Review



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*Review*

# Calcium Route in the Plant and Blossom-End Rot Incidence

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**Abstract:** Calcium ($Ca^{2+}$) is a macro-mineral essential for the growth, development, yield, and quality of vegetables and fruits. It performs structural, enzymatic, and signaling functions in plants. This review outlines $Ca^{2+}$ translocation from soil to fruit via the plant xylem network, emphasizing the importance of $Ca^{2+}$ compartmentalization within fruit cell organelles in developing the $Ca^{2+}$-deficiency disorder, blossom-end rot (BER). The causes and possible control measures of BER are also discussed. Soil available $Ca^{2+}$ enters the root apoplast with the water flow and moves towards the xylem via apoplastic or symplastic routes. The transpiration force and growth of organs determine the movement of $Ca^{2+}$-containing xylem sap to aerial plant parts, including fruits. The final step of fruit-$Ca^{2+}$ regulation is the partitioning among cellular compartments, which determines susceptibility to $Ca^{2+}$-deficiency disorders such as BER. Depleting cytosolic and apoplastic $Ca^{2+}$ due to excessive deposition in organelles such as the vacuole may lead to disintegration of the plasma membrane, resulting in BER, even at high $Ca^{2+}$ availability at the blossom end of the fruit. BER management requires cultural and physiological practices that ensure $Ca^{2+}$ translocation to the fruit and proper $Ca^{2+}$ compartmentalization. The use of BER-resistant and $Ca^{2+}$-efficient cultivars may also help in BER management. Therefore, a comprehensive understanding of $Ca^{2+}$ dynamics in plants is crucial for managing BER, reducing production costs, minimizing environmental impact, and enhancing crop productivity.

**Keywords:** BER; $Ca^{2+}$-deficiency disorder; cellular organelles; $Ca^{2+}$-transportation; $Ca^{2+}$-compartmentalization

## 1. Introduction

Calcium ($Ca^{2+}$) as a macro-mineral is essential for plant growth and development [1–3], and is found within plants in higher quantities than any other divalent inorganic cations. When hydrated, $Ca^{2+}$ is a big cation with a 41.2 Å ionic radius [4]. Among the macronutrients, $Ca^{2+}$ is third after nitrogen (N) and potassium (K) in terms of quantity in a plant body, which reflects its essentiality for the plant. The plant requires 1-3 mM $Ca^{2+}$ for proper growth and development [5]. It plays roles in membrane and cell wall stabilization, cell function, signal transduction, growth and development, gene expression, and stress resistance [6–10]. $Ca^{2+}$ is required for every cellular compartment, such as cell wall, apoplast, plasma membrane, cytosols, and organelles suspended within cytosols, e.g., vacuoles, endoplasmic reticulum (ER), plastid, Golgi apparatus, and nucleus (Fig.1). $Ca^{2+}$ concentration varies from $10^{-7}$ M to $10^{-3}$ M across the cell organelles. Though cytoplasm contains $10^{-7}$ M calcium at the resting stage, it increases to $10^{-5}$ to $10^{-4}$ M in the storage organelles and $10^{-3}$ M in the extracellular milieu [11,12]. The $Ca^{2+}$ content of mitochondrial and nuclear matrices is similar to that of the cytosol. The free $Ca^{2+}$ content in cytosol and vacuoles is 100–200 nM and 1–10 mM, respectively [13,14], and 60% of the plant $Ca^{2+}$ remains as calcium pectate. $Ca^{2+}$ content in plants varies greatly; shoot-$Ca^{2+}$ ranges from 0.1%-5%, while fruit-$Ca^{2+}$ varies from 0.2%-0.3% of total dry mass [15]. The proportion of $Ca^{2+}$ in specific tissues can be more than 10% without affecting plant growth and development [4].

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Calcium is essential for cell wall integrity, membrane permeability, and stability and plays a role in the signaling route as a messenger [15–18]. The available $Ca^{2+}$ enters the root apoplast with the water flow [19], and moves towards the xylem, following either apoplastic or symplastic routes [20]. Along the xylem water flow, $Ca^{2+}$ is transported to the leaves and fruits based on their transpiration strength during the daytime, while at night, root pressure does the job [21]. Poor $Ca^{2+}$ translocation to the fruit or leaf tip can result in $Ca^{2+}$ deficiency disorders, e.g., blossom-end rot (BER), tip burn, blackheart, brown heart, bitter pit, empty pod, and fruit cracking (Table 1) [20,22–26].

Among numerous $Ca^{2+}$-deficiency disorders, BER is most prevalent and causes substantial economic losses worldwide. BER often initiates at the fruit blossom part (away from the peduncle) during early fruit growth stages (2-3 weeks following anthesis) [27–37]. Insufficient $Ca^{2+}$ supply to the rapidly growing fruit tissue causes the disintegration of the plasma membrane and lysis of the middle lamella, resulting in cell plasmolysis and a water-soaked appearance. Subsequent drying develops sunken, brown, and black spots that are limited to the fruit blossom end or can encompass the entire fruit [38]. BER incidence is positively correlated with root's relative water content, fruit number per plant, potassium ($K^+$) and vitamin C levels of fruits and negatively correlated with plant height, leaf chlorophyll content, total yield, and fruit $Ca^{2+}$ content in tomato [39,40].

Though agricultural soils are not usually low in $Ca^{2+}$, $Ca^{2+}$-deficiency disorders are numerous and cause significant economic losses worldwide. $Ca^{2+}$ deficiency is usually not manifested by the unavailability of soil $Ca^{2+}$; instead, it is the soil's inability to supply sufficient $Ca^{2+}$ to the affected plant parts [23]. By being phloem immobile, $Ca^{2+}$ cannot be translocated from the available sources (mature leaves and peduncle-end of fruits) to the deficient sinks (young-growing leaves and blossom-end of fruit). Therefore, $Ca^{2+}$ fertilization generally does not overcome these physiological disorders, and thus, these disorders (e.g., BER) are complex and challenging to solve. Though the genes linked to calcium deficiency disorders are not well-documented, expression of $Ca^{2+}$ /$H^+$ antiporters (*CAX*s) such as *CAX1* and s*CAX1* [41–45], and calreticulin (*CRT*) [44,46] may play a role in this regard [47]. There is no straightforward solution for these disorders. Moreover, the uncertainty of the onset of these deficiency disorders complicates the issues regarding their workable solutions [23]. The present study discusses the route of calcium translocation within plants and the causes and control of BER, the most devastating $Ca^{2+}$-deficiency disorder.

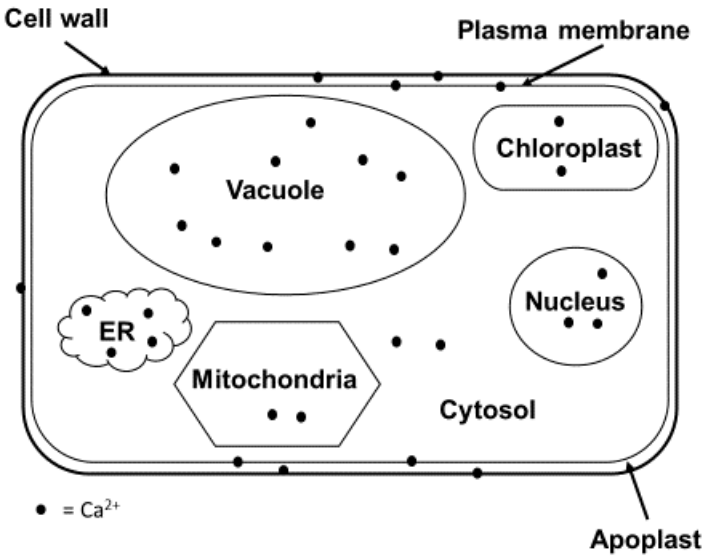

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**Figure 1.** Calcium compartmentalization within a plant cell. Calcium is required for every part of a plant cell and is an essential mineral. Calcium plays a pivotal role in cell structures such as the cell wall pectin and the plasma membrane, as a signaling molecule in the cytosol, and as a cofactor for several enzymes. Most calcium is found in the vacuoles, which function as an ion balance and pH mediator.

## 2. Function of Calcium in Plants

$Ca^{2+}$ performs numerous plant functions [15] as a structural component of cells, enzymatic regulation, and signal transduction [48]. $Ca^{2+}$ contributes to growth and development via cellular growth, metabolism, and signaling [49].

### *2.1. Structural Role of Calcium*

Calcium maintains cell wall integrity [15,50], cell division and cell elongation [51,52], cell expansion [53], membrane permeability and membrane stability [50,54], and assembly of microtubules [55].

The plant cell wall contains carbohydrates (cellulose, hemicellulose, pectin), proteins, particularly structural ones, and lignin (secondary cell wall). Cell wall $Ca^{2+}$ mainly represents $Ca^{2+}$ bindings in the middle lamella that glue adjacent cells and maintain cell wall integrity. $Ca^{2+}$ is unique among other inorganic elements, which are not usually integral components of cell walls except nitrogen (N). Cell wall $Ca^{2+}$ generally binds with pectin, a polymer of a diverse group of pectic polysaccharides, including homogalacturonans, rhamnogalacturonan I, and rhamnogalacturonan II. Homogalacturonans are polymers of galacturonic acid in a fashion of $\alpha$(1–4) linkage. $Ca^{2+}$ forms a tight linkage between the charged carboxyl ($COO^-$) group of galacturonic acid [56], through which it provides cell wall strength. Pectin forms a gel-like structure by binding with $Ca^{2+}$ molecules in a reversible fashion that aids in tightening (binding with $Ca^{2+}$) and loosening ($Ca^{2+}$ removal) of the cell wall. During the biosynthesis of sugar residues in the Golgi apparatus, the charged carboxyl group can be esterified with methyl, acetyl, or unknown groups that prevent the binding of $Ca^{2+}$ [56], and keep the cell wall loose. Cell walls are also loosened by the degradation of $Ca^{2+}$-pectate by polygalacturonase (PG), and the activity of PG is inhibited by high $Ca^{2+}$ concentration [57]. The PG activity is increased in $Ca^{2+}$-deficient tissue, leading to middle lamella disintegration, primary cell wall degradation, and cell death. $Ca^{2+}$ cannot bind to methylated pectin residue. Pectin methyl esterase (PME) removes the methyl group from methylated pectin and opens up free binding sites for $Ca^{2+}$, where $Ca^{2+}$ binds to form a strong electrovalent bond [58]. Demethylation of pectin by PME action favors the further degradation of pectin by enzymes like endo-polygalacturonase, exo-polygalacturonase, β-galactosidases, and pectate lyases [59,60].

Besides strengthening the cell wall, $Ca^{2+}$ plays a role in stabilizing and functioning of the plasma membrane. The plasma membrane is composed of phospholipid bilayers held together by proteins. $Ca^{2+}$, on the apoplast side of the plasma membrane, binds to the carboxylic group of protein and the phosphate group of phospholipid and thus stabilizes the membrane, allowing a proper membrane selective permeability. The requirement of $Ca^{2+}$ increases due to an increase in heavy metals [61], aluminum ($Al^{+3}$), sodium ($Na^+$) [62], and protons ($H^+$) in the external environment. Other cations can replace the $Ca^{2+}$, but their role is not in proper membrane functioning. $Ca^{2+}$ in high concentration is required to restrain the unfavorable effects. For plants growing in soil with a higher concentration of other cations, the $Ca^{2+}$ requirement increased substantially to ensure optimum plant growth and development [63]. Replacement of $Ca^{2+}$ with $Na^+$, heavy metals, or $Al^{+3}$ can cause salinity, heavy metal, or aluminum toxicity, respectively [64,65]. Membrane instability is prevalent under freezing, low temperature, and anaerobiosis [4]. Unstable membranes are prone to loss of low molecular weight solutes, such as potassium ($K^+$) and sugars. It can also cause an influx of toxic ions (e.g., the heavy metal $Al^{+3}$) in the cytosol. A high concentration of free $Ca^{2+}$ in the apoplast prevents the loss of solutes and helps to avoid potential toxicity from toxic elements. Lack of $Ca^{2+}$ results in a leaky membrane that causes loss of cell material, impairment of cell metabolism, and subsequent cell death.

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$Ca^{2+}$ stabilizes the cell wall by binding with pectin, the cell membrane, and the proteins and lipids at the membrane surfaces [48,66]. $Ca^{2+}$ influences vesicles - full of materials and enzymes for cell wall and membrane construction - incorporation into the plasma membrane [53]. Moreover, $Ca^{2+}$ is required for regulating ion uptake, pH, carbohydrate translocation, the activity of the oxygen-evolving complex, and as a counteraction in the vacuoles for all types of anions [4,16].

### *2.2. Enzymatic Role of Calcium*

$Ca^{2+}$ can promote or demote enzyme activity essential for cell growth and development. The activity of $\alpha$-amylase is stimulated by high $Ca^{2+}$ concentration during starch breakdown in germinating cereal seeds, in which $Ca^{2+}$ ion stabilizes amylase [67]. However, high $Ca^{2+}$ concentrations may inhibit enzyme activity [23], as has been shown with cytosolic enzyme fructose-1,6-bisphosphatase (FBPase) (Figure 2), which regulates sucrose synthesis from triosephosphate (TP) in the cytosol. A slight increase in $Ca^{2+}$ concentration can markedly inhibit the activity of that particular enzyme [4]. $Ca^{2+}$ is also a cofactor of several enzymes, e.g., 1,4-lactonase, phosphoinositide phospholipase C, N-acetylgalactoseaminyltransferase, and affects the synthesis and transport of those enzymes [68].

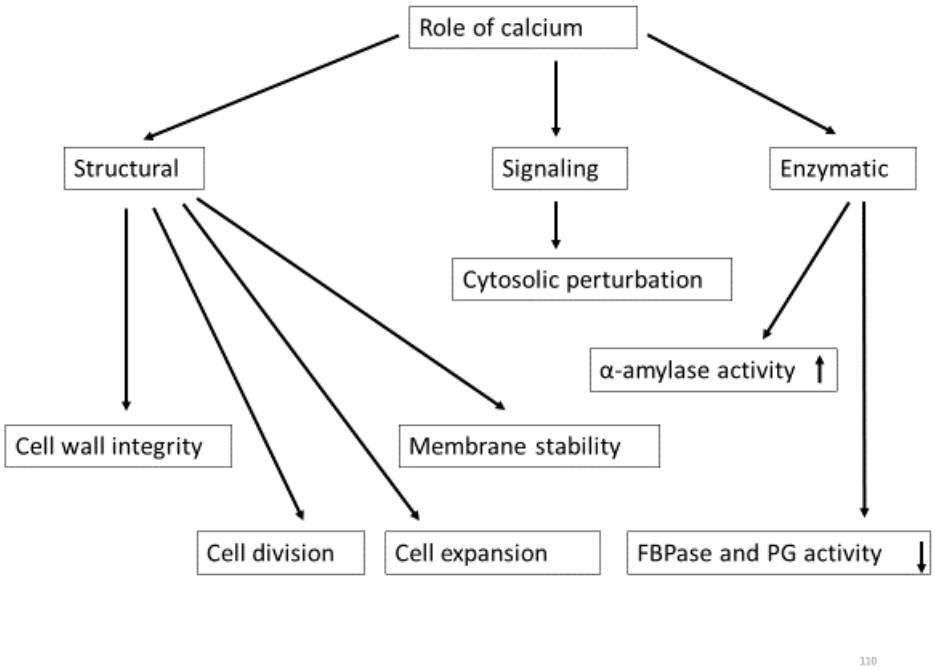


**Figure 2.** Calcium plays structural, enzymatic, and signaling functions. Activities of $\alpha$-amylase are seen during seed germination; fructose 1,6 bisphosphatase (FBPase) is a regulatory enzyme in the sucrose biosynthesis pathway; polygalacturonase (PG) hydrolyzes the alpha-1,4 glycosidic bonds between galacturonic acid residues of pectin.

### *2.3. Calcium and Signal Transduction*

The interest in $Ca^{2+}$ in recent years has gained momentum due to its role as a secondary messenger, particularly for developmental and environmental cues [4]. It plays a crucial role as a signaling molecule for signaling pathways [69]. $Ca^{2+}$ acts as a universal signaling molecule [70], and plays a role in plants' growth, development, and stress management. Environmental stressors initiate cytosolic $Ca^{2+}$ spikes, activating downstream gene expression and adaptation in adverse conditions [70]. Understanding $Ca^{2+}$ dynamics may help develop and engineer climate-smart crop varieties [70]. $Ca^{2+}$ is a stress-response element. Upon sensing stresses, it conveys signals to the downstream protein kinases, leading to phenotypic responses that may result in stress tolerance [71–74]. It also contributes

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to immunity by activating immune responses [75]. Characterizing the $Ca^{2+}$ channels, pumps, and binding proteins is required to comprehend the role of stress signals on $Ca^{2+}$ homeostasis and adaptive responses [76]. It will improve understanding of how specific stress signals modulate $Ca^{2+}$ homeostasis to orchestrate adaptive responses [76].

In response to stimuli, $Ca^{2+}$ transduces signals to the other end upon binding with calmodulin, a calcium protein in the cytosol [17,77–79]. Plants maintained a very low (100 to 200 nM) cytosolic [$Ca^{2+}$], which skyrocketed up to 2 μM at the stimulated state [80]. Plants maintain very low cytosolic [$Ca^{2+}$ ] to serve as a messenger, to prevent precipitation of inorganic phosphate, and to minimize competition for binding sites with magnesium [4]. The role of $Ca^{2+}$ as a messenger is possible due to very low cytosolic [$Ca^{2+}$] and chemistry [20]. Any signal - intensity of light, day length, temperature, salinity, drought, osmotic and oxidative stresses, aluminum toxicity, mechanical injury, anoxia, nodulation, and pathogen attack - exerts an abrupt change in cytosolic [$Ca^{2+}$] and initiates a $Ca^{2+}$-signaling pathway [20,78,81]. Besides, the pathway is also activated by various developmental cues, such as germination, cell division and elongation, circadian rhythms, tropic responses, senescence, and apoptosis [4]. The $Ca^{2+}$-signaling event is location- and time-specific and vital for encoding specific cellular responses [82]. This signaling is subject to judicious regulations as a marked increase in $Ca^{2+}$-concentration activates $Ca^{+}$-dependent enzymes, which are harmful to a cell. Therefore, very tight regulation is in place for $Ca^{2+}$-signaling processes through the coordinated activities of calcium proteins, calcium channels, and efflux systems.

### 2.3.1. Calcium Proteins

Changes in cytosolic [$Ca^{2+}$] are detected by specific proteins that either relay or respond to the messages. Upon binding with $Ca^{2+}$, relay proteins such as calmodulin undergo conformational changes that enable them to interact with a target protein to regulate its function [83,84]. Response proteins such as $Ca^{2+}$-dependent protein kinases (CDPKs) bind with $Ca^{2+}$, followed by a conformational change that initiates their intrinsic kinase activity. Cytosolic $Ca^{2+}$-binding proteins include calmodulins (CaMs), CaM-like proteins, annexins, calcineurin B-like (CBL) proteins, and CDPKs. Calmodulins bind with CaM-binding transcription activators (CAMTAs) and are responsible for gene expression [85,86]. Calmodulins and similar proteins initiate responses to developmental or environmental cues and pathogen attack; CBL to cold, drought, salinity, and wounding; and CDPKs to various stimuli [4]. Plant annexins are associated with cell elongation, membrane repair, the secretory process, salinity, and drought stresses [87]. Several $Ca^{2+}$-binding proteins, e.g., calreticulin, calnexin, calsequestrin, and BiP (Binding Immunoglobulin protein), are found in the ER and are responsible for protein folding, $Ca^{2+}$ homeostasis, and modifications at the post-translational stage [4].

### 2.3.2. Calcium Channels

The membrane-bound calcium channels channel $Ca^{2+}$ in the cell cytosol from the apoplast space, vacuoles, and ER. The channels are voltage-sensitive and are called depolarization-activated calcium channels (DACCs), hyperpolarization-activated calcium channels (HACCs), and voltage-insensitive calcium channels (VICCs) [84,88,89]. The membrane-bound $K^{+}$ channel [outward-rectifying ($Ca^{2+}$ -permeable) $K^{+}$ channel, KORC] is also considered a calcium-permeable DACC [90]. Calcium channels are activated and perform specific roles to different environmental and developmental signals, such as DACCs, which are activated by stresses such as low temperatures [20,91]; HACCs by pathogen attack, oxidative stresses, cell elongation, and tropism [20,78,92]; and VICCs maintains steady-state cytosolic $Ca^{2+}$ at resting stage of cell [20]. $Ca^{2+}$ channels are also found in the tonoplast and ER membrane, allowing Ca2+ to enter the cytosol. Tonoplast-bound channels include HACC, SV (slow-vacuolar), inositol phosphates (IP3, Inositol-1,4,5-triosephosphate; IP6), and cADPR (cyclic ADP-ribose). Tonoplast IP3 may be involved in turgor regulation, cell elongation, tropism, salt stress, and hyperosmotic stress [20,93–95], and cADPR in cold adaptations, desiccation tolerance, stomatal

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behavior, circadian rhythms, and pathogen attack [4]. The IP3, cADPR, and NAADP receptors are also found in the membrane of the ER [20].

2.3.2. Calcium Efflux Systems

Plant cells tightly regulate cytosolic [$Ca^{2+}$]. Therefore, extra $Ca^{2+}$ is expelled out to the vacuoles, apoplast, ER, and plastid through active transporters such as $Ca^{+}$-ATPases and $H^{+}$/ $Ca^{2+}$-antiporters (CAX) [20] to aid in proper metabolism in cytoplasm, to restore intra- and extracellular $Ca^{2+}$-stores, and to remove divalent cations [96–101]. As $Ca^{2}$-transporters, $Ca^{2+}$-ATPases have high-affinity but low-capacity attributes, and $H^{+}/Ca^{+}$-antiporters are the opposite [102]. $Ca^{2+}$-ATPases are located in organelle membranes such as vacuolar membranes (tonoplast), ER membranes, plastid, and cell [103–109], and ER-type calcium ATPases are found in the Golgi, ER, and endosomes [110–112]. CAX are found in the plasma membrane and the vacuolar membrane (tonoplast) [84,102,113–115]. $Ca^{2+}$ serves as the coordinator for cell wall and cytoplasm communication [116].

## 3. Plant Calcium Uptake by the Root System

The soil $Ca^{2+}$ may not ensure its availability for the plant unless it has a vigorous system to uptake available $Ca^{2+}$. The $Ca^{2+}$ in solution enters with water into the young, un-suberized root. $Ca^{2+}$ generally enters through the root tip where the suberized endodermis (Casparian strip) [117] is absent, and where the suberized endodermis is broken due to new root growth [52,118]. The Casparian strip is a barrier to apoplastic solute movement, while suberization blocks $Ca^{2+}$ transfer in endodermal cells [52,118,119]. Thus, the root is the first gateway of entering $Ca^{2+}$ from the soil solution into the plant system. Intact roots with profuse new growth (volume and size) may exploit more soil volume, which favors higher $Ca^{2+}$ uptake. Impaired root systems due to hard soil, waterlogged conditions, pathogen attacks, damage by insects and animals, and nematode infestation can reduce $Ca^{2+}$ uptake.

## 4. Calcium Uptake Through Foliar Application

Foliar application of $Ca^{2+}$ can increase leaf- and fruit-$Ca^{2+}$ and reduce deficiency disorders. Foliar application of $Ca^{2+}$ strengthens cell walls [120], and tomato leaves absorb 90% of foliar calcium chloride application [121]. Foliar application of $Ca^{2+}$ @ 0.9% increases fruit $Ca^{2+}$ and decreases BER in tomatoes [122]. Foliar application of 150% of the recommended dose of calcium nitrate decreases cabbage tip burn [123]. Tip burn of two mini Chinese cabbages (QYH and HN) disappears due to applications of 4-6 $mmol.L^{-1}$ $Ca^{2+}$ [124]. Foliar spray of eggshell solutions increases the $Ca^{2+}$ content on the aerial part of tomato plants and decreases BER in fruits [125]. Calcium foliar application increases defense mechanisms against diseases [120]. Spraying with $CaCl_2$ or $Ca(NO_3)_2$ controls blackheart, a $Ca^{2+}$ deficiency disorder in celery [126,127]. $Ca^{2+}$-spraying in the form of 'Calciogreen' or '$CaCl_2$' or with other calcium formulations either decreases or effectively controls deficiency disorders, including BER in tomato and bell pepper [128–130]. However, $Ca^{2+}$ has minimal mobility within the phloem [131], and thus foliar $Ca^{2+}$-application may not improve fruit-$Ca^{2+}$ status [132]. Therefore, foliar absorption and $Ca^{2+}$ transport are yet to be clarified [121].

## 5. Calcium Uptake Through the Fruit

One of the leading causes of $Ca^{2+}$ application is to increase fruit $Ca^{2+}$ content to reduce deficiency disorders. Soil or foliar applications, decreasing competition at the root zone, and ameliorating plant and environmental issues are all indirect approaches to increasing fruit $Ca^{2+}$ content. Applying $Ca^{2+}$ directly to the fruit surface can be another approach. However, it is complex to maintain sufficient fruit $Ca^{2+}$ [133]. It is noteworthy to recall a four-decade-old comment about the BER complexity - ''the number of possible interactions that can affect $Ca^{2+}$ uptake and distribution is so great that in the near future, we are unlikely to see the development of cultural practices that will eliminate $Ca^{2+}$ deficiency, without a direct application of $Ca^{2+}$ to the susceptible organ''[134]. $Ca^{2+}$ applied to the apple fruit

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surface may penetrate the fruit epidermis [135,136], preferably at 40-50 days after full bloom [137], probably through trichomes and stomata. Young apple fruitlets take up less exogenous $Ca^{2+}$ than mature fruit [138]; penetration of $Ca^{2+}$ into the fruit depends on the retention time of the solution on the fruit surface and the concentration of the applied solution [137]. Applying 1.33 g calcium-nitrate/polybag decreases BER in tomatoes [139]. $Ca^{2+}$ @100 ppm reduces BER incidence in susceptible tomato accession (*Solanum lycopersicum lycopersicum*) by 5-11% [40]. Bone meal decreases BER in tomato 'Cobra F1' by increasing fruit $Ca^{2+}$ content [140]. $Ca^{2+}$ application improves the quality of cucumber, $Ca^{2+}$ content in leaves and peels in pomegranate, and reduces phenolics and flavonoids in cherries [141–143]. Instead of fruit calcium (pedicel, proximal half, and distal half) content, $Ca^{2+}$/ $K^{+}$ and their relative % in the pedicel are associated with the BER in peppers [144]. BER-resistant peppers express defense responses against calcium-deficient stressors [144]. However, direct $Ca^{2+}$ application can decrease BER symptoms but cannot control the disorder completely; moreover, exogenous $Ca^{2+}$ may leak out easily [145,146]. Detection of BER at early stages may lead to appropriate control measures to minimize postharvest losses, as the success rate of detecting BER by fluorescence and image analysis at this stage is above 86% [147].

## 6. Calcium Translocations

### *6.1. Calcium Translocations Within the Plant*

The soil available $Ca^{2+}$ enters the root apoplast with the water flow and moves through the xylem following either apoplastic or symplastic routes [19,20]. The apoplastic routes consist of cell walls and intercellular spaces, with $Ca^{2+}$ traveling following water potential gradients [52,148], and, for the symplastic pathway – made up of cytoplasm – $Ca^{2+}$ moves through plasmodesmata [52,148]. The apoplastic route is considered the principal route of $Ca^{2+}$ translocation [20,24,148]. However, the Casparian strip along this route restricts further $Ca^{2+}$ movement to the xylem. Therefore, $Ca^{2+}$ enters the root either through the unrubberized endodermis of the root tip [117], or where the endodermis has been broken due to the growth of a new root [24,52,118,148]. Once $Ca^{2+}$ is in the xylem sap, its further movement within the shoot is regulated by the xylem flow of water, xylem water potential [149–151], and cation exchange capacity (CEC) of the xylem cell wall. $Ca^{2+}$ moves mainly with the xylem flow of water from root to shoot [24]. The canopy's transpiration force and plant growth drive the xylem water flow. Xylem water potential depends on dissolved solutes, and xylem cell wall CEC on available binding sites for $Ca^{2+}$ in the xylem cell wall. Along with xylem water flow, $Ca^{2+}$ is transported to the fruits and storage tissues, and this movement is aided by the leaf transpiration strength during the day time and root pressure at night [21].

### *6.2. Leaf or Fruit?*

What determines whether the xylem sap containing $Ca^{2+}$ will move toward the leaf or the fruit? It is the transpiration force and organ growth [149–151]. Transpiration from leaf and fruit surfaces triggers water flow towards them. The leaf, being a strong transpiring organ and a higher mass accumulator than fruit [53,149], results in most of the $Ca^{2+}$ being deposited in the leaf. Fruit sap uptake can be facilitated by either reducing leaf transpiration or enhancing stomatal closure, leading to better $Ca^{2+}$ uptake and thus minimizing BER [24,149,150,152]. $Ca^{2+}$ content does not decrease in the leaves by being phloem-immobile; instead, it may increase due to dehydration during senescence [153,154].

### *6.3. Calcium Translocation Within the Fruit*

Xylem sap $Ca^{2+}$ enters the fruit through the peduncle and is distributed within the fruit based mainly on the xylem network. Being phloem immobile, $Ca^{2+}$ accumulation within the fruit depends on fruit transpiration. Fruit transpiration rate is lower than that of leaves, resulting in a low $Ca^{2+}$ supply to the fruit. High N causes fruit expansion, leading to reduced $Ca^{2+}$ availability to fruit through dilution [33,155], resulting in BER. Though high $[K^{+}]$ and $[Mg^{2+}]$ may replace plasma membrane $Ca^{2+}$,

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they cannot substitute the function of $Ca^{2+}$ in the membrane [156,157], which may also lead to loss of membrane permeability and make the fruit susceptible to $Ca^{2+}$ deficiency disorders. Compared to the total fruit $Ca^{2+}$, the relative $Ca^{2+}$ contents, such as the ratios $N/Ca^{2+}$, $K^{+}/Ca^{2+}$, $Mg^{2+}/Ca^{2+}$, $(K^{+}+Mg^{2+})/Ca^{2+}$, are better predictors of $Ca^{2+}$ deficiency disorders such as BER [158–160]. Fruits can also regulate $Ca^{2+}$ translocation by altering aquaporin activity and cell wall properties [161].

## 7. Calcium Compartmentalization Within the Cell

Partitioning of $Ca^{2+}$ within cellular compartments is the final step of $Ca^{2+}$ regulation. Fruit sensitivity to $Ca^{2+}$ deficiency disorders is triggered by modifying cellular $Ca^{2+}$-partitioning [41,162]. $Ca^{2+}$ compartmentalization is regulated by the capacity of binding $Ca^{2+}$ to the cell wall and the presence of $Ca^{2+}$ channels, ATPases, and exchangers in the membranes of organelles [163]. The cellular plasma membrane is located between the apoplast and cytosol. Apoplastic $Ca^{2+}$ includes water-soluble plasma membrane and cell wall $Ca^{2+}$ [53]. Water-soluble $Ca^{2+}$ stabilizes the plasma membrane by binding phosphate and carboxylate from phospholipids and proteins, respectively, and keeps it functional [48,65]. A certain threshold of water-soluble $Ca^{2+}$ is always maintained in the apoplast to avoid membrane damage and leakiness [65,164] and replacement of $Ca^{2+}$ with other ions can damage the membrane [60,165]. Cell wall $Ca^{2+}$ binds with the pectin matrix to obtain the rigidity of the cell wall. Newly synthesized pectic polysaccharides are highly methyl-esterified. Removal of a methyl group by pectin methyl esterases (PMEs) creates a carboxylate group with which $Ca^{2+}$ binds strongly [57,166].

The $Ca^{2+}$ concentration of organelles varies greatly, and the cell maintains a certain $Ca^{2+}$ threshold level for its function. The vacuole is the biggest store of $Ca^{2+}$, maintaining 1-10 mM $Ca^{2+}$ [20,167]. Other $Ca^{2+}$ storage sites are the ER (1-5 mM) [168], chloroplast (0.1-10 μM) [169,170], mitochondria (0.2-1.2 μM) [169,170], nucleus (0.1-0.2 μM) [171], and cytosol (100-200 nM) [80] (Figure 1).

## 8. Calcium Deficiency Disorders

$Ca^{2+}$ deficiency in crop plants can cause numerous disorders that are responsible for significant crop losses. An economically crucial $Ca^{2+}$-related disorder is BER. Symptoms of BER include the development of dry, brown/black, sunken spots at the blossom end of fruits, leading to rotting that may cover a significant part of fruits in peppers, tomatoes, watermelon, eggplant, and squash [20,22,24,172] (Figure 3). $Ca^{2+}$ deficiency leads to physiological disorders in tomatoes, peppers, apples, and watermelons [173,174]. $Ca^{2+}$ deficiency causes cell death in the apical meristem [175], and weakens the cell wall, leading to disease and pest susceptibility [176].

Tip burn is another relevant physiological disorder. It is characterized by necrosis of rapidly growing young leaves in cabbage, Chinese cabbage, Brussels sprouts, lettuce, chervil, chicory, escarole, onion, fennel, and potatoes [23,25,177–180] (Table 1). Other disorders include bitter pit – the development of brown/black depressed spots on the blossom end of fruit – in apples [20,134,158]; blackheart – collapsing of young leaf tissue that turned black, usually at the center (heart) of the plant – in celery [126,181]; brown heart – necrosis of the tip of young leaves that cover the entire leaf later – in leafy vegetables [20]; empty pod – poor or no development of seed kernel results in empty pod/shell – in peanut [20]; and fruit cracking – splitting of skin or cuticle – in apple, tomatoes, and cherry [20] (Table 1). Besides deficiency, $Ca^{2+}$-toxicity is reported in crop plants such as gold spot/yellowish flecks – tiny flecks develop around the calyx and shoulder of fruit – in tomato [184], and $Ca^{2+}$-toxicity halted germination and growth of vegetables [23].

 doi:10.20944/preprints202506.0305.v1



**Figure 3.** Blossom-end rot (BER) in tomato and bell pepper fruits. Blossom-end rot affects the distal end of the fruit and occurs during the first few weeks of fruit growth. BER is considered a calcium deficiency disorder that other environmental conditions can exacerbate. In advanced stages, dry, sunken, black/brown symptoms appear that can cover the entire blossom part of the fruit. Upper panel: BER in tomato; middle panel: development of BER in bell pepper while in the plant; and bottom panel: different stages of development of BER symptoms in bell pepper (from left to right: very low, low, moderate, high, and very high BER). Photos are from the first author's experiments conducted in Athens, GA, USA, from December 2015 to April 2018.

Tip burn is another relevant physiological disorder. It is characterized by necrosis of rapidly growing young leaves in cabbage, Chinese cabbage, Brussels sprouts, lettuce, chervil, chicory, escarole, onion, fennel, and potatoes [23,25,177–180] (Table 1). Other disorders include bitter pit – the development of brown/black depressed spots on the blossom end of fruit – in apples [20,134,158]; blackheart – collapsing of young leaf tissue that turned black, usually at the center (heart) of the plant – in celery [126,181]; brown heart – necrosis of the tip of young leaves that cover the entire leaf later – in leafy vegetables [20]; empty pod – poor or no development of seed kernel results in empty pod/shell – in peanut [20]; and fruit cracking – splitting of skin or cuticle – in apple, tomatoes, and cherry [20] (Table 1). Besides deficiency, $Ca^{2+}$-toxicity is reported in crop plants such as gold spot/yellowish flecks – tiny flecks develop around the calyx and shoulder of fruit – in tomato [184], and $Ca^{2+}$-toxicity halted germination and growth of vegetables [23].

**Table 1.** Calcium deficiency disorders of crops.

| Deficiency symptoms | Crops | Description | Reference |
|---|---|---|---|
| Blossom-end rot | Bell pepper, tomato, watermelon, | Blossom-end rot in fruit and vegetables develops dry, brown/black, sunken spots, | [20,22,24,172,182,183] |

 doi:10.20944/preprints202506.0305.v1



| | | | |
|---|---|---|---|
| | eggplant, Squash | leading to rotting that may cover most of the fruit. | |
| Blackheart | Celery | Young leaf tissue collapsed and turned black, usually at the center (heart) of celery. | [126,181] |
| Bitter pit | Apple | Development of brown/black depressed spots on the fruits. | [20,134,158] |
| Empty pod | Peanut | Poor or no development of the seed kernel results in an empty pod/shell of the peanut | [20] |
| Tip burn | Cabbage, Chinese cabbage, other cabbages | The tips of rapidly growing young leaves become necrotic | [23,25,178] |
| | Brussels sprouts, lettuce | Necrosis of the tip of rapidly growing young leaves | [23,179,180] |
| | Chervil | Tip of rapidly growing young leaves become necrotic | [23,177] |
| | Chicory, escarole, onion, fennel, potatoes | Necrosis of tip of rapidly growing young leaves | [23] |
| Brown heart | Leafy vegetables | Necrosis of tip of young leaves that cover the entire leaf later | [20] |
| Fruit cracking | Tomato, cherry, apple | Splitting of skin or cuticle | [20] |

*8.1. Genesis of Blossom-End Rot Development*

BER is a costly $Ca^{2+}$ deficiency disorder that decreases the yield and quality of the produce, leading to significant economic losses. The $Ca^{2+}$ content of the Earth's crust is 3.64%, higher than most minerals [185]. The inadequacy of soil $Ca^{2+}$ for plant growth is rare [1]. Soil $Ca^{2+}$ is found in bound, exchangeable, and soluble forms. Bound $Ca^{2+}$ occurs in the form of $Ca^{2+}$-minerals, e.g., calcite, dolomite, and apatite [1,185], and is not readily available to the plant. Exchangeable $Ca^{2+}$ remains bound with the soil in negative sites (cation exchange capacity, CEC). It can be available to the plants based on soil pH and the presence of other competing cations. The soil solution $Ca^{2+}$ is readily available for the plants to be taken up. Soil solution $Ca^{2+}$ depends on the weathering of parent rock material, the mineralization of primary minerals and soil organic matter, soil pH, fertilization, and diffusion along the gradient [2]. Just the presence of $Ca^{2+}$ in the soil may not ensure its availability to the plant. $Ca^{2+}$ depends on water availability, the competition of $Ca^{2+}$ with other cations, medium pH, salinity, root growth, anoxia, root zone temperature, and root damage by pathogens, insects, and nematodes [186]. $Ca^{2+}$ can be supplied to the plants by applying $Ca^{2+}$ in the soil and on leaf and fruit surfaces.

Cations decrease while anions increase $Ca^{2+}$ uptake by the plants. The presence of cations such as $K^+$, manganese ($Mn^{2+}$), magnesium ($Mg^{2+}$), ammonium ($NH_4^+$), $Al^{3+}$, and $Na^+$ antagonize [24,34,187], and anions such as nitrate ($NO_3^-$) and phosphate ($PO_4^{3-}$) synergize $Ca^{2+}$ uptake by the plant root system. Although soil contains about 10 times more $Ca^{2+}$ than $K^+$, the uptake of $Ca^{2+}$ is lower than that of $K^+$ [65], which might be due to the higher valency of $Ca^{2+}$ [15]. $NH_4^+$ competes with $Ca^{2+}$ to be taken up by the plants. Moreover, high N fertilization promotes shoot growth, which diverts absorbed $Ca^{2+}$ to the leaf instead of the fruit because of the higher leaf transpiration rate than the fruit [53,188]. At high soil calcium availability, fruit $Ca^{2+}$-deficiency disorder may not appear. However, at

 doi:10.20944/preprints202506.0305.v1



a low calcium availability, BER can appear due to depletion of apoplastic $Ca^{2+}$ content. BER may also occur at high calcium availability due to improper $Ca^{2+}$ compartmentalization (Figure 4).

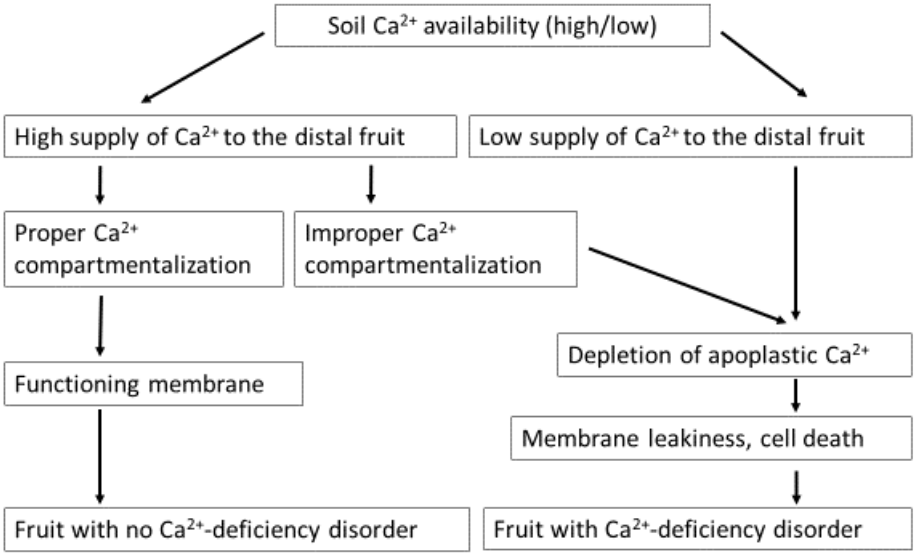


**Figure 4.** Calcium availability affects the development of calcium deficiency disorder blossom-end rot (BER). At high soil calcium availability, fruit $Ca^{2+}$-deficiency disorder may not appear. However, at low calcium availability, deficiency disorder appears through depletion of apoplastic calcium and subsequent membrane leakiness. However, BER may occur at high calcium availability due to improper calcium compartmentalization.

*8.2. Incidence of BER Based on Variety, Season, and Truss*

The incidence of BER may vary from variety to variety, as reported from cultivated peppers and tomatoes. $Ca^{2+}$ deficiency, differential fruit growth rate, and variation in xylem development are considered the basis of this variability [189]. $Ca^{2+}$-efficient cultivars absorbed $Ca^{2+}$ more efficiently than $Ca^{2+}$-inefficient cultivars when the availability of $Ca^{2+}$ is low on the substrate. Thus, $Ca^{2+}$-efficient cultivars could be selected against BER, but the difficulty is that they yield poorly. However, no significant difference in BER susceptibility has been found between efficient and inefficient varieties [189]. Tomato varieties 'Calypso' and 'Spectra' showed higher incidences of BER than 'Counter' [190]; 'Petomech II' higher than 'IPA-L' [191]; 'Celebrity' higher than 'Rutgers,' 'Mountain Pride', and 'Mountain Spring' [192]; STEP 158 (breeding line) higher than 'Rutgers' and 'Doublerich' [193], and 'Boludo' higher than 'Daniela' [194]. Elongated tomato varieties are more susceptible to BER [195]. BER never occurs in small-fruit and wild tomato cultivars [53]. Lack or excess of minerals may cause deficiency disorders [196,197]. A low fruit apoplastic calcium, which results in leaky cell membranes, leads to BER development [198]. BER occurs in tomatoes if the calcium concentrations of the lateral ends of green fruits are < 0.2 μmol $g^{-1}$ [199]. The fruit growth rate of the BER-susceptible tomato accession (*Solanum lycopersicum lycopersicum*) is higher than the BER-resistant one (*Solanum lycopersicum cerasiforme*), however, it is not clear whether this may contribute to BER development [186]. Phytohormones may also affect BER incidence. Foliar spray of ABA reduces BER incidence by increasing calcium availability [38,199], and gibberellins increase it through increasing oxidative stresses in plants [200]. ABA increases, and GA decreases calcium accumulation [144]. Thus, applying ABA or GA inhibitors (paclobutrazol and prohexadione-$Ca^{2+}$) may decrease BER in peppers [144]. Transport and homeostasis of calcium ions are crucial for preventing BER in peppers [144].

 doi:10.20944/preprints202506.0305.v1



Pepper varieties with larger final fruit sizes and faster growth rates, such as 'Marconi' and 'J27', had higher sensitivity to BER than 'Jerid', which produces a smaller final fruit size [202]. No incidence of BER has been reported from wild-type tomatoes (small fruit size). This observation indicates that BER might be associated with larger fruits under favorable growth conditions that influence rapid fruit growth. 'Marmande' tomatoes had a higher BER incidence than cherry, cocktail, or round tomatoes [203]. Pygmy fruits, having no rapid growth phase, usually do not develop BER [204]. BER-affected tomatoes ripen earlier and are smaller than healthy fruit [205].

The onset of BER can vary based on trusses and seasons. Frequent incidence of BER was observed with the first truss [36] followed by a subsequent decrease [31,206,207], or increase [207,208]. Basal fruits of a truss had more severe BER than the others [28]. Based on seasons, BER incidence can increase or decrease from the first to the upper trusses [151,190].

## 9. Control of BER

BER, a critical physiological disorder for several vegetables, has been studied for over a century, although the mechanism is unclear. Most researchers agree that this condition is a $Ca^{2+}$-deficiency disorder, and supplying sufficient $Ca^{2+}$ to the fruit may prevent the symptom development. However, the $Ca^{2+}$ route from soil to the target organ, i.e., fruit, is not straightforward. Many factors are in action on the journey of $Ca^{2+}$ from the soil to the fruit. Moreover, it is not the effect of a single factor, rather a combined effect of one or more factors [24], such as low soluble soil $Ca^{2+}$, high $Mg^{2+}$, $NH^{4+}$, and $K^{+}$ concentrations, high salinity, inconsistent soil moisture (high, low or fluctuating), rapid fruit growth rate, poor xylem network towards the blossom end of the fruit, high temperature, and high or low transpiration of the target organ [24]. Cultural management approaches should favor $Ca^{2+}$ translocation to the fruit to control BER. However, the transport of $Ca^{2+}$ to the fruit may not ensure BER control, as cellular $Ca^{2+}$ partitioning is the final and most crucial control level for this disorder [163]. Reports show severe BER incidence in the distal part of the fruit despite high $Ca^{2+}$-concentration [211]. Moreover, no BER symptoms when the $Ca^{2+}$-supply was low in the case of slow-growing plants [212] (Figure 3). The relatively high $Ca^{2+}$ concentration in the BER-affected fruit might be explained through abnormal cellular partitioning, such as transport of abundant $Ca^{2+}$ to the vacuoles, which may cause $Ca^{2+}$ deficiency in other sub-cellular organelles and may develop BER. Moreover, an unanticipated change in cellular $Ca^{2+}$ concentration response to environmental stimuli or hormonal effects may cause localized $Ca^{2+}$ deficiency, leading to BER development [163].

BER occurs due to abiotic stresses, high temperature, drought, salinity, water logging, higher transpiration, production of ROS, and low availability of ascorbic acid [195,214]. Control of BER is complicated due to the involvement of many changing and unpredictable abiotic factors, and proper management can only reduce the incidence rate [36]. Spraying calcium on the fruit during the developmental stage reduces BER incidence [53]. Spraying should be started at the early stage of the fruit and, of course, before the onset of BER, and it needs to be continued for the entire development stage. However, spraying may not be effective [195]. Balanced fertilizer and avoiding vigorous foliage growth may help reduce BER [195]. Shade net may also reduce BER incidence [215–219]. However, the incidence of BER in tomatoes is positively correlated with fruit plant$^{-1}$, K content, root relative water content, firmness, vitamin C, titratable acidity, and peroxidase, and negatively correlated with calcium content [39]. A high calcium concentration (e.g., 20 mmol.L$^{-1}$) decreases growth [plant height, diameter, biomass production (leaf, stem, root, and total)], physiological attributes [photosynthesis, stomatal conductance, transpiration, and chlorophyll content (chlorophyll a; chlorophyll b)], enzyme levels (superoxide dismutase, catalase, and peroxidase), and water use efficiency in poplar seedlings [49]. Therefore, correctly understanding calcium nutrition helps reduce crop cultivation costs, minimize environmental pollution, and boost crop production [220].

Cultural and physiological crop management that ensures $Ca^{2+}$ transportation to the fruit and appropriate cellular distribution may reduce the incidence of BER. The selection of BER-resistant and $Ca^{2+}$-efficient cultivars may also help to reduce BER incidence. Controlling or skipping calcium deficiency disorders may include modifications of watering, light, temperature, transpiration, and

 doi:10.20944/preprints202506.0305.v1



application of mulching and growth regulators. Using resistant varieties and customization of planting and harvesting (early planting and harvesting) may help skip calcium deficiency disorders [23]. Understanding the molecular mechanisms of BER may help in better managing the disorder [221].With the combination of appropriate management practices and BER-resistant cultivars, the incidence of this disorder may be eliminated in the future [53].

## 10. Conclusions

Calcium is a crucial element for plant growth and development. Though soils worldwide are not typically deficient in $Ca^{2+}$, $Ca^{2+}$-deficiency disorders in crops are numerous and cause substantial yield loss. Among the $Ca^{2+}$-deficiency disorders, BER is widely prevalent worldwide. The complex route of $Ca^{2+}$ from soil to the appropriate cellular compartments, such as the cytosol depends on multiple factors, e.g., soil (moisture availability, the competition of $Ca^{2+}$ with other cations, pH, anoxia, and salinity), plant [genotypes, growth habit (dwarf, tall), xylem network, root and shoot growth, yield, root damaged by pathogens, insects, and nematodes], and environment [temperature (air and root zone), relative humidity, vapor pressure deficit, and transpiration] that renders it difficult to control BER. Moreover, the cellular $Ca^{2+}$ compartmentalization, particularly in the vacuoles, depletes cytosolic $Ca^{2+}$ levels and may disintegrate the plasma membrane, leading to BER development despite having high $Ca^{2+}$ content in the blossom end of the fruit. Therefore, possible ways of minimizing and controlling BER include i) an integrated approach that ameliorates soil, plant, and environmental factors towards supplying sufficient $Ca^{2+}$ into the cells; ii) appropriate cultural and physiological management of crops; iii) a favorable environment; and iv) BER-resistant and $Ca^{2+}$-efficient cultivars. Other approaches that include agronomic, physiological, breeding, and molecular methods may also contribute to minimizing BER occurrence.

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