Boron

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Boron (B) is a chemical element with atomic number 5. It has two isotopes, ^{10}B and ^{11}B, with a relative abundance of 20% and 80%, respectively, giving an atomic weight of 10.81. Together with Silicon and Germanium, B is considered a metalloid because it has intermediate properties between metals and non-metals. Its requirement for plant growth has been known for one century. Plants take up B mainly in the form of boric acid and its deficiency causes a plethora of symptoms. The biological functions of B are associated with its capacity to form borate cross-links with polysaccharides, glycoproteins and glycolipids.

boron ligands cell wall cell membranes plant development

1. Boron and the Cell Wall: The Only Demonstrated Primary Role

The attention of '*boronists*' very soon focused on the cell wall. Katherine Warrington already indicated that B is fixed by the plant [1], and later, Skok provided evidence that B's role was related to the complexing capacity of borate ions [2]. Mazurek & Perlin described boric acid/borate complexes with diols containing compounds [3], and Loomis & Durst reported that 90% of B is associated with the cell wall fraction [4]. Years later, Matoh et al. raised the amount of B in the cell wall up to 98% [5], and Hu & Brown localized B associated with cell wall pectin and proposed that "…B plays a critical, although poorly defined, role in the cell wall structure of higher plants" [6].

That poorly understood role of B in the cell wall began to be defined when Findeklee & Goldbach showed that the elasticity of the cell wall is reduced under B-deficiency [7], which pointed to a B function in anchoring cell wall macromolecules and to the capacity of borate cross-linking two chains of rhamnogalacturonan II (RGII) through diol-ester bonds [8]. Matoh et al. found later that such RGII-dimer (dRGII) was ubiquitous in the cell wall of higher plants [9]. Finally, O'Neill et al. convincingly demonstrated that the growth of *Arabidopsis thaliana* dwarf mutants *mur1* relies on the presence of apiose-borate complexes mediating the dimerization of RGII [10][11]. Although RGII is a minor part of the pectin fraction [12], more than 90% appear dimerized, which is critical for plant growth. Specifically, this dRGII-borate complex is important to determine pore size and cell wall mechanic properties influencing cell wall expansion and, therefore, plant growth [13][14].

Although the basic structure of RGII and the B-mediated dRGII-B are highly conserved in vascular plants, some variability has been described among different plant species [15]. The relative abundance of the pectin fraction is lower in monocots than in dicots, likely explaining its lower B requirement [16]. Primitive plants contain traces of

dRGII-B also with the same conserved structure, which suggests that genes involved in RGII biosynthesis appeared early in the evolution of land plants and that RGII dimerization was crucial for such evolution [17].

2. The Fine-Tuning of Boron Homeostasis. Does It Support Other Primary Roles of This Micronutrient?

Being the structural role the only demonstrated primary role of B in plants, it is proposed that many of the B deficiency symptoms result from the drastic changes in the cell wall structure and properties resulting from the decrease in dRGII-borate complexes [11]. Even more, the rapid cell death after inducing B deficiency has been attributed to defects in newly forming cell walls [18]. Nevertheless, it should not preclude the possibility that other primary functions of B may exist, particularly those related to the maintenance of membrane activities or/and regulation of developmental events, perhaps influencing cell signalling and transduction pathways [19][20][21].

Although there is still a lack of convincing evidence supporting those mechanisms underlying alternative roles of B in plant cells, the discovery of different transporters involved in B uptake and inner transport may support that this micronutrient´s role goes beyond being a cell wall structural element.

Indeed, although most B is associated with RG-II in land plants [9], it can be found in the cytosol and vacuole [22] or associated with the plasma membrane [23][24]. Independently of the concentration of B in soils, its concentration in cell walls is kept almost constant, while B cytosolic levels vary with soil concentrations. This has resulted in the interesting hypothesis that a minimal level not bound to RG-II is needed to avoid several early B-deficiency symptoms [25] and that the reaction of the non-RG-II-linked B with other ‘ligands’ might be responsible for roles of B beyond the cell wall [26].

The uptake of B by plants was considered an unregulated passive process until the discovery of the presence of a complex transport system that was acting to maintain B homeostasis [27]. Today, B passive diffusion through the plasma membrane is considered to occur only for B uptake from soil to roots [28], but it is accompanied by transport mediated by channels [29] that have also been described in growing shoot tissues [30] and in reproductive organs [31][32]. In summary, plants sense internal and external conditions of B and rapidly regulate the expression of channels of the NIP (*Nodulin26-like Intrinsic Protein*) subfamily of MIPs (MAJOR INTRINSIC PROTEIN) family and transporters of the Borate Exporter family (BOR) to control B homeostasis [33]. In *Arabidopsis*, AtNIP5;1 is responsible for facilitating the uptake of B from soil to roots [34], and the exporter BOR1, which is localized toward the stele, is key for xylem loading under low-B conditions [35], and BOR2 seems to export B from the symplast to the apoplast to ensure efficient RG-II cross-linking [36]. Meanwhile, BOR4 is induced under high-B concentrations and is involved in the exclusion of B, enhancing B toxicity tolerance [37].

Additionally, other borate channels have been described to be expressed in different tissues and at particular developmental stages. *AtNIP6;1* is expressed mainly in the node region of shoots and is involved in xylem-phloem translocation of B to growing leaves [30]; AtNIP7;1 seems to be required for pollen development [32]; and *AtNIP4;1* and *AtNIP4;2* expressions are related to pollen tube elongation [38]. Moreover, evidence that X Intrinsic Proteins (-

XIPs), another subfamily of MIPs, can facilitate B transport to young tissues has also been reported [39]. Altogether, the complex mechanism of B uptake and translocation must guarantee continuous supply to grow cell walls, but, at the same time, it is preferentially distributed to developing meristems, which supports that B is not a merely structural element but plays a potentially key role in developmental processes.

Reinforcing the importance of B for plant development, orthologs and paralogs of Arabidopsis BORs and NIPs have been described in many plant species [40][41][42][43][44][45][46]. Sequences similarities indicate that these transporters belong to conserved gene families that show developmental stage-dependent expression patterns in different tissues to reach B requirements that ensure the proper execution of blueprints for the plant building [33].

3. Boron and Cell Membranes

Before demonstrating B function in RGII crosslinking, researchers focused their attention on cell membranes describing that B deficiency impairs membrane transport, membrane-associated enzymatic activities, or membrane composition, and interestingly, during the last years, the evidence of B roles as a linker element in the cell membrane and endomembranes has re-emerged.

Robertson & Loughman demonstrated a reduced absorption of phosphate under B deficiency [47], and Goldbach showed that phosphate and glucose uptake and efflux rates were decreased under B-limiting conditions [48]. Also, K-Cl stimulated ATPase [49], and ATP-dependent H^+ pumping and vanadate-sensitive ATPase activities were demonstrated to be inhibited by B deficiency [50]. In all cases, these effects were quickly reverted by B addition, suggesting that the membrane properties rely on B nutrition. Furthermore, B has also been involved in redox activities and the maintenance of membrane potential [51].

A structural role of B in the membrane was proposed by several researchers to explain a large number of reported effects of B on membrane processes. For instance, the fluidity of liposomes prepared from low B treated cells of sunflower was lower than in liposomes coming from B-sufficient cells [50]; deficiency reduced both total lipid and phospholipid contents in roots and leaves of *Lycopersicon esculentum* (tomato) and *Abelmoschus esculentus* (okra) [52], and Cakmak et al. (1995) showed increments of solutes´ leakage under B deficiency [53]. Altogether, these findings indicate that B could primarily affect membrane function by playing a structural role that protects membrane integrity. This is supported by Tanada analyses that showed a major part of B localized in membranes [54]. Nevertheless, although membranes harbour glycans as good candidates for binding B, the difficulty identifying B complexes formed with membrane components does not allow us to discard that the observed effects are secondary events of the affected cell wall.

At the end of the 20th and the beginning of the 21st Century, new techniques, and new biological models for plants, animals, and prokaryotes, have allowed the development of new approaches to unravel membrane-related B functions. For instance, the use of phenylboronic acid (PBA) as a competitor for B-binding sites caused the disassembly of transvacuolar cytoplasmic strands and cell collapse [55], suggesting either a structural role of B in the cytoskeleton or, more likely, a disruption of cytoskeletal proteins anchored to membrane glycolipids or/and

glycoproteins. Also, following a similar experimental procedure, it was shown that PBA induced abnormal internalization of PIN1, blocking auxin transport and generating abnormal Arabidopsis embryo early development, which supported that PBA competes with B for membrane proteins [56].

A large amount of information relating B with membrane glycans has come from the research performed on the legume-rhizobia symbioses [57][58][59]. This symbiotic interaction triggers the development of a new organ, the root nodule, that follows a unique process of organogenesis characterized by events in which exists an intense membrane synthesis. It is estimated that the membrane synthesis rate is about 30 to 50-fold higher than in other plant-growing organs [60]. Matching with the analyses reported by Tanada, the content of B in nodules is higher than in roots or shoots, likely because it is demanded by such an amount of membrane synthesis [61]. Therefore, the legume-rhizobia symbioses is a very suitable model to investigate the role of B in membrane-located processes. The bacteria proliferate inside the nodule and differentiate into N_2-fixing bacteroids enclosed by a plant-derived peribacteroid membrane (PBM), which differentiates a glycocalyx composed of new glycolipids and glycoproteins [62][63] involved in bacteria-plant cell surface interactions important to ensure the success of the symbiosis [64]. Some of those components are either abnormally glycosylated [57] or not detected [58] in B-deficient nodules, resulting in cell cycle and the cell division-cell differentiation transition misregulation that leads to a tumour-like development [59]. Recently, an abnormal N-glycosylation during early development under B deficiency has been described in pea nodules, Arabidopsis roots, and *Dario rerio* [65]. Like in nodules, aberrant root apical meristem in Arabidopsis and a failure of zebrafish organogenesis occurred. Although the described aberrant development could be due to defects in the cell wall structure, the effects observed in the animal model support a primary role of B in membranes, likely related to the synthesis and stability of glycan moieties of glycoproteins and glycolipids. As mentioned above, *cis*-diol-containing sugar residues, harboured by the cell glycocalyx in membranes and matrices, are potential candidates to be ligands of B. Several of them have already been identified [23][24][66].

4. Boron and Developmental Events

Back in 1985, Lovatt published an interesting hypothesis stating that the evolution of the xylem resulted in the acquisition of the essentiality of B for apical meristem activity and conferred the advantage of preventing B toxicity [67], coinciding with the recent consideration of Lewis that affirmed that B is a toxic element for vascular plants [68]. Lovatt suggested that a threshold concentration of B must reach meristematic cells to promote division and subsequent expansion to ensure growth, preventing the accumulation of B in meristems to a toxic concentration. The evolution of the xylem ensured a gradient of B, lowering its concentration in the growing cells in the elongation zone and reaching a critical minimum content in the meristematic cells to elicit mitosis. The hypothesis was supported by the fact that B is toxic to most organisms at relatively very low concentrations, being vascular plants the most tolerant, and by observing that DNA synthesis, cell division, and elongation are inhibited under B deprivation and soon reversed after B supply. The review also proposed that regulation of cell division by B may be potentially common to other organisms than vascular plants.

More recent studies have shown that induced B deficiency or loss of function mutations on B translocators alters cell cycle regulatory pathways, cell differentiation, and the development of vegetative and reproductive structures [40][69][70][71][72]. Particularly, responses to deficiency described in those and other studies recently reviewed [73][74] suggest that B can induce molecular pathways that determine meristem fate and place B nutrition as a key regulator of developmental processes.

Indeed, the earliest defects following B-starvation are growth arrest and aberrant meristem formation [71][75][76]. This growth arrest can be attributed to defects in cell elongation or differentiation due to abnormal cell wall architecture [11][71][77], although evidence of cell division inhibition has also been reported. Actually, at the initial steps of B nutrition research, the works performed by Sommer and Sorokin supported that B deficiency caused root tip malformations and impairment of cell division [78]. And as mentioned above, using PBA to mimic B-deficiency, it was shown that root apical meristem (RAM) formation was disrupted in embryos as early as the first asymmetric cell division of the hypophysis appeared [56], placing B as crucial for embryo formation. Furthermore, Poza-Viejo et al. reported a reduction of cell division 4h after transferring Arabidopsis seedlings to severe B deficiency media due to inhibition of the G1-DNA replication phase transition [72]. Cell division inhibition was accompanied by a later loss of identity of the quiescent centre (QC) that could be attributed to the down-regulation of *CCS52A2* that controls QC and the maintenance of surrounding stem cells [79].

As previously stated, the interaction of legumes with soil rhizobia triggers an interesting process of plant organogenesis in which cell division, cell elongation, and cell differentiation must be finely regulated [80][81]. Particularly crucial is the activation by CCS52A of the transition from mitosis to endoreduplication to gain polyploidy required for cell elongation prior to bacteria invasion [82]. Interestingly, *CCS52A* is also downregulated during early organogenesis of B-deficient nodules, leading to failure of cell elongation and cell differentiation [69]. Both in the QC and in nodule cells, expression of *CCS52A(A2)* promotes ubiquitination and proteolysis of the anaphase-promoting complex, resulting in cell polyploidy. This is crucial to maintain QC identity and mitotic activity of surrounding stem cells of RAM and to induce nodule cell elongation, bacterial invasion/spreading, and cell differentiation, respectively.

Development of reproductive organs is often more sensitive to B deficiency than vegetative growth [75]; therefore, it is not surprising that specific mechanisms of B homeostasis are induced at a particular moment in which shoot meristems transition from vegetative to floral development [83]. Apparently, BRAHMA (BRM) protein, which is degraded in response to high B [84], maintains the juvenile phase [85]. The transition to a mature phase prior to reproductive development may be the consequence of the reduction in BRM activity in response to the B translocation increase driven by B transporters [74].

Altogether, it seems that this micronutrient could play central roles in molecular regulatory pathways of the embryo and post-embryo plant development.

5. Boron, Cell Signaling Mechanisms, and Gene Expression Regulation

Recent studies using microarrays or RNAseq have provided increasing evidence supporting B nutrition's effect on the regulation of gene expression affecting metabolism, cell wall, and membrane integrity and function, stress response, or micronutrient homeostasis [70][86][87][88][89][90]. As summarized in the following lines, almost every signalling and transduction pathway is activated in response to B deficiency, which allows us to hypothesize that the micronutrient may be involved in cell signalling. Nevertheless, many experimental data support that most of the responses are linked to the effect on cell walls. Briefly, based on our and other author´s studies, Kobayashi et al. [18] proposed that the disturbed pectin network decreases the tensile strength of the cell wall leading to an increase of turgor pressure that stretches the cell membrane triggering a stress response that resembles hypersensitive responses to pathogens. Transduction of such mechanical signal involves a rapid influx of Ca^{2+}, ROS production, and MAPK cascades that result in auxin/ethylene-mediated cessation of growth and cell death [77][91][92][93][94]. This is supported by the fact that blocking Ca^{2+} channels in B-deprived cells largely inhibited the expression of stress-responsive genes [92] or adding antioxidant reagents can prevent the death of B-deficient cells [91] by restoring cell elongation. Similarly, blocking ethylene biosynthesis or perception or using mutants defective in ethylene or auxin response can restore B deficiency molecular responses [77]. Furthermore, destabilization of the cell wall under Ca deficiency but not under other nutrient deficiencies such as K or Mg that are also involved in pectin cross-linking triggers a similar response [93]. On the contrary, preincubating B-deficient cells with a supplement of extra Ca increases cell wall strengthening, attenuating the expression of B-deficiency-responsive genes [93]. Similarly, the addition of Ca partially restored the impaired development of B-deficient legume nodules [61] and the expression of 75% of genes affected by stress [70].

In roots, Ca influx, ROS production, and cell death occur preferentially in the elongation zone [18]. In line with this, maize mutants affected in the synthesis of B transporters, which are involved in the transition to the reproductive phase, develop defective inflorescences with reduced RG-II dimerization, which is largely restored by adding B into the media [40]. Therefore, failure of meristem formation and functioning can also be explained by the mechanosensitive response to B deficiency. But is that all?

Based on the comparison among different gene expression profiles, the mechanosensitive hypothesis proposes a pathogen-like response under B deficiency. However, B-deficiency disturbs cell wall structure, and it is expected that the upregulation of genes is involved in cell wall functioning. Nevertheless, while genes related to the cell wall structure, included in different transcriptomic analyses, are upregulated after pathogen attack [95], they appear downregulated under B deficiency [18][87], suggesting that B might be required to induce the expression of cell wall synthesis and assembly-related genes [22]. Using the legume nodule model, the first visible symptoms of B deficiency appear early after root inoculation with rhizobia [96], and although pathogenesis-related proteins were synthesized [97], oxidative damage was not detected even 3 weeks post-inoculation [98]. At this developmental stage, cell death is not observed, but it appears an abnormal cell division resembling tumour behaviour [59], indicating an early failure of development and suggesting that B deficiency is not necessarily associated with cell death. Furthermore, low-B results in abnormal embryonic development in animals [99], also leading to a tumour-like amorphous structure when B deprivation occurs at the early cleavage stage [65]. Interestingly, boric acid can inhibit cell proliferation in different cancer cell lines [100][101]. Such implications of B nutrition in animal

physiology/development, together with the organogenesis failure in plants and animals, claim for alternative or/and additional sensing/response mechanisms to low B conditions.

The existence of a putative B sensor molecule in plants able to detect external B concentration remains still unknown. Certainly, the loss of cell wall integrity that can alter turgor pressure could be the cellular signal triggering the B stress response. In agreement with such a hypothesis, the literature offers other possible sensing mechanisms associated with soluble, not necessarily external, B. Regarding B deprivation sensing mechanisms, a computational model for B distribution in roots localized the highest concentration of soluble B around the QC, which might be likely used to keep RAM activity [102]. Also, the fact that the 5′-untranslated region (UTR) of NIP5;1 responds to the increase in cytosolic B promoting mRNA degradation [103] led to the assumption of a sensor mechanism acting in the cytosol resulting in the development of biosensors of cytosolic B [104]. Interestingly, this 5′-UTR response to B seems to function also in animal cells [103]. Additionally, there are different cell wall receptors that act in response to stresses regulating cell wall dynamics [105]. Also, the arabinogalactan-proteins (AGP) have been proposed as sensors of soluble periplasmic B because, as researchers described later, they contain sugar residues susceptible to interaction with B [106]. Afterwards, the signal would be conveyed to the nucleus, and several cell signalling transduction pathways can be implicated. In agreement with this hypothesis, Dumont et al. demonstrated that the inhibition of root cell elongation induced by the fucose analogue 2-fluoro 2-L-fucose (a chemical inhibitor of RG-II biosynthesis) was partially restored by boric acid supplementation without rescuing RG-II synthesis nor dimerization [107]. These observations suggest that B itself, rather than the RG-II dimer, is an essential component of the cell wall integrity-sensing mechanism that controls cell elongation, perhaps due to its ability to bind to the *cis*-diol motifs of signalling molecule(s).

The potential role of phytohormones in the regulation of B stress responses has been widely studied. By applying pharmacological approaches combined with reverse genetics using mutant lines affected in hormone synthesis or hormonal perception/transduction pathways, it was described that B deprivation alters plant development affecting synthesis, transport, or/and reception of auxins [56][108], ethylene [97][109], cytokinins [71][72][110], brassinosteroids [111], jasmonic acid [112], and the cross-talk among hormones [113]. Thus, phytohormone regulatory pathways are considered crucial in regulating cell signalling in B nutrition, although other cell signalling mechanisms common in plants and animals must also be important.

As previously mentioned, blocking sites of B binding with PBA led to cytoskeleton disruption [55], and levels of actin and tubulin increased in response to short-term B starvation due to the altered cytoskeleton polymerization [114]. Therefore, B might be involved in signalling through a cascade of signals via the cell wall-plasma membrane-cytoskeleton continuum [20], through endocytosis of signalling elicitors. Supporting this hypothesis, the abundance of homogalacturonan and RGII rapidly increased in cell walls shortly after B-deprivation in *Zea mays*, and their endocytosis was also inhibited [115]. In animals, the maintenance of the membrane-cytoskeleton continuum and, hence, endocytosis-mediated signalling also support the essentiality of B in the development of these organisms.

Also, the fact that the increase of cytosolic Ca^{2+} (cytCa^{2+}) is a rapid response to B deficiency could explain why B may be involved in signalling through transduction pathways activated by Ca^{2+} [106], which could also be extended

to animals. Many abiotic and biotic stresses induce an increase of cytCa^{2+} following the activation of cyclic nucleotide-gated Ca^{2+} channels (CNGCs) [116]. A plasma membrane-localized CNGC was found to be upregulated in Arabidopsis in response to B deficiency [117]. Therefore, it has been proposed that membrane sensors of B-deficiency could induce activation of CNGCs resulting in Ca^{2+} increments that could activate Ca-related proteins, such as calmodulin (CaM). Ca-CaM regulates then different transcription factors and B-responsive genes [106].

Right after discovering that B is part of a signalling molecule in bacteria (AI-2 *quorum-sensing* autoinducer) that interacts with the sensor protein LuxP [118], another tentative working hypothesis to explain the possible function of B is that it is a cellular signal itself or that it is implicated in a soluble B-complex that interacts with different transcription factors. In line with this, Kasajima et al. described that WRKY6 is a transcription factor involved in response to B deficiency in Arabidopsis [119], which also plays an important role in embryogenesis [120]. Besides, B could interact with -hydroxyl groups (OH) of amino acid residues (as serine or threonine) of different transcription factors, as in the bacterial LuxP. However, there is no evidence to date of this type of binding.

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