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The Role of Xylem in Bitter Pit Incidence of Apple: A Review

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Abstract

Bitter pit is a physiological disorder in pome fruit which causes substantial losses for commercial apple producers. Bitter pit is purportedly associated with calcium (Ca) deficiency, but other nutrients and their ratios with Ca, i.e. magnesium (Mg) and potassium (K), exacerbate the disorder. Symptoms of bitter pit are similar to corking disorders resulting from boron (B) deficiency, resulting in frequent and considerable confusion, especially in the early literature. Ca and B transport in apple, as in other fruits, becomes increasingly more limited as fruit develop by gradual and progressive xylem dysfunction. While the mechanisms that regulate and impair xylem function are still poorly understood, xylem dysfunction of bitter pit-sensitive cultivars occurs earlier than in resistant cultivars. Although phloem plays a compensatory role for water transport to growing apples, phloem tissue is void of free Ca. The focus of this review therefore is on new insights in xylem differentiation, function, and transport and the consequent effects on nutrient deliveries to fruit. We propose a novel approach to mitigate bitter pit and increase xylem differentiation and function via exogenous applications of auxin. The process by which xylem tissue is maintained, and the mechanisms which govern its dysfunction require further elucidation.

Bitter pit is a physiological disorder of pome fruit characterized by dark circular lesions which develop just beneath the surface of fruit and extend to the peel. Lesions typically develop in storage or after storage, but fruit can develop lesions while still on the tree in extreme cases (Brooks 1908). The lesions vary from cultivar to cultivar and typically range from 2 to 10 mm in diameter. Jones (1899) described the pits as "extending into the flesh for a distance about equal to its diameter", implying pits have a hemispherical shape which originate from a single point in the outer cortex just beneath the fruit surface. The lesions extend into the cortical tissue of fruit, comprising corky brown areas of necrotic cells. Visible symptoms appear on the fruit surface, typically displaying first at the calyx end of the fruit.

Susceptibility to bitter pit varies dramatically among apple cultivars. Several notable cultivars in the 19th and first half of the 20th century, such as 'Baldwin' and 'Northern Spy', were prone to bitter pit and have been replaced by less susceptible alternatives. Among modern cultivars, 'Cortland', 'Honeycrisp', 'Braeburn', 'Mutsu', 'Jonagold', and 'Ida Red' are susceptible. 'Honeycrisp' is particularly susceptible to bitter pit, which

Fig. 1: Presentation of bitter pit in 'Honeycrisp' fruit

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partially accounts for increased packout costs in the face of high consumer demand. Some widely consumed cultivars such as 'Gala' and 'McIntosh' appear to be almost completely resistant to the disorder, however.

Historically, symptoms of bitter pit were difficult to differentiate from alternative corking disorders or pathogen-induced skin lesions such as those associated with (*Venturia inaequalis)* apple scab, which was first understood as a fungal infection in the late 1800s (Lamson 1897). Because of its conflation with apple scab, bitter pit was previously imputed to fungal infection; the efficacy of Bordeaux mixture facilitated a distinction of bitter pit from apple scab. Bitter pit was also misdiagnosed as chemical toxicity, viral infection, insect predation, B deficiency, and excessive transpiration (McAlpine 1912). The condition was finally understood to be the result of calcium deficiency through fruit ash analysis by DeLong (1936). Attempts to correct the disorder with exogenous applications of Ca have not eliminated bitter pit. Therefore, a better understanding of bitter pit and its causes are necessary to further manage the condition.

This review seeks to synthesize previous research and horticultural reviews on bitter pit to form a cohesive narrative from which we advance new areas of inquiry to mitigate the disorder. Brooks (1908) was among the first to synthesize early bitter pit research in an effort to clearly define the presentation of the disorder and its relation to both apple anatomy and physiology. McAlpine's review (1912) rejected previously suggested causes of bitter pit and narrowed the list of probable causes. Faust and Shear (1968) separated bitter pit from other corking disorders and advanced recommendations to limit incidence, and Perring and Pearson (1986) examined the physiology of postharvest bitter pit incidence and its mitigation. Ferguson and Watkins (1989) produced a comprehensive overview, which included the history of bitter pit research and current scientific understanding of Ca movement in fruit. Most recently, Fallahi and Mahdavi (2020) described the effects of common horticultural practices on bitter pit incidence, synthesized research pertinent to bitter pit prediction, and recommended methods of control. This review will primarily focus on the role and limits of nutrient transport on bitter pit incidence. Based on a greater understanding of vascular anatomy, we propose novel strategies to extend xylem function within fruit tissues.

Bitter Pit as a Calcium Deficiency Disorder

Historical Linkages between Calcium and Bitter Pit. As early as 1912, McAlpine, while still unsure of the exact cause of bitter pit, noted that "where the soil and subsoil is calcareous, the most susceptible apples are comparatively free from bitter pit". McAlpine suggested the addition of lime to orchard soils to mitigate the condition and described a difference in the ash of pitted and healthy fruits. In 1932, DeLong analyzed the ash of two 'Fallawater' apples, one affected by "blotchy cork" (an older synonym for bitter pit) and one unaffected. Among the different ash constituents, Ca varied between the two fruits—0.071 g/kg versus 0.052 g/kg in clean and pitted fruit, respectively. In 1934, DeLong returned to the orchard to conduct a more comprehensive study on fruit of trees receiving variable horticultural treatments. Among this fruit, Ca concentration again explained differences between pitted and clean fruit (DeLong 1936). This study was the first notable account to ascribe bitter pit incidence to the concentration of Ca in fruit.

Improving Ca concentration in fruit, however, has proven difficult. Bangerth (1979) proposed that bitter pit may result from deficiencies in the uptake of Ca, not soil Ca levels since soil Ca is typically in excess of apple tree demand. Although bitter pit can be induced by growing trees in Ca-depleted media (Ferguson and Watkins 1989), container culture in combination with nutrient withholding confirmed the association of bitter pit with localized Ca deficiency and not

total tree deficiency (Martin et al. 1962). Ca concentrations vary markedly within a tree, decreasing with height and at branch apices (Saure 2005). Even within individual fruit, total Ca is not a sufficiently robust predictor of bitter pit incidence (Perring and Pearson 1986). Plots of bitter pit incidence and Ca concentration in fruit have been described as "wedge-shaped" with some fruit remaining healthy despite low Ca concentrations (Perring 1986; Ferguson and Watkins 1989). Recently, bitter pit has been connected to apoplastic Ca pools, indicating that bitter pit is 1) a localized Ca deficiency and 2) that transport is an important regulatory contributor to bitter pit incidence (de Freitas et al. 2010; Falchi et al. 2017). This is consistent with the fact that Ca is unique among the macronutrients (N, P, K, Mg, Ca, S) *in planta* as it is exclusively xylem-mobile, with only trace amounts found in living phloem tissue (Ferguson et al. 1979).

Calcium Function in Apple. Sixty percent of intracellular Ca is located in the form of cross-linkages between pectins in cell walls (de Freitas et al. 2010). These cross-linkages determine many of the physical characteristics of plant cells, including rigidity and selectivity of membranes with physical changes in cells being associated with modification and/or solubilization of pectins (Hocking 2016). The majority of these pectins occur in the middle lamella, the location of cell-to-cell junctions. Ca and B are unique in their suitability for forming these bonds; Ca bonding with homogalacturans and B between rhamnogalacturonan II units (Pérez-Castro et al. 2012; Funakawa and Miwa 2015). Bangerth (1973) linked bitter pit with the replacement of Ca in these bonds by either K, Mg, or H, resulting in weak linkages that eventually leak pectins to the surrounding tissue. Pectin methylesterase expression was higher in the calyx of bitter pit affected fruit, and the pectins present in pits are shorter than those found in healthy cells (Faust and Shear 1968; Zúñiga et al. 2017). As a result, bitter pit lesions stain intensely when treated with solutions that bind to pectins; McAlpine attributed the brown color of the pits themselves to pectins (McAlpine 1912; MacArthur 1940). Because of the role of Ca in maintaining cell wall integrity, Ca has been firmly linked with storage quality in fruit, with Ca-deficient fruit having decreased firmness and increased susceptibility to postharvest disorders (Ferguson and Watkins 1989).

Among organelles, the vacuole is by far the largest site for Ca storage (Peiter 2011) where the remaining 40% of Ca in apple cells was quantified (de Freitas et al. 2010). Vacuolar Ca concentration varies significantly among different plant species and cells of different tissues within the same plant. Ca transport across the tonoplast of the vacuole is mediated by Ca^{2+}-ATPase pumps and Ca^{2+}/H^{+} antiporters. Higher concentrations of Ca in vacuoles was associated with a greater expression of Ca^{2+}/H^{+} antiporters, decreased apoplastic Ca, reduced plasma membrane stability, and a greater incidence of blossom end rot (BER), a Ca-deficiency disorder in tomato (de Freitas et al. 2011). Knockout of these Ca^{2+}/H^{+} antiporters in *Arabidopsis* resulted in higher apoplastic Ca (Conn et al. 2011). Apoplastic Ca is available to link with pectins to maintain cell stability. As a result, bitter pit resistance was associated with the total amount of Ca in the apoplast rather than the fruit as a whole (Turner et al. 1977; Falchi et al. 2017). De Freitas et al. (2010) determined that cystolic Ca in apple cells is extremely low (0.1-0.2 μM), and that proper cell function requires maintenance of at least 0.1 mM free Ca in the apoplastic pool. By assessing the leakage of Ca from fruit discs, Ferguson and Watkins (1983) found that exogenously applied Ca does not migrate into cells to help perform cell functions, but instead remains in this apoplastic space between cells.

Calcium Deficiency vs. Boron Deficiency

Like Ca, B is considered to be relatively immobile in plant tissues and its uptake is driven by transpiration (Raven 1980). However, B is freely transported in the phloem

in the form of sorbitol-B complexes in sorbitol-rich species such as *Malus domestica* B. (Brown and Hu 1995). B is important in maintaining cell wall integrity through bonding with pectins (Fang et al. 2016), and is important in facilitating auxin transport (Quiles-Pando et al. 2019). Because of the similarities in uptake and function, B deficiency in apple manifests in a similar fashion to Ca deficiency.

Early researchers struggled to differentiate bitter pit from other corking disorders such as internal cork, corky core, cork spot, blotchy pit, and drought spot, all of which have been reclassified as B deficiency disorders. One of the key differences between these disorders and bitter pit is that these conditions always occur distal to the fruit's surface, with lesions only reaching the surface in the most severe of cases (Mix 1916). B deficiency disorders also do not worsen during postharvest storage like those associated with Ca disorders (Ferguson and Watkins 1989).

While perhaps not playing a direct role in bitter pit lesion formation, bitter pit is exacerbated by B deficiency, (Biggs and Peck 2015; Fallahi 2020) and fruit Ca was positively associated with B concentration (Dixon et al. 1973; Fazio et al. 2017). This association has been contentious, historically, with many earlier studies contradicting any association between B and Ca (Wallace and Jones 1941) and later studies finding the link more definitive. Korban and Swiader (1984) identified two genes which potentially conferred bitter pit resistance and noted higher B and Ca and lower Mg and K in resistant fruit. The mode of action by which B affects fruit Ca uptake is not clear, but B plays a role in Ca partitioning, sugar formation, and root growth, the latter of which is critical to bitter pit prevention (Atkinson 1935; DeLong 1936; Garman and Mathis 1956). Unlike fruit K, which remains constant throughout the growing season, or Ca and Mg which decrease as fruit mature, B concentrations increase as fruit ripen (Cheng and Raba 2009). While B deficiency disorders are effectively treated by exogenous B application, bitter pit is not completely prevented by Ca sprays (Wallace and Jones 1941). The incomplete effect of Ca sprays on bitter pit suggest that bitter pit may result from the interplay of Ca with other cations.

Magnesium and Potassium Excess

McAlpine (1912) noted that pitted tissue contained more ash minerals than healthy tissue, and this ash was more alkaline, indicating a proportionately higher concentration of cations. Among the metals, K, Ca, and Mg are present in markedly higher concentrations than other elements.

Mg deficiency can cause leaf spotting and early fruit drop, but Mg deficiency is not common in most production regions and can even be withheld (Garman and Mathis 1956; Martin et al. 1962). Elevated levels of both Mg and K in mature fruit were associated with higher incidence of bitter pit (Ferguson et al. 1999), and treatment of trees and their harvested fruit with Mg actually induced bitter pit (Witney et al. 1991; Amarante et al. 2005). However, labeled, exogenously applied Mg was only recovered in peel, not the cortex (Cooper and Bangerth 1976). This finding is potentially paradoxical to the understanding of bitter pit as a condition that begins in the outer cortex of fruit, rather than the peel. However, in 'Honeycrisp', peel nutrient status has proven a better predictor of bitter pit susceptibility than flesh nutrients (Baugher et al. 2017). Zúñiga et al. (2017) used Fourier transform infrared and X-ray spectrometers to demonstrate significant differences in healthy vs pitted tissue in the amounts of Mg and K present, with both being significantly higher in pitted tissue. Askew et al. (1960) showed Mg to be present in pits at up to four times the concentration in healthy tissue. Mg is detrimental to the postharvest storage quality of apple fruit, negatively affecting firmness (Marcelle 1995); thus, producers are encouraged to limit Mg sprays.

While Mg concentration in fruit correlates

to greater incidence of bitter pit, the ratio of K/Ca in fruit tissue is a far more effective indicator of bitter pit development (Wills 1976). Like Mg, high levels of K negatively affect storage characteristics in apple, but K is positively correlated with both sugars and acid in fruit (Garman and Mathis 1956; Marcelle 1995). Like Ca and Mg, K is present at its highest concentrations in the skin and core and at its lowest concentration in the outer cortex where bitter pit is believed to originate (Ferguson and Watkins 1983). 'Honeycrisp', a cultivar highly susceptible to bitter pit, contains higher amounts of K and lower concentrations of Ca in fruit tissue relative to 'Gala', a resistant (Cheng and Sazo 2018). Lightly cropped trees frequently exhibit higher levels of bitter pit from the dilution of Ca but also because fruit serve as a major K sink (Cheng and Raba 2009). The optimal level of K in apple fruit and leaves varies with cultivar. Producers are encouraged to limit supplemental K while maintaining sufficient levels to support healthy fruit growth and development since K is mobile in soil and easily depleted (Fallahi and Mahdavi 2020).

Mg and K are believed to initiate bitter pit in fruit by replacing Ca ions in cell membranes, resulting in a loss of rigidity and selective permeability and eventual cell death (Bangerth 1979). The high concentrations of these ions in bitter pit lesions are likely the result of the disintegration of cell walls containing these elements in abundance. Because K and Mg are phloem-mobile, unlike Ca, these cations accumulate in areas where xylem has become dysfunctional, leading to bitter pit lesion formation. Thus, much of the relationships among nutrients that associate with bitter pit are issues of mobility and transport.

The Role of Xylem

Unlike phloem, xylem becomes gradually dysfunctional during the fruit growth period in apple (Lang 1990) but also in *Vitis vinifera* (grape) (Düring et al. 1987; Findlay et al. 1987), *Actinidia deliciosa* (kiwifruit) (Dichio et al. 2003), *Prunus avium* L. (sweet cherry) (Grimm et al. 2017), *Prunus domestica* (European plum) (Winkler and Knoche 2021), and *Solanum lycopersicum* (tomato) (Ho et al. 1987).Thus water delivery to fruit is limited to phloem transport as development advances. Because functional xylem tissue is required for Ca transport into and throughout fruit, Ca concentrations become diluted with growth. In contrast, phloem-mobile ions such as Mg and K continue to accumulate in tissues and cell walls which rely on Ca for cell integrity (Jones et al. 1983; Cheng and Sazo 2018). Xylem tissue becomes dysfunctional in stages; fine, secondary vasculature nearest the fruit surface is the earliest to lose functionality followed by the ventral, dorsal, and primary bundles located beside the carpels, at the tips of the carpels, and on the cortex line, respectively (Dražeta 2004). The rate of dysfunction of primary bundles exceeds that of dorsal bundles. Interestingly, xylem function in the pedicel was retained throughout the entire growth period implicating xylem tissue in the fruit, rather than the pedicel, as the weak link to transport (Dražeta 2003).

The rate of xylem dysfunction is not constant across cultivars. Notably, the relative susceptibilities of various cultivars to bitter pit has been connected to the onset and rate of xylem dysfunction (Lang 1990). The rate at which primary bundles become dysfunctional varies more widely among cultivars than either dorsal or ventral bundles, with primary bundles typically becoming completely dysfunctional at maturity (Dražeta 2004). Dysfunction of primary bundles is problematic for rapidly growing fruit, as primary bundles supply nutrients to the flesh (MacDaniels 1940). The primary bundles of bitter pit sensitive cultivars such as 'York Imperial' (Barden and Thompson 1963), 'Braeburn' (Dražeta 2004), 'Catarina' (Amarante et al. 2013), and 'Honeycrisp' (Griffith et al. unpublished; Fig. 2) become dysfunctional significantly earlier than resistant cultivars. Amarante et al. (2013) determined primary bundles became mostly dysfunctional just 40

days after full bloom (DAFB) in the highly susceptible 'Catarina' compared to 100 DAFB in the less susceptible 'Fuji'.

While some differences in bitter pit susceptibility between cultivars can be explained by variability in fruit size, a genetic component exists as well. Volz et al. (2006) determined that genetic variability explained bitter pit susceptibility better than either fruit size or Ca content of fruit, suggesting Ca delivery may vary between cultivars. The open-pollinated seedlings used by Volz et al. (2006) had significantly less bitter pit overall than the cultivars selected for commercial production, with less than half of seedlings exhibiting symptoms, and the seedlings that were affected had an average bitter pit incidence of under 10%. This rate of incidence is significantly less than the 50% observed in 'Honeycrisp' (Rosenberger et al. 2004). This information suggests bitter pit is not a common disorder in apple—rather, bitter pit is common among the large-fruited cultivars which have been chosen for widespread cultivation which tend to experience earlier vascular dysfunction.

Regardless of the timing of dysfunction, primary bundles become dysfunctional following an exponential decay model. Consequently, nearly all Ca uptake in apple fruit occurs early in the growing season (Saure 2005). In fact, under periods of stress, xylem dysfunction may be a programmed action to limit the backflow of water and solutes from fruit to tree (Dražeta 2003; Winkler and Knoche 2021). Loss of xylem functionality has been attributed to a variety of different perturbations in cells, including an increase and/or elongation of parenchyma cells which constrict xylem elements (Lang and Ryan 1994), cessation of cambial activity by which xylem cells are generated (Dražeta 2003), or simple stretch-induced dysfunction of xylem tissue (Findlay et al. 1987).

These suggestions may fail to account for the persistence of phloem function throughout the season as xylem becomes progressively dysfunctional. Dražeta suggests three potential pathways by which the phloem may retain its functionality:

1. Greater flexibility of sieve tube elements in phloem compared to tracheary elements in xylem (Lee 1981)
2. The existence of a "continuous symplastic pathway through parenchyma cells that occupy the voids between the broken structures"
3. The potential for sieve tubes to be constantly differentiated to replace broken elements over the course of the season

The latter of these suggestions is possible because phloem can differentiate without the presence of xylem (the inverse is not true) and can differentiate at lower auxin levels than xylem (Aloni 1980). This suggestion points to a potentially new insight in xylem dysfunction—auxin deficiency.

Differentiation of tracheary elements is the result of the action of multiple phytohormones. Auxin, in the form of the native indole-3-acetic acid (IAA), controls xylogenesis through the expression of xylogenetic genes (Jacobs 1952; Pesquet et al. 2005). These xylogenetic genes result in the maintenance of a population of procambial cells from which tracheary elements can differentiate (Turner et al. 2007). GAs and cytokinins function synergistically with auxins in the early stages of xylem differentiation by expanding the xylem cells initiated by auxin (Israelsson et al. 2005). Through canalization, a process whereby local auxin accumulation creates a positive feedback loop, auxins create vascular pathways in plant tissues (Yoshimoto et al. 2016). Canalization requires an initial auxin source. This source is the endosperm of growing seeds, beginning around 25 days after petal fall, a period corresponding to the cessation of the post-bloom drop (Luckwill 1948). The role of seeds as auxin producing agents and promoters of xylem differentiation explains the observation that apple fruit with fewer seeds than average contain less Ca (Buccheri and Di Vaio 2004). Moreover, maximum IAA concentration in apple cortical tissue occurs at 45 DAFB and

then declines, a period which correlates well with the rapid decline in xylem functionality of bitter pit sensitive cultivars (Devoghalere et al. 2012). Griffith et al., applied successive exogenous auxins at 30, 45 and 60 DAFB to 'Honeycrisp' apple trees and noted significant improvements in both xylem functionality of fruit and bitter pit incidence (Griffith et al. *in-press)*.

Incidentally, this time frame also corresponds to the period in which xylem elements are lignified through secondary cell wall deposition, providing structural support to the apple fruit but also rendering xylem prone to breakage (Dražeta 2003). This process was promoted by gibberellins (GAs) and antagonized by auxins, which reduced cell wall thickness, allowing xylem elements to remain flexible (Johnsson 2018). However, application of GAs in the form of whole-tree GA_3 sprays produced no differences in the number of functional vascular bundles relative to control fruit (Griffith et al. unpublished). In tomato, abscisic acid (ABA), led to an increase in the abundance of functional vascular bundles in fruit (de Freitas et al. 2011). Additionally, ABA reduced transpiration in tomato, reducing the gradient in water potential between leaves and fruit (Falchi et al. 2017). Because Ca transport is driven by transpiration, treatment with ABA increased Ca in tomato fruit and reduced BER incidence. Preliminary research by the authors has reproduced these findings in apple with whole-tree ABA sprays and significantly reduced bitter pit (Griffith et al. *in-press*).

Another potential avenue of bitter pit research may be to focus on prevention of symptoms rather than addressing Ca deficiency itself. The breakdown of cell membranes is initiated by the enzyme phospholipase D. Hexanal slows the synthesis of the enzyme phospholipase D which is responsible for cell membrane breakdown (Aghdam 2012). Recently, DeBrouwer et al. (2020) found that whole-tree applications of hexanal were remarkably effective in limiting phospholipase D, increasing structural integrity of cells, and decreasing bitter pit by as much as 70%. As the mechanisms by which bitter pit symptoms manifest are better understood, other treatments focusing on symptoms may be discovered and adopted to use alongside Ca deficiency treatments.

The above recent findings agree with and draw new connections between older anatomical studies and observations. Early researchers observed bitter pit lesions to always occur at the terminations of vascular bundles (Jones 1899; Brooks 1908). Smock and Van Doren (1937) corroborated these observations, but did not detect a difference in the size, shape, or number of vascular bundles associated with pits from those that were not. It is likely, however, that while the presence of vascular elements in conjunction with pits

Fig. 2: Gradual dysfunction of xylem in 'Honeycrisp.' Fruit were stained with acid fuchsin (1% w:w solution in DI H_2O) to demonstrate functionality. Fruit were harvested at 86, 107, and 136 DAFB (top to bottom, stem end of fruit on left, calyx end on right). At 86 DAFB, primary vascular bundles at the core line and dorsal bundles at the tips of carpels can be observed. As fruit develop, xylem dysfunction gradually increases (Griffith, unpublished).

appeared normal, that they were likely dysfunctional, a characteristic only observable upon treatment of fruit with a lignin-specific dye. Nevertheless, early researchers such as McAlpine (1912) and Mix (1916) still facilitated the hypothesis that bitter pit was the result of vascular dysfunction as the result of rapid fruit growth. It appears that the unseen action of hormones such as auxins, GAs, and ABA may be the connection between older hypotheses and newer research.

Conclusion

In their comprehensive bitter pit review in 1989, Ferguson and Watkins stated that, at the time, "the main advances have been in control rather than understanding of the disorder." While this is still somewhat true over 30 years later, the breadth of control methods which have been explored combined with a better understanding of apple fruit physiology has allowed for a narrowing of focus into treatments which maintain apoplastic Ca and therefore cell membrane integrity. Bitter pit's status as a Ca-deficiency disorder is complicated because Ca sprays have not proven to entirely prevent the disorder. Because Ca is only mobile in xylem and because xylem is differentiated via auxins and lignified via GAs, it is conceivable that bitter pit could more accurately be described as a hormone-imbalance disorder. While many disparate studies have been conducted on the role of hormones in mitigating related disorders of different species, there have been fewer studies that directly address their effects on bitter pit. The authors' own preliminary research has demonstrated a significant effect of exogenous auxin (Griffith et al. 2021) and ABA (Griffith et al. *in-press*) on reducing bitter pit incidence and increasing the longevity of vasculature, especially in the distal end of fruit. Further research will be required to elucidate the modes of action of these compounds and to identify efficacious concentrations and timings.

Bitter pit is a complex disorder as its incidence is related not just to mineral status but also to hormonal balances, cellular functions, and horticultural techniques. As a result, a perfect understanding of the disorder is not yet close at hand. However, the combined research of scholars in each of these specialties has already produced methods of mitigation which have prevented substantial fruit losses for growers. Further research is needed to hone and connect these facets of understanding. It is our hope that this review may illuminate gaps in understanding so these connections can be made.

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