Host Defenses at the Wound Site on Harvested Crops

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Postharvest diseases of fruits and vegetables often develop at injuries that occur during harvesting, processing, and packaging. Injuries such as punctures, cuts, abrasions, and bruises are commonplace during harvest and handling of the commodity, and most pathogens are only capable of causing decay through such injuries. Inherent injuries, such as those that occur at the point of separation of the crop from the plant or those that occur during abscission of attached floral parts, can also be sites of infection. Propagules of decay bacteria and fungi are commonly abundant on fruit and vegetable surfaces and are stimulated to germinate where injuries occur because of the presence of nutrients and moisture. High populations of decay pathogens can also accumulate in packing facilities, either in water used during handling of the commodity, or as airborne propagules in packing or storage environments.

Entry of decay pathogens through injuries depends on many factors. Some of the most important ones are the physiological changes that occur at the site of injury which affect susceptibility to infection. In many instances, susceptibility of injuries to infection can be manipulated through the use of certain cultural and handling practices. The purpose of this paper is to review those instances where physiological changes in injured tissue have influenced susceptibility of crops to postharvest diseases caused by wound pathogens, and to discuss how management decisions can alter susceptibility.

ORGANIC CONSTITUENTS OF INJURED TISSUE THAT ALTER HOST SUSCEPTIBILITY

Injuries to harvested fruits and vegetables often trigger metabolic processes in cells close to the site of injury that assist in the repair process. Phenylpropanoid metabolism occurs in stressed but unbroken cells near the injury, leading to the formation of various phenolic compounds that play a role in protecting the injury from invasion by postharvest pathogens. Three types of response in phenolic metabolism have been reported (46). First, endogenous phenolics may be oxidized to form quinones, which in turn are condensed to form polyquinoid structures or to react with proteins or amino acids to form melanin substances. These melanins are analogous in many ways to lignin, and may function in a similar manner in defense. Quinones and melanins may also be relatively nonspecific enzyme inhibitors, and thus inhibit wall-degrading enzymes of postharvest pathogens. Second, endogenous or additional phenolic compounds may be synthesized and accumulated at injury sites (14,33,40). Examples of such compounds include cinnamic acid derivatives chlorogenic and caffeic acid. In addition to these compounds, isocoumarins, related chromones, and coumarins may form in response to injury (7,31). Third, the polymeric polyphenols, lignin and suberin, are often synthesized in response to wounding. Lignin is commonly found in injured plant tissue and is composed of polymerized phenylpropanoids, principally of *p*-coumaryl origin with small amounts of coniferyl units. During lignification, the polymerized lignin complex is covalently bound to plant cell walls or related matrices. Other phenylpropanoids, such as *p*-coumaric, ferulic, and *p*-hydroxybenzoic acids, are usually esterified to the polymer. Lignin also forms ester linkages with fatty acid polyesters, similar to those in cutin, to yield suberin. The major aliphatic components of suberin are ω-hydroxy acids, the corresponding dicarboxylic acids, very long chain ($>C_{20}$) acids, and corresponding alcohols. The deposition of suberin is often associated with renewed cell division during the formation of wound periderm (wound cork) that helps to seal off injured surfaces. Waxes associated with the suberin polymer are the major barrier to water loss from injured tissue. Callose has occasionally been observed in injured tissue in relation to host resistance. It is a β1-3 glucan, which is usually electron transparent in normal staining but often varying in electron density when formed in response to stress. It may be associated with other components such as lignin, protein, and phospholipids.

ROLE OF ORGANIC CONSTITUENTS IN HOST DEFENSE AT THE WOUND SITE

Toxicity of constituents to postharvest pathogens. Phenolic compounds associated with suberization and lignification may be implicated in disease resistance. Chlorogenic acid accumulates in cells adjacent to wounds in sweet potatoes and correlates closely with suberization and wound periderm formation (40). Its presence, however, did not inhibit infection of sweet potato by *Rhizopus* spp. Gans (22) reported that resistance in potato cultivars to gangrene caused by *Phoma exigua* Desmaz. was associated with high constitutive levels of chlorogenic acid in the tissue and an increase in this compound after wounding. Rishitin, phytuberin, and solavetivone were produced in potato tubers inoculated with *Erwinia carotovora*, the cause of bacterial soft rot, and incubated under aerobic conditions (37). Rishitin was bactericidal (ED_{50} = 106 μg/ml in 0.1% peptone water) and may retard development of bacterial soft rot (37). McKee (42) observed a marked increase in the level of solanine in wounded potato tuber tissue and evaluated its activity against *Fusarium coeruleum* Lib. ex Sacc., which causes dry rot. Solanine played no decisive role in preventing infection, since no correlation was demonstrated between susceptibility of tubers to dry-rot infection and their ability to accumulate solanine near wounds. Umbelliferone (31) and scoparone (7), associated with lignification in injuries to citrus exocarp, were toxic to *Penicillium digitatum* (Pers.:Fr.) Sacc., they may retard development of green mold.

Wounded carrot root tissues are particularly resistant to gray mold, caused by *Botrytis cinerea* Pers.:Fr., and to liquorice rot, caused by *Mycocentrospora acerina* (Hartig) Deighton (36). Three compounds inhibitory to *M. acerina* were recovered from stored carrot roots (18). These compounds were 6-methoxymellein, *p*-hydroxybenzoic acid, and falcarindiol. ED_{50} values for inhibition of chlamydospore germination by these compounds were 79 μg/ml, 292 μg/ml, and 31.8 μg/ml, respectively (18,25), indicating that 6-methoxymellein and falcarindiol were the more toxic compounds involved in the resistance response to *M. acerina*. Other antifungal compounds, such as 6-hydroxymellein, eugenin, and benzoic acid were less important individually, but may be synergistic with the more potent antifungal compounds (18). Falcarindiol was present in large oil droplets that accumulated on the injured surface of phloem parenchyma tissue during healing (23). Droplets containing falcarindiol were derived from oil or

canal ducts present in the phloem parenchyma tissue which were exposed by an injury. Accumulation of these antifungal substances was one of the early events of wound healing. Suberin and lignin barriers were formed after 2 and 6 days, respectively. Falcarindiol, being present at injury, is probably the main barrier to infection during the first 16 hr. After this initial period, the inhibitory effect is probably reinforced by inducible antifungal compounds, particularly 6-methoxymellein, which was detected after 16 hr (36). Harding and Heale (29) reported activity of 6-methoxymellein (ED_{50} = 104 μg/ml), *p*-hydroxybenzoic acid (ED_{50} = 607 μg/ml), and falcarinol (ED_{50} = 9.27 μg/ml) against *B. cinerea*. They did not detect falcarindiol because very little phloem parenchyma, which contains this compound, was included in their samples of root tissue.

Wound healing. Barriers formed at injury sites by suberization, lignification, or callose formation have been observed in many crops. Formation of these barriers has been extensively studied in potatoes (1,44,53), sweet potatoes (2,40), and yams (39,45). Other crops, where healing has been observed, are apple (35), carrot (24), cassava (9), citrus (6,10,12), and vegetables such as beet, parsnip, squash, and turnip (14). Wound healing has been associated with resistance of potatoes to bacterial soft rot (50), dry rot (15), and gangrene (22), and resistance of sweet potatoes and yams to storage rots caused by species of *Rhizopus* and *Fusarium* (39,40). Green mold (10) and sour rot (6) of citrus and gray and blue mold of apples (35) were significantly reduced when surface injuries became lignified. Suberin and lignin were involved in resistance of injured carrot roots to gray mold and liquorice rot, but antifungal substances were more significant in providing resistance (24). Type and severity of the injury affect the extent of healing and subsequent degree of protection to infection. In citrus, deep injuries into the mesocarp heal less effectively (6,10), and, in yams, injuries consisting of surface abrasions, bruises, or deep cuts did not heal as effectively as minor cuts (39,45). Varietal differences in rate of wound healing were observed in potatoes (41), but they were not correlated with resistance to the common storage diseases.

Though wound barriers are responsible for resistance to many postharvest diseases, efforts to identify the exact mechanism(s) involved in resistance to penetration by the pathogens have been very limited. Lignified cell walls are more resistant to enzyme attack (12), and if lignification of hyphae of the pathogen at the injury site can occur (28), hyphal extension would possibly be prevented. Wound barriers may contain compounds that directly inhibit fungal growth. Other possible mechanisms not yet specifically identified are structural barriers to physical penetration, inhibitors of enzymes or toxins of the pathogen, and restriction of movement of nutrients, water, enzymes, or toxins (47).

MANAGEABLE FACTORS THAT ALTER HOST DEFENSE AT THE WOUND SITE

Temperature, water, and water vapor. Temperature and relative humidity are the two most important conditions affecting the wound healing process. Temperatures must be sufficiently high to encourage rapid development of the metabolic reactions involved in healing and adequate moisture must be present to prevent desiccation and death of the tissues surrounding the damaged cells. Generally, temperatures above 10 C and relative humidities above 85% are required. Artschwager (1) observed suberization in potatoes in 3 and 2 days at 10 and 15 C, respectively, but only in 1 day at 21–35 C. Suberization was a prerequisite to wound periderm formation (1), which usually occurred 1–2 days later. At 20 C, healing was most rapid between 70 and 100% RH, and at 10 C between 80 and 100% RH (53). High humidity appeared not quite so important at 20 C, where the effects of surface drying were negated by the rapid rate of healing. Relative humidities close to 100% sometimes caused cell proliferation, which prevented periderm formation (53). Seven days of wound healing at 20 C and high relative humidity before inoculation were necessary to provide protection against *Fusarium*, and 2 days were required for protection against *Erwinia*. In sweet potatoes (2), yams (45), and cassava (9), suberization and wound periderm formation occurred readily between 22 and 35 C at high relative humidities. A temperature of 25 C and near 100% relative humidity was optimum for carrot roots (24).

Optimum temperatures for healing are not necessarily those that are most effective for disease control, particularly if the temperature is also optimal for growth of the pathogen. Infection may occur before healing can convey resistance. For this reason, temperatures near 10 C, less than optimal for wound healing of potato, were used for the most effective control of bacterial soft rot (50). Degreening of citrus fruit with ethylene is done commercially in Florida at 30 C and 90–96% RH, conditions that cause the most rapid removal of chlorophyll. Lignification of minor injuries in the rind occurs optimally under these conditions (10), but growth of *P. digitatum* is retarded. Significant reductions of green mold occur during degreening in Florida, but not in California where degreening is conducted at 25 C, a temperature more favorable for growth of the green mold fungus. Control of green mold has also been accomplished by wrapping fruit in plastic wraps to maintain high relative humidities at 36 C during curing to induce healing and prevent growth of *P. digitatum* (7).

Addition of water to healed injuries has increased bacterial soft rot of potato (43) and sour rot of citrus fruit (6). Free liquid in the intercellular space may enhance the diffusion and activity of macerating enzymes or increase enzyme production (19). In contrast, desiccation of injured tissue to water potentials below those required for fungal growth has reduced Alternaria rot and gray mold rot of blueberries (13) and green mold of citrus (20). Green mold will develop in desiccated, unhealed injuries if the citrus fruit are returned to a humid environment (10).

Chemicals. Various chemicals, such as sprout inhibitors, sanitizers, bactericides, and fungicides, have been applied to harvested crops, particularly potato seed pieces. Some of these chemicals have affected wound healing processes and subsequent disease development. The sprout inhibitor, CIPC (isopropyl-*N*-3-chlorophenyl carbamate), delayed suberin and wound periderm formation, and at the higher rates of application, it inhibited periderm formation (3). Even though very little or no periderm developed after treatment with higher concentrations of CIPC, all injuries eventually developed enough suberin to retard or prevent infection by *E. carotovora* (3). The sprout inhibitor, methyl ester of naphthaleneacetic acid, delayed healing. It essentially prevented periderm formation and caused higher incidences of dry rot caused by *Fusarium* spp. in treated tubers (15). Chlorogenic acid (32) and catechol (49) stimulated suberization more than other orthodihydric phenols. Resorcinol inhibited suberization, and this was attributed to its deleterious effect on tyrosinase, which reportedly was involved in suberin synthesis (49). Similarly, cycloheximide blocked suberization by its action on phenylalanine ammonia-lyase, thereby rendering potato tubers susceptible to soft rot (54). Dichlorophen, a phenolic compound, exhibited in vitro activity against *E. carotovora*, but poor control in vivo because of its inhibitory action on wound healing (30). Metiram stimulated suberization in cut potatoes, but wound periderm was not stimulated as efficiently (16). Hydrated lime and chloranil favored suberization and wound periderm production (48). Ferbam and sulphur delayed them slightly, while mercuric chloride and phenylmercury acetate were obviously detrimental (48). Of the various fungicide and antibiotic formulations tested by Nolte et al (44), none exhibited stimulatory effects on wound healing, and streptomycin and captan-rhodamine were detrimental. Chlorine caused a thicker suberin layer to form, but this barrier was less effective in preventing moisture loss and infection by *Fusarium sambucinum* Fuckel than the barrier formed on untreated potatoes (52). Chlorine inhibited deposition of the wax, which may play a role in preventing pathogen entry until the suberin layer is completely formed.

Growth regulators. Treatments with ethylene or ethylene-generating compounds have enhanced accumulation of phenolics, 6-methoxymellein, eugenin, and lignin in carrot roots and phen-

olics and lignin in turnip (8). However, increased disease control resulting from enhancement of 6-methoxymellein accumulation is of little practical value because it creates off-flavors (27). In citrus fruit, pretreatment with ethylene has enhanced resistance to subsequent inoculations with *Penicillium italicum* (blue mold) (21), and sensitized the rind to lignify more extensively upon subsequent injury (11). In contrast, ethylene has enhanced development of stem-end rot by suppressing the accumulation of phenolic and lignin compounds in cells at injury sites exposed during abscission (4). Time of exposure of injured tissue to ethylene apparently influences whether healing is enhanced or suppressed. Suberization in cut potato disks is stimulated by application of abscisic acid, which apparently plays a role in inducing the enzymes involved in suberin biosynthesis (51). Of various growth-regulating substances added to injured potato, traumatic acid markedly stimulated wound periderm, but it did not increase resistance to bacterial soft rot (43).

Atmosphere. Lower oxygen and higher carbon dioxide levels than those in the atmosphere had a progressively inhibitory effect on suberin and periderm formation in injured potato tubers (53). However, under aerobic conditions, resistance to early bacterial soft rot infections may be more dependent on factors that protect pectate substrates in the middle lamella from pectic enzymes than on suberization, which occurs later (38).

Cultural and handling practices. Production, harvesting, and storage practices influence wound healing and development of some postharvest diseases. Carrot roots were more susceptible to liquorice rot with advanced maturity at harvest and increased time in storage (17). Young roots (127 days old at harvest) were most resistant, suggesting that sowing date could be timed to harvest roots at the stage of maximum resistance. Young roots had the greatest potential for effective formation of structural and chemical barriers. This potential apparently also decreased with time in storage. Loss of resistance of carrot roots to grey mold with increasing periods in cold storage was related to decreasing ability of the roots to synthesize 6-methoxymellein (27). Increased weight loss due to inadequate humidity control during storage also increased susceptibility to the disease (26). Increased susceptibility of potatoes to gangrene was related to less effective wound healing as harvesting was delayed (41). Turgid lemons, picked in the morning or after rainfall, developed more sour rot because of the effect of high water potential on increased susceptibility of injured tissue to infection (5).

CONCLUSIONS

Susceptibility of injured fruit and vegetables to infection by various postharvest pathogens is altered by the accumulation of toxic compounds at the injury site and by healing of the injury primarily through suberization or lignification. Various factors, as discussed in this review, can affect the extent to which resistance can be manifested. In addition to these factors, another area of study, which has been largely neglected, is that of induced resistance through the use of elicitors. Basic studies, particularly in the area of phytoalexins and host resistance, have shown that some of the compounds responsible for resistance in injured tissue, such as rishitin or lignin, can be elicited by specific fungal or plant constituents (34). These include rather innocuous compounds, such as specific fatty acids, polysaccharides, such as β1-3 glucans of certain sizes, chitin, chitosan, cellulase, pectin, and polygalacturonic acid. Some of these elicitors or others could possibly enhance resistance in injured tissue if applied in postharvest treatments.

Management practices that use wound healing as a basis for controlling postharvest diseases of potato, sweet potato, and carrot roots have been significant, and will continue to be important with these particular commodities. Disease control in other crops through wound repair is currently less important. Effective management of wound repair should be combined with careful handling, sanitation, proper storage, and proper application of effective biocides to assure good keeping quality of harvested crops.

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