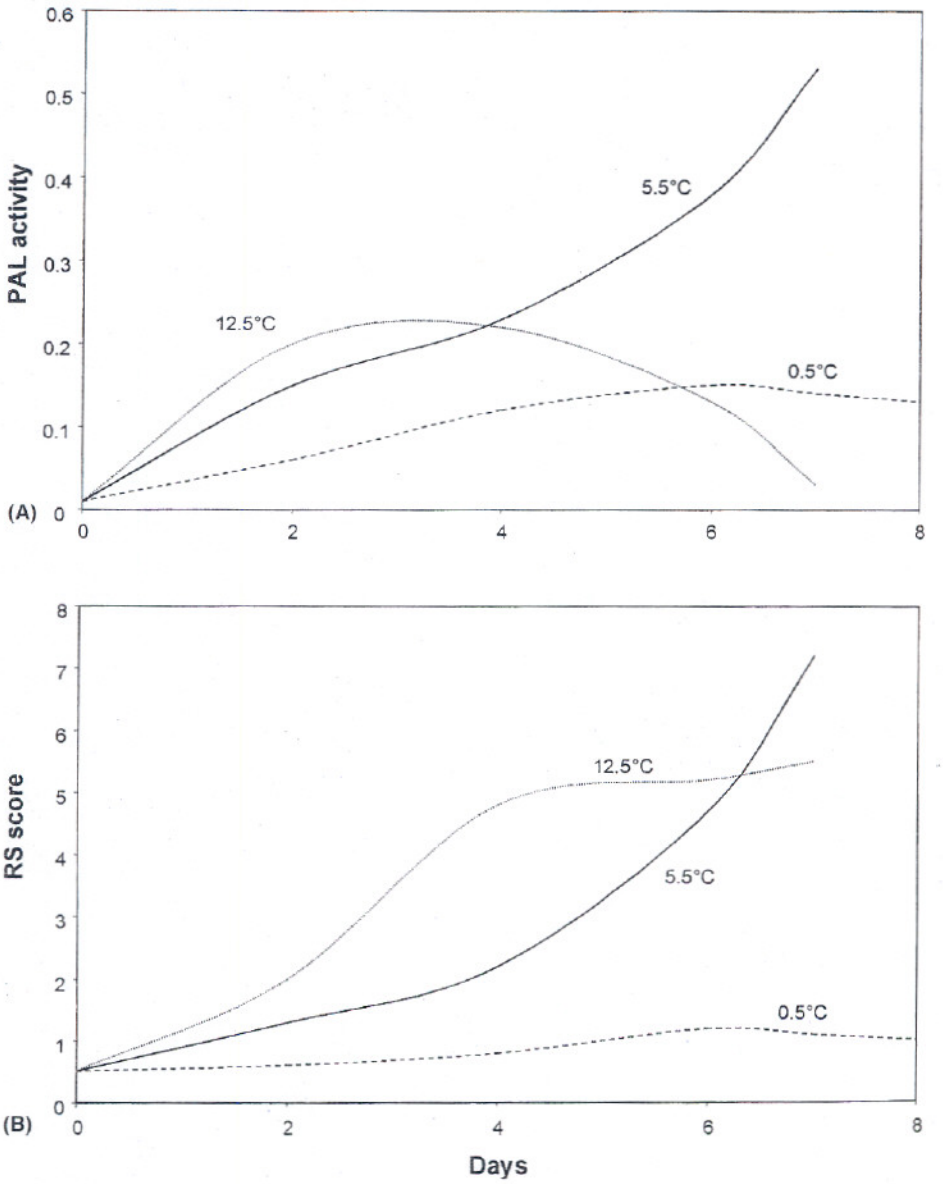

Figure 2 Activity of PAL (A) and RS scores (B) of lettuce tissue stored for up to 8 days at 0.5, 5.5, or 12.5°C with 10 uL L^{-1} ethylene in air. (Redrawn from Hyodo et al., 1978.)

L.) are significantly influenced by temperature, being maximally induced at 5°C and to lesser extents at 0 and 12.5°C. Minimal induction at 0°C reflects the slower rate of metabolism at this low temperature, while the low induction of activity at 12.5°C is the result of the concomitant induction of an inactivity factor that is maximal at 12.5°C. As this example illustrates, some reactions have different temperature optima while others have different Q_{10}'s.

D. Temperature and Physiological Time

There is a difference between chronological and physiological time. In humans, being warm-blooded animals, most biological processes proceed at a relatively constant rate, and humans experience time as a linear flow of equal-spaced intervals. However, biochemical reactions in cold-blooded animals and plants vary with temperature; the perception of time, therefore, also fluctuates with temperature. Given a Q_{10} of 3, a time interval at 25°C would be three times as long physiologically as the same time interval at 15°C (Fig. 3). For example, during daily fluctuations in temperature from 15 to 30°C, a 6-h period during the warmest part of the day would be 12.7 physiological hours long, while a 6-h period during the coldest period would be only 3.2 physiological hours long. If the Q_{10} values for interconnected developmental reactions are significantly different from one another, the coordinated process of development could be impeded when temperatures fluctuate into the extreme range.

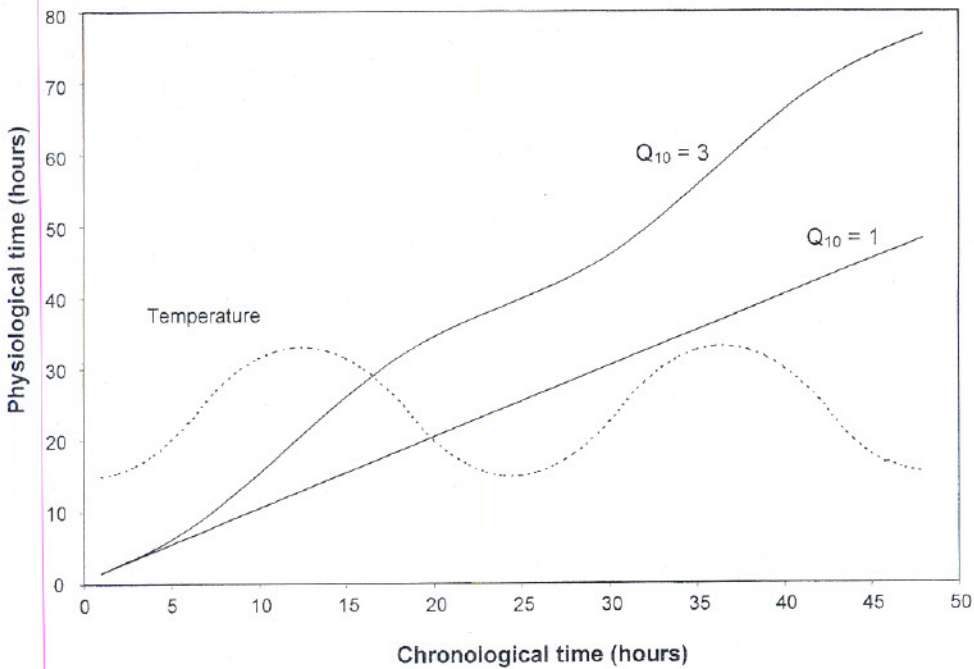

Figure 3 Comparison between chronological and physiological time for a reaction with a Q_{10} of 3.

II. SUSCEPTIBILITY TO TEMPERATURE DAMAGE

A. Sensitivity to Temperature Extremes

Susceptibility to injury by temperature extremes varies not only with the species and cultivar but also with the geographical location and season, cultural practices, time of harvest during the day, and other factors (Levitt, 1972, 1990). The ability of environmental factors and cultural practices to alter temperature sensitivity implies that plants have means of adapting to both long- and short-term temperature changes. Obvious short-term adaptive changes include reorientation of leaves to minimize solar exposure and increased transpiration to cool leaves, while longer-term adaptive changes include the shading of sensitive tissue and physiological hardening of tissues.

The perception of a stress, the selection of the appropriate adaptive response, and the means to implement this response are all under genetic control. Since the genetic information in any cell is limited, it is not surprising that there is some overlap and that when a plant adapts to one stress, it also adapts to other stresses as well. For example, cucumber (*Cucumis sativus* L.) and tomato plants adapted to chilling during low-temperature hardening can simultaneously become resistant to high-temperature injury (Saltveit and Morris, 1990; Wang, 1990). These responses differ in that adaptation to the primary stress is logistical while the secondary adaptation is asymmetrical; i.e., a rapid increase in resistance to heat stress at the beginning of the hardening period is followed by a gradual decline in resistance to heat stress as the primary hardening continues.

However, one stress can also increase the injury caused by another stress or a subsequent exposure to the stress. Partial chilling in the field before harvest increases the susceptibility of tomato fruit to subsequent chilling (Morris, 1954). Mechanical wounding and bruising disrupts the cuticle barrier and enhances water loss under conditions of water stress (Walter et al., 1990). This interaction of stresses is especially characteristic of fresh-cut fruits and vegetables (Saltveit, 1996).

B. Temperature Optima

The optimal temperature for enzyme activity can be influenced by substrate availability and effector concentration. Purified phosphoenolpyruvate (PEP) carboxylase shows this type of response when assayed at 10 to 45°C (Caldwell, 1990). Enzyme sensitivity to stress (e.g., chilling) also varies according to species. Depending on the species, PEP can either be stable or lose up to 50% of its activity when incubated at 0°C for 60 min (Krall and Edwards, 1993). Pyruvate kinase extracted from leaves of cold-sensitive cucumber plants had different pH, ion, and substrate requirements than the enzyme extracted from cold-hardened plants (Sobczyk et al., 1984). Plants appear to be able to modify enzyme response to temperatures by producing different isoenzymes and modifying the immediate environment.

C. Growth, Yield, and Quality

Temperature not only affects the rate of plant growth and maturation but also influences the total yield of marketable crops, their quality, and their composition and nutritive value (Hall, 1989; Kader, 1992; Swiader, et al., 1992). The ascorbic acid (vitamin C) content is highest in broccoli (*Brassica oleracea* L. Italica group) grown at 10 to 15°C as compared with higher or lower temperatures. Tomatoes grown at 26°C contain more ascorbic acid than fruit grown at 17°C. Higher acidity and solids are found in tomato fruit grown at a

day temperature of 35°C, but lycopene synthesis is inhibited at temperatures above 30°C (Orzolek and Angell, 1975). The optimal temperature for the highest yield of asparagus is 28°C, but the highest yield of first-quality spears is optimal at 25°C (Lipton, 1990). The same temperature can also produce dramatically different results in various developmental systems within the same commodity. For example, tomato yield was optimized by a 25/18°C diurnal cycle, while shoot growth was optimal at 30/15°C and mineral uptake by a constant 24°C root temperature (Swiader, et al., 1992).

Temperature can influence the activity of plant hormones. In the presence of CO_2, 10 mM ABA induced stomata closure in *Phaseolus vulgaris* leaves at 22°C but not at 5°C. Removal of CO_2 at 22°C increased stomata aperture, while it induced closure at 5°C (Eamus and Wilson, 1984). Ethylene exposure causes the browning disorder called russet spotting in lettuce at 5°C, but development of this disorder is significantly reduced at either higher or lower temperatures (Hyodo et al., 1978).

Plant growth and development is regulated not only by the absolute temperature but also by the various temperature patterns to which plants are constantly exposed. Some temperature fluctuations are needed to regulate long-term development. For example, most plants grow best when the day/night temperatures are not identical. A day/night cycle of 23/17°C gave optimal growth of peppers (*Capsicum annuum* var. *annuum* L.) (Bakker, 1989; deKoning, 1992). The highest yield of greenhouse cucumbers was produced by plants grown under 24/17°C, partially because the plants were larger and leafier (deKoning, 1992). Absolute and cycles of temperature also affect such morphological changes as periderm formation in potatoes (*Solanum tuberosum* L.), shape of carrot (*Daucus carota* L.) roots, fiber content of celery [*Apium graveolens* L. var. *dulce* (Mill.) Pers.] and asparagus, pithiness of radishes (*Raphanus sativus* L.), and color development in carrots (Rubatzky and Yamaguchi, 1997). Although it appears that plants are adapted to grow and develop best when exposed to specific variations in temperature, harvested plants are almost universally stored at a constant temperature—a condition plants never experience in their native environment.

Commodities may store better under fluctuating temperatures. This is definitely the case for chilling-sensitive crops in which the development of chilling injury symptoms is prevented when a period of cold storage that would be damaging is interspersed with periods at temperatures around 5°C above the threshold for chilling (Cabrera and Saltveit, 1990). Other, non-chilling-sensitive crops may also benefit from periodic changes in temperatures that could, for example, allow repair of oxidative damage accumulating at the "optimum" storage temperature and prolong storage life. However, a number of problems appear to preclude commercial development of this technology—e.g., the cost of cycling the temperature and water condensation or excessive water loss on crops as they are repeatedly warmed and cooled. In practice, there is also the formidable problem of doing the multifactorial experiments that would be needed to arrive at an optimal storage regime.

III. FREEZING TEMPERATURES

A. Exposure to Freezing Temperatures

Temperatures cold enough to freeze plant tissue are infrequently encountered in the production of most vegetable crops. If encountered, they usually occur at the beginning of the season in the early spring and affect the young plants or at the end of the season in the late fall and may damage the harvestable commodities. If postharvest freezing does

occur, it is usually caused by the malfunctioning of the mechanical refrigeration system or by the improper placement of commodities, so that they receive subzero air coming directly from the evaporator of the refrigeration system.

Refrigeration systems normally do not run continuously but cycle on and off to minimize wear and tear to the equipment. This cycling maintains the air temperature within a few degrees of a set temperature. The difference between the higher air temperature that initiates the refrigeration cycle and the lower temperature that terminates the cycle is often 1 to 2°C. If the desired storage temperature for the commodity is close to 0°C, normal fluctuations in the operation of the refrigeration system can periodically cause the evaporator to produce air a degree or two below zero. The thermal mass of the commodity and its storage environment usually buffer this transient subzero dip in air temperature, so that the temperature of the commodity never drops below freezing. However, freezing can occur if the temperature is set too low and the inherent fluctuations in the system are large, if there is icing of the evaporator coils (icing lowers the temperature of the air exiting the evaporator), or if there is insufficient thermal mass to buffer the fluctuations in the air temperature. Eliminating these sources of postharvest freezing simply requires prudent maintenance and oversight of the refrigeration system and the proper placement of commodities within the storage and transport environment to prevent their exposure to the subzero temperatures that will cause freezing.

B. Symptoms of Freezing Injury

Vegetable tissue freezes at around -0.8 ± 0.4°C. The actual freezing point is governed by the solute concentration within the aqueous milieu of the tissue. Freezing temperatures range from −0.2°C for lettuce to −0.4°C for watermelon [*Citrullus lanatus* (Thunb.) Matsum. & Nakai] to −0.8°C for cabbage (*Brassica oleracea* L. Capitata group) and Brussels sprouts (*Brassica oleracea* convar oleracea), to −1.4°C for carrots and −1.8°C for winter squash (*Cucurbita maxima* Duchesne) (Hardenburg et al., 1986; Whiteman, 1957). However, since the solute concentration within a commodity varies among the tissues, those tissues that are more hydrated will freeze before tissue with higher solute concentrations. For example, freezing in carrot roots will proceed from the crown, where sugars are less plentiful, to the tip, where they are more abundant. Freezing will also occur in the vascular tissue earlier than in storage tissue. This differential susceptibility to freezing among tissues within a given commodity can produce paradoxical effects in which freezing-damaged tissue is surrounded by undamaged tissue.

Upon thawing, frozen tissues become water-soaked and flaccid. General tissue collapse rapidly follows thawing, with microbial infestation quickly proliferating throughout the entire commodity. If the freeze was slight and/or the tissue is freezing-tolerant, the extracellular solution can be reabsorbed with little permanent damage to the tissue. If cells have been killed, however, they can no longer retain water and solutes. Localized water loss is accelerated and the flaccid and waterlogged tissue develops surface pits or internal voids. Browning and general discoloration of vascular tissue, which is more sensitive than surrounding tissue, often produces characteristic patterns of necrosis after freezing.

The relative susceptibility to freezing damage is not directly related to the freezing point of the tissue (Fig. 4). For example, both tomatoes and parsnips (*Pastinaca sativa* L.) have freezing points between −1.1 to − 0.6°C, yet parsnips can be frozen and thawed several times without apparent injury, while tomatoes are ruined after one freeze (Hardenburg et al., 1986). Many late-season vegetables (e.g., cabbage, Brussels sprouts) can toler-

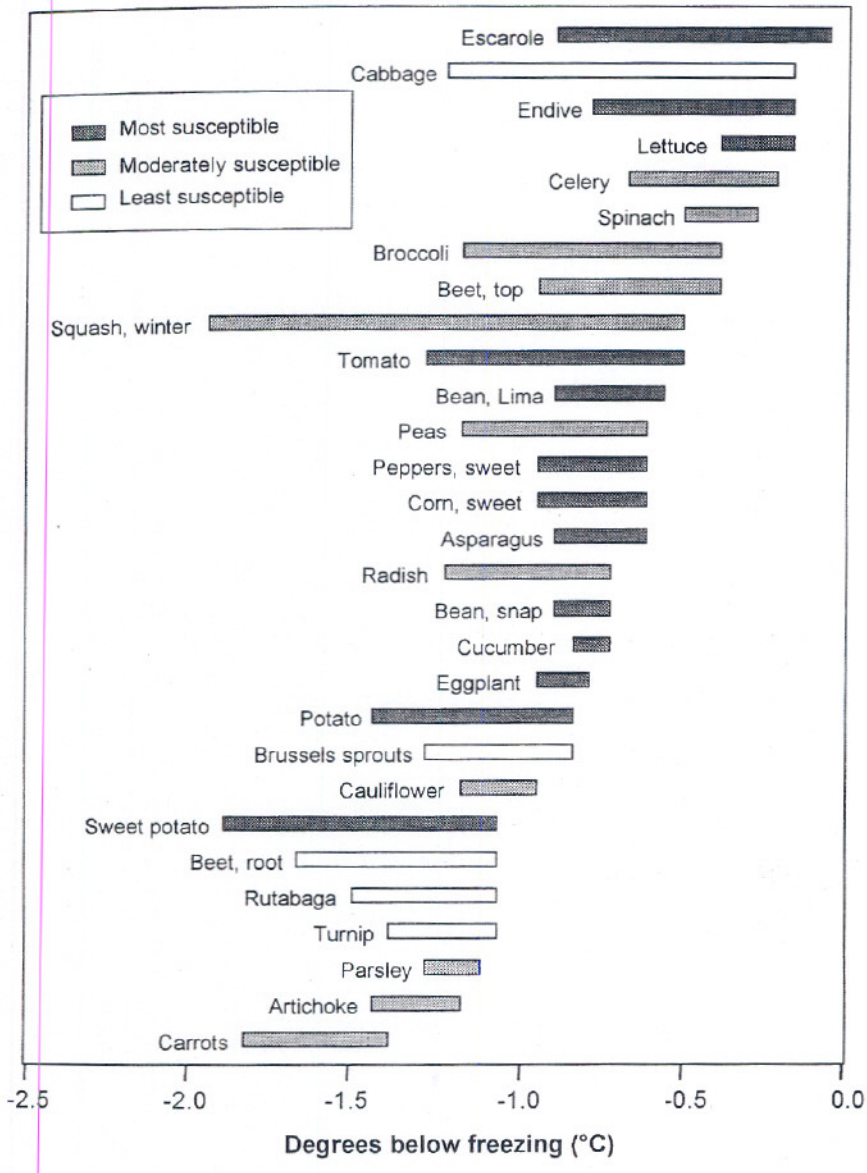

Figure 4 Range of freezing temperatures and susceptibility to freezing injury for some major vegetable crops. (Data from Hardenburg et al., 1986; Whiteman, 1957.)

ate slight freezes (Moline, 1987). Cabbage has one of the highest freezing points (−0.6°C) of any vegetable, yet it can tolerate some ice formation and thawing without injury. In contrast, sweet potatoes [*Ipomoea batatas* (L.) Lam.] have a much lower freezing point (−1.6°C) yet are very susceptible to freezing. Slightly frozen sweet potatoes develop a characteristic brownish discoloration of the vascular ring and internal vascular tissue (Whiteman, 1957).

C. Physiological Cause of Freezing Injury

Since ice nucleation will first appear in the "purest" water, it usually occurs extracellularly in the xylem, intercellular spaces, or cell wall. Supercooling denotes the capacity of water to be cooled below its freezing point before the formation of ice crystals occurs. In undisturbed tissue, supercooling can be a few degrees below the freezing point, but agitation during transport usually precludes supercooling. The amount of energy that must be removed to freeze water (80 kcal kg^{-1}) is great in comparison with that needed to cool water (~1.0 kcal $kg^{-1}°C^{-1}$) or ice (~0.5 kcal $kg^{-1}°C^{-1}$). If energy is being removed from a commodity at a constant rate, its temperature will steadily fall until freezing is initiated, at which point its temperature will remain constant until all the available water is frozen. This retarding of the temperature drop under moderate freezing conditions of a few degrees below zero can protect sensitive tissues from freezing injury. If supercooling has occurred, the temperature of the freezing commodity will actually increase as it freezes (e.g., going from −2 to 0°C).

Ice crystals rarely form intercellularly during slight or slow freezing because extracellular solutions are usually lower in solutes and thus are the first to freeze (Moline, 1987; Palva and Heino, 1997). As extracellular ice crystals grow during slow cooling, the solution in which they are forming becomes more concentrated and water is osmotically drawn from the surrounding cells to the centers of ice nucleation. This progressive dehydration of the cells is the primary cause of most freezing injury. If cooling is quite rapid, water movement from the cell may be insufficient to maintain extracellular ice formation and the temperature of the tissue will drop to the point where intracellular ice crystals will form. This is usually lethal to the cell because of protein denaturation and membrane disruption due to the removal of water and the concentration of the resultant solution.

Freezing injury is not the result of ice crystal formation per se but of the change in water activity in the freezing tissue because of the exclusion of solutes from the ice crystal and the resulting concentration of the remaining aqueous solution. Ice crystals appear sharp and able to puncture delicate cellular membranes. However, an ice crystal forms by the deposition of water molecules on its surface and therefore cannot exert any force to puncture or mechanically disrupt the cell. Although frozen tissue is more prone to mechanical injury, cellular dehydration, not mechanical perturbation, is the cause of most freezing injury.

D. Alleviation of Freezing Symptoms

The avoidance of freezing temperatures is the best prevention and can easily be accomplished after harvest by proper maintenance and supervision of refrigeration facilities (Moline, 1987). Once frozen, most vegetable crops are ruined. A few crops like cabbage can tolerate some ice formation and thawing without injury (Hardenburg et al., 1986). However, even these crops are sensitive to mechanical injury when frozen, and frozen commodities should be left undisturbed until they have been slowly rewarmed. It may be economically feasible to divert large quantities of frozen vegetables to alternate uses, but small quantities are usually simply discarded as unusable.

IV. CHILLING INJURY

The advantage of storing crops at low temperatures is to slow metabolic processes and prolong the market life of the commodity. Low temperatures usually prolong the storage life of most commodities. Some crops, however, cannot tolerate temperatures below ~10°C without developing severe physiological disorders that are grouped together under

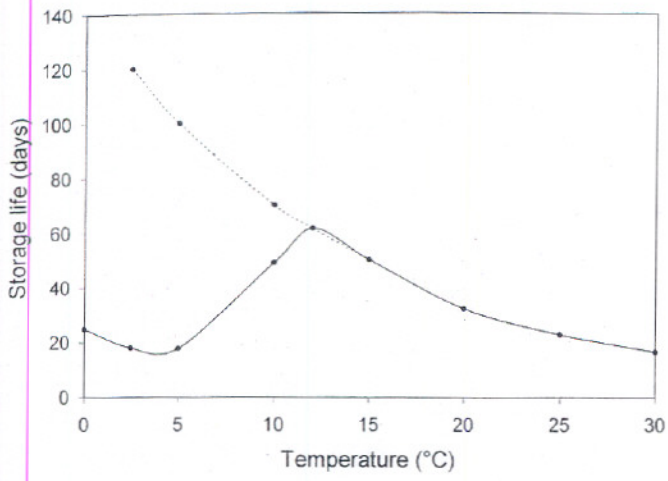

Figure 5 Storage life of cucumber fruit held at various temperatures. Actual data (solid line), likely storage life if cucumber fruit were not chilling sensitive. (Data from Eaks and Morris, 1956.)

the term *chilling injury* (Lyons and Breidenbach, 1987; Saltveit and Morris, 1990). For example, the storage life of cucumber fruit is prolonged at low temperatures (Fig. 5), but lowering the temperature below ~12.5°C shortens the storage life (Eaks and Morris, 1956). If we assume an approximate Q_{10} of 2, then the dashed line shows what the storage life would probably be if cucumber fruit were not chilling sensitive. Plants sensitive to chilling are usually of tropical or subtropical origin, but some temperate crops (e.g., asparagus and potatoes) also experience physiological disorders at low but nonfreezing temperatures. There is considerable variation among chilling-sensitive crops in the critical temperature and the length of exposure that will induce chilling injury (Table 2).

A. Exposure to Chilling Temperatures

Chilling-sensitive tissues are usually injured at temperatures below ~10°C but above freezing. The severity of symptom expression is dependent on many factors, such as species, cultivar, growing location and conditions, past temperature exposure, and postchilling treatments. Within a specific lot of produce, the severity of chilling injury is also highly dependent on the temperature and the duration of exposure (Fig. 6). Chilling symptoms are usually most clearly expressed upon transfer to a warm, nonchilling environment. However, some symptoms (e.g., mealiness in tomatoes, pitting of cucumbers, and sweetening in potatoes) are exhibited during chilling. The postchilling environment (e.g., relative humidity) can significantly affect the severity and speed of symptom development.

Although prolonged exposure and lower temperatures exacerbate the level of injury, a certain duration of chilling is usually necessary before symptoms become apparent. For example, cucumber fruit must be chilled at 2.5°C for more than 3 days to induce visual symptoms of chilling injury such as yellowing and increased disease susceptibility (Cabrera and Saltveit, 1990). If the exposure is shorter, other, more subtle symptoms may appear and the tissue may recover from chilling without exhibiting any overt symptoms. Interspersing subinduction durations of exposure with short periods to recover at a nonchilling temperature can permit storage for cumulative exposures that would otherwise

Table 2 Vegetables Susceptible to Chilling Injury

Commodity	Lowest safe temperature (°C)	Symptoms
Asparagus	0–2	Dull, gray-green, limp tips
Bean (lima)	1–4.5	Rusty brown specks or spots
Bean (snap)	7	Pitting and russeting
Cucumber	7	Pitting, water-soaked spots, decay
Eggplant	7	Surface scald, *Alternaria* rot, seed blackening
Melons		
Cantaloupe	2–5	Pitting, surface decay
Honeydew	7–10	Reddish-tan discoloration, pitting, surface decay, failure to ripen
Casaba	7–10	Same an honeydew but no discoloration
Crenshaw	7–10	Same as above
Watermelon	4.5	Pitting, objectionable flavor
Okra	7	Discoloration, water-soaked areas, pitting
Pepper	7	Sheet pitting, *Alternaria* rot, seed darkening
Potato	3	Mahogany browning, sweetening
Squash (winter)	10	Decay, especially *Alternaria* rot
Sweet potato	13	Decay, pitting, internal discoloration, hard core
Tomato		
Mature-green	13	Slowed ripening, pitting, poor color, *Alternaria* rot
Ripe	7–10	Waterlogging, softening, decay
Yam	16	Tissue discoloration and waterlogging, decay

Source: Hardenburg et al., 1986.

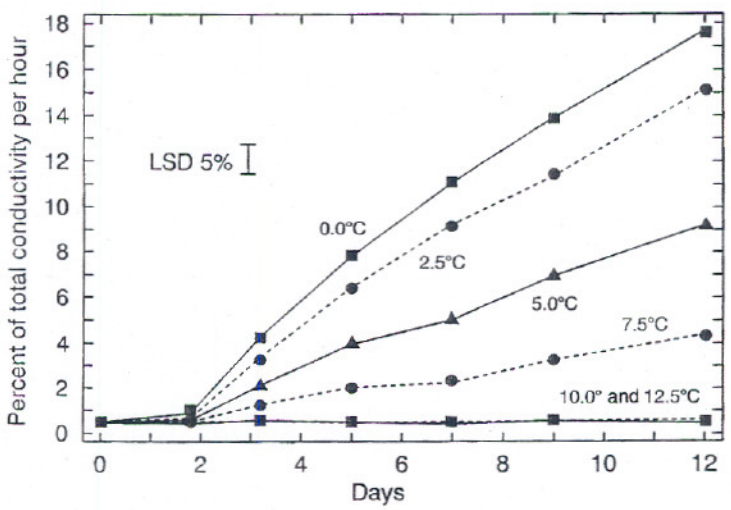

Figure 6 Effect of duration of exposure and temperature on the rate of ion leakage from cucumber cotyledons. The vertical bar is the calculated 5% LSD value. (From Saltveit, 1994.)

be injurious. The duration necessary to induce injury varies with many factors, foremost among which is the method used to measure chilling injury (e.g., visual changes, electrolyte leakage, or enhanced respiration). Also important are the species, cultivar, growing conditions, and atmosphere composition during exposure.

Chilling is usually cumulative. Exposure to chilling temperatures in the field or during storage or transit may be individually insufficient to cause injury, yet they may be injurious in combination. For example, chilling in the field as the season comes to an end and night temperatures become cold causes severe problems by reducing quality and the market life of some fruit. Mature-green tomato fruit loses quality and market potential the longer it has been exposed to low temperatures in the field. Such tomatoes may not have received enough chilling in the field to develop overt symptoms, but they may have accumulated enough chilling that they will be injured when handled in the normal manner (Morris, 1954). The chilling sensitivity of tomatoes, squash, cucumbers, and peppers grown in the fall, when they are exposed to cold nights, is greater than for the same cultivars grown in the summer (Hardenburg et al., 1986). Here the effect is the additive cold units of the fall-grown plants.

In contrast to the accumulation of chilling in the field during the fall, plants grown in cool climates and in slightly stressed environments (e.g., water stress) are often more resistant to chilling than are plants grown in warm climates and under more luxurious conditions (e.g., high nitrogen fertilization) (Graham and Patterson, 1982). Summer-grown temperate crops are sometimes not only injured by shorter exposures to chilling temperatures than crops grown during cooler periods but also have much higher critical temperatures for chilling injury.

The physiological effects of growing temperature are not manifest only in modulating chilling susceptibility over extended periods of months but are also effective over shorter periods. For example, tomatoes harvested in the morning, when cool, are more chilling-resistant than fruit from the same plant harvested in the afternoon, when warm (Fig. 7) (Saltveit and Cabrera, 1987). Fruit harvested on cool, cloudy days, during which it was roughly the same temperature throughout the day, did not show significant differences in chilling sensitivity with time of harvest. In a follow-up study, holding mature-green tomato fruit at temperatures ranging from 0 to 37°C for 6 h significantly affected its subsequent chilling sensitivity (Fig. 8) (Saltveit, 1991). Tomato plants also exhibit diurnal changes in chilling resistance, but these are more related to the light and dark cycles than to temperature (King et al., 1982). The cause of the variation in chilling sensitivity of tomato fruit is unknown, but such changes could be economically important if used to decide when to harvest a chilling sensitive commodity or how to best handle it after harvest.

B. Symptoms of Chilling Injury

Symptoms of chilling injury are usually not visually apparent at the chilling temperature but are fully expressed upon subsequent exposure to nonchilling temperatures (e.g., ~20°C for ripening tomato tissue). Common horticultural symptoms of chilling injury are diverse and range from enhanced water loss and surface lesions to reduced growth and viability of seedlings (Table 3). In addition to the conditions present during chilling, both the pre- and postchilling environment also affects symptom expression. Depending on a number of factors, stresses experienced by the tissue can either increase or decrease its subsequent sensitivity to chilling and the expression of chilling injury symptoms.

Maturity affects chilling sensitivity. Tomato fruit is particularly susceptible to chilling injury at the mature-green stage, at which it is often harvested and shipped (Autio

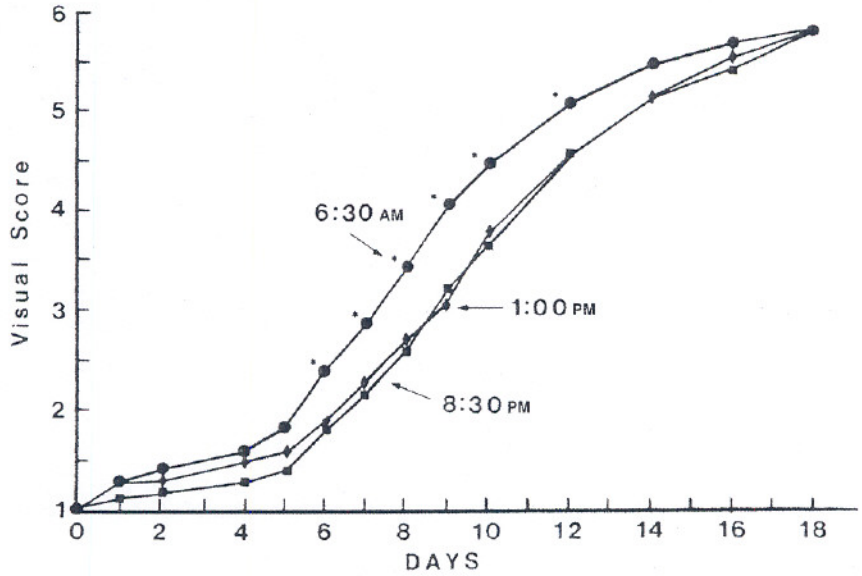

Figure 7 Effect of chilling at 7°C for 7 days on the subsequent ripening at 20°C of mature-green tomato fruit harvested during a sunny day at sunrise (6:30 AM) when cool (19°C), at 1:00 PM when hot (32°C) and at sunset (8:30 PM) when warm (29°C). Ripeness scores of the sunrise harvest with asterisk are significantly different at the 5% level from observations on the same day for the other harvest times. A subjective scale of ripeness was used where 1 equaled mature-green and 6 equaled red-ripe. The x-axis represents the days after transfer to 20°C. (From Saltveit and Cabrera, 1987.)

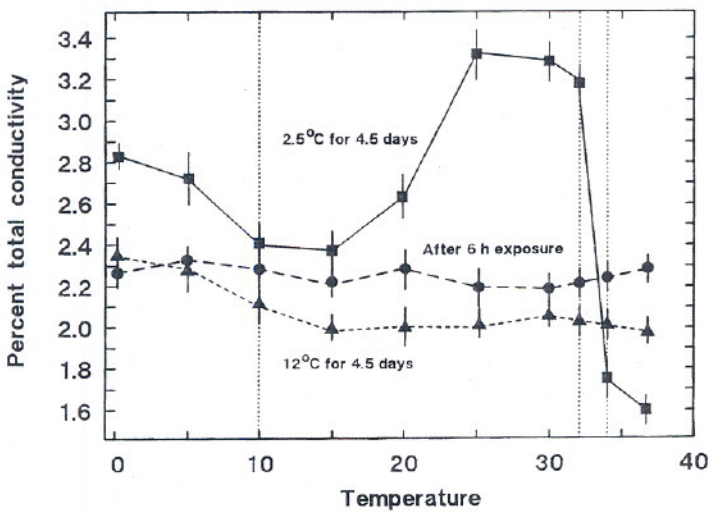

Figure 8 Effects of prior temperature exposure for 6 hours of mature-green tomato pericarp discs on their subsequent rates of ion leakage immediately after conditioning (●) and after holding for 4.5 days at 12°C (▲) or at 2.5°C (■). Data represent triplicate sampling. The vertical error bars represent the SE of the mean. (From Saltveit, 1991.)

Table 3 General Symptoms of Chilling Injury

- Surface lesions—pitting, large sunken areas, and discoloration
- Water-soaking of tissue
- Internal browning of flesh and seeds
- Breakdown of tissue structure; e.g., in apples and peaches
- Failure to ripen normally; e.g., in bananas and tomatoes
- Accelerated senescence but with normal appearance
- Compositional changes with altered flavor or taste
- Increased susceptibility to decay
- Shortened storage life
- Loss of growth capacity (i.e., sprouting) in propagules

and Bramlage, 1986). Chilled tomatoes exhibit increased rates of respiration and ethylene production, slow and abnormal ripening, and increased disease susceptibility (Saltveit and Morris, 1990). Increased rates of solute and electrolyte leakage occur from chilled tissue and have been used as a measure of the increased permeability of the plasmalemma membranes following chilling (King and Ludford, 1983). Red-ripe fruit is also chilling-sensitive (Saltveit, 1991), but since it has ripened, it exhibits fewer ripening-related symptoms of chilling injury and is therefore thought to be more chilling-tolerant than mature-green fruit. Although not all chilling-sensitive fruit shows increased ion leakage following chilling (McCollum and McDonald, 1991), enhanced leakage has been highly correlated with chilling injury of tomato and cucumber fruit (King and Ludford, 1983; Cabrera and Saltveit, 1990).

C. Physiological Cause of Chilling Injury

The physiological cause of chilling injury is unknown. Since there are many symptoms of chilling injury in different species and tissues and they occur after varying periods of exposure to different chilling temperatures, the possible physiological causes are many. Some of those proposed include phase changes in critical membrane lipid domains, physical changes in proteins (e.g., enzymes), increases or decreases in enzyme activity, unbalancing of critical reactions in metabolic pathways, altered metabolism and the accumulation of toxins or depletion of substrates, changes in cytosolic levels of calcium, changes in gas solubility, and decreased water activity and hydration. The physiological changes associated with chilling injury can be grouped into those that happen rapidly after chilling and those that are slower in appearance (Table 4).

In general, it is currently thought that the chilling temperature is transduced into a physiological change by a primary event that may involve a phase change in some cellular membrane or enzyme system (Saltveit, 2000). The resultant alteration in metabolism produces the myriad of symptoms that are grouped together under the term *chilling injury*. Some of the major theories of chilling injury are described below.

1. Phase Change in Membrane Lipids

Lyons (1973) proposed that, at chilling temperatures, a portion of the cell membrane of sensitive plants undergoes a phase transition from liquid-crystalline to solid gel. This conformational change would then give rise to the physiological changes associated with chilling injury (Fig. 9).

Table 4 Physiological Responses of Vegetables to Chilling

Rapid responses
• Phase change in membranes and/or proteins
• Changes in membrane organization
• Cessation of protoplasmic streaming
• Depolymerization of microtubles
• Increased solute leakage (K^+ and amino acids)
Slower responses
• Increased membrane permeability
• Altered respiration
• Accumulation of toxic substances
• Failure of essential reactions
• Stimulation of respiration and ethylene production
• Interference with energy production (reduced ATP levels)
• Changes in cellular structure
• Changes in protein structure and enzyme activity
• Synthesis or activation of enzymes

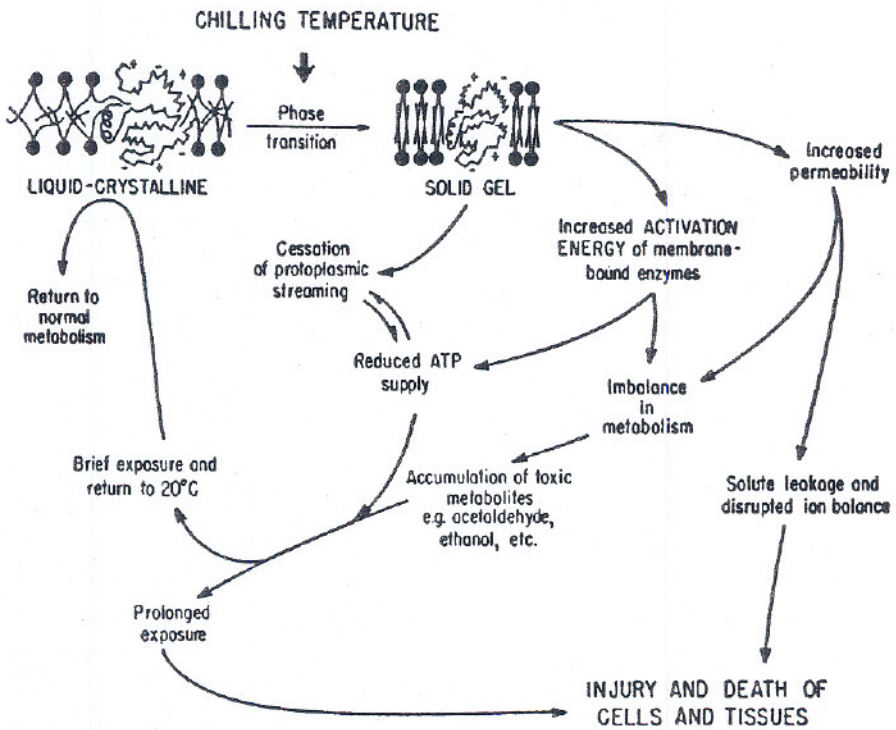

Figure 9 Schematic pathway of the events leading to chilling injury in sensitive plant tissue. (From Lyons, 1973.)

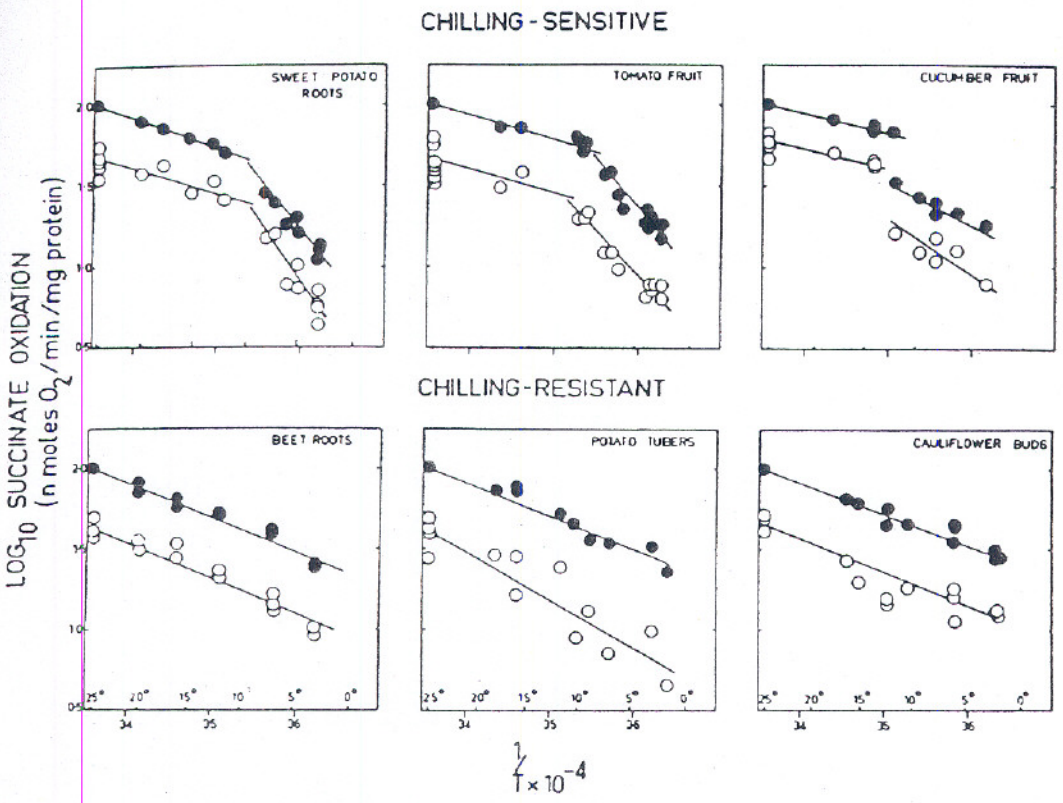

Figure 10 Arrhenius plots of succinate oxidation by plant mitochondria isolated from chilling sensitive (sweet potato roots, tomato fruit, and cucumber fruit) and chilling resistant (beet roots, potato tubers, and cauliflower buds) tissue. Each plot shows state 3 (●) and state 4 (○) respiration. (From Lyons and Raison, 1970.)

The "break" in an Arrhenius plot of temperature versus reaction rate (Fig. 10) is interpreted as the temperature at which the phase transition or separation of the membrane occurs (Lyons and Raison, 1970). Chilling-sensitive vegetables (e.g., sweet potato roots, tomato fruit, and cucumber fruit) exhibit breaks in Arrhenius plots of succinate oxidation by isolated mitochondria at temperatures similar to those that produce chilling injury. In contrast, chilling-resistant vegetables [e.g., beet (*Beta vulgaris* L. Crassa group) roots, potato tubers, and cauliflower (*Brassica oleracea* L. Botrytis group) buds] do not exhibit breaks in Arrhenius plots over the same 0-to-25°C range of temperature.

It is hypothesized that physical changes in the membranes of chilling-sensitive tissue are reflected in changes in their biological function (Saltveit and Morris, 1990). Two consequential effects on membranes would be changes in membrane permeability and the activity of associated enzymes (see below). Lyons (1973) originally proposed that membrane permeability would increase at chilling temperatures, yet the increases in ion leakage that are highly correlated with symptoms of chilling injury increase significantly only after days of chilling for most vegetable crops (Fig. 6) (Saltveit, 2000). Permeability changes would result in changes in cytosolic levels of calcium, since the calcium concentration in the cell wall and vacuole are orders of magnitude greater than in the cytosol. Calcium is

involved in many important regulatory mechanisms in the plant cell, and an unregulated influx of calcium could cause serious injury. There are similarities between the effect of treatments that raise the cytosolic levels of calcium and the effects of chilling injury in chilling-sensitive plants For example, chilling causes microtubule depolymerization, as does artificially raising the cytosolic calcium concentration (Bartolo and Carter, 1992; Gunning and Hardham, 1982).

Since first proposed, the membrane-phase change theory has continually undergone revisions to accommodate new information concerning how low temperatures might realistically be affecting membranes. However, there has been a lack of consistent correlation between membrane lipid composition and chilling sensitivity (Nishida and Murata, 1996). In response to these observations, it has been argued that minor compositional components of the membrane, and not the bulk lipids, may dictate the level of chilling sensitivity.

Recent studies using molecular biology and genetic engineering to change the saturation of fatty acids in membranes have failed to definitively show that the phase transition of a membrane is the primary cause of chilling injury. Although decreasing the saturation of fatty acids in chloroplast membranes reduced the level of injury in chilling sensitive tobacco leaves (*Nicotiana tabaccum* L.) exposed to chilling temperatures in the light (Nishida and Murata, 1996), the corollary of increasing the saturation of fatty acids in membranes did not increase the sensitivity of chilling tolerant *Arabidopsis* plants (Wu and Browse, 1995).

2. Increased or Decreased Enzyme Activity

Cold-sensitive enzymes are known to exist in plants. Phosphofructokinase (PFK), a key enzyme in glycolysis, exists in an active tetrameric form that is dissociated into inactive dimers at low temperatures (Dixon et al., 1981). This inactivation has been suggested as one of the mechanisms responsible for sweetening in cold-stored potatoes (Wismer et al., 1995). Other enzymes associated with low-temperature sweetening of potatoes increase during tuber storage at chilling temperatures (Cottrell et al., 1993).

Certain enzymes have thresholds for activation or synthesis near the chilling threshold temperature. For example, development of caffeoyl-CoA:quinic acid o-caffeoyltransferase (CQT) activity has a threshold temperature of 2 to 5°C in potatoes (Fig. 11) and 10 to 12°C in tomato fruit (Fig. 12) (Rhodes and Wooltorton, 1977, 1978). This enzyme catalyzes the formation of chlorogenic acid. Increased activity appears to be the result of de novo synthesis, since cycloheximide inhibits increases in enzyme activity. In addition to the effect of low temperatures on enzyme induction, low temperatures are also known to slow the decay of enzymes.

3. Altered Metabolism

Thermal displacement of equilibrium can lead to a shift in metabolism. The rate of enzymatic activity usually declines with declining temperature. The extent of the decline differs for each enzyme. In biological systems, there may be several series of reactions competing for the same substrate. If these reactions have different activation energies or Q_{10} values, a change in temperature could markedly shift the proportion of substrate being converted into the various products, leaving the cell devoid of sufficient product from one series of reactions or producing toxic levels of product by another pathway.

As a result of these rate imbalances induced by chilling temperatures, there may also be a change in the pool size of metabolites that serve as metabolic regulators or protein protectants. Loss of feedback inhibition could accelerate breakdown of metabolites

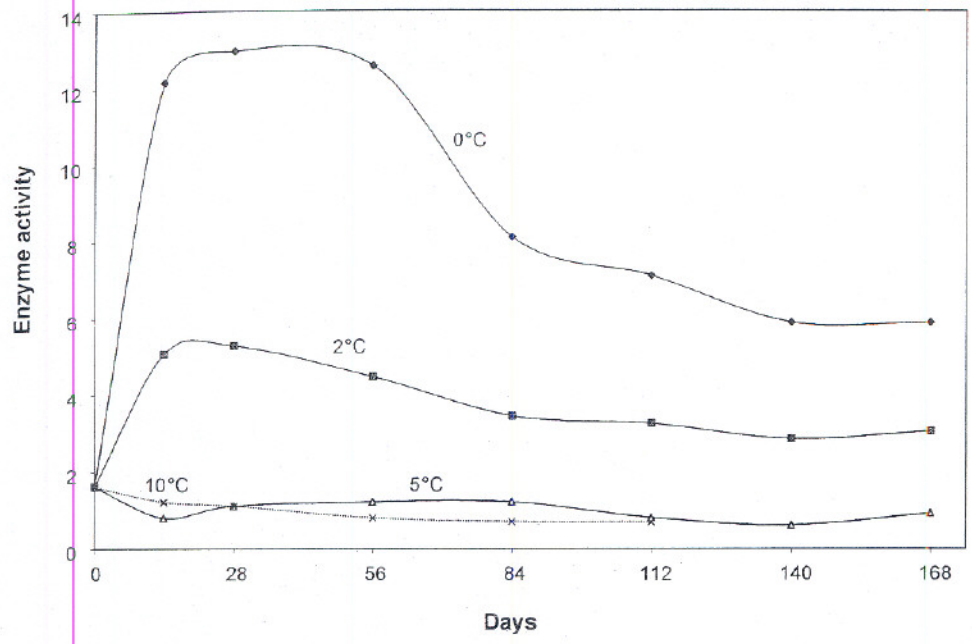

Figure 11 Changes in the activity of caffeoyl-CoA:quinic acid o-caffeoyltransferase in potatoes stored at 0 to 10°C for 170 days. (Redrawn from Rhodes and Wooltorton, 1978.)

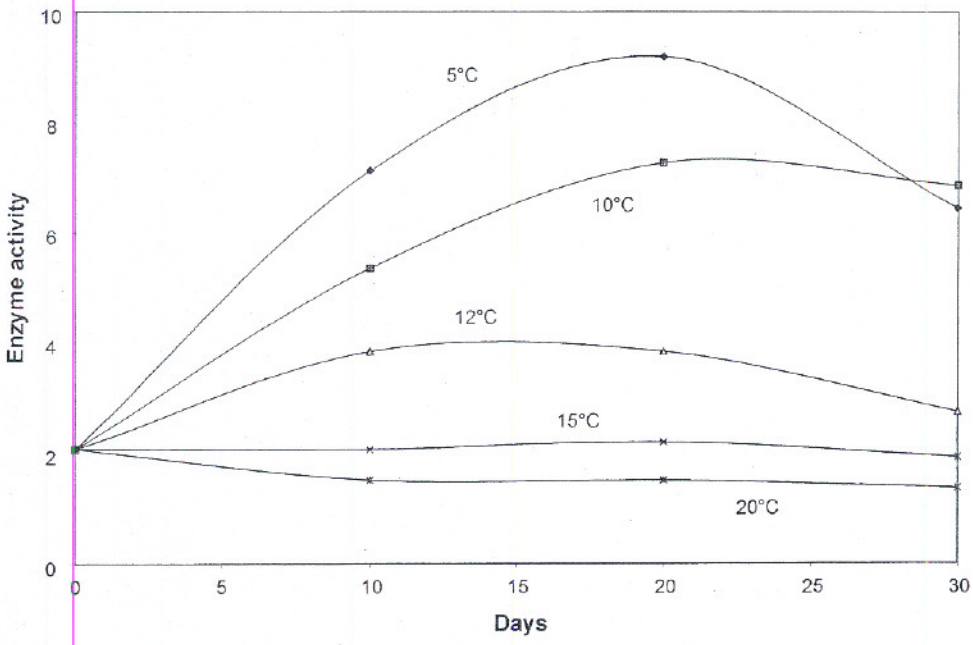

Figure 12 Changes in the activity of caffeoyl-CoA:quinic acid o-caffeoyltransferase in "breaker" tomatoes stored at 5–20°C for 30 days. (Redrawn from Rhodes and Wooltorton, 1977.)

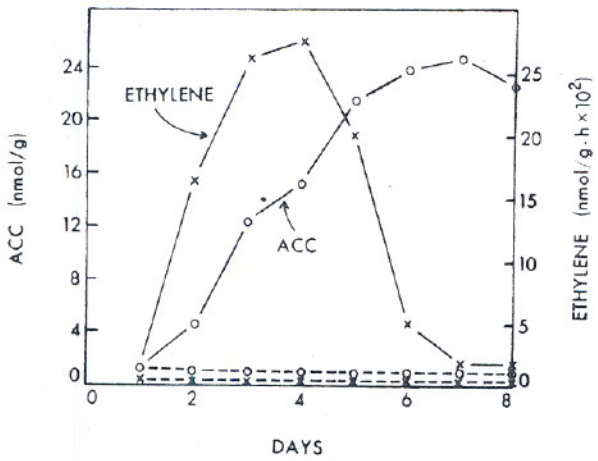

Figure 13 ACC content (○) and ethylene production (×) of cucumber 7 hours after transfer to 25°C following various periods at 2.5°C (continuous line) and 13°C (dashed line.) (From Wang and Adams, 1982.)

and enzymes. It has frequently been noted that many enzymes are more stable in the presence of their substrates (Kays, 1991). Unregulated alterations in substrate concentration could also affect metabolism by altering the rate of enzyme stability.

Ethylene is an important plant hormone, the synthesis of which is actively regulated by the plant (Abeles, et al., 1992). Many postharvest processes are influenced by the biological activity of ethylene. During chilling, the synthesis of the immediate precursor of ethylene (ACC) is often stimulated, while the ability of the plant to convert ACC to ethylene is progressively diminished (Fig. 13) (Wang and Adams, 1982). *Intermittent warming* refers to the periodic interruption of an injurious chilling exposure with periods at nonchilling temperatures (Fig. 14). Enhanced respiration and ethylene production occur during intermittent warming and are thought to reflect the dissipation of toxic products in the chilled cells, so that they do not reach injurious levels (Cabrera and Saltveit, 1990).

D. Alleviation of Chilling Symptoms

The most direct method to reduce chilling injury is to avoid chilling temperatures. When this is not possible, cultivars can be used that are chilling-resistant. While cultivars exhibit differences in chilling sensitivity, genetic modification by breeding or genetic engineering for such a complicated trait has not produced any spectacular results (Saltveit, 2000; Wang, 1990). Chilling resistance can also be increased by exposing sensitive harvested commodities to hardening conditions, such as brief warm or cold storage treatment (Kays, 1991; Wheaton and Morris, 1967). Warm storage allows healing of harvest-related wounds, while cold storage physiologically alters the tissue to better tolerate chilling (Wang, 1993).

Another method to reduce chilling injury is to suppress symptom development. Since symptoms of chilling injury often develop during holding and marketing at a warmer, nonchilling temperature after chilling, rapid use of the commodity can prevent symptom expression. Storage in high relative humidity can also reduce symptoms, since many symptoms appear to entail increased water loss (Wang, 1993).

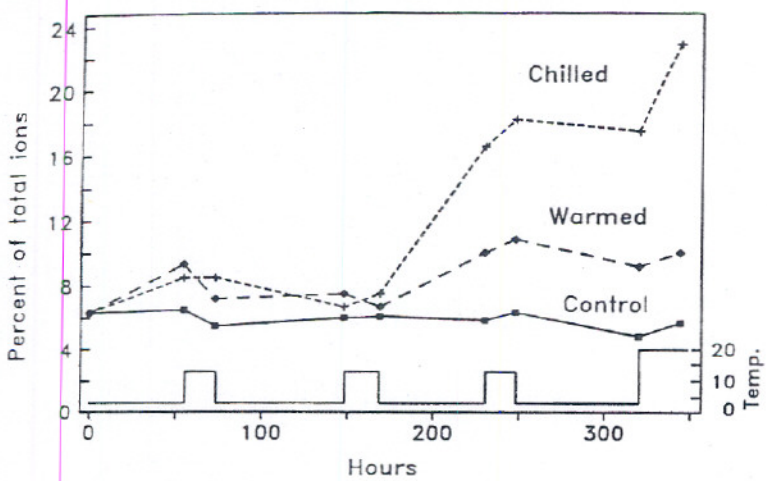

Figure 14 Effect of chilling and intermittent warming on ion leakage from 4 × 9-mm-thick discs excised from cucumber fruit held at 2.5°C (chilled) or 12.5°C (control) for 320 hours. Fruit held at 2.5°C were warmed to 12.5°C for 18 hours every 2 to 3 days (warmed). (From Cabrera and Saltveit, 1990.)

Symptoms of chilling injury can also be reduced by a variety of treatments before and during chilling (Forney and Lipton, 1990; Jackman et al., 1989; Wang, 1993). These treatments include intermittent warming during chilling (Artes and Escriche, 1994; Cabrera and Saltveit, 1990), controlled or modified atmospheres (Forney and Lipton, 1990), treatments with calcium (Wang, 1993) or ethylene (Abeles et al., 1992), and conditioning near the chilling temperature (Jackman et al., 1989; Wang, 1993; Wheaton and Morris, 1967). Using short exposures to high temperatures to induce chilling resistance through the synthesis of heat-shock proteins could add to the list of effective treatments (Collins et al., 1995; Sabehat et al., 1996; Saltveit, 1991).

V. HEAT STRESS TEMPERATURES

A. Exposure to Heat Stress Temperatures

Injury from exposure to high temperatures has received much less study than cold-induced injury, and it actually should rarely occur during storage or transport of harvested vegetables. In this respect, a distinction should be made between exposing the commodity to warm temperatures around 10°C above the recommended storage temperature (e.g., storing lettuce at 10°C), which is unfortunately all too common an occurrence, and exposing the commodity to true heat stress temperatures above 30°C (Fig. 1).

The energy required to vaporize water (540 kcal kg^{-1}) is enormous compared with that required to warm water (1 kcal $kg^{-1}°C^{-1}$), so that the evaporation of small amounts of water can have a significant cooling effect. Evaporation is an effective cooling mechanism for leafy vegetables. However, many fruit vegetables are bulky, and evaporation is limited because they have a low surface-to-volume ratio and are covered with a cuticle that is relatively impermeable to water loss.

High-temperature stress can occur in the field due to solar radiation, where direct

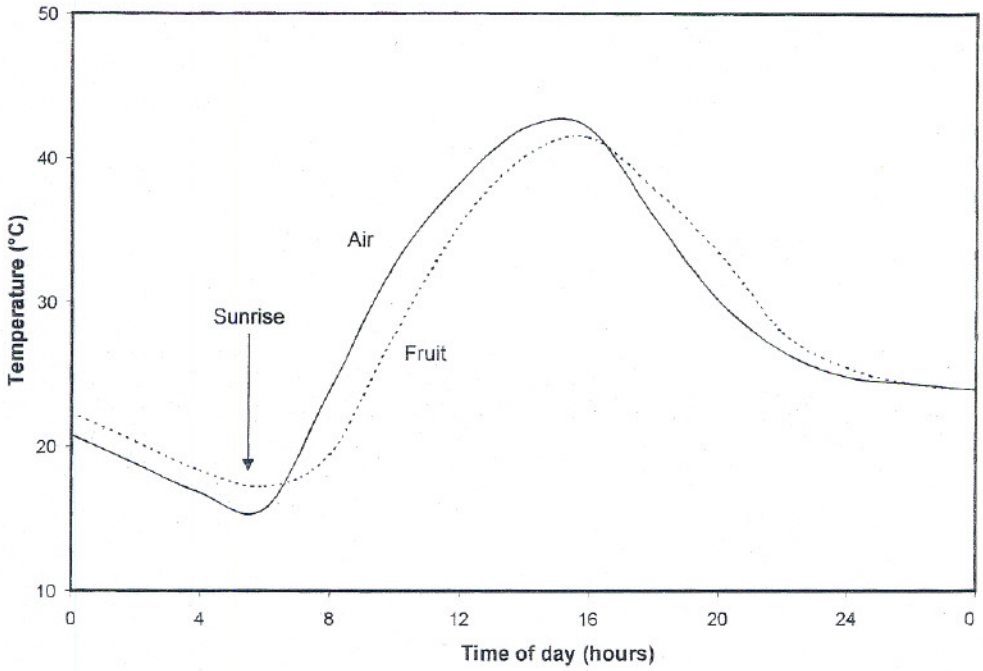

Figure 15 Typical air temperature profile during the day in a desert valley in southern California and the temperature within a cantaloupe melon. (Redrawn from Fairbank et al., 1987.)

exposure can easily cause exposed plant organs with limited transpiration capacity to reach temperatures in excess of 40°C (Fig. 15) (Fairbank et al., 1987). Such high temperatures can affect fruit ripening and the activity of plant growth regulators, such as ethephon on processing tomatoes (Fritz, 1990). Apart from the heating effects of sunlight, ultraviolet radiation can also be damaging (Teramura, 1986).

After harvest, commodities are removed from the shade and water supplied by the parent plant and are often directly exposed to solar radiation. This usually occurs during the steps in transporting the harvested commodity to the packing shed. The upper layer of commodities in field bins or in open gondolas is often exposed to full sunlight for a few hours, which can rapidly heat the expose tissue. Providing shade in the field where bins are temporarily stored or in packinghouse parking lots where trucks wait to be unloaded can significantly reduce damage to the exposed commodities.

Apart from these unintentional exposures to high temperatures, the intentional application of a temperature stress has been used for many years to control physiological disorders and pathogenic organisms in harvested vegetables (Couey, 1989). With the phasing out of many chemical pesticides and the increase in international trade, there is renewed interest in using heat treatments as part of insect disinfestation protocols and quarantine treatments (Paull and Armstrong, 1994). All of these applications depend on the presumption that high temperatures are more deleterious to the pest or pathogen than to the commodity. It is often necessary to significantly alter the environment (e.g., prior conditioning, moist heat, reduced oxygen, elevated carbon dioxide, etc.) to ensure that this assumption is realized.

B. Symptoms of Heat Stress Injury

High-temperature disorders include failure to ripen normally in tomatoes, poor storability and browning of cabbage, discoloration in melons (*Cucumis melo* L.), and cell death in exposed peppers and melons. Ripening characteristics of pigment synthesis, volatile production, and tissue softening are very susceptible to high-temperature stress. Lycopene synthesis is greatly curtailed in tomatoes stored continuously at 32°C, with the fruit developing only yellow pigmentation (Hall, 1964). Shorter durations of exposure, even to higher temperatures, have minimal effect. Storage at 37°C for 3 days reduced the ripening of pink tomato fruit sufficiently to allow marketing at 21°C for 7 days (Shewfelt et al., 1989). Exposure to 50 to 60°C for 0.5 to 1.5 min has been used as a postharvest treatment on peppers and cantaloupes (*Cucumis melo* L. Reticulatus group) (Couey, 1989). The duration of exposure that is lethal usually varies inversely with the temperature and is often an exponential function.

C. Physiological Cause of Heat Stress Injury

Heat stress is often accompanied by water stress. Lack of sufficient soil water can reduce transpiration, reduce evaporative cooling, and cause overheating of the plant. The plant may also be unable to transport enough water to meet the needs of rapidly transpiring leaves. Wilting and the resulting increase in ABA content and ethylene production can reduce quality and promote senescence (Abeles et al., 1992).

High temperatures cause enzyme inactivation and protein denaturation. The deleterious temperature is specific for each enzyme, and the range of temperatures over which enzymes become inactive is rather wide. Moderately high temperatures (35 to 40°C) inactivate some enzymes, while others withstand rather high temperatures (100°C) for short periods. The order for thermal inactivation of some enzymes present in potato tubers is lipase, lipoxygenase, monophenol oxidase, and peroxidase (Belitz and Grosch, 1987). Peroxidase was relatively stable at 100°C, while lipase was quickly inactivated at 70°C.

Since metabolic pathways comprise a sequence of enzymatically driven reactions, each of which has its own inactivation temperature, the lowest inactivation temperature will be the limiting temperature for the whole sequence. In a developing commodity, the many linked and independent pathways must interact to yield an acceptable product. In tomato fruit ripening, ethylene synthesis is inhibited at a temperature conducive to tissue softening yet incompatible with pigment and volatile synthesis (Abeles, et al., 1992; Hall, 1964).

D. Alleviation of Heat Stress Symptoms

Since most heat stress is unintentional and rarely encountered during postharvest handling, a protective procedure is not ordinarily applied to increase commodity tolerance to heat. The common use of shading and rapid transport to packing facilities should eliminate most instances of heat stress. In contrast, when heat stress is intentionally used to reduce postharvest disorders or in connection with disinfestation protocols, prior conditioning of the commodity can significantly reduce adverse effects (Couey, 1989; Paull and Armstrong, 1994). A prior heat stress can induce the synthesis of heat-shock proteins, which protect against subsequent heat stress (Vierling, 1990). The ability of a heat shock to redirect protein synthesis away from the synthesis of wound-induced enzymes of phenylpropanoid metabolism has been used to reduce the browning of lightly processed lettuce

(Loaiza-Velarde et al., 1997). High-temperature stress may have other beneficial effects on the postharvest storability and quality of vegetables.

VI. METHODS TO ALLEVIATE DELETERIOUS EFFECTS OF EXTREME TEMPERATURES

1. Avoid extreme temperatures. The most obvious method to alleviate the deleterious effects of extreme temperatures is simply to avoid them. This is difficult to do with crops in the field or greenhouse, but it is easily accomplished with harvested vegetable commodities. The proper maintenance and oversight of cold storage rooms and transport containers would do much to eliminate the postharvest exposure of vegetables to adverse temperatures.

2. Use product before symptoms develop. In many cases, the commodity can still be used after exposure to extreme temperatures if symptom development takes time or alternate uses are available. Frozen tomatoes or okra [*Abelmoschus esculentus* (L.) Moench] may be unsuitable for the fresh market but still suitable for processing. Chilled cucumbers and tomatoes are acceptable in salads and on sandwiches if used within a few days.

3. Make sure the commodity is harvested at the proper stage of maturity. Maturity has a significant effect on the response of commodities to temperature stresses. Immature fruit vegetables are often less susceptible after they have matured.

4. Handle to minimize physical injury and reduce water loss. Since many symptoms involve increased water loss, the use of high humidity, waxing, and plastic films can reduce these symptoms. Physical injury accelerates water loss, and wound stimulation of respiration and ethylene production can exacerbate the effects of temperature stresses.

5. Harden crops before exposure. Exposure to temperatures near the extremes can induce low levels of resistance in some crops. However, the effect is limited to a few degrees protection. Conditioning to protect against one stress (e.g., heat) often confers resistance to other stresses (e.g., chilling).

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