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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 macronutrient essential for the growth, development, yield, and quality of vegetables and fruits. It performs structural, enzymatic, and signaling functions in plants. This review examines Ca^{2+} translocation from soil to the fruit via the plant xylem network, emphasizing the importance of Ca^{2+} compartmentalization within fruit cell organelles in the development of calcium deficiency disorders such as blossom-end rot (BER). The underlying causes of BER and potential control measures are also discussed. Soil-available Ca^{2+}, transported by water flow, enters the root apoplast through membrane channels and moves toward the xylem via apoplastic or symplastic routes. The transpiration force and the growth of organs determine the movement of Ca^{2+}-containing xylem sap to aerial plant parts, including fruits. At the fruit level, the final step of Ca^{2+} regulation is intracellular partitioning among organelles and cellular compartments. This distribution ultimately determines the fruit's susceptibility to Ca^{2+}-deficiency disorders such as BER. Excessive sequestration of Ca^{2+} into organelles such as vacuoles may deplete cytosolic and apoplastic Ca^{2+} pools, compromising membrane integrity and leading to BER, even when overall Ca^{2+} levels are adequate at the blossom end. Effective BER management requires cultural and physiological practices that promote Ca^{2+} uptake, translocation to the fruit, and appropriate intracellular distribution. Additionally, the use of BER-resistant and Ca^{2+}-efficient cultivars can help mitigate this disorder. Therefore, a comprehensive understanding of Ca^{2+} dynamics in plants is critical for managing BER, minimizing production loss and environmental impacts, and maximizing overall crop productivity.

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

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1. Introduction

Calcium (Ca^{2+}) as a macronutrient is essential for plant growth and development [1–3] and is found in higher quantities (0.5%) than any other divalent inorganic cations [4]. When hydrated, Ca^{2+} is a big cation with a 41.2 Å ionic radius [5]. Among the macronutrients, Ca^{2+} is third after nitrogen (N) and potassium (K) in terms of quantity in plant tissue, which reflects its indispensability for the plant. The plant requires 1–3 mM Ca^{2+} for proper growth and development [6]. It plays roles in membrane and cell wall stabilization, cell function, signal transduction, growth and development, gene expression, and stress resistance [7–15]. Ca^{2+} concentration varies from 10^{-7} M to 10^{-3} M across the cell organelles (Figure 1). Though the 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 [16,17]. 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 [18,19], 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 [11]. The proportion of Ca^{2+} in specific tissues can be more than 10% without affecting plant growth and development [4].

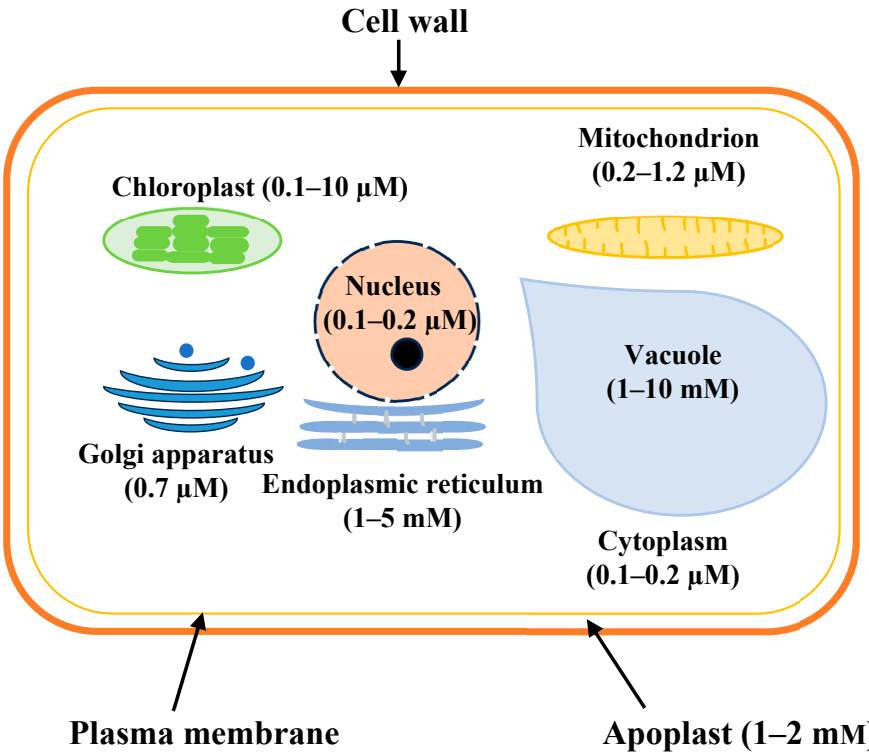


Figure 1. Calcium compartmentalization within a plant cell. Calcium (Ca^{2+}) is an essential macronutrient involved in diverse structural, regulatory, and signaling processes within plant cells. Its structural role is most evident in the cell wall, where Ca^{2+} cross-links pectic polysaccharides to maintain cell wall stability, and in the plasma membrane, where it contributes to membrane integrity and modulates permeability. In the cytosol, calcium functions as a fundamental second messenger, mediating intracellular signaling cascades in response to both biotic and abiotic stimuli. Although transient increases in cytosolic Ca^{2+} concentrations are critical for signaling, the bulk of cellular calcium is sequestered in the vacuole. This compartment serves as the principal intracellular calcium reservoir, contributing to the regulation of ion homeostasis and buffering cytoplasmic pH. The tight spatial and temporal regulation of calcium distribution is essential for maintaining cellular function and avoiding calcium toxicity, given the cytotoxic potential of sustained elevated cytosolic Ca^{2+} levels.

The available Ca^{2+}, carried by water flow, enters the root apoplast through membrane channels [20], and moves toward the xylem, following either apoplastic or symplastic routes [21]. 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 [22]. 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) [21,23–27].

Among numerous Ca^{2+}-deficiency disorders, BER is most prevalent and causes up to 50% economic loss in extreme conditions [25]. BER often initiates at the fruit blossom part (away from the peduncle) during early fruit growth stages (2–3 weeks following anthesis) [28–38]. 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 [39]. BER incidence is positively correlated with root 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 [40,41].

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 [24]. By being phloem-immobile, Ca^{2+} cannot be translocated from the available sources (mature leaves and the peduncle end of the fruit) to the deficient sinks (young-growing leaves and the blossom end of the 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* [42–46], and calreticulin (*CRT*) [45,47] may play a role in this regard [48]. 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 [24]. Although calcium's dual role as a nutrient and signal transducer is well established, significant knowledge gaps persist. The molecular identity and regulation of many calcium channels, pumps, and sensors remain uncharacterized [49]. Moreover, the interconnectedness of calcium's roles in nutrient uptake versus signaling pathways is poorly understood. It remains unclear whether distinct or overlapping sets of genes mediate these functions, such as those involved in calcium uptake in roots versus those regulating signal transduction in leaves. Therefore, the present study discusses the route of calcium translocation within plants, the causes and control of BER, and recent advances and future directions in calcium research.

2. Function of Calcium in Plants

The roles of Ca^{2+} in cellular integrity and growth are well documented [50,51].

2.1. Structural Role of Calcium

Calcium maintains cell wall integrity [11,52], cell division and elongation [53–55], cell expansion [55], membrane permeability and membrane stability [52,56], and assembly of microtubules [57].

The plant cell wall contains carbohydrates (cellulose, hemicellulose, pectin), proteins, particularly structural ones, and lignin (secondary cell wall) [58]. Cell wall Ca^{2+} mainly represents Ca^{2+} bindings in the middle lamella that glue adjacent cells and maintain cell wall integrity [59]. 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 α(1–4) linkage. Ca^{2+} forms a tight linkage between the charged carboxyl (COO^-) group of galacturonic acid [4], 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+} [4] 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 [60]. The PG activity is increased in Ca^{2+}-deficient tissue, leading to middle lamella disintegration, primary cell wall degradation, and cell

death [61]. 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 [62]. Demethylation of pectin by PME action favors the further degradation of pectin by enzymes like endo-polygalacturonase, exo-polygalacturonase, β-galactosidases, and pectate lyases [63,64].

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 proper membrane selective permeability. The requirement of Ca^{2+} increases due to an increase in heavy metals [65], aluminum (Al^{3+}), sodium (Na^{+}) [66], 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 increases substantially to ensure optimum plant growth and development [67]. Replacement of Ca^{2+} with Na^{+}, heavy metals, or Al^{3+} can cause salinity, heavy metal, or aluminum toxicity, respectively [68,69]. Membrane instability is prevalent under freezing, low temperature, and anaerobiosis [5]. 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+}) into 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.

Ca^{2+} stabilizes the cell wall by binding with pectin, the cell membrane, and the proteins and lipids at the membrane surfaces [50,70]. Ca^{2+} influences vesicles—full of materials and enzymes for cell wall and membrane construction—in their incorporation into the plasma membrane [55]. 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 [5,12].

2.2. Enzymatic Role of Calcium

Ca^{2+} can promote or demote enzyme activity essential for cell growth and development. The activity of α-amylase is stimulated by high Ca^{2+} concentration during starch breakdown in germinating cereal seeds, in which the Ca^{2+} ion stabilizes amylase [71]. However, high Ca^{2+} concentrations may inhibit enzyme activity [24], as has been shown with cytosolic enzyme fructose-1,6-bisphosphatase (FBPase) (Figure 2), which regulates sucrose synthesis from triosephosphate (TP) in the cytosol. 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 [72].

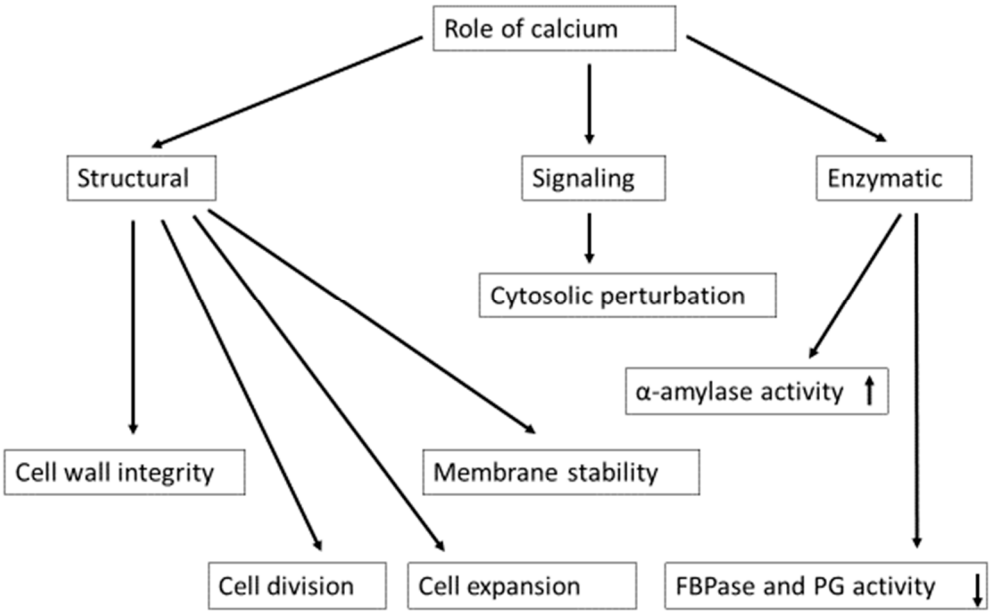


Figure 2. Calcium plays structural, enzymatic, and signaling functions. Alpha-amylase works during seed germination; fructose 1,6 bisphosphatase (FBPase) is a regulatory enzyme in the sucrose biosynthesis pathway; polygalacturonase (PG) hydrolyzes the alpha-1,4 glycosidic bonds between galacturonic acid residues of pectin. An upward arrow (↑) indicates an increase, and a downward arrow (↓) shows a decrease in enzyme activity.

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 [5]. It plays a crucial role as a signaling molecule in signaling pathways [73]. Ca^{2+} acts as a universal signaling molecule [74], 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 [74]. Understanding Ca^{2+} dynamics may help develop and engineer climate-smart crop varieties [74]. 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 [75–78]. It also contributes to immunity by activating immune responses [79]. 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 [80]. It will improve understanding of how specific stress signals modulate Ca^{2+} homeostasis to orchestrate adaptive responses [80].

In response to stimuli, Ca^{2+} transduces signals to the other end upon binding with calmodulin, a calcium protein in the cytosol [13,81–83] (Figure 3). Plants maintain a very low (100 to 200 nM) cytosolic [Ca^{2+}], which can rise to 2 µM in the stimulated state [84]. Plants maintain very low cytosolic [Ca^{2+}] to serve as a messenger, prevent precipitation of inorganic phosphate, and minimize competition for binding sites with magnesium [5]. The role of Ca^{2+} as a messenger is possibly due to this low cytosolic [Ca^{2+}] concentration and its chemistry [21]. Any signal—changes in light intensity, 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 [21,82,85]. Additionally, 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 [86]. This signaling is subject to strict 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.

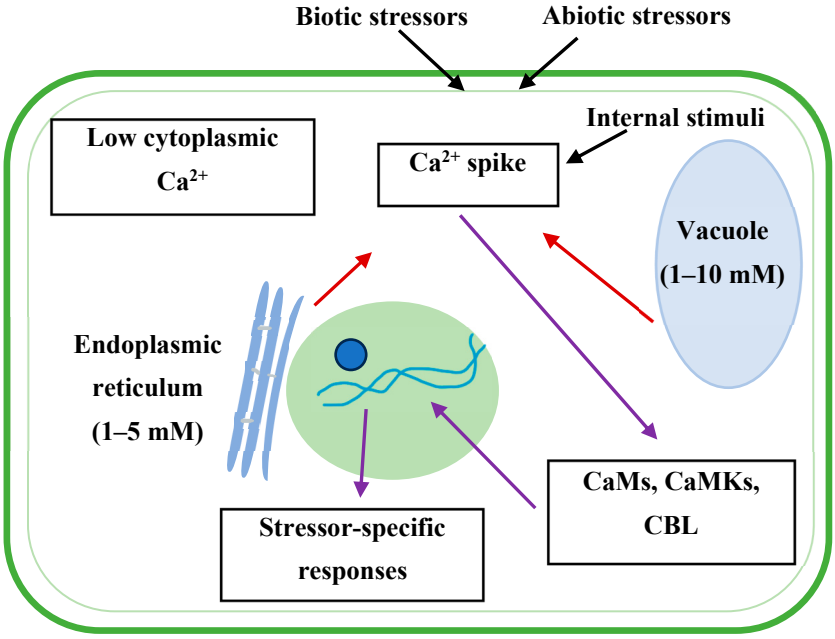


Figure 3. Though cytoplasm maintains a low state of Ca^{2+}, the cytoplastic Ca^{2+} spikes due to the release of more Ca^{2+} principally from the vacuoles and endoplasmic reticulum—the large storage of Ca^{2+} (dark red arrows). Ca^{2+} binds with calmodulins (CaMs), Ca^{2+}/calmodulin-dependent protein kinases (CaMKs), or calcineurin B-like (CBL) proteins, which cause stressor-specific responses through binding to the transcription factors (purple arrows).

2.3.1. Calcium Proteins

Changes in cytosolic [Ca^{2+}] are detected by specific proteins that either relay or respond to 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 [87,88]. 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 [89,90]. 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 [5]. Plant annexins are associated with cell elongation, membrane repair, the secretory process, salinity, and drought stresses [91]. 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 [5].

2.3.2. Calcium Channels

The membrane-bound calcium channels move Ca^{2+} into 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) [88,92,93]. The membrane-bound K^+ channel [outward-rectifying (Ca^{2+}-permeable) K^+ channel, KORC] is also considered a calcium-permeable DACC [94]. Calcium channels are activated and per-

form specific roles to different environmental and developmental signals, such as DACCs, which are activated by stresses such as low temperatures [21,95]; HACCs by pathogen attack, oxidative stresses, cell elongation, and tropism [21,82,96]; and VICCs maintains steady-state cytosolic Ca^{2+} at the cells' resting stage [21]. Ca^{2+} channels are also found in the tonoplast and ER membrane, allowing Ca^{2+} 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 [21,97–99], and cADPR in cold adaptation, desiccation tolerance, stomatal behavior, circadian rhythms, and pathogen attack [5]. The IP3, cADPR, and NAADP receptors are also found in the membrane of the ER [21].

2.3.3. Calcium Efflux Systems

Plant cells tightly regulate cytosolic [Ca^{2+}]. Therefore, excess Ca^{2+} is expelled out to the vacuoles, apoplast, ER, and plastids through active transporters such as Ca^{+}-ATPases and H^{+}/ Ca^{2+}-antiporters (CAX) [21] to aid proper metabolism in the cytoplasm, to restore intra- and extracellular Ca^{2+}-stores, and to remove divalent cations [100–105]. As Ca^{2+} transporters, Ca^{2+}-ATPases have high-affinity but low-capacity attributes, and H^{+}/Ca^{+}-antiporters are the opposite [106]. Ca^{2+}-ATPases are located in organelle membranes such as vacuolar membranes (tonoplast), ER membranes, plastids, and cell [107–113], and ER-type calcium ATPases are found in the Golgi, ER, and endosomes [114–116]. CAX proteins are found in the plasma membrane and the vacuolar membrane (tonoplast) [88,106,117–119]. Ca^{2+} serves as the coordinator for cell wall and cytoplasm communication [120].

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 take up available Ca^{2+}. Ca^{2+} enters the root apoplast of young, unsuberized roots through the membrane channels. Ca^{2+} generally enters through the root tip, where the suberized endodermis (Casparian strip) [121] is absent and where the suberized endodermis is broken due to new root growth [56,122]. The Casparian strip is a barrier to apoplastic solute movement, while suberization blocks Ca^{2+} transfer in endodermal cells [56,122,123]. Thus, the root is the first gateway for Ca^{2+} entry 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 [124], and tomato leaves absorb 90% of foliar calcium chloride application [125]. Foliar application of Ca^{2+} at 0.9% increases fruit Ca^{2+} and decreases BER in tomatoes [126]. Foliar application of 150% of the recommended dose of calcium nitrate decreases cabbage tip burn [127]. Tip burn of two mini Chinese cabbages (QYH and HN) disappears due to the applications of 4–6 $mmol \cdot L^{-1}$ Ca^{2+} [128]. Foliar spray of eggshell solutions increases the Ca^{2+} content on the aerial part of tomato plants and decreases BER in fruits [129]. Calcium foliar application increases defense mechanisms against diseases [124]. Spraying with $CaCl_2$ or $Ca(NO_3)_2$ controls blackheart, a Ca^{2+} deficiency disorder in celery [130,131]. 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 [132–134]. However,

Ca^{2+} has minimal mobility within the phloem [135], and thus foliar Ca^{2+}-application may not improve fruit-Ca^{2+} status [136]. Therefore, foliar absorption and Ca^{2+} transport are yet to be clarified [125].

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+} [137]. 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" [138]. Ca^{2+} applied to the apple fruit surface may penetrate the fruit epidermis [139,140], preferably at 40–50 days after full bloom [141], probably through trichomes and stomata. Young apple fruitlets take up less exogenous Ca^{2+} than mature fruit [142]; 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 [141]. Applying 1.33 g calcium nitrate/polybag decreases BER in tomatoes [143]. Ca^{2+} at 100 mg L^{-1} reduces BER incidence in susceptible tomato accession (*Solanum lycopersicum lycopersicum*) by 5–11% [41]. Bone meal decreases BER in tomato 'Cobra F1' by increasing fruit Ca^{2+} content [144]. Ca^{2+} application improves the quality of cucumber, Ca^{2+} content in leaves and peels in pomegranate, and reduces phenolics and flavonoids in cherries [145–148]. 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 [148]. BER-resistant peppers express defense responses against calcium-deficient stressors [148]. However, direct Ca^{2+} application can decrease BER symptoms but cannot control the disorder completely; moreover, exogenous Ca^{2+} may leak out easily [149,150]. 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% [151].

6. Calcium Translocations

6.1. Calcium Translocations Within the Plant

The solution Ca^{2+} enters the root apoplast through membrane channels and moves through the xylem following apoplastic or symplastic routes [20,21]. In the apoplastic route, Ca^{2+} travels following water potential gradients, and in the symplastic route, Ca^{2+} moves through plasmodesmata [54,152]. Though the apoplastic route is considered the principal pathway of Ca^{2+} translocation, there are debates about it [152,153]. The Casparian strip along this route restricts further Ca^{2+} movement to the xylem. Once Ca^{2+} is in the xylem sap, its further movement within the shoot is directed by the xylem flow of water, xylem water potential [154–156], and cation exchange capacity (CEC) of the xylem cell wall. The canopy's transpiration force and plant growth drive the xylem water flow. Along the flow of xylem water, Ca^{2+} is transported to the fruits and storage tissues.

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 [154–156]. Transpiration from leaf and fruit surfaces triggers water flow toward them. The leaf, being a strong transpiring organ and a higher mass accumulator than fruit [55,154], 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 [25,154,155,157]. Ca^{2+} content does not decrease in the leaves by being phloem-immobile; instead, it may increase due to dehydration during senescence [158,159].

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. However, phloem may contribute to the transport of Ca^{2+} as it contains higher Ca^{2+} in the pedicel, and xylem functionality is lost at maturity [160]. High N causes fruit expansion, leading to reduced Ca^{2+} availability to fruit through dilution [34,161], resulting in BER. Though high $[K^+]$ and $[Mg^{2+}]$ may replace plasma membrane Ca^{2+}, they cannot substitute the function of Ca^{2+} in the membrane [162,163], 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 [164–166]. Fruits can also regulate Ca^{2+} translocation by altering aquaporin activity and cell wall properties [153].

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 [42,167]. 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 [168]. The cellular plasma membrane is located between the apoplast and cytosol. Apoplastic Ca^{2+} includes water-soluble plasma membrane and cell wall Ca^{2+} [55]. Water-soluble Ca^{2+} stabilizes the plasma membrane by binding phosphate and carboxylate from phospholipids and proteins, respectively, and keeps it functional [50,69]. A certain threshold of water-soluble Ca^{2+} is always maintained in the apoplast to avoid membrane damage and leakiness [69,169], and replacement of Ca^{2+} with other ions can damage the membrane [64,170]. 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 [60,171].

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+} [21,172]. Other Ca^{2+} storage sites are the ER (1–5 mM) [173], chloroplast (0.1–10 µM) [174,175], mitochondria (0.2–1.2 µM) [174,175], Golgi apparatus (0.7 µM), nucleus (0.1–0.2 µM) [176], and cytosol (100–200 nM) [84] (Figure 1).

8. Calcium Deficiency Disorders

Ca^{2+} deficiency leads to physiological disorders like BER in tomatoes, peppers, watermelon, eggplant, squash, and apples [177–179] (Figure 4). It weakens cell walls, leading to disease and pest susceptibility and cell death [180,181]. 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 [24,26,182–185] (Table 1). Other disorders include bitter pit—the development of brown/black depressed spots on the blossom end of fruit—in apples [21,138,164]; blackheart—collapsing of young leaf tissue that turns black, usually at the center (heart) of

the plant—in celery [130,186]; brown heart—necrosis of the tip of young leaves that covers the entire leaf successively—in leafy vegetables [21]; empty pod—poor or no development of seed kernel resulting in an empty pod/shell—in peanut [21]; and fruit cracking—splitting of skin or cuticle—in apple, tomatoes, and cherry [21] (Table 1).



Figure 4. 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 on 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.

Table 1. Calcium deficiency disorders of crops.

Deficiency Symptoms	Crops	Description	Reference
Blossom-end rot	Bell pepper, tomato, watermelon, eggplant, squash	Blossom-end rot in fruit and vegetables develops dry, brown/black, sunken spots, leading to rotting that may cover most of the fruit.	[21,23,25,177,187,188]
Blackheart	Celery	Young leaf tissue collapses and turns black, usually at the center (heart) of celery.	[132,186]

Table 1. *Cont.*

Deficiency Symptoms	Crops	Description	Reference
Bitter pit	Apple	Development of brown/black depressed spots on the fruits.	[21,138,164]
Empty pod	Peanut	Poor or no development of the seed kernel, resulting in the empty pod/shell of the peanut.	[21]
Tip burn	Cabbage, Chinese cabbage, other cabbages	The tip of rapidly growing young leaves becomes necrotic.	[24,26,183]
	Brussels sprouts, lettuce	Necrosis of the tip of rapidly growing young leaves.	[24,184,185]
	Chervil	The tip of rapidly growing young leaves becomes necrotic.	[24,182]
	Chicory, escarole, onion, fennel, potatoes	Necrosis of the tip of rapidly growing young leaves.	[24]
Brown heart	Leafy vegetables	Necrosis of the tip of young leaves that covers the entire leaf successively.	[21]
Fruit cracking	Tomato, cherry, apple	Splitting of skin or cuticle.	[21]

In addition to deficiency, Ca^{2+} toxicity is reported in crop plants, such as gold spot/yellowish flecks—tiny flecks that develop around the calyx and shoulder of fruit—in tomato [189], and Ca^{2+} toxicity halts germination and growth of vegetables [24].

8.1. Genesis of Blossom-End Rot Development

Although Ca^{2+} is the most abundant mineral (3.64%) in the Earth's crust, it may not be available to plants [190]. Among the three forms (bound, exchangeable, and soluble) of soil Ca^{2+}, soluble Ca^{2+} is readily available; availability of exchangeable Ca^{2+} depends on pH, and bound Ca^{2+} remains unavailable. 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. In acidic soil (pH < 5.0), Ca^{2+} can be leached out with rainwater, and in alkaline soil (pH > 7.5), it can be precipitated with phosphorus, resulting in less availability. Ca^{2+} availability also depends on the presence of other cations, salinity, root growth, anoxia, and root zone temperature [191]. 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 [25,35,192], 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^+ [69], which might be due to the higher valency of Ca^{2+} [11]. NH_4^+ competes with Ca^{2+} to be taken up by 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 [55,193]. At high soil calcium availability, fruit Ca^{2+} deficiency disorder may not appear. However, at 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 through disruptions to calcium transport and signaling pathways (Figure 5).

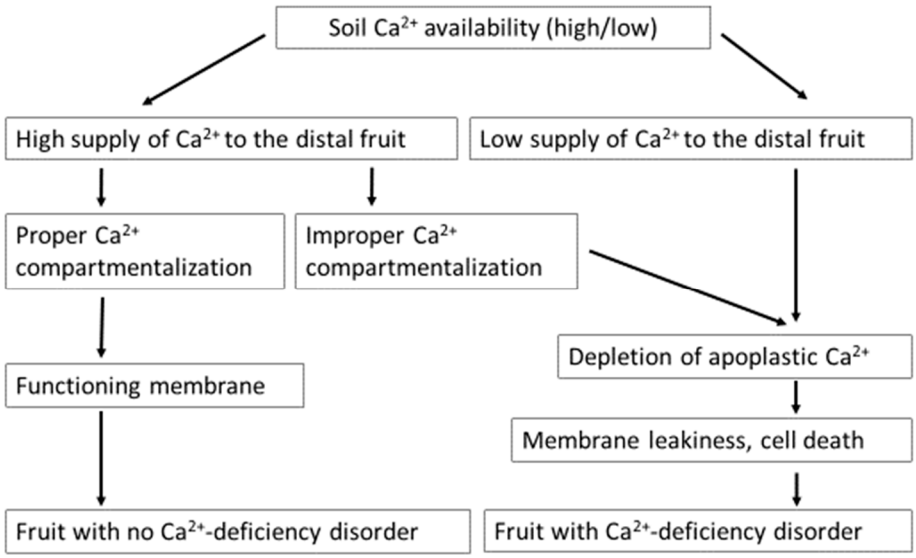


Figure 5. Calcium availability affects the development of the calcium deficiency disorder blossom-end rot (BER). At high soil calcium availability, fruit Ca^{2+} deficiency disorder may not appear. However, at low calcium availability, the deficiency disorder appears through depletion of apoplastic calcium 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 [194]. 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 [194]. Tomato varieties 'Calypso' and 'Spectra' showed higher incidences of BER than 'Counter' [195]; 'Petomech II' higher than 'IPA-L' [196]; 'Celebrity' higher than 'Rutgers,' 'Mountain Pride', and 'Mountain Spring' [197]; STEP 158 (breeding line) higher than 'Rutgers' and 'Doublerich' [198], and 'Boludo' higher than 'Daniela' [199]. Elongated tomato varieties are more susceptible to BER [200]. BER never occurs in small-fruit and wild tomato cultivars [55]. Lack or excess of minerals may cause deficiency disorders [201,202]. A low fruit apoplastic calcium, which results in leaky cell membranes, leads to BER development [203]. BER occurs in tomatoes if the calcium concentrations of the lateral ends of green fruits are <0.2 μmol g^{-1} [204]. 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 [191]. Phytohormones may also affect BER incidence. Foliar spray of ABA reduces BER incidence by increasing calcium availability [39,204], and gibberellins increase it through increasing oxidative stresses in plants [205]. ABA increases, and GA decreases calcium accumulation [148]. Thus, applying ABA or GA inhibitors (paclobutrazol and prohexadione-Ca^{2+}) may decrease BER in peppers [148]. Transport and homeostasis of calcium ions are crucial for preventing BER in peppers [148].

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 [206]. 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 [207]. Pygmy fruits, having no rapid growth phase, usually do not develop BER [208]. BER-affected tomatoes ripen earlier and are smaller than healthy fruit [209].

The onset of BER can vary based on trusses and seasons. Frequent incidence of BER was observed with the first truss [37], followed by a subsequent decrease [32,210,211], or increase [211,212]. Basal fruits of a truss had more severe BER than the others [29]. Based on seasons, BER incidence can increase or decrease from the first to the upper trusses [156,195].

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 act 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 [25], 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 toward the blossom end of the fruit, high temperature, and high or low transpiration of the target organ [27,69,210,213–217]. 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 [168]. Reports show severe BER incidence in the distal part of the fruit despite high Ca^{2+}-concentration [215]. Moreover, no BER symptoms occurred when the Ca^{2+} supply was low in the case of slow-growing plants [216]. 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 subcellular 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 [168].

BER occurs due to abiotic stresses, high temperature, drought, salinity, water logging, higher transpiration, production of ROS, and low availability of ascorbic acid [200,218]. 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 [37]. Spraying calcium on the fruit during the developmental stage reduces BER incidence [55]. 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 [200]. Balanced fertilizer and avoiding vigorous foliage growth may help reduce BER [200]. Shade net may also reduce BER incidence [219–223]. 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 [40]. 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 [50]. Therefore, correctly understanding calcium nutrition helps reduce crop cultivation costs, minimize environmental pollution, and boost crop production [224].

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 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 [24]. Understanding the molecular mechanisms of BER may help in better managing the disorder [225]. With the combination of appropriate management practices and BER-resistant cultivars, the incidence of this disorder may be eliminated in the future [55].

10. Climate Change and Calcium Availability

Climate change, characterized by temperature extremes, prolonged drought, elevated atmospheric CO_2 levels, and episodes of heavy precipitation (Figure 6), poses significant challenges to crop productivity and quality. Understanding calcium's role in enhancing crop resilience to such environmental stressors is increasingly vital [226]. Elevated temperatures increase leaf transpiration relative to fruit transpiration, thereby limiting calcium transport to fruit tissues and promoting the onset of blossom-end rot (BER) [223]. Additionally, high air and root-zone temperatures impair calcium uptake and exacerbate deficiency-related disorders [227]. Elevated temperatures also enhance the incidence of plant diseases and pest infestations, further restricting nutrient availability.

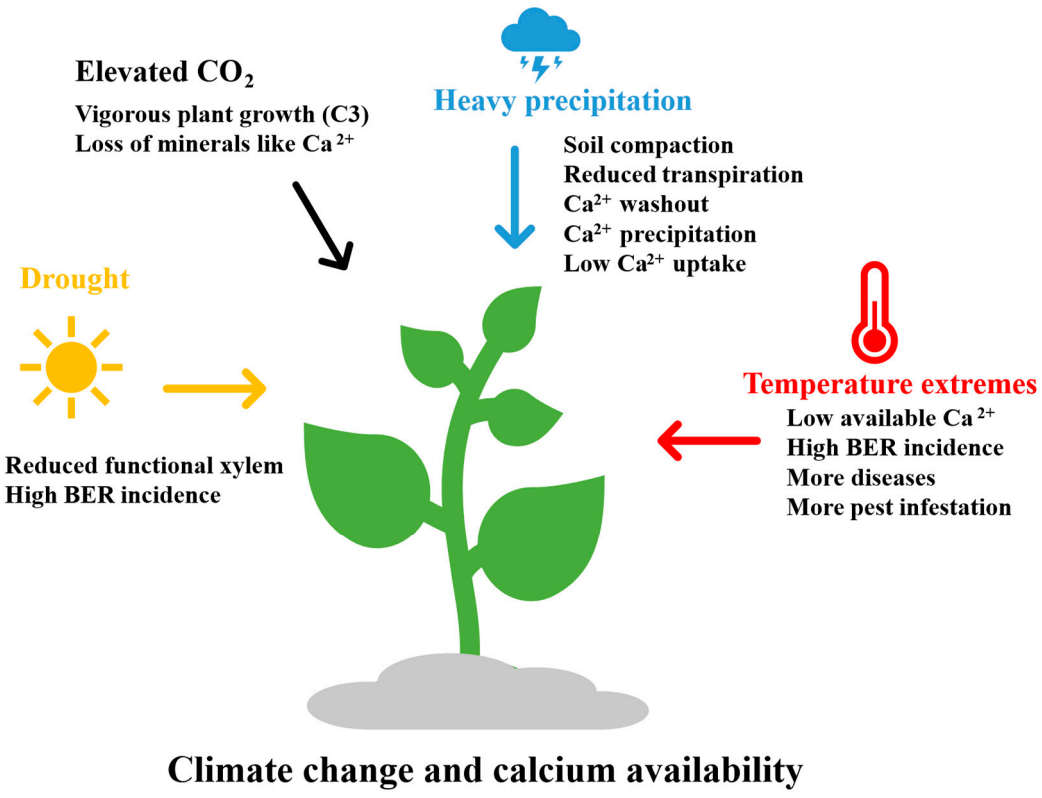


Figure 6. A diagram illustrating how climate change affects calcium availability in plants.

Drought stress limits xylem functionality, diminishing calcium translocation and contributing to BER development [228]. Since calcium is primarily transported via the xylem in the transpiration stream, water scarcity significantly impairs calcium delivery to actively growing tissues such as fruits, as observed in tomatoes and bell peppers [220,222].

Conversely, excessive precipitation can compact soil, reduce transpiration, leach soluble calcium, and induce calcium precipitation with phosphates—factors that collectively lower calcium bioavailability and increase the prevalence of disorders like BER [229]. Waterlogged or compacted soils, damaged root systems (caused by pathogens or insects), and

inconsistent soil moisture regimes all further impede calcium uptake. Elevated atmospheric CO_2 can enhance the growth of C_3 plants, yet this often coincides with reduced mineral concentrations in plant tissues [230]. Enhanced vegetative growth may dilute essential minerals, including calcium, magnesium, iron, and zinc. It is estimated that elevated CO_2 levels can reduce mineral content in food crops by 5% to 15% [231].

11. Recent Advances and Future Directions

Calcium (Ca^{2+}) plays a central role in plant physiology, acting both as a structural nutrient critical for cell wall integrity and membrane stabilization and as a versatile secondary messenger in a wide range of physiological processes [50]. Recent progress in plant science has introduced promising approaches to enhance calcium delivery, engineer calcium transporter genes, and monitor intracellular calcium dynamics with high spatial and temporal resolution. Key emerging areas include: (1) nanotechnology-based calcium delivery systems, (2) CRISPR-mediated modification of calcium transporter genes, and (3) advanced imaging and biosensor tools for real-time calcium monitoring.

11.1. Nanotechnology-Based Calcium Delivery Systems

Conventional calcium fertilization methods often face limitations, such as low solubility, leaching losses, and inefficient uptake, particularly in calcareous soils or under abiotic stress conditions. Nanotechnology offers a novel solution by enabling the formulation of nanoscale calcium carriers that enhance solubility, mobility, and targeted delivery to plant tissues [224]. Calcium-based nanoparticles (Ca-NPs), including calcium carbonate, calcium phosphate, and hydroxyapatite forms, have demonstrated improved foliar and root uptake due to their high surface area and controlled-release properties. For example, foliar application of calcium phosphate nanoparticles has been shown to enhance calcium mobility and mitigate blossom-end rot in tomato by improving calcium allocation to distal fruit tissues [232]. Nanoencapsulation—embedding calcium salts in polymeric or lipid nanocarriers—further improves nutrient bioavailability and ensures sustained release. These carriers protect calcium ions from premature precipitation or interaction with soil constituents, thereby enhancing availability in the rhizosphere or phyllosphere [233]. In addition to nutrient delivery, Ca-NPs may influence stress signaling by modulating calcium-dependent cascades, thus simultaneously correcting deficiencies and enhancing stress resilience [49,232]. Calcium transients, whether transient, sustained, or oscillatory, serve as signals that are decoded into physiological responses [81]. Despite promising results, the agricultural application of calcium phosphate nanomaterials remains in its infancy. Critical gaps persist in understanding the physiological and molecular effects, cost-effectiveness, and scalability of these approaches. Further research is needed to elucidate plant responses to these formulations and to tailor nanoparticle design for crop-specific needs [234]. For instance, nano-calcium carbonate at 200 mg L^{-1} significantly improved wheat seedling growth, enhanced photosynthetic pigments, antioxidant enzyme activity, Rubisco function, and stomatal conductance [235].

11.2. CRISPR-Mediated Gene Manipulation of Calcium Transporters

Calcium homeostasis in plants is orchestrated by a group of transporters, including Ca^{2+}-ATPases, Ca^{2+}/H^{+} exchangers, and cyclic nucleotide-gated channels (CNGCs) [236]. Genome editing via CRISPR-Cas9 has emerged as a tool for functional dissection and targeted manipulation of these transporters. For instance, the CRISPR-based knockout of the vacuolar Ca^{2+}/H^{+} antiporter gene CAX1 in Arabidopsis altered cytosolic calcium levels and increased sensitivity to abiotic stress, underscoring its regulatory role in calcium partitioning and stress responses [50]. Furthermore, CRISPR-dCas9 systems enable pre-

cise transcriptional regulation without altering genomic sequences, allowing fine-tuned modulation of calcium influx via CNGC genes [237]. These strategies not only improve calcium-use efficiency but also provide a broader understanding of the regulatory networks underlying calcium signaling. Calcium transients in the cytosol ($[Ca^{2+}]$cyt) are triggered by diverse stimuli, including light, touch, hormones, pathogens, and abiotic stresses like salinity and drought. These transients result from a dynamic interplay of calcium influx through channels and efflux via pumps, primarily mediated by Ca^{2+}-ATPases and H^+/Ca^{2+} antiporters. Calcium-binding proteins and calcium-dependent protein kinases decode these signals, regulating gene expression and conferring stress tolerance [238]. The use of CRISPR tools is accelerating insights into calcium-regulated stress responses. For example, their application in dissecting plant adaptation to drought and salinity has proven effective, particularly when combined with genomic-assisted breeding strategies [239]. Additionally, CRISPR systems have expanded into phytometabolic editing, targeting regulatory mechanisms such as upstream open reading frames (uORFs), post-translational modifications, and protein–protein interactions [240]. However, regulatory uncertainty remains a barrier to the widespread adoption of CRISPR-edited crops [241].

11.3. Imaging and Biosensors for Calcium Dynamics

Deciphering calcium signaling requires real-time monitoring of transient and spatially specific cytosolic Ca^{2+} changes [242]. Genetically encoded calcium indicators (GECIs) have enabled in vivo imaging of calcium dynamics with high resolution. These biosensors comprise calcium-binding domains fused to fluorescent proteins, which emit signals upon binding to calcium. Each physiological process—ranging from growth and differentiation to stress and defense—exhibits unique "calcium signatures" defined by amplitude, frequency, and duration. Understanding how transporter activity shapes these signatures remains a significant research challenge [242]. Recent biosensor advancements include red-shifted indicators for deep-tissue imaging, multiplex systems for co-detection of additional analytes, and microfluidic integration for real-time environmental monitoring. Beyond imaging, calcium acts as a signaling nexus across various physiological processes, including cell wall maintenance and development, enzyme regulation, and programmed cell death. GECI-based imaging has revealed refined subcellular dynamics and is now supported by artificial intelligence tools for analyzing complex datasets, as well as miniaturized systems for in-field applications [243]. Improvements such as calibration-free sensors, quantum dot incorporation, and bio-orthogonal activation will further advance live-cell calcium imaging. In Arabidopsis, GECIs have been utilized to monitor calcium responses to various stimuli, including mechanical stress, gravity, cold, osmotic changes, reactive oxygen species, and hormones [244].

11.4. Future Directions

Key areas requiring further investigation include: (1) limited understanding of long-distance calcium transport mechanisms [153]; (2) incomplete characterization of calcium transport and intracellular compartmentalization [20]; (3) poor resolution of how calcium signatures encode specific responses [245]; and (4) the complex interactions between calcium signaling and hormonal or nutritional networks [238]. To bridge these gaps, interdisciplinary research integrating molecular biology, systems imaging, nanotechnology, and agronomic studies is essential. Such integration is crucial for developing crops with optimized calcium nutrition and enhanced stress resilience.

12. Conclusions

Calcium is an essential element for plant growth, development, and productivity. While most soils worldwide are not typically deficient in Ca^{2+}, calcium-deficiency disorders in crops are prevalent and lead to substantial yield losses in several horticultural crops. 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, and yield], and environment [temperature (air and root zone), relative humidity, vapor pressure deficit, and transpiration, which 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 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 toward 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, such as agronomic, physiological, breeding, and molecular methods, may also contribute to minimizing BER. Although calcium is well studied, the molecular identity and regulation of many calcium channels, pumps, and sensors remain uncharacterized. The relation between calcium as a nutrient and secondary messenger is also poorly understood. Therefore, future investigations should explore how calcium signatures encode specific responses and how the complex interactions between calcium signaling and nutritional or hormonal networks interplay.

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