Chapter 7

PHYSIOLOGY OF BORON STRESS IN COTTON

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INTRODUCTION

Boron is the most deficient essential micronutrient in cotton (*Gossypium hirsutium* L.) fields (Rosolem *et al*., 2001). Cotton has a relatively high requirement for B (Zhao and Oosterhuis, 2002), requiring an average of 340 g B ha^{-1} and exporting in seedcotton around 12% of the B accumulated in the plant (Rochester, 2007). Nutritional disorders caused by B deficiency in cotton are quite common in tropical soils, where soil organic matter and/or clay content are low (Rosolem *et al*, 2001), and in other areas of the world where B availability is significantly reduced in calcareous soils (Shorrocks, 1997). For instance, it was estimated that 37% of Indian soils are B deficient (Singh, 2009). Boron is prone to leach through the soil profile, depending on soil texture, another factor leading to B deficiency (Communar and Keren, 2006; Rosolem and Biscaro, 2007) as well as posing an environmental threat to water tables. Conversely, B toxicity to crops is most commonly found in arid and semi-arid regions or soils developed from marine sediments, as a consequence of high B concentration in irrigation water and use of high B compost material or fly ash (Nable *et al*., 1997). Hence, B deficiency or toxicity can be found throughout cotton growing regions worldwide.

The range between boron deficiency and toxicity is very narrow. It is known that B deficiency can significantly limit cotton yields without any visible foliage and flower symptoms, characterizing the occurrence of "hidden hunger" (Satya *et al*., 2009). Boron deficiency is not easily recognized even in foliar diagnosis, since cotton plants showing 11 mg kg^{-1} of B in the most recently mature leaves yielded the same dry matter as non-deficient plants, but the number of reproductive structures was lower (Rosolem *et al*., 1999). Reported B sufficiency levels in cotton leaves range from 16 to 80 mg kg^{-1} (Rosolem *et al*., 2001; Zhao and Oosterhuis, 2002), and toxicity may be observed with B concentrations over 100 mg kg^{-1} (Bergmann, 1992). Because of this narrow range, plant analysis is not a highly effective tool for monitoring plant B nutritional status and estimating plant response to fertilizers.

Despite positive yield responses to B applied either to the soil or sprayed directly on cotton leaves, a controversy remains as to when to apply B, as well as the best means of doing so. The low mobility of B in cotton phloem is an additional complication in this equation, because a temporary deficiency may lead to some yield loss. In this chapter the onset, development and physiology of B deficiency in cotton will be discussed aiming at a better understanding of the role of B in cotton production.

FUNCTIONS OF BORON IN PLANTS

The unusual nature of boron chemistry suggests the possibility of a wide variety of biological functions for the micronutrient. However, the exact metabolic functions are not yet fully understood (Hansch and Mendel, 2009). Boron is essential for the formation of meristematic tissues: its uptake is fast but its mobility in cotton plants is low. Most of the currently known processes involving B are based on its role in the formation of reversible diester bonds with *cis*-diol containing molecules, but it may play a role in membrane stabilization crosslinking glycoproteins, and may be also involved in their recruiting to membrane domains (Wimmer *et al*., 2009). Boron may stimulate or inhibit enzymes and participate in phenol metabolism avoiding toxicity problems (Römheld and Marschner, 1991).

One of the primary functions of B in higher plants is based on the formation of borate esters with apiose residues of rhamnogalacturonan II (RG-II) in the cell wall (Kobayashi *et al*., 1996), which is essential to its structure and function and contributes significantly to the control of cell wall porosity and strength (Fleischer *et al*., 1999, Ryden *et al*., 2003). Boron was reported to be involved in sugar transport, plant respiration, metabolism of RNA, carbohydrates and plant hormone (indole acetic acid) metabolism (Camacho-Cristótal *et al*., 2008). It promotes structural integrity of bio-membranes and the formation of lipid rafts. Since all these functions are fundamental to meristematic tissues, boron deficiency is predominantly damaging in actively growing organs such as shoot and root tips (Hansch and Mendel, 2009). The transport of chlorine and phosphorus are increased as a result of plasmalemma ATPase induction, and it has been shown that boron can stimulate proton pumping that causes hyperpolarization of the membrane potential (Camacho-Cristóbal, 2008). Hence, B may affect ionic absorption and its deficiency would decrease the uptake of several nutrients (Dugger, 1983).

No membrane-bound molecules interacting with B have been isolated so far, but deficiency symptoms point to additional functions of B in cell membranes. Binding of mitochondrial ATP synthase, several beta-glucosidases, a luminal binding protein and fructose bisphosphate aldolase to B was significantly reduced with B deprivation (Wimmer *et al*., 2009).

Boron is particularly important during the plant reproductive phase as pollen germination and growth of the pollen tube are impaired when B is deficient (Agarwala *et al*., 1981). In cotton, B deficiency during flowering and fruit formation increases shedding, decreasing fiber yields and also fiber quality (Miley *et al*., 1969; Rosolem and Costa, 2000). Given the rather high proportion of B present in the non-cell wall fraction of pollen and silk, the high B requirement for plant reproduction suggests an additional role for B other than in cell wall formation. However, the identity of non-cell wall B binding substrates in pollen and carpel tissue awaits further study. The higher sensitivity of plant reproduction to B deficiency is also related to weaker B transport into floral organs, especially where transpiration is suppressed in reproductive plant parts by enclosure of sheaths (e.g. wheat ear) or husks (e.g. maize ear) during the critical stage of development (Huang *et al*., 2009).

There is increasing evidence that B is required for the maintenance of the structure and functions of membranes and, especially, plasma membrane (Camacho-Cristobal *et al*., 2008). For example, B deficiency altered the membrane potential and reduced the activity of proton-pumping

ATPase in roots (Ferrol and Donaire 1992), and it has been also reported that B deficiency alters plasma membrane permeability for ions and other solutes (Cakmak *et al*., 1995). Therefore, B action in membranes might not be restricted to stabilizing membrane molecules with *cis*-diol groups, but also by regulating the expression of genes involved in membrane structure and function.

BORON UPTAKE AND MOBILITY

Boron is present in soil solution in several forms. However, at common soil pH values, the most abundant is the undissociated boric acid. It is accepted that it is the only essential nutrient that plants take up from soil as an uncharged molecule (Marschner, 1995). Boron in soil solution moves towards plant roots mainly through mass flow (Barber, 1966), then its uptake can be carried out by three different molecular mechanisms, depending on B availability: (i) passive diffusion across lipid bilayers, where B can cross membranes by a passive process to satisfy plant B requirements (Brown *et al*., 2002); (ii) facilitated transport by major intrinsic protein (MIP) channels; and (iii) an energy-dependent high affinity transport system induced in response to low B supply, which is mediated via BOR transporters (Tanaka and Fujiwara, 2008).

The first experimental evidence suggesting the involvement of channel proteins in B transport was provided by Dordas *et al*. (2000), when they described that B permeation across root plasma-membrane vesicles was partially inhibited by channel blockers. Another boric acid channel has been identified in *Arabidopsis* (AtNIP5;1), which belongs to the nodulin 26-like intrinsic proteins (NIP), subfamily of the MIPs family (Takano *et al*., 2006). At-NIP5;1 is localized and expressed in the plasma membrane of root epidermal, cortical, and endodermal cells, and it is upregulated in B-deficient roots, suggesting a crucial role of this channel for B uptake under low availability (Takano *et al*., 2006).

Physiological studies also have shown the occurrence of active B uptake by roots under low B conditions (Dannel *et al*., 2002). One BOR transporter (OsBOR1 in rice) has been suggested to be involved in the efficient uptake of B into root cells under B deficiency (Nakagawa *et al*., 2007). A BOR transporter was also indentified as capable of increasing B toxicity tolerance by pumping excess boric acid out of the cell (Miwa *et al*., 2007). Plants may be tolerant to B excess or deficiency through the expression of these transporters. Most of the results were obtained in model plants but could be applied to other plant species and may be helpful in developing crops tolerant either to B toxicity or deficiency (Miwa and Fujiwara, 2010).

More recently, accumulating evidence suggests that non-sugar-alcohol-producing plants can transport boric acid preferentially to young tissues. This translocation was detected under B limitation, but not under conditions of normal B supply, and B transporters and channels may be involved. The fact that this translocation occurs only under boron limitation suggests that plants are capable of sensing boron levels and regulating boron transport (Tanaka and Fujiwara, 2008).

The mobility of B within the plant is an important characteristic and is determined by the plant species and B availability. Knowledge of B mobility in plants is useful for the management of B application in agricultural systems where nutrient supply may be limiting or excessive. The remobilization is generally defined as the movement of nutrients from a plant tissue to another, through the phloem.

BORON IN COTTON

More than 90% of B in plants is found in cell walls, and if there is any B remobilization in cotton phloem, it is low (Rosolem and Costa, 2000). When cotton was exposed to a temporary deficiency and B was sprayed on new or old leaves, the responses varied. Boron applied to young immature leaves increased B concentration locally, with no further effects. However, despite the effects of B deficiency inhibiting meristematic growth and its low mobility within the plant, there was a positive response to B application to mature leaves. As there was no new development of cell walls to incorporate the nutrient, it could eventually be available for mobilization (Rosolem and Costa, 2000). The authors argued that foliar application of B to mature leaves may have prevented, at least in part, xylem malformation, and when the nutrient was replaced in the solution, the preservation of a better vascular system allowed for near-normal plant growth. Bogiani and Rosolem (unpublished) observed that B remobilization in cotton was low, but there were differences among cotton cultivars in mobilizing B from roots, stems and leaves to reproductive structures. Furthermore, when B concentration in the nutrient solution was low, but enough to avoid severe shedding, the reproductive structures received more B from other plant parts, but when there was plenty of B for plant growth, most of the nutrient accumulated in cotton leaves. Hence, B remobilization occurred under low B supply, and under high B supply, the nutrient was transported mainly in the transpiration stream, accumulating in organs with high transpiration rates. In China, it has been shown that B uptake by cotton roots is faster than B uptake by leaves and translocation (Xie *et al*., 1992). During vegetative growth, B is mobilized mainly to growing points and young leaves, whereas during reproductive growth, it is mobilized preferentially towards the main-stem leaves and leaves subtending reproductive structures. Though B is not easily remobilized from old leaves, it may be remobilized from photosynthetic active leaves (Xie *et al*., 1992).

These results are consistent with the findings of Tanaka and Fujiwara (2008) on B transport in non-sugar-alcohol-producing plants, mediated by B transporters and channels. For instance, OsBOR1, a B efflux transporter in rice was found to mediate efficient B translocation from root to shoot under B deficiency (Uraguchi *et al*., 2009). Conversely, a temporary deficiency of B leads to xylem malformation, which may decrease the translocation of B, carbohydrates, etc, to new tissues in cotton (Oliveira *et al*., 2006).

The possibility of some B translocation out of the leaves would explain some responses of cotton to foliar B application observed in the field in Brazilian acidic soils (Carvalho *et al*., 1996; Ferreira and Carvalho, 2005) and in calcareous soils in Greece (Dordas, 2006), among others.

Boron Deficiency

The appearance and severity of B deficiency symptoms in cotton are a function of soil nutrient availability, time of plant exposure to deficiency and cultivar (Silva *et al*., 1982; Rosolem *et al*., 1999). Considering the role of B in cell wall and membrane formation and in carbohydrate transport (Tanada, 1983; Agarwala *et al*., 1981), the first symptoms appear in young parts of

the plant, in vessel tissues and reproductive organs (Hinkle and Brown, 1968). As a result of the critical role of B in expanding tissues and its limited mobility in cotton, it must be supplied continuously throughout the plant's life. If it is withdrawn from the nutrient medium, even for a short period, a deficiency is established and reproductive structures shed (Rosolem and Costa, 2000; Oliveira *et al*., 2006). When B is replaced in the nutrient solution after a temporary deficiency, full growth recovery does not occur and, therefore, a temporary B deficiency causes permanent damage to the plant (Rosolem and Costa, 2000). This is important in the field because B uptake and transport to new tissues depends on the transpiration stream, which may be impaired by a very low evaporative demand, stomata closure in hot, dry days, low temperatures, etc. This may lead to a temporary B deficiency in cotton, even when there is plenty of soil B available.

Boron deficiency can result in shorter fruit branches and poor fruit set, deformed, chlorotic leaves and development of dark green bands (often excessively hairy) on the petioles and stems (Hinkle and Brown, 1968; Rosolem and Bastos, 1997). The pith in such regions of the petioles is characteristically necrotic, the terminal bud often dies and many lateral branches develop, which have short internodes and enlarged nodes. Under B deficiency there is significant square and boll shedding (Zhao and Oosterhuis, 2002; Rosolem and Bastos, 1997, Oliveira *et al*., 2006). Abnormal fibers have also been observed in cultured ovules (Birnbaum *et al*., 1974) and shorter fibers in the field (Sankaranarayanan *et al*., 2010). The petals are frequently crumpled and misshapen. Discoloration of the extra-floral nectaries is quite common. Cracks may develop on the stems, at the base of the squares or bolls, and there may be some exudation (Shorrocks 1997). The accumulation of chlorogenic and caffeic acids caused by B deficiency inhibits the *enzyme auxi*n oxidase, resulting in auxin accumulation in the plant tissue (Gupta, 2006), over proliferation of the cambium (Oliveira *et al*., 2006), and a fast and unproportional elongation and collapse of the nearby cells (Srivastava and Gupta, 1996). Therefore, morphological changes during B deficiency development may be due to auxin accumulation in the tissue.

Although B deficiency decreases photosynthesis (Zhao and Oosterhuis, 2003), sugars and starch accumulate in leaves of deficient plants (Dugger, 1983). According to Dugger (1983), B deficiency decreases photosynthesis by decreasing the activity of nitrogenous compounds such as uracil, a precursor of UDPG (uridine diphosphate glucose), which is involved in sucrose synthesis (Birnbaum *et al*., 1977). With less UDPG, translocation is decreased and starch and photoassimilate accumulate. However, it is worth noting that while B deficiency increased nonstructural carbohydrates in deficient cotton leaf blades and depressed photosynthate export from leaves, leaf intercellular CO_2 concentration of cotton plants changed little with increasing leaf-blade B concentrations (Zhao and Oosterhuis, 2003).

It has often been observed that reproductive growth, especially flowering, fruit and seed set and seed yield, is more sensitive to B deficiency than vegetative growth. This is due to several reasons such as: each flower develops over a very narrow window of time, some reproductive structures (e.g., pollen chamber, embryo sac) have poorer access to the vascular system than any vegetative organ (van Iersel *et al*., 1994), and sexual reproduction involves a large number of specialized cell types, many of which have distinctive cell walls (Huang *et al*., 2009). Phloem elements in the peduncle vascular cylinder of B-deficient plants have no clear differentiation and the number of vascular bundles of the petiole and peduncle is decreased in B-deficient

cotton and the few xylem elements formed are disorganized. Moreover, in B-deficient cotton plants, the xylem vessel walls were thickened and vessels were observed in lower number, with an irregular perimeter (Oliveira *et al*., 2006). Boron deficiency during the early growth of cotton has been reported to decrease the leaf CO_2-exchange rate, increase leaf blade non-structural carbohydrate concentration, and decrease photosynthate export out of leaves (Zhao and Oosterhuis, 2002). The decrease in carbohydrate transport to fruiting sites results in square and flower abscission (Rosolem *et al*., 2001; Zhao and Oosterhuis, 2002, 2003). Squares remaining are deformed, with chlorotic bracts and stunted corolla (Silva *et al*., 1982).

With fewer reproductive structures the sink for carbohydrates is decreased and excess carbohydrate is available for vegetative growth, resulting in rank-growth, self-shading, delayed maturity and less yield.

Boron Toxicity

Boron toxicity is a serious concern for sustainable crop production in irrigated agriculture throughout the world. Boron is transported within the plant mainly in the transpiration stream through the xylem and accumulates at the leaf tips and margins of older leaves (Bennet, 1993; Sestren and Kroplin, 2009). Hence, toxicity symptoms (yellowing and necrosis in patches between veins and tips and margins of leaves) first appear on older leaves. As severity of the disorder increases, the chlorotic areas later become necrotic, and the necrosis progresses from the leaf tips and margins towards the midrib and base of the leaf (Ahmed *et al*., 2008). This gives the leaf a scorched appearance and eventually the entire leaf dies and falls from the plant (Silva *et al*., 1979). Cassman (1993) reported that in cotton the necrotic areas of the leaves suffering B toxicity contained 2700-6400 mg B kg^{-1}, and Silva *et al*. (1979) observed that cotton plants showing symptoms of B toxicity contained over 590 mg B kg^{-1}. Boron concentration may vary 100 fold within a single leaf, hence, results of foliar diagnosis represent only an average of the actual concentration. Boron concentration usually increases with leaf age (Brown and Shelp, 1997) and in some cases may reach toxic levels in old leaves and be deficient in newly developed leaves (Oertli, 1994).

Boron accumulation in old leaves could unbalance cell wall constituents leading to tissue necrosis and death (Sestren and Kroplin, 2009). In excess, B concentration increases in the cytosol, causing metabolic dysfunctions through the formation of complexes with NAD^+ and eventually affecting the RNA structure (Loomis and Durst, 1992). However, toxicity of mature tissues may be due rather to the accumulated retardation of many cellular processes, enhanced in light by photo-oxidative stress (Reid *et al*., 2004).

Boron toxicity negatively affects very diverse processes in vascular plants, such as photosynthetic rates, leaf chlorophyll contents, root cell division and lignin and suberin levels (Reid, 2007). Accordingly, a reduced growth of shoots and roots is typical of plants exposed to high B levels (Nable *et al*., 1990). According to Camacho-Cristóbal (2008) three main causes have been proposed taking into account our knowledge of B chemistry (i.e. the ability of B to bind compounds with two hydroxyl groups in the *cis*-configuration): (i) alteration of cell wall structure; (ii) metabolic disruption by binding to the ribose moieties of molecules such as adenosine

triphosphate (ATP), nicotinamide adenine dinucleotide, (reduced form) (NADH) or nicotinamide adenine dinucleotide phosphate, (reduced form) (NADPH); and (iii) disruption of cell division and development by binding to ribose, either as the free sugar or within RNA (Reid *et al*., 2004).

There are genotypic differences in tolerance to high B, e.g. in wheat, characterized by a decreased B concentration in leaf tissues (Nable *et al*., 1990), probably due to a reduced uptake of B. The basis for B-tolerancein plants has been explained by plant ability to efflux B, and two models have been proposed for this mechanism: borate exchange or an anion channel (Hayes and Reid, 2004). BOR1 is an efflux-type borate transporter required for the transport of B from roots to shoots under low B supply (Takano *et al*., 2002). However, in the presence of toxic levels of B, BOR1 is degraded via endocytosis (Takano *et al*., 2005), and its' over expression does not result in better plant growth (Miwa *et al*., 2006), suggesting that BOR1 is not involved in B tolerance. More recently it was found that overproduction of another B transporter in *A. thaliana*, BOR4-GFP, improved growth under conditions of B toxicity through B efflux (Miwa *et al*., 2007). This enhanced B efflux from the roots of crop plants is expected to result in improved crop productivity in B-toxic soils. Another gene, Bot1 (a BOR1 ortholog), has been identified as responsible for B-toxicity tolerance in barley (Sutton *et al*., 2007), and it has been suggested that the BOR2 gene encodes an efflux type borate transporter responsible for tolerance to B toxicity in wheat and barley (Reid, 2007).

SUMMARY

Boron deficiency and toxicity can be observed in many cotton regions worldwide. Considering the low remobilization of B within cotton plants, even a temporary deficiency occurring with enough available B in soil may lead to some degree of reproductive structure shedding, either decreasing cotton yields or delaying plant maturity and increasing costs. Although foliar fertilization has not been regarded as effective in correcting B deficiency in low B soils, it may help to overcome a temporary B deficiency, with some improvement in cotton yields in tropical soils (Rosolem *et al*., 2001) and significant increases in Mediterranean soils (Dordas, 2006). This would only be possible as a consequence of some B translocation in cotton. Over 90 % of B is bound to cell walls and membranes, while some of the remaining 10 % could be available for remobilization. In addition, B applied to mature leaves does not bind to the previously formed cell walls and could also be available for mobilization within the plant. Therefore, some B could be mobilized from mature leaves into actively growing reproductive organs via phloem, as recently demonstrated in white lupin (Huang *et al*., 2008). This remobilization was promoted by specific boron transporters. This was not demonstrated in cotton, but accumulating evidence suggests that non-sugar-alcohol-producing plants can transport boric acid preferentially to young tissues, which would explain the observed responses of cotton to foliar applied B. Moreover, some differences have been observed in B remobilization among cotton cultivars. In addition to B fertilization, the selection of cultivars or the introduction of the ability to remobilize B would be important steps in better dealing with B deficiency and toxicity in cotton. The natural genetic variability in this trait and the introduction of B transporter genes are tools to be used in plant breeding towards improved B use in cotton.

REFERENCES

Agarwala, S.C., P.N. Sharma, C. Charttejee, C.P. Sharma. 1981. Development and enzymatic changes during pollen development in boron deficient maize plants. J. Plant Nutr. 3:329-336.

Ahmed, N., M. Abid, F. Ahmad. 2008. Boron toxicity in irrigated cotton (*Gossypium hirsutum* L.). Pakistan J. Bot. 40:2443-2452.

Barber, S.A. 1966. The role of root interception, mass-flow and diffusion in regulating the uptake of ions from soils. pp.39-45. *In:* International Atomic Energy Agency, Limiting steps in ion uptake by plants from soil, Vienna, (IAEA. Technical Report Series, 65).

Bennet, W.F. 1993. Nutrient deficiencies and toxicities in crop plants. p. 202. APS Press, St. Paul, Minn.

Bergmann, W. 1992. Colour Atlas: Nutritional disorders of Plants. pp. 204-239. Gustav Fisher, N.Y.

Birnbaum, E.H., W.M. Dugger, C.A. Beasley. 1977. Interaction of boron with components of nucleic acid metabolism in cotton ovules culture *in vitro*. Plant Physiol. 59:1034-1038.

Birnbaum, E.H., C.A. Beasley, W.M. Dugger. 1974. Boron deficiency in underfertilized cotton (*Gossypium hirsutum*) ovules grown *in vitro*. Plant Physiol. 54:931–935.

Blevins, D.G. 2009. More hidden hunger: Special nutrient needs of plants based on their structure and function. UC Davis: Proc. International Plant Nutrition Colloquium XVI. Retrieved from: http://escholarship.org/uc/item/7pz1g68s

Brown, P.H., B.J. Shelp, 1997. Boron mobility in plants. Plant Soil. 193:85–101.

Brown, P.H, N. Bellaloui M.A. Wimmer, E.S. Bassil, J. Ruiz, H. Hu, H. Pfeffer, D. Dannel, V. Römheld. 2002. Boron in plant biology. Plant Biol. 4:205–223.

Cakmak, I., H. Kurz, H. Marschner. 1995. Short-term effects of boron, germanium and high light intensity on membrane permeability in boron deficient leaves of sunflower. Physiol. Plant. 95:11–18.

Camacho-Cristóbal, J.J., J. Rexach, A. González-Fontes. 2008. Boron in Plants: Deficiency and Toxicity. J. Integrative Plant Biol. 50:1247–1255.

Carvalho, L.H., N.M. Silva, M.O.C. Brasil Sobrinho, J.I. Kondo, E.J. Chiavegato. 1996. Aplicação de boro no algodoeiro, em cobertura e em pulverização foliar. Revista Brasileira de Ciência do Solo. 20:265-266.

Cassman, K.G. 1993. Cotton. p. 202. *In:* W.F. Bennet (ed.). Nutrient Deficiencies and Toxicities in Crop Plants. ASP Press, Amer. Phytopath. Soc., St. Paul, Minn.

Communar, G. and R. Keren. 2006. Rate-limited boron transport in soils: the effect of soil texture and solution pH. Soil Sci. Soc. Amer. J. 70:882-892.

Dannel, F., H. Pfeffer, V. Römheld. 2002. Update on boron in higher plant-uptake, primary translocation and compartmentation. Plant Biol. 4:193–204.

Dordas, C., M.J. Chrispeels, P.H. Brown. 2000. Permeability and channel-mediated transport of boric acid across membrane vesicles isolated from squash roots. Plant Physiol. 124:1349–1361.

Dordas, C. 2006. Foliar boron application affects lint and seed yield and improves seed quality of cotton grown on calcareous soils. Nutr. Cycl. Agroecosyst. 76:19–28.

Dugger, W.M. 1983. Boron in metabolism. p. 626 *In*: Encyclopedia of Plant Physiol. Springer--Verlag, N.Y.

Ferreira, G.B. and M.C.S. Carvalho. 2005. Adubação do algodoeiro no Cerrado: Com resultados de pesquisa em goiás e Bahia. Campina Grande: Embrapa Algodão, 71p. (Documentos, 138).

Ferrol, N. and J.P. Donaire. 1992. Effect of boron on plasma membrane proton extrusion and redox activity in sunflower cells. Plant Sci. 86:41–47.

Fleischer, A., M.A. O'Neill, R. Ehwald. 1999. The pore size of nongraminaceous plant cell walls is rapidly decreased by borate ester cross-linking of the pectic polysaccharide rhamnogalacturonan II. Plant Physiol. 121:829–838.

Gupta, U.C. 2006. Boron. pp.241-277. *In*: A.V. Barker and D.J. Pilbean, (eds.). Handbook of Plant Nutrition. Taylor and Francis Publications, Boca Raton, Fla.

Hansch, R. and R.R. Mendel. 2009. Physiological functions of mineral micronutrients (Cu, Zn, Mn, Fe, Ni, Mo, B, Cl). Current Opinion in Plant Biology. 12:259–266.

Hayes, J.E. and R.J. Reid. 2004. Boron tolerance in barley is mediated by efflux of boron from the roots. Plant Physiol. 136:376–3382.

Hinkle, D.A. and A.L. Brown. 1968. Secondary nutrients and micronutrients. pp.281–320. *In*: F.C. Elliot *et al*. (eds.), Advances in Production and Utilization of Quality Cotton: Principles and Practices. Iowa State University Press, Ames, Iowa.

Huang, L., R.W. Bell, B. Dell. 2008. Evidence of phloem boron transport in response to interrupted boron supply in white lupin (*Lupinus albus* L. cv. Kiev Mutant) at the reproductive stage. J. Exp. Bot. 59:75–583.

Huang, L., R.W. Bell, B. Dell. 2009. Exploring the Physiological Basis for High Reproduction Sensitivity to Boron Deficiency in Plants. Proc. International Plant Nutrition Colloquium XVI, DavisCalif. Retrieved from: http://escholarship.org/uc/item/lmz458kn

Kobayashi, M., T. Matoh, J. Azuma. 1996. Two chains of rhamnogalacturonan II are cross-linked by borate-diol ester bonds in higher plant cell walls. Plant Physiol. 110:1017–1020.

Loomis, W.D. and R.W. Durst. 1992. Chemistry and biology of boron. Biofactors. 3:229-239.

Marschner H. 1995. Mineral nutrition of higher plants, 2nd Ed. Academic Press, London. p. 889.

Miley, W.N., G.W. Hardy, M.B. Sturgis, F.E. Sedberry Jr. 1969. Influence of boron, nitrogen and potassium on yield, nutrient uptake and abnormalities of cotton. Agron. J. 61:9-13.

Miwa, K. and T. Fujiwara. 2010. Boron transport in plants: co-ordinated regulation of transporters. Ann. Bot. 105:1103–1108.

Miwa, K., J. Takano, T. Fujiwara. 2006. Improvement of seed yields under boron-limiting conditions through over expression of BOR1, a boron transporter for xylem loading, in A*rabidopsis thaliana*. Plant J. 46:1084–1091.

Miwa, K., J. Takano, H. Omori, M. Seki, K. Shinozaki, T. Fujiwara. 2007. Plants Tolerant of High Boron Levels. Science 318:1417.

Nable, R.O., G.S. Banueuels, J.G. Paull. 1997. Boron toxicity. Plant Soil. 193:181-198.

Nable, R.O., B. Cartwright, R.C. Lance. 1990. Genotypic differences in boron accumulation in barley: relative susceptibilities to boron deficiency and toxicity. pp.243–251. *In*: N. El Bassam, M. Dambroth, B. Loughman (eds.). Genetic Aspects of Plant Mineral Nutrition. Kluwer Academic Publishers, Dordrecht, The Netherlands.

Nakagawa, Y., H. Hanaoka, M. Kobayashi, K. Miyoshi, K. Miwa, T. Fujiwara. 2007. Cell-type specificity of the expression of Os BOR1, a rice efflux boron transporter gene, is regulated in response to boron availability for efficient boron uptake and xylem loading. Plant Cell. 19:2624–2635.

Oertli, J.J. 1994. Non-homogeneity of boron distribution in plants and consequences for foliar diagnosis. Commun. Soil Sci. Plant Anal. 25:1133-1147.

Oliveira, R.H., R.S.D. Milaneze, M.A. Moraes-Dallaqua, C.A. Rosolem. 2006. Boron Deficiency Inhibits Petiole and Peduncle Cell Development and Reduces Growth of Cotton. J. Plant Nutr. 29:2035-2048.

Reid, R. 2007. Update on boron toxicity and tolerance in plants. pp. 83–90. *In*: F. Xu, H.E. Goldbach, P.H. Brown, R.W. Bell, T. Fujiwara, C.D. Hunt, S. Goldberg, and L. Shi, (eds.). Advances in Plant and Animal Boron Nutrition. Springer, Dordrecht, The Netherlands.

Reid, R.J., J.E. Hayes, A. Post, J.C.R. Stangoulis and R.D. Graham. 2004. A critical analysis of the causes of boron toxicity in plants. Plant Cell and Environ. 25:1405-1414.

Rochester, I. 2007. Nutrient uptake and export from an Australian cotton field. Nutrient Cycling in Agroecosystems. 77:213–23.

Römheld, V. and H. Marschner. 1991. Function of micronutrients in plants. pp. 297-328. *In*: J.J. Mortvedt (ed.). Micronutrients in Agriculture, 2nd Ed. Soil Sci. Soc. Amer. Madson, Wisc.

Rosolem, C.A and T. Bíscaro. 2007. Adsorção e lixiviação de boro em Latossolo Vermelho-Amarelo. Pesquisa Agropecuária Brasileira. 42:1473-1478.

Rosolem, C.A. and A. Costa. 2000. Cotton growth and boron distribution in the plants as affected by a temporary deficiency of boron. J. Plant Nutr. 23:815-825.

Rosolem, C.A., J.A.F. Esteves, L. Ferelli. 1999. Resposta de cultivares de algodoeiro ao boro em solução nutritiva. Scientia Agricola. 56:705-711.

Rosolem, C.A., J.A. Quaggio, N.M. Silva. 2001. Algodão, Amendoim e Soja. pp.321-354. *In*: Ferreira, M.E., Cruz, M.C.P., Raij, van. B., Abreu, C.A. (eds.). Micronutrientes e elementos tóxicos na agricultura. Jaboticabal: CNPq/FAPESP/POTAFOS.

Rosolem, C.A and G.B. Bastos. 1997. Deficiências minerais no cultivar de algodão IAC 22. Bragantia 56:377-387.

Ryden, P., K. Sugimoto-Shirasu, A.C. Smith, K. Findlay, W.D. Reiter, M.C. McCann. 2003. Tensile properties of *Arabidopsis* cell walls depend on both a xyloglucan cross-linked microfibrillar network and rhamnogalacturonan II-borate complexes. Plant Physiol. 132: 1033–1040.

Sankaranarayanan, K., C.S. Praharaj, P. Nalayini, K.K. Bandyopadhyay, N. Gopalakrishnan. 2010. Effect of magnesium, zinc, iron and boron application on yield and quality of cotton (*Gossypium hirsutum*). Indian J. Agric. Sci. 80:699–703.

Satya, S., J.G. Pitchai, R. Indirani. 2009. Boron nutrition of crops in relation to yield and quality- A review. Agric. Rev. 30:139–44.

Sestren, J.A., and R. Kroplin. 2009. Sintomas de toxicidade de boro no algodoeiro. *In*: VII Congresso Brasileiro de Algodão. ABRAPA/IAPAR, Foz do Iguaçu.

Shorrocks, V.K. 1997. The occurrence and correction of boron deficiency. Plant Soil. 193:121–148.

Silva, M.N., L.H. Carvalho, O.C. Bataglia, R. Hiroce. 1979. Efeitos do boro em algodoeiro cultivado em condições de casa de vegetação. Bragantia. 38:153-164.

Silva, N.M., L.H. Carvalho, E.J. Chiavagato, N.P. Sabino, H. Rúter. 1982. Efeitos da dose de boro aplicadas no sulco de plantio do algodoeiro em solo deficiente. Bragantia. 41:181-191.

Singh, S.K. 2009. Management of micronutrients for increasing crop productivity. Indian J. Agric. Chem. 42:17–41.

Srivastava, P.C., U.C. Gupta. 1996. Essential trace elements in crop production. pp.73-173. *In*: P.C. Srivastava, and U.C. Gupta (eds.). Trace Elements in Crop Production. New Delhi, India: Oxford & IBH Publishing Cop. Pvt. Ltd.

Sutton, T., U. Baumann, J. Hayes, N.C. Collins, B.J. Shi, T. Schnurbusch, A. Hay, G. Mayo, M. Pallotta, M.P. Tester, Langridge. 2007. Boron-toxicity tolerance on barley arising from efflux transporter amplification. Science. 318:1446–1449.

Takano, J., K. Miwa, L.X. Yuan, N. von Wiren, T. Fujiwara. 2005. Endocytosis and degradation of BOR1, a boron transporter of *Arabidopsis thaliana*, regulated by boron availability. Proc. Natl. Acad. Sci., USA. 102:12276–12281.

Takano, J., K. Noguchi, M. Yasumori, M. Kobayashi, Z. Gajdos, K. Miwa, H. Hayashi, T. Yoneyama, T. Fujiwara. 2002. *Arabidopsis* boron transporter for xylem loading. Nature. 420:337–340.

Takano, J., M. Wada, U. Ludewig, G. Schaaf, N. Von Wirén, T. Fujiwara. 2006. The Arabidopsis major intrinsic protein NIP5, 1 is essential for efficient boron uptake and plant development under boron limitation. Plant Cell. 18:1498–1509.

Tanada, T. 1983. Localization of boron in membranes. J. Plant Nutr. 6:743-749.

Tanaka, M. and T. Fujiwara. 2008. Physiological roles and transport mechanisms of boron: perspectives from plants. Eur J. Physiol. 456:671–677.

Uraguchi, S., H. Hanaoka, K. Aizawa, Y. Kato, Y. Nakagawa, T. Fujiwara. 2009. Boron deficiency in rice and the potential of up-regulated rice boron transporter in improving boron deficient symptoms. UC Davis: The Proceedings of the International Plant Nutrition Colloquium XVI. Retrieved from: http://escholarship.org/uc/item/3rr3s095

Xie, Q., W.X. Wei, Y.H. Wang. 1992. Studies on absorption, trasnlocation and distribution of boron in cotton (*Gossypium hirsutum* L.). Acta Agronomica Sinica, 18:31-37.

Van Iersel, M.W., D.M. Oosterhuis, W.M. Harris, 1994. Apoplastic water flow to cotton leaves and fruits during development. J. Exp. Bot. 45:163-169.

Wimmer, M.A, G. Lochnit, E. Bassil, K.H. Muehling, P.H. Brown, H.E. Goldbach. 2009. Identification of boron-binding proteins supports a function of boron at the cell membrane. UC Davis: Proc. International Plant Nutrition Colloquium XVI, Davis, Calif. Retrieved from: http://escholarship.org/uc/item/7v68m82b

Zhao, D., and Oosterhuis, D.M. 2002. Cotton carbon exchange, nonstructural carbohydrates, and boron distribution in tissues during development of boron deficiency. Field Crops Res. 78:75-87.

Zhao, D. and Oosterhuis, D.M. 2003. Cotton growth and physiological responses to boron deficiency. J. Plant Nutr. 26:855-867.