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Guarding Your Tomatoes: A Seeking Attention Against Blossom End Rot

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Introduction

Blossom end rot (BER) is one of the most devastating physiological disorder or an abiotic disease affecting tomato production worldwide. Physiological or abiotic diseases predominantly arise from alterations in environmental conditions, encompassing factors such as fluctuations in temperature and moisture levels, high pH, imbalances in soil nutrient composition, inadequate or excessive concentrations of specific soil minerals, and inadequate drainage (Khavari-Nejad et al., 2009).

Symptoms

Initial symptoms appear as a pale tan lesion with a water-soaked appearance, which subsequently enlarges, darkens, and transforms into a leathery texture (Gangadhara et al., 2021). While the primary damage is localized, the lesion often serves as an entry point for secondary pathogens like saprophytic species of *Alternaria* fungi, leading to complete fruit decay (Topcu et al., 2022).

Fig 1 Disease symptoms of Blossom end rot

Key Physiological Causes of Blossom End Rot

Ca^{2+} in BER:

Blossom-end rot arises from a localized deficiency of calcium at the distal end of the fruit. Ca^{2+} plays an essential role in plant growth and development, but its abnormal regulation and unequal distribution in cellular compartments (cytosol, vacuole and apoplast),

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arenegatively associated to BER development.The altered Ca^{2+} distribution disrupts cellular membrane integrity and function, potentially leading to solute leakage and BER (Fig 1). Pectin, a major component of the middle lamellae, interacts with Ca^{2+} electrostatically, facilitating cross-linking of pectin molecules and cell wall stiffening. Using gene silencing approach, such as antisense expression of pectin methylesterase (LePME3) reduced BER incidence by increasing water-soluble Ca^{2+} concentration in tomato fruits (De Freitas et al. 2012). However, the role of free apoplastic Ca^{2+} concentration in maintaining proper Ca^{2+} homeostasis among different cellular compartments and preventing membrane leakage, thus reducing BER incidence.

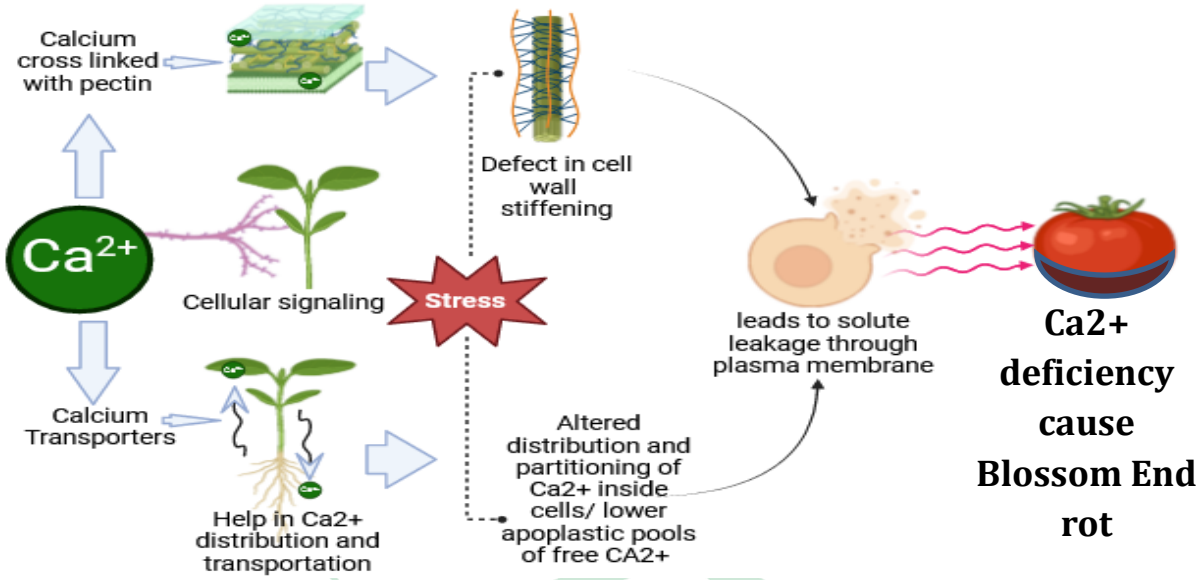

Fig 2 Diagrammatic representations of Blossom end rot disease association with Ca^{2+} deficiency

Reactive oxygen species (ROS):

Ca^{2+} and ROS both serve as intertwined secondary messengers, responding to various environmental stresses. Ca^{2+} modulates ROS production, while ROS influences Ca^{2+} homeostasis. In plants, ROS primarily originate from electron transport reactions in the plasma membrane, endoplasmic reticulum, chloroplast, and mitochondria, yielding free radicals like superoxide anion and hydroxyl radicals, as well as non-radical molecules such as hydrogen peroxide and singlet oxygen. Excessive ROS levels, resulting in lipid and protein oxidation,

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enzyme inhibition, and cell membrane leakage, and are closely associated with BER.

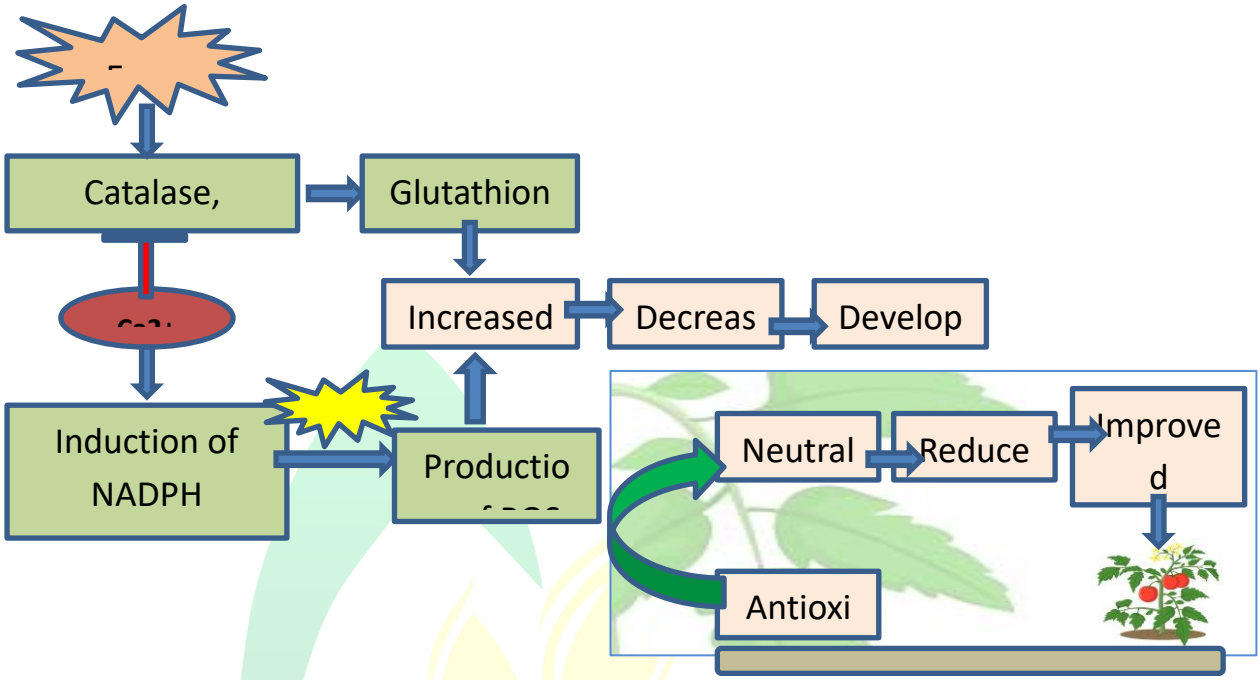

Fig 3 Diagrammatic representations of Blossom end rot disease association with ROS

The antioxidant genes like catalase, ascorbate peroxidase, and glutathione reductase are down regulated in calcium-deficient tomatoes, resulting ROS accumulation and BER incidence (Fig 2). The elevated levels of antioxidants during BER's critical stage exhibit greater resistance to the disorder (Topcu et al. 2022). Overall, the interaction among, ROS enzymes, antioxidants, and calcium levels significantly influences BER development, particularly under conditions of insufficient calcium and abiotic stress, where deregulation of ROS production and scavenging pathways leads to membrane damage and increased BER incidence.

Other physiological factors responsible for BER development

Elevated concentrations of monovalent cations in soils, including potassium (K^+), magnesium (Mg^+), sodium (Na^+), and ammonium (NH_4^+), negatively influence the uptake of divalent cation Ca^{2+}, thereby increasing BER susceptibility. An increase in NH_4 concentration relative to nitrate/ammonium ratio suppresses Ca^{2+} uptake and leads to increasing BER development. Hormonal regulation, particularly auxins, cytokinins and gibberellin, plays a significant role in cell division and expansion during fruit development, increasing BER incidence by reducing Ca^{2+} concentration and enhancing cell membrane permeability. Conversely, the application of growth retardants like abscisic acid and Apogee inhibits GA biosynthesis, resulting in reduced BER by increasing pericarp Ca^{2+} concentration and

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promoting antioxidant production to counter ROS activity. Slower initial fruit growth rates are also associated with reduced BER incidence, indicating that rapid growth following pollination or growth regulator application may induce stress in fruit, leading to lower Ca^{2+} concentrations and compromised cell wall stabilization and membrane integrity.

Genetic basis of BER

With the advancement of sequencing techniques, understanding the genetic basis of BER in tomatoes has become significantly easier. Previously, the lack of molecular markers constrained our ability to fully comprehend the genetic basis of BER. However, the advent of the full genome sequence of tomato (Tomato Genome Consortium, 2012) has revolutionized this field. The availability of the complete genome and numerous resequencing projects has enabled the discovery of single nucleotide polymorphisms (SNPs) between closely related tomato parents. This has led to the development of new approaches for mapping BER loci, such as QTL-seq approach (Topcu et al. 2021). The *Solanum lycopersicum* var. cerasiforme (SLC) and *S. lycopersicum* var. lycopersicum (SLL) were used as parents to generate the mapping population, resulting in identification of four loci associated with BER such as BER3.1 and BER3.2 on chr03, BER4.1 on chr04 and BER11.1 on chr11 (Topcu et al. 2021). Fine mapping further refined the locations of two loci namely, BER3.2 and BER11.1 to 1.58 Mb and 1.13 Mb regions, respectively. Additionally, BER11.1 was also mapped in another population derived from crosses between Ailsa Craig and Kentucky Beefsteak tomato cultivars (Prinzenberg et al. 2021). In future, the cloning the genes within these loci are expected to provide novel comprehensions into the onset and early developmental stages of BER.

Management to confer resistance against Blossom End Rot

Different strategies can be used to mitigate the incidence of blossom end rot in tomato in home gardens. The most critical point is to ensure uniform soil moisture level during the growing season through regular irrigation and mulching. The selection of suitable site with well drained fertile soil having good aeration and enriched with organic matter helps to ensure good moisture in dry period which reduces the development of disease. The sowing of tomato early in cold soil increases the development of blossom end rot while, less incidence was observed in warmers soil if tomatoes are planted early in the season. Keep the soil pH between 6-6.5. If a soil test indicates calcium deficiency in soil then gypsum, superphosphate, or high-calcium limestone should be applied to complete the requirement of Ca in soil. Avoid the

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excessive use of nitrogen fertilizer including ammonical forms during fruiting period. Calcium is not well absorbed by plants due to low efficiency so foliar application is advised to reduce BER in fruits. Marginal leaf burn has been linked with calcium spray leading to phytotoxicity. The tender feeder roots responsible for water and nutrient uptake should be destroyed while cultivating. Selection of suitable cultivar with varying rate of susceptibility helps to minimize BER. Tomato cultivars with a plum or pear shape have been observed to be more vulnerable to blossom-end rot.

Conclusion

Blossom End Rot (BER) significantly challenges tomato production due to factors like calcium deficiency, water imbalance, and oxidative stress. Effective management includes maintaining consistent soil moisture, ensuring adequate calcium levels, and avoiding excessive nitrogen fertilization. Advanced genomic research has identified genetic markers associated with BER resistance, paving the way for developing resilient tomato varieties. Integrating these physiological insights and genetic advancements can help devise better strategies to mitigate BER, ensuring healthier and more productive tomato crops.

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