ELSEVIER

Scientia Horticulturae 90 (2001) 193–208

SCIENTIA
HORTICULTURÆ

www.elsevier.com/locate/scihorti

Review

Blossom-end rot of tomato (*Lycopersicon esculentum* Mill.) — a calcium- or a stress-related disorder?

M.C. Saure*

Chamber of Agriculture, Dorfstr. 17, 21647 Moisburg, Germany

Accepted 5 December 2000

Abstract

Many researchers have attributed the occurrence of blossom-end rot (BER) in tomato to a deficiency of Ca^{2+} in the fruit or parts of the fruit in connection with the uptake of nutrients by the roots, the transport of Ca^{2+} to and within the fruit or a varying demand for Ca^{2+} depending on the growth rate of fruits. However, a critical concentration of Ca^{2+} in the fruit has not yet been found, and the influence of favourable or unfavourable growing conditions on the development of BER is still poorly understood. The symptoms of BER are caused by a disintegration of the cell membranes and an increased ion permeability. It is proposed that not a single factor but the sequence of two equally important factors are involved: (1) a higher susceptibility to various stresses due to an increase in physiologically active gibberellins (GAs) and a resulting decrease in Ca^{2+}, causing the enhanced permeability of cell membranes, (2) some kind of stress, e.g. by soil water deficit, high salinity, or high NH_4^+ activity, causing the deterioration of cell membranes with subsequent loss of turgor and leakage of cell liquids. BER seems to occur when stress exceeds stress tolerance, most frequently in young fruit at the beginning of cell enlargement. At this stage, usually the highest amount of physiologically active GAs and the lowest amount of Ca^{2+} are found, i.e. stress tolerance is lowest. Ca^{2+} per se is considered neither a primary nor an independent factor in the development of BER. This hypothesis is advanced with the objective of stimulating further research into the causes of a physiological disorder that, as yet, remains stubbornly obscure. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Ammonium; Blossom-end rot; Calcium deficiency; Cell membrane; Environment; Gibberellin; Growth rate; Salinity; Stress; Tomato

---

* Tel./fax: +49-4165-6277.
E-mail address: msaure@t-online.de (M.C. Saure).

PII: S0304-4238(01)00227-8

1. Introduction

Blossom-end rot (BER) of tomato was first identified as a physiological disorder more than 100 years ago (Selby, 1896). In susceptible cultivars, it may cause severe economic losses in some seasons and under certain environmental conditions. Since Lyon et al. (1942) and Raleigh and Chucka (1944) found a correlation between the occurrence of BER and Ca^{2+} nutrition, BER is now generally attributed to an inadequacy of Ca^{2+} in the fruits and it is therefore called a 'calcium-related disorder' (Shear, 1975). Indeed, most modern textbooks and technical papers hold that Ca^{2+} deficiency is the primary cause of BER. Adams and Ho (1993) specified that BER is a local deficiency of Ca^{2+} in tomato fruit, or in the distal end of tomato fruit, respectively (Adams and Ho, 1995). However, it is not possible to predict the occurrence of BER on this basis (Ho, 1989; Vielemeyer and Weissert, 1990). Adams and Ho (1995) pointed out that the causes of BER are still not fully understood, despite over 100 years of research. Neither the mechanism by which the external factors affect the Ca^{2+} content of the fruits and the development of BER nor the kind of relationship between Ca^{2+} deficiency and BER are unequivocally identified. Kinet and Peet (1997) confirmed that for many physiological disorders of tomato the causes are poorly understood both in terms of why cultivars differ in susceptibility and why certain environments or cultural practices predispose plants to the disorder. They observed that in BER, despite some progress in understanding at the physiological level, causes and control measures are not always apparent at the practical level. Therefore, it was considered necessary to rethink and to integrate the wealth of already available but inconsistent information in order to find a new approach for prospective research.

2. Characteristics of BER

BER becomes visible first as one or more tiny lesions slightly depressed below the surface at or near the blossom-end of the fruit, close to the base of the style. Later, the affected tissue appears watery and darker green. It reaches its full size in about 1 week from inception. Finally it shrinks or collapses, forming a depressed, leathery necrosis of the distal part of the placenta and the adjacent pericarp that gradually turns from brown to nearly black (Robbins, 1937; Evans and Troxler, 1953; Maynard et al., 1957; Spurr, 1959; van Goor, 1968). Tomatoes with BER ripen earlier and are generally smaller than healthy fruit (DeKock et al., 1979). Additional to the described 'external' BER an 'internal' BER may occur, affecting only tissues inside the outer wall of the fruit including some seeds (Spurr, 1959; Adams and Ho, 1992; Petersen and Willumsen, 1992; Willumsen et al., 1996).

The incipient stages of BER have been observed by Spurr (1959) only in fruit ranging from 12 to 15 days after anthesis. This period is characterised by maximal relative growth rate of fruit volume and the beginning of cell enlargement (Monselise et al., 1978; Manishi et al., 1996). In fruit older than 15 days, Spurr (1959) found only the more advanced stages of BER. Many other authors agree that the incidence of BER mostly occurs at about the second week after anthesis (Westerhout, 1962; Barke, 1968; Guttridge and Bradfield, 1983; Banuelos et al., 1985; El-Gizawy and Adams, 1986; Adams and El-Gizawy, 1988; Sonneveld and Voogt, 1991; Wui and Takano, 1995b; Wada et al., 1996; Cho et al., 1998). Occasionally occurring pygmy fruits have no phase of rapid growth and do not develop BER (Robbins, 1937). However, there are several reports that BER can be induced experimentally at any stage of fruit development (Maynard et al., 1957; Chiu and Bould, 1976; Černe, 1990; Barker and Ready, 1994).

The incidence of BER was mainly confined to the first truss in experiments of Westerhout (1962). Others observed that it may increase for successive trusses (van Goor, 1974; Chiu and Bould, 1976; Nukaya et al., 1995; Cho et al., 1998; Paiva et al., 1998b) or decrease (Pill and Lambeth, 1980; El-Gizawy and Adams, 1986; Adams and Ho, 1993). In the same cultivars both an increase or decrease of the incidence from the first to the upper trusses may occur, depending on the season (Adams and Ho, 1992; Ho et al., 1993). Banuelos et al. (1985) reported that within a truss, the fruit from the basal part were affected most severely with BER.

3. Relations between Ca^{2+} deficiency and BER

Evidence for Ca^{2+} deficiency as the primary cause of BER has been derived from observations that the blossom end has always the lowest content of Ca^{2+} within tomato fruits (Ward, 1973; Ehret and Ho, 1986; Adams and Ho, 1993; Franco et al., 1994; Nonami et al., 1995) and that the Ca^{2+} content in fruit affected by BER is lower than in healthy fruit. Since the early work of Lyon et al. (1942) and Raleigh and Chucka (1944), most of these observations are based on experiments with insufficient Ca^{2+} nutrition. Tomatoes grown in soilless media containing no or extremely low amounts of Ca^{2+} frequently produce various proportions of fruit showing symptoms of BER, sometimes together with or after the appearance of other symptoms of Ca^{2+} deficiency such as chlorosis or necrosis at the margins of expanding leaves, inhibition of root and shoot elongation, necrosis of the root and shoot tips, etc. (e.g. Geraldson, 1957; Maynard et al., 1957; Ward, 1973; Bangerth, 1973; Chiu and Bould, 1976; Bradfield and Guttridge, 1984; Drews and Frank, 1987; Adams and El-Gizawy, 1988; Sonneveld and Voogt, 1991; Mu et al., 1992; Cardoso et al., 1995; de Kreij, 1996; Hadi et al., 1996; Paiva et al., 1998a). Accordingly, spray applications of Ca-salts to fruiting plants, especially if grown in Ca^{2+}-deficient substrates, may reduce BER (Evans and Troxler, 1953; Geraldson, 1957; Bangerth, 1973; Al-Dolimy and Al-Ani, 1987; Vielemeyer and Weissert, 1990; Wada et al., 1996; Franco et al., 1998; Plese et al., 1998; Ho, 1999). Several authors, such as Raleigh and Chucka (1944), Wiersum (1966) and Bradfield and Guttridge (1984), established threshold values below which BER is likely to occur, but they all differ.

The occurrence of BER at insufficient supply of Ca^{2+} has formerly been attributed to lacking rigidity of cell walls (Evans and Troxler, 1953), but Spurr (1959) observed the earliest changes in tissues affected by BER before any collapse of the cells. van Goor (1968), Bangerth (1973) and Simon (1978) stated that BER is associated with the disintegration and increased ion permeability of cell membranes due to Ca^{2+} deficiency, resulting in loss of turgor and cell fluids invading the intercellular air space, thus causing the watery appearance in the early stages of BER. It is well known that an adequate supply of Ca^{2+} maintains membrane integrity and selectivity (Grattan and Grieve, 1999).

However, several authors could not find significant differences in Ca^{2+} content of tissues from healthy and affected fruit or of healthy fruit from affected and non-affected plants, respectively (Spurr, 1959; Millikan et al., 1971; Murray et al., 1972; Ward, 1973; Pill and Lambeth, 1980; Petersen and Willumsen, 1992; Barker and Ready, 1994; Nonami et al., 1995; Pulupol et al., 1996; Franco et al., 1999). Because of the discrepancies in the published values for Ca^{2+} in fruit with and without BER, Ward (1973) and Nonami et al. (1995) had some doubts regarding Ca^{2+} deficiency being the (only) cause of BER. Franco et al. (1999) observed serious BER incidence despite fairly high levels of Ca^{2+} in the distal part of the fruit, and at reduced Ca^{2+} supply, Chiu and Bould (1976) found no BER when plants were growing slowly. Regarding the susceptibility of cultivars, some authors observed a lower Ca content in BER-susceptible than in less susceptible cultivars (Sánchez Conde and Felipe, 1983; Cardoso et al., 1995) but others found no such relationship (Greenleaf and Adams, 1969; Adams and Ho, 1992; Brown and Ho, 1993; Ho et al., 1995).

According to DeKock et al. (1982b), the K/Ca ratio is a better indicator for BER than Ca alone, but Wada et al. (1996) and Bar-Tal and Pressman (1996) found no clear relationship between the K/Ca ratio and the incidence of BER. Gerard and Hipp (1968) noted that tomatoes with a low Ca content and a high K/Ca ratio can have a very low incidence of BER.

4. Influence of external factors on BER

4.1. Growth inhibiting factors

Increased incidence of BER may be associated with reduced plant and fruit growth due to stresses induced in the root zone such as salinity, soil water stress, and NH_4^+-toxicity. The inhibiting effect of *salinity* on fruit growth, especially during the cell expansion phase, and on plant growth has been summarised by Cuartero and Fernández-Muñoz (1999). Since Robbins (1937) established a relation between increased nutrient salt concentration, less fruit and plant growth and a high percentage of BER, an increased occurrence of BER at high salinity has frequently been confirmed (e.g. Cerda et al., 1979; Cho et al., 1997; van Ieperen, 1996), but even in susceptible cultivars, the percentage of BER may be quite low at high salinity (e.g. Adams and El-Gizawy, 1986; Adams and Ho, 1992; Willumsen et al., 1996), especially when NaCl was used instead of the major nutrients to increase salinity (Adams and Ho, 1993). Recently, Chrétien et al. (2000) stated that increasing salinity by NaCl resulted neither in an increase of BER incidence nor in a decrease of fruit Ca^{2+} concentration of rockwool-grown tomatoes. Certainly, there is no linear relationship between salinity and BER, and the responses of susceptible cultivars to the same environmental stress differ (Adams and Ho, 1992).

The role of *soil water stress* has been widely investigated and controversially discussed, since Selby (1896) suggested that BER was associated with insufficient soil moisture. Evans and Troxler (1953) concluded from the literature that BER is most serious during the initial phase of the harvest season and in periods of limited soil moisture supply. Others stated that BER is not due entirely to soil water stress but can be aggravated by it (Shaykewich et al., 1971; van Goor, 1974; Pill and Lambeth, 1980). BER increased only occasionally as water stress increased in the experiments of Obreza et al. (1996) and little or no influence of reduced irrigation on incidence of BER was observed by Sperry et al. (1996) and Pulupol et al. (1996). Differences in BER incidence during the course of the season were not related to moisture level treatment in the experiments of Gerard and Hipp (1968) and DeKock et al. (1982a), and the incidence of BER was increased by higher irrigation levels in experiments of Westerhout (1962), Caliandro (1971) and Mohamed et al. (1989). Adams and Ho (1992), who stated that water stress was the most common cause of BER, also noticed that BER frequently occurred when the moisture content of the substrate was fully adequate (Adams and Ho, 1993). Robbins (1937) claimed that not only a restricted absorption of water by the plant but also a greatly increased rate of transpiration of water will increase the probability that BER occurs. This has been confirmed by Gerard and Hipp (1968) and, as a tendency, by Bradfield and Guttridge (1984), de Kreij (1996), and Paiva et al. (1998a) but does not fit the promotion of BER by high relative humidity (RH) as reported by Banuelos et al. (1985) and Cho et al. (1998).

It is well documented that *NH_4^+-N nutrition* under certain conditions such as high temperature may cause reduced plant and fruit growth and increased ethylene evolution, leaf epinasty, etc. (e.g. Puritch and Barker, 1967; Hartman et al., 1986; Kafkaki, 1990; Barker and Ready, 1994). Therefore, applications of $NH_4{}^+$-N fertilisers or higher $NH_4{}^+/NO_3{}^-$ ratios in the nutrient solution are often associated with increased incidence and severity of BER (DeKock et al., 1979, 1982a; Pill et al., 1978; Pill and Lambeth, 1980; Ikeda and Osawa, 1988; Terabayasi et al., 1988; Vielemeyer and Weissert, 1990; Lee et al., 1991; Albahou, 1999). $NH_4{}^+$-N application during fruiting of tomato plants in sand cultures may cause symptoms of BER within 7 days (Wilcox et al., 1973). However, the effects of increasing $NH_4{}^+$-N may vary. Adams (1986) summarised that $NH_4{}^+$-N usually does not have an adverse effect in summer weather due to its rapid transformation into nitrate and to vigorous plant growth.

The influence of these factors on the incidence of BER has been frequently considered as indirect by interfering with the supply of Ca^{2+} to the fruit, causing a localised inadequacy of Ca^{2+} (Shear, 1975), for which various mechanisms have been proposed:

- Restricted uptake of Ca^{2+} by the roots, e.g. due to either restricted water supply, increased salinity or interactions with other nutrients such as $NH_4{}^+$-N in the feeding solution, or poor aeration in the root zone (Adams and Ho, 1995). Adams and Ho (1992) summarised that water stress, which reduces Ca^{2+} uptake, is the most common cause of BER. The role of transpiration, as affected by RH, in the uptake of Ca^{2+} and incidence of BER is disputed (Ehret and Ho, 1986; Ho and Adams, 1989).
- Restricted transport of Ca^{2+}, e.g. due to substantially retarded xylem tissue development in the pedicel and within the fruit at high salinity (Ehret and Ho, 1986; Belda and Ho, 1993), but the restriction of Ca^{2+} transport inside the fruit cannot be the sole cause of BER (Belda et al., 1996).
- Competition for Ca^{2+} between leaves and fruit, distracting Ca^{2+} flux from the fruit to the leaves at high transpiration (Gerard and Hipp, 1968; Ho, 1989).

On the other hand, there are several indications that stress factors and BER are more directly related. Differences in Ca^{2+} uptake, in the percentage of Ca^{2+} and in total Ca^{2+} content of the fruit caused by differences in salinity are not always related to the incidence of BER (Adams and Ho, 1993). Shaykewich et al. (1971) observed that the incidence of BER increased at increasing soil water stress, but the concentration of Ca^{2+} was not significantly affected. Ward (1973) noticed that BER was produced by subjecting plants to water stress in the presence of adequate supplies of Ca^{2+} and argued that there may be two physiological conditions resulting in identical symptoms, and that there may be a different sequence of physiological events in plants that have suffered water stress rather than Ca^{2+} deficiency. Similarly, Franco et al. (1999) stated that at a higher level of irrigation fewer fruit were affected by BER although the Ca^{2+} concentration in the stylar portion of mature fruit remained virtually the same, and that BER might be a serious problem even in the presence of fairly high levels of Ca^{2+} at the blossom end.

Regarding the effects of NH_4^+-N, Hartman et al. (1986) observed that the incidence of BER increased and fruit size was severely reduced by increasing NH_4^+/NO_3^- ratios in the nutrient solution, but the percentage of Ca^{2+} in the fruit was unaffected by increments of NH_4^+. In experiments by Vielemeyer and Weissert (1990), the application of N-fertilisers containing NH_4^+-N coincided with the highest incidence of BER, but the Ca^{2+} uptake of fruits was not inhibited. Pill et al. (1978) and Barker and Ready (1994) found a lower content of Ca^{2+} in the fruit of NH_4^+ grown plants as compared to NO_3^- grown plants, but no difference in percentage of Ca^{2+} at different NH_4^+ levels, and no difference in the Ca^{2+} concentration between fruit with and without BER, respectively.

4.2. Growth promoting factors

Quite in contrast, an increased incidence of BER may also be associated with conditions favouring vigorous growth and fruit development. Spurr (1959) concluded from his extensive review of the literature that vigorously growing tomato plants under a high level of available nitrogen were especially susceptible to BER, and Westerhout (1962) stated that vigorous growth before anthesis of the first flower was responsible for the incidence of BER. Moreover, he suggested that the prevalence and severity of BER depended on the growth rate of tomato fruit being closely related to the vigour of the plant; slowly growing fruit were not affected. This has been confirmed, e.g. by Wiersum (1966), Chiu and Bould (1976) and DeKock et al. (1982b). Various cultural practices and environmental factors have been reported to increase the incidence of BER by promoting the rate of fruit growth, such as fruit thinning (DeKock et al., 1982b), increased daily radiation (Sonneveld and Voogt, 1991); increased temperature or a combination of warm weather and high irradiance, especially during the period of rapid fruit growth (Ho et al., 1993; Wui and Takano, 1995a,b). In this context, it appears reasonable that a reduction of plant and fruit growth, e.g. by foliar applications of high levels of the growth inhibitor daminozide during flowering (Kheshem et al., 1988), by repeated foliar applications of the GA biosynthesis inhibitor uniconazole (Wui and Takano, 1995b), or by reducing N uptake through application of compost (Manishi et al., 1996) may reduce the incidence of BER.

Some authors have attributed the increase of BER in vigorously growing plants to a higher requirement for Ca^{2+} for cell membrane biosynthesis. At an increased rate of fruit enlargement and accelerated cell growth, it may exceed the supply of Ca^{2+} (Chiu and Bould, 1976; Adams and Ho, 1992; Paiva et al., 1998a), but this does not explain why BER does not occur regularly in rapidly growing plants and fruit of susceptible cultivars.

4.3. Interaction of various external factors

From the preceding overview it becomes obvious that the incidence of BER cannot be reliably attributed to a single growth inhibiting or growth promoting factor. Few authors have doubted a role for Ca^{2+}, but many conceded that various other factors are additionally involved. BER may be aggravated by the interaction of stress factors causing reduced fruit growth, such as soil water stress together with high transpiration (Obreza et al., 1996), with high temperature (Gerard and Hipp, 1968), with high NH_4^+ (Pill and Lambeth, 1980), or with high salinity (Nonami et al., 1995); high NH_4^+ together with high temperature (Ikeda and Osawa, 1988), and high salinity together with high transpiration (Robbins, 1937; Bradfield and Guttridge, 1984; Stanghellini et al., 1998). However, Albahou (1999) reported that high NaCl salinity reduced the incidence of BER in NH_4^+-fed tomatoes.

BER may also result from an interaction between stress factors and factors causing rapid fruit growth. According to Adams and Ho (1993), the cause of BER is usually the result of an interaction between the effects of irradiance and ambient temperature on fruit growth and the effects of environmental stress on Ca^{2+} uptake and distribution within the whole plant. Ho et al. (1993) suggested that temperature may be the major environmental factor which induces BER, regardless of cultivars and salinity treatment. An increased incidence of BER associated with high temperature and rapid growth rate in plants grown at high nutrient supply and high salinity has been confirmed by Wui and Takano (1995a).

Additionally, there are some indications that the effects of environmental factors are time-dependent, i.e. the sequence of growth promotion and inhibition and their relation to plant development may be important. Large losses from BER were observed when initially well irrigated, luxuriantly grown plants were subject to drought, or when vigorous plants with well-developed fruit were moved from a shaded to an unshaded glasshouse (Chamberlain, 1933). Robbins (1937) observed that the initial symptoms of BER appeared most often shortly after a sudden change from cool, dark weather with low evaporation rates to light, warm weather and high evaporation rates. Ho et al. (1993) suggested that more sunshine and higher temperatures were required to induce BER after a period of predominantly sunny weather than after a less sunny period.

5. Discussion and conclusions

This review has provided very little evidence that certain environmental factors reliably produce BER every time they occur. Moreover, most of the available information on the effects of environment on the incidence of BER and on the role of Ca^{2+} deficiency is based on correlative relationships. Taylor and Smith

(1957) warned that correlations between Ca^{2+} content and incidence of BER do not show a cause and effect relationship between nutrient element composition and plant performance, and Pill et al. (1978) pointed out that BER is a qualitative phenomenon which implies that quantitative plant parameters are merely associative and not causal. Additionally, many experiments have been carried out by withholding or strictly limiting Ca^{2+} in the growing media, thus creating an overall deficiency rather than localised Ca^{2+} deficiency in rapidly growing tissue at the distal end of tomato fruit, for which BER is said to be a symptom (Ho and Adams, 1989). These experiments may induce symptoms of BER, but they do not reflect the situation in commercial tomato production where insufficient Ca^{2+} supply is hardly ever a problem (Kirkby, 1979).

The reduced Ca^{2+} concentration in rapidly growing tissues as well as an increased K^+/Ca^{2+} ratio is an essential physiological parameter reflecting growth rate, not only in tomato (DeKock et al., 1982b), but also in other crops (e.g. Göring and Mardanov, 1976; Richards, 1978). Further, rapid growth is associated with an increase of physiologically active gibberellins (GAs) (e.g. Suge, 1978; Rood et al., 1990; Grindal and Moe, 1997; Grünzweig et al., 2000). Externally applied GAs have been shown to increase the incidence of BER substantially (Bangerth, 1973). They interfere with Ca^{2+} uptake and transport, and consequently the application of GA biosynthesis inhibitors frequently results in increased Ca^{2+} levels (for references cf. Saure, 1998, p.140). Whereas Ca^{2+} is known to protect the integrity of cell membranes, i.e. to reduce their permeability and to prevent ion leakage caused by stress (for references cf. Marschner, 1983; Saure, 1998; Grattan and Grieve, 1999), GAs have the opposite effect (for references cf. Saure, 1998, p.139). Therefore the susceptibility to stress is increased by low concentrations of Ca^{2+}, but also by increased levels of GA (Vettakkorumakanav et al., 1999; Fletcher et al., 2000). Low Ca^{2+} concentrations may yet be physiologically reasonable: Marschner (1983) pointed out that in rapidly growing tissue high membrane permeability is required for solute flux. This means that the frequently postulated demand for higher Ca^{2+} supply at rapid growth may not exist. High Ca^{2+} supply may even reduce shoot and fruit growth and yield (Raleigh and Chucka, 1944; Barke and Menary, 1971; Hall, 1977; Hadi et al., 1996). The highest amount of physiologically active GA (El-Beltagy et al., 1976) and the lowest concentration of Ca^{2+} (Wada et al., 1996), i.e. the lowest stress tolerance, have been found in young tomato fruit at the beginning of cell enlargement, when BER is most likely to occur (cf. Section 2).

However, vigorous growth with low Ca^{2+} concentration in the tissues does not necessarily imply increased incidence of BER, because the loss of turgor, and the leakage of solutes observed in connection with the development of BER (cf. Section 3), may occur only if followed by a disintegration of membranes as a response to various stressors, such as soil water deficit, high salinity, or high NH_4^+ activity. Only stress above a certain threshold — relative to the actual stress tolerance — causes damage by affecting the fluidity and permeability of the membranes, thus enhancing ion leakage by an inhibition of membrane pumps and degradation of membrane lipids (Palta, 1990). Considering the indications in Section 4.3, it is tempting to speculate that it is the sequence of stress-free growth conditions, resulting in luxuriant growth but high susceptibility to BER, and the subsequent occurrence of severe stress that causes large losses from BER. This may also explain the differing observations regarding the position of the most severely affected trusses, or fruits within a truss (cf. Section 2). Investigations of El-Gizawy and Adams (1986), Adams and El-Gizawy (1988) and Sonneveld and Voogt (1991) indicate that the incidence of BER depends on where susceptible young fruits are situated when a triggering event occurs.

In contrast, mild stress may increase stress tolerance — and thus avoid incidence of BER — by temporarily promoting the formation of abscisic acid (ABA), known as a ‘stress hormone’, which reduces GA activity and thus promotes Ca^{2+} import (for references cf. Saure, 1998, p.140). Various stresses may substitute for each other, establishing some kind of ‘cross-adaptability’ to different stresses, a phenomenon widely recognised in plants (Wright, 1978). Unfortunately, most reports dealing with BER do not reveal the course of growing conditions during the experiment.

High temperature may act both by reducing stress tolerance because of a promotion of GA activity and, together with high irradiance and low RH, by increasing stress (Nilsen and Orcutt, 1966), especially when occurring during rapid fruit development (Sonneveld and Voogt, 1991; Ho et al., 1993).

As a result, the role of Ca^{2+} in the development of BER must be reassessed. Although being instrumental, it can no longer be considered a primary or an independent factor. This may explain why several authors found differing concentrations of Ca^{2+} associated with the incidence of BER, as noticed by Ward (1973) and Nonami et al. (1995), and supports the suggestion of Nonami et al. (1995) that Ca^{2+} deficiency in fruits may not be the direct cause of the occurrence of BER in tomato plants. Consequently, control of BER should not further focus mainly on attempts to keep the Ca^{2+} level in the fruit as high as possible, but should explore other avenues such as trying to maintain moderate shoot growth and to avoid or counteract sudden environmental stresses.

References

Adams, P., 1986. Mineral nutrition. In: Atherton, J.G., Rudich, J. (Eds.), The Tomato Crop: A Scientific Basis for Improvement. Chapman & Hall, London, pp. 281–334.

Adams, P., El-Gizawy, A.M., 1986. Effect of salinity and watering level on the calcium content of tomato fruit. Acta Hort. 190, 253–259.

Adams, P., El-Gizawy, A.M., 1988. Effect of calcium stress on the calcium status of tomatoes grown in NFT. Acta Hort. 222, 15–22.

Adams, P., Ho, L.C., 1992. The susceptibility of modern tomato cultivars to blossom-end rot in relation to salinity. J. Hort. Sci. 67, 827–839.

Adams, P., Ho, L.C., 1993. Effects of environment on the uptake and distribution of calcium in tomato and on the incidence of blossom-end rot. Plant Soil 154, 127–132.

Adams, P., Ho, L.C., 1995. Uptake and distribution of nutrients in relation to tomato fruit quality. Acta Hort. 412, 374–387.

Albahou, M., 1999. Alternative greenhouse tomato production system. Ph.D. Thesis. Oregon State University, Oregon, 223 pp.

Al-Dolimy, I.M., Al-Ani, A.M., 1987. Effects of calcium and nitrogen on blossom-end rot of tomato fruit under unheated plastic houses. Iraqi J. Agric. Sci., Zanco 5, 65–75 (from Hort. Abstr. 58 (1988) 335).

Bangerth, F., 1973. Investigations upon Ca related physiological disorders. Phytopath. Z. 77, 20–37.

Banuelos, G.S., Offermann, G.P., Seim, E.C., 1985. High relative humidity promotes blossom-end rot on growing tomato fruit. HortScience 20, 894–895.

Barke, R.E., 1968. Absorption and translocation of calcium foliar sprays in relation to the incidence of blossom-end rot in tomatoes. Queensland J. Agric. Anim. Sci. 25, 179–197.

Barke, R.E., Menary, R.C., 1971. Calcium nutrition of the tomato as influenced by total salts and ammonium nutrition. Aust. J. Exp. Agric. Anim. Husbandry 11, 562–569.

Barker, A.V., Ready, K.M., 1994. Ethylene evolution by tomatoes stressed by ammonium nutrition. J. Am. Soc. Hort. Sci. 119, 706–710.

Bar-Tal, A., Pressman, E., 1996. Root restriction and potassium and calcium solution concentrations affect dry-matter production, cation uptake, and blossom-end rot in greenhouse tomato. J. Am. Soc. Hort. Sci. 121, 649–655.

Belda, R.M., Ho, L.C., 1993. Salinity effects on the network of vascular bundles during tomato fruit development. J. Hort. Sci. 68, 557–564.

Belda, R.M., Fenlon, J.S., Ho, L.C., 1996. Salinity effects on the xylem vessels in tomato fruit among cultivars with different susceptibilities to blossom-end rot. J. Hort. Sci. 71, 173–179.

Bradfield, E.G., Guttridge, C.G., 1984. Effects of night-time humidity and nutrient solution concentration on the calcium content of tomato fruit. Scientia Hort. 22, 207–212.

Brown, M.M., Ho, L.C., 1993. Factors affecting calcium transport and basipetal movement in tomato fruit in relation to blossom-end rot. J. Exp. Bot. 44, 1111–1117.

Caliandro, A., 1971. Influenca del volume stagionale, variato in funzione del turno irrigua, sulla produzione del pomodoro da pelati. Riv. Agronomia 5, 215–222 (from Hort. Abstr. 42 (1972) 7912).

Cardoso, M.O., de Queiróz, M.A., Souza, R.F., 1995. Incidência de podridão apical em cinco cultivares de tomateiro cultivados em solo com três níveis de cálcio. Hort. Brasileira 13, 172–175 (from Hort. Abstr. 66 (1996) 7789).

Cerda, A., Bingham, F.T., Labanauskas, C.K., 1979. Blossom-end rot of tomato fruit as influenced by osmotic potential and phosphorous concentrations of nutrient solution media. J. Am. Soc. Hort. Sci. 104, 236–239.

Černe, M., 1990. Different kinds and levels of nitrogen nutrition in tomatoes. Acta Hort. 277, 179–182.

Chamberlain, E.E., 1933. Blossom-end rot of tomatoes. NZ J. Agric. 46, 293–296.

Chiu, T.-F., Bould, C., 1976. Effects of shortage of calcium and other cations on ^{45}Ca mobility, growth and nutritional disorders of tomato plants (*Lycopersicon esculentum*). J. Sci. Food Agric. 27, 969–977.

Cho, I.-H., Woo, Y.-H., Lee, E.-H., Kim, H.-J., 1997. Changes in cuticular transpiration and calcium content of tomato fruits and prevention of blossom-end rot through environmental control. J. Korean Soc. Hort. Sci. 38, 98–102.

Cho, I.-H., Lee, E.-H., Kim, T.-Y., Woo, Y.-H., Kwon, Y.-S., 1998. Effects of high humidity on occurrence of tomato blossom-end rot. J. Korean Soc. Hort. Sci. 39, 247–249.

Chrétien, S., Gosselin, A., Dorais, M., 2000. High electrical conductivity and radiation-based water management improve fruit quality of greenhouse tomatoes grown in rockwool. HortScience 35, 627–631.

Cuartero, J., Fernández-Muñoz, R., 1999. Tomato and salinity. Scientia Hort. 78, 83–125.

de Kreij, C., 1996. Interactive effects of air humidity, calcium and phosphate on blossom-end rot, leaf deformation, production and nutrient contents of tomato. J. Plant Nutr. 19, 361–377.

DeKock, P.C., Hall, A., Inkson, R.H.E., Robertson, R.A., 1979. Blossom-end rot in tomatoes. J. Sci. Food Agric. 30, 508–514.

DeKock, P.C., Hall, A., Boggie, R., Inkson, R.H.E., 1982a. The effect of water stress and form of nitrogen on the incidence of blossom-end rot in tomatoes. J. Sci. Food Agric. 33, 509–515.

DeKock, P.C., Inkson, R.H.E., Hall, A., 1982b. Blossom-end rot of tomato as influenced by truss size. J. Plant Nutr. 5, 57–62.

Drews, M., Frank, R., 1987. Der Einfluß des Kalziumgehaltes des Wassers auf die Pflanzenentwicklung von Tomate und Gurke beim NFT-Verfahren. Archiv Gartenbau 35, 417–424.

Ehret, D.L., Ho, L.C., 1986. Translocation of calcium in relation to tomato fruit growth. Ann. Bot. 58, 679–688.

El-Beltagy, A.S., Patrick, J.P., Hewett, E.W., Hall, M.A., 1976. Endogenous plant growth regulator levels in tomato fruits during development. J. Hort. Sci. 51, 15–30.

El-Gizawy, A.M., Adams, P., 1986. Effect of temporary calcium stress on the calcium status of tomato fruit and leaves. Acta Hort. 178, 37–43.

Evans, H.J., Troxler, R.V., 1953. Relation of calcium nutrition to the incidence of blossom-end rot in tomatoes. Proc. Am. Soc. Hort. Sci. 61, 346–352.

Fletcher, R.A., Gilley, A., Sankhla, N., Davis, T.D., 2000. Triazoles as plant growth regulators and stress protectants. Hort. Rev. 24, 55–138.

Franco, J.A., Bañón, S., Madrid, R., 1994. Effects of protein hydrolysate applied by fertigation on the effectiveness of calcium as a corrector of blossom-end rot in tomato cultivated under saline conditions. Scientia Hort. 57, 283–292.

Franco, J.A., Pérez-Saura, P.J., Durán, A., 1998. Calcio para corregir la necrosis apical en tomate. Horticultura 126, 90–92 (from Hort. Abstr. 69 (1999) 2226).

Franco, J.A., Pérez-Saura, P.J., Fernández, J.A., Parra, M., García, A.L., 1999. Effect of two irrigation rates on yield, incidence of blossom-end rot, mineral content and free amino acid levels in tomato cultivated under drip irrigation using saline water. J. Hort. Sci. Biotechnol. 74, 430–435.

Geraldson, C.M., 1957. Control of blossom-end rot of tomatoes. Proc. Am. Soc. Hort. Sci. 69, 309–317.

Gerard, C.J., Hipp, B.W., 1968. Blossom-end rot of 'Chico' and 'Chico Grande' tomatoes. Proc. Am. Soc. Hort. Sci. 93, 521–531.

Göring, H., Mardanov, A.A., 1976. Beziehungen zwischen dem K^+/Ca^{++} — Verhältnis im Gewebe und der Wirkung von Cytokininen in höheren Pflanzen. Biol. Rundschau 14, 177–189.

Grattan, S.R., Grieve, C.M., 1999. Salinity-mineral nutrient relations in horticultural crops. Scientia Hort. 78, 127–157.

Greenleaf, W.H., Adams, F., 1969. Genetic control of blossom-end rot disease in tomatoes through calcium metabolism. J. Am. Soc. Hort. Sci. 94, 248–250.

Grindal, G., Moe, R., 1997. The role of gibberellin and phytochrome in DIF mediated stem elongation. Acta Hort. 435, 87–104.

Grünzweig, J.M., Rabinowitch, H.D., Katan, J., Wodner, M., Ben-Tal, Y., 2000. Involvement of endogenous gibberellins in the regulation of increased tomato shoot growth in solarized soil. Plant Growth Regul. 30, 233–239.

Guttridge, C.G., Bradfield, E.G., 1983. Root pressure stops blossom-end rot. Grower 100 (6), 25–26.

Hadi, M.S., Conway, W.S., Sams, C.E., 1996. Effects of calcium on yield and incidence of blossom-end rot of three tomato cultivars (abstract). HortScience 31, 672.

Hall, D.A., 1977. Some effects of varied calcium nutrition on the growth and composition of tomato plants. Plant Soil 48, 199–211.

Hartman, P.L., Mills, H.A., Jones Jr., J.B., 1986. The influence of nitrate: ammonium ratios on growth, fruit development, and element concentration in 'Floradel' tomato plants. J. Am. Soc. Hort. Sci. 111, 487–490.

Ho, L.C., 1989. Environmental effects on the diurnal accumulation of ^{45}Ca by young fruit and leaves of tomato plants. Ann. Bot. 63, 282–288.

Ho, L.C., 1999. The physiological basis for improving tomato fruit quality. Acta Hort. 487, 33–40.

Ho, L.C., Adams, P., 1989. Calcium deficiency — a matter of inadequate transport to rapidly growing organs. Plants Today (November/December), 202–207.

Ho, L.C., Belda, R., Brown, M., Andrews, J., Adams, P., 1993. Uptake and transport of calcium and the possible causes of blossom-end rot in tomato. J. Exp. Bot. 44, 509–518.

Ho, L.C., Adams, P., Li, X.Z., Shen, H., Andrews, J., Xu, Z.H., 1995. Responses of Ca-efficient and Ca-inefficient tomato cultivars to salinity in plant growth, calcium accumulation and blossom-end rot. J. Hort. Sci. 70, 909–918.

Ikeda, H., Osawa, T., 1988. The effects of NO_3/NH_4 ratios and temperature of the nutrient solution on growth, yield and blossom-end rot incidence in tomato. J. Jpn. Soc. Hort. Sci. 57, 62–69.

Kafkaki, U., 1990. Root temperature, concentration and the ratio NO_3^-/NH_4^+ effect on plant development. J. Plant Nutr. 13, 1291–1306.

Kheshem, S.A., Kochan, W.J., Boe, A.A., Everson, D.O., 1988. Calcium translocation and tomato plant and fruit responses to molybdenum and daminozide. HortScience 23, 582–584.

Kinet, J.M., Peet, M.M., 1997. Tomato. In: Wien, H.C. (Ed.), The Physiology of Vegetable Crops. CAB International, Wallingford, UK, pp. 207–258.

Kirkby, E.A., 1979. Maximizing calcium uptake by plants. Commun. Soil Sci. Plant Anal. 10, 89–113.

Lee, E.H., Park, S.K., Kim, K.Y., 1991. The effect of NO_3-N to NH_4-N ratio on the growth of several garden fruits in hydroponics. 2. The effect of NO_3-N to NH_4-N ratio on the growth, mineral content and yield of tomato. Res. Rep. Rural Develop. Administr. Hort. 33, 1–6 (from Hort. Abstr. 63 (1993) 2760).

Lyon, C.B., Beeson, K.C., Barrentine, M., 1942. Macro-element nutrition of the tomato plant as correlated with fruitfulness and occurrence of blossom-end rot. Bot. Gaz. 103, 651–667.

Manishi, K., Fukumoto, Y., Yoshida, T., 1996. Effects of application of compost on growth and quality of tomatoes under water stress due to root-zone restriction. J. Jpn. Soc. Hort. Sci. 67, 257–264.

Marschner, H., 1983. General introduction to the mineral nutrition of plants. In: Läuchli, A., Bieleski, R.L. (Eds.), Encyclopedia of Plant Physiology NS, Vol. 15A. Springer, Berlin, pp. 4–60.

Maynard, D.N., Barham, W.S., McCombs, C.L., 1957. The effect of calcium nutrition of tomatoes as related to the incidence and severity of blossom-end rot. Proc. Am. Soc. Hort. Sci. 69, 318–322.

Millikan, C.R., Bjarnason, E.N., Osborn, R.K., Hanger, B.C., 1971. Calcium concentration in tomato fruits in relation to the incidence of blossom-end rot. Aust. J. Exp. Agric. Anim. Husbandry 11, 570–575.

Mohamed, A.A., Hanna, K.R., Omar, K.A., 1989. Effect of soil water regime and calcium level on growth, yield and blossom-end rot incidence of tomato plant (*Lycopersicon esculentum* Mill.). Mesopotamia J. Agric. 21, 65–78.

Monselise, S.P., Varga, A., Bruinsma, J., 1978. Growth analysis of the tomato fruit, *Lycopersicon esculentum* Mill. Ann. Bot. 42, 12145–12147.

Mu, Y.H., Lu, D.Z., Rao, L.H., 1992. Studies on the relation between Ca-deficiency during flowering and blossom-end rot in tomato fruits. Acta Hort. Sinica 19, 251–255 (from Hort. Abstr. 64 (1994) 6751).

Murray, S.A., Spurr, A.R., Paulson, K.N., 1972. Electron probe analysis of Ca, Mg, and K in the fruit of the tomato, *Lycopersicon esculentum*, with reference to blossom-end rot. Agron. J. 64, 586–588.

Nilsen, E.T., Orcutt, D.M., 1966. Physiology of Plants Under Stress. Wiley, New York.

Nonami, H., Fukuyama, T., Yamamoto, M., Yang, L., Hashimoto, Y., 1995. Blossom-end rot of tomato plants may not be directly caused by calcium deficiency. Acta Hort. 396, 107–114.

Nukaya, A., Goto, K., Jang, H., Kano, A., Ohkawa, K., 1995. Effect of NH_4-N level in the nutrient solution on the incidence of blossom-end rot and gold specks on tomato fruit grown in rockwool. Acta Hort. 401, 381–388.

Obreza, T.A., Pitts, D.J., McGovern, R.J., Spreen, T.H., 1996. Deficit irrigation of micro-irrigated tomato affects yield, fruit quality, and disease severity. J. Prod. Agric. 9, 270–275.

Paiva, E.A.S., Martinez, H.E.P., Casali, V.W.D., Padilha, L., 1998a. Occurrence of blossom-end rot in tomato as a function of calcium dose in the nutrient solution and air relative humidity. J. Plant Nutr. 21, 2663–2670.

Paiva, E.A.S., Sampaio, R.A., Martinez, H.E.P., 1998b. Composition and quality of tomato fruit cultivated in nutrient solutions containing different calcium concentrations. J. Plant Nutr. 21, 2653–2661.

Palta, J.P., 1990. Stress interactions at the cellular and membrane levels. HortScience 25, 1377–1381.

Petersen, K.K., Willumsen, J., 1992. Effects of root zone warming and season on blossom-end rot and chemical composition of tomato fruit. Danish J. Plant Soil Sci. 96, 489–498.

Pill, W.G., Lambeth, V.N., 1980. Effects of soil water regime and nitrogen form on blossom-end rot, yield, water relations, and elemental composition of tomato. J. Am. Soc. Hort. Sci. 105, 730–734.

Pill, W.G., Lambeth, V.N., Hinckley, T.M., 1978. Effects of nitrogen form and level on ion concentrations, water stress, and blossom-end rot incidence in tomato. J. Am. Soc. Hort. Sci. 103, 265–268.

Plese, L.P.M., Tiritan, C.S., Yassuda, E.I., Prochnow, L.I., Corrente, J.E., Mello, S.C., 1998. Efeitos das aplicações de cálcio e de boro na ocorrência de podicão apical e produção de tomate em estufa. Scient. Agric. 55, 144–148 (from Hort. Abstr. 69 (1999) 3188).

Pulupol, L.U., Behboudian, M.H., Fisher, K.J., 1996. Growth, yield, and postharvest attributes of glasshouse tomatoes produced under deficit irrigation. HortScience 32, 926–929.

Puritch, G.S., Barker, A.V., 1967. Structure and function of tomato leaf chloroplasts during ammonium toxidity. Plant Physiol. 42, 1229–1238.

Raleigh, S.M., Chucka, J.A., 1944. Effect of nutrient ratio and concentration on growth and composition of tomato plants and on the occurrence of blossom-end rot of the fruit. Plant Physiol. 19, 671–678.

Richards, D., 1978. Root–shoot interactions: functional equilibria for nutrient uptake in peach (*Prunus persica* L. Batsch.). Ann. Bot. 42, 1039–1043.

Robbins, R., 1937. Relation of nutrient salt concentration to growth of the tomato and to the incidence of blossom-end rot of the fruit. Plant Physiol. 12, 21–50.

Rood, S.B., Zanewich, K.P., Bray, D.F., 1990. Growth and development of *Brassica* genotypes differing in endogenous gibberellin content. II. Gibberellin content, growth analyses and cell size. Physiol. Plant. 79, 679–685.

Sánchez Conde, M.P., Felipe, M.R., 1983. Composición mineral y estudio estructural de los tejidos sanos y afectados de podedumbre apical en dos variedades de tomate (*Lycopersicum esculentum* L.). Anales Edafología y Agrobiol. 42, 831–846 (from Hort. Abstr. 54 (1984) 6255).

Saure, M.C., 1998. Causes of the tipburn disorder in leaves of vegetables. Scientia Hort. 76, 131–147.

Selby, A.D., 1896. Investigations of plant diseases in forcing house and garden. Ohio Agric. Exp. Sta. Bull. 73, 146–221(from Spurr, 1959).

Shaykewich, C.F., Yamaguchi, M., Campbell, J.D., 1971. Nutrition and blossom-end rot of tomatoes as influenced by soil water regime. Can. J. Plant Sci. 51, 505–511.

Shear, C.B., 1975. Calcium-related disorders of fruits and vegetables. HortScience 10, 361–365.

Simon, E.W., 1978. The symptoms of calcium deficiency in plants. New Phytol. 80, 1–15.

Sonneveld, C., Voogt, W., 1991. Effect of Ca-stress on blossom-end rot and Mg-deficiency in rockwool grown tomato. Acta Hort. 294, 81–88.

Sperry, W.J., Davis, J.M., Sanders, D.C., 1996. Soil moisture and cultivar influence cracking, blossom-end rot, zippers, and yield of staked fresh-market tomatoes. HortTechnol. 6, 21–24.

Spurr, A.R., 1959. Anatomical aspects of blossom-end rot in the tomato with special reference to calcium nutrition. Hilgardia 28, 269–295.

Stanghellini, C., van Meurs, W.T.M., Corver, F., van Dullemen, E., Simonse, L., 1998. Combined effect of climate and concentration of the nutrient solution on a greenhouse tomato crop. II. Yield quantity and quality. Acta Hort. 458, 231–237.

Suge, H., 1978. Growth and gibberellin production in *Phaseolus vulgaris* as affected by mechanical stress. Plant Cell Physiol. 19, 1557–1560.

Taylor, G.A., Smith, C.B., 1957. The use of plant analysis in the study of blossom-end rot of tomato. Proc. Am. Soc. Hort. Sci. 70, 341–349.

Terabayasi, S., Miyaoi, Y., Takahata, T., Namiki, T., 1988. Calcium concentration in tomato fruits grown in water culture in relation to the incidence of blossom-end rot. Scient. Rep. Kyoto Prefect. Univ. Agric. 40, 8–14.

van Goor, B.J., 1968. The role of calcium and cell permeability in the disease blossom-end rot of tomatoes. Physiol. Plant. 21, 1110–1121.

van Goor, B.J., 1974. Influence of restricted water supply on blossom-end rot and ionic composition of tomatoes grown in nutrient solution. Comm. Soil Sci. Plant Nutr. 5, 13–24.

van Ieperen, W., 1996. Effects of different day and night salinity levels on vegetative growth, yield and quality of tomato. J. Hort. Sci. 71, 99–111.

Vettakkorumakanav, N.N., Falk, D., Saxena, P., Fletcher, R.A., 1999. A crucial role for gibberellins in stress protection of plants. Plant Cell Physiol. 40, 542–548.

Vielemeyer, H.-P., Weissert, P., 1990. Diagnose des Ca-Ernährungszustandes und der Blütenendfäule bei Buschtomate. Gartenbauwiss. 55, 209–212.

Wada, T., Ikeda, H., Ikeda, M., Furukawa, H., 1996. Effects of foliar application of calcium solutions on the incidence of blossom-end rot of tomato fruit. J. Jpn. Soc. Hort. Sci. 65, 553–558.

Ward, G.M., 1973. Causes of blossom-end rot of tomatoes based on tissue analysis. Can. J. Plant Sci. 53, 169–174.

Westerhout, J., 1962. Relation of fruit development to the incidence of blossom-end rot of tomatoes. Neth. J. Agric. Sci. 10, 223–234.

Wiersum, L.K., 1966. Calcium content of fruits and storage tissues in relation to the mode of water supply. Acta Bot. Neerlandica 15, 406–418.

Wilcox, G.E., Hoff, J.E., Jones, C.M., 1973. Ammonium reduction of calcium and magnesium content of tomato and sweet corn leaf tissue and influence on incidence of blossom end rot of tomato fruit. J. Am. Soc. Hort. Sci. 98, 86–89.

Willumsen, J., Petersen, K.K., Kaack, K., 1996. Yield and blossom-end rot of tomato as affected by salinity and cation activity ratios in the root zone. J. Hort. Sci. 71, 81–98.

Wright, S.T.C., 1978. Phytohormones and stress phenomena. In: Letham, D.S., Goodwin, P.B., Higgins, T.J.V. (Eds.), Phytohormones and Related Compounds — A Comprehensive Treatise, Vol. 2. Elsevier/Biomedical Press, Amsterdam, pp. 495–536.

Wui, M., Takano, T., 1995a. Effect of temperature and concentration of nutrient solution during the stage of the fruit development on the incidence of blossom-end rot in fruits of tomato, *Lycopersicon esculentum* L. Environ. Contr. Biol. 33, 7–14.

Wui, M., Takano, T., 1995b. Effect of air temperature, air flow and uniconazole spray on the incidence of blossom-end rot of tomato fruit. Environ. Contr. Biol. 33, 49–57.