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REVIEW

Plant Cortical Microtubules Are Putative Sensors under Abiotic Stresses

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Abstract—In this article, we review current knowledge on the dynamic changes and roles of microtubule (MT) arrays under abiotic stresses. The results emphasize the existence of highly dynamic changes, complex regulatory networks, and the vitally important role of MTs in the response to abiotic stresses. In particular, some findings indicate that cortical microtubules (CMTs) underlying the plasma membrane play an important role in abiotic stress-induced signaling pathways. Therefore, we also discuss the relationship between CMTs and abiotic stress signaling. The data show that at least three early response mechanisms, namely, Ca^{2+} signaling, abscisic acid biosynthesis, and the formation of plant cell walls, follow CMT reorganization and are mediated by dynamic changes in the CMTs. Consequently, we propose that the CMTs are not only part of the plant response to abiotic stresses but might also serve as a type of cell wall membrane-bound sensor that perceives the stress stimuli to generate adaptive signals and responses of cells.

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Key words: microtubules, abiotic stresses, response mechanisms, signaling pathway, sensors

Microtubules (MTs), which are highly dynamic structures, are the main integrants of the cytoskeleton. They are assembled from heterodimers of α- and β-tubulin subunits to form four unique types of array—cortical MTs (CMTs), pre-prophase band, spindle, and phragmoplast—throughout the plant cell cycle [1, 2]. From the time when they were first discovered in 1963, the important functions of MT arrays have been identified in almost every intracellular activity from cell division to cell movement, cell morphogenesis, and even cell signal transduction [3-5]. Recently, research on MT arrays has expanded to encompass the responses to abiotic stresses, primarily those of high salt, drought, and low temperature [6-9], constituting a new topic in the field of abiotic stress tolerance mechanisms in plants. The effects of high salinity, drought, and low temperature on plants have traditionally received considerable attention because they are major stress factors that adversely affect crop productivity and quality [10, 11]. Furthermore, the organization and dynamics of MTs are likely to play a vital role in enhancing plant tolerance through various cell activities. Therefore, MT arrays have become a key focus area in studies on plant abiotic stress tolerance. However, the role of MTs in the response to abiotic stresses has not previously been reviewed as a separate subject. In this article, we review current information on the dynamic changes in MT organization and our present understanding of the role of MTs in plant responses to abiotic stress. Some results indicate that the CMTs play an important role in abiotic stress-induced signal transduction. Therefore, the relationship between the CMTs and abiotic stress signaling pathways is also discussed in this article. It is well known that rapid calcium ion (Ca^{2+}) influx and significant accumulation of abscisic acid (ABA) in plant cells are the earliest and most obvious physiological events that stimulate adaptive responses to abiotic stresses, and that the plant cell wall is the first cell structure to perceive environmental stimuli. However, the current findings suggest that dynamic changes in CMT organization can be an important upstream event that regulates the three main response

Abbreviations: ABA, abscisic acid; CESA, cellulose synthase; (C)MTs, (cortical) microtubules; CSCs, CESA complexes; MAP, microtubule-associated protein; ROS, reactive oxygen species; SOS, Salt Overly Sensitive.

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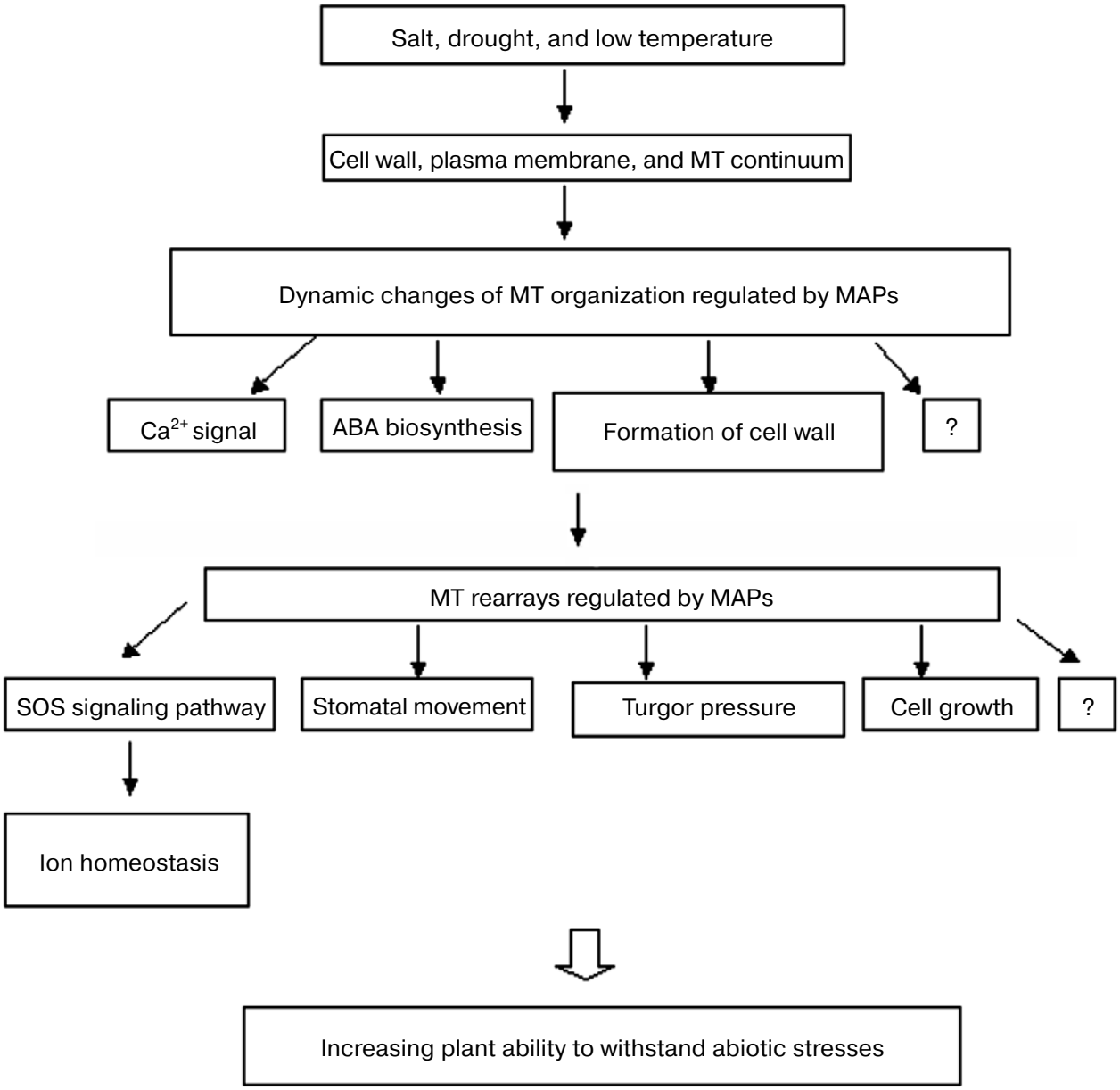

Overview of putative pathways for regulatory mechanisms of microtubule organization under abiotic stresses. The earlier changes of MT organization may be responsible for perceiving environmental stimuli and regulating the intercellular abiotic stress signaling transduction, such as Ca^{2+} signal, ABA biosynthesis, and formation of plant cell wall. The abiotic stress-induced subsequent MT rearrangement may play a vital role in regulating cell biological functions, such as ion homeostasis, stomatal movement, turgor pressure, and cell growth. These MAPs may include SPIRAL1, PFD3, PFD5, MPB2C, MAP18, MAP65-2, PLDδ

mechanisms, namely, Ca^{2+} signaling, ABA biosynthesis, and the formation of plant cell walls under abiotic stresses (Scheme). Thus, we propose that CMTs underlying the plasma membrane are not only part of the plant response to abiotic stresses but may also be associated with the cell wall and plasma membrane as a headstream responsible for perceiving stress stimuli, thereby enhancing plant adaptation to abiotic stresses.

MICROTUBULES AND SALT STRESS

The main effect of salt stress in plants is to cause an intracellular ion imbalance [11]. In *Arabidopsis*, tolerance to ion imbalance is mediated by a signaling pathway based on the SOS (Salt Overly Sensitive) genes, notably SOS1 (a plasma membrane Na^+/H^+ antiporter), SOS2 (a serine/threonine protein kinase), and SOS3 (a calcineurin

B-like calcium-binding protein) [10, 11]. When plants are stressed by salt, the pathway is mediated through calcium signals to an SOS2/SOS3 kinase complex that phosphorylates and enhances transcription of SOS1, which in turn transports Na^+ into the apoplast to prevent plants experiencing damage from Na^+ accumulation [11].

Recently, several studies have provided evidence for the existence of a close relationship between the SOS pathway and CMTs [9, 12]. *Arabidopsis sos1* and *sos2* mutants are hypersensitive to NaCl because of an intracellular Na^+ imbalance [10]. It has been demonstrated that *sos1* and *sos2* mutants display CMT disruption [9]. When treated with 50 mM NaCl, the roots of *sos1* mutants display a pattern of right-handed growth, and the CMTs are more disordered than in wild-type *Arabidopsis* cells [12]. By using a pharmacological approach, *sos* mutants have been shown to have abnormal responses to low doses of MT-interacting drugs [9]. Stabilization of CMTs with paclitaxel (a MT stabilizing drug) results in greater *sos1* seedling death, whereas disruption of CMTs with oryzalin (a MT disrupting drug) rescues *sos1* seedlings from death in salt stress treatments [12]. Additionally, *sos1* and *sos2* mutants suppress the right-handed root growth phenotype of *Arabidopsis* plants with mutations in SPIRAL1, which is a plant-specific protein that localizes to MTs *in vivo* [9, 13, 14]. These results imply that the SOS signaling pathway could contain CMT regulatory mechanisms, and that CMTs play an active role in the salt tolerance of plants, partly via the SOS signaling pathway.

What are the dynamic changes of salt stress-induced MTs that have been observed? In addition to CMT disruption in salt hypersensitive *sos1* and *sos2* mutants, *Arabidopsis* plants with mutations in PFD3 and PFD5 subunits, which are implicated in the correct folding of tubulin and actin, display changes in their developmental patterns and have less abundant and organized CMT arrays [15]. *Pfd3* and *pfd5* mutants have reduced levels of α- and β-tubulin compared with the wild-type *Arabidopsis* under both control and salt-stress treatments, whereas *pfd* mutants are hypersensitive to salt stress, indicating that PFD3 and PFD5 can be key MT-associated proteins involved in plant salt tolerance.

In addition, the results of a long-term observational study have shown that CMTs depolymerize and then repolymerize when salt stressed [12]. The initial depolymerization of CMTs appears in both low and high NaCl concentration treatments. However, there are a considerably lower number of cells exhibiting CMT repolymerization with high salt concentration treatment than with low salt concentration treatment [12]. The number of cells undergoing CMT repolymerization coincides with the number of surviving seedlings under salt stress. Thus, salt-induced CMT recovery could reflect the ability of plants to withstand salt stress. Interestingly, if the initially disrupted CMTs are further induced by oryzalin, the number of cells exhibiting CMT recovery is obviously increased [12]. This implies that the initial CMT disruption is closely related to the following CMT reorganization. Using a pharmacological approach, more wild-type *Arabidopsis* seedlings die when treated with an MT stabilizing drug and more survive when treated with MT disrupting drugs under salt stress, whereas almost all seedlings in which the CMTs are reorganized can withstand salt stress [12]. These results further emphasize that both depolymerization and reorganization of CMTs are important events in a plant's ability to withstand salt stress. In particular, salt-induced destruction of the CMT network is not the result of cell damage, but it does play a vital role in the response to salt stress, possibly as a regulator or even as a sensor.

It is well known that transient Ca^{2+} influx into the cytoplasm can be induced under salt stress. By using yellow cameleon (YC 3.6) as a calcium reporter, notable increases in the free cytosolic calcium concentrations ($[Ca^{2+}]_{cyt}$) after initial salt stress-induced depolymerization of the CMTs are found, and these increases are inhibited when paclitaxel is added [12]. Further, the salt-induced recovery of CMTs is regulated by increases in the levels of $[Ca^{2+}]_{cyt}$ [12]. This implies that CMT depolymerization triggers a Ca^{2+} influx and that this has a regulatory effect on CMT reassembly under salt stress.

Salt stress mainly causes ion imbalance and hyperosmotic stress in plants [11]. Thus, it is of interest to know which factor(s) might play a role in MT reorganization in response to salt stress. The functions of MTs are associated with the SOS signaling pathway, showing that ion imbalance is the major factor responsible for dynamic changes of MT in salt-stressed plants. *Pfd* mutants are also hypersensitive to NaCl but not to LiCl or mannitol [15]. Additionally, when grown on medium containing 50 and 100 mM NaCl, seedlings exhibit a pattern of right-skewed root growth. However, seedlings display a normal pattern of root growth when treated with either 100 or 200 mM mannitol [12]. The CMTs in root epidermal cells exhibit abnormalities when treated with 50 mM NaCl, whereas they remain intact and have normal orientation after treatment with 100 mM mannitol [12]. There are thus obvious differences in the pattern of root growth and arrangement of CMTs following the treatments with NaCl and mannitol. Taken together, these results suggest that the highly dynamic changes in MT organization are possibly triggered by an ion-specific, but not by an osmotic-specific, factor.

MICROTUBULES AND DROUGHT

Drought stress can cause water deficit and hyperosmotic stress in plant cells [14]. To study the response of MT arrays to water or osmotic stress, researchers have examined changes in MT organization in plant cells exposed to sorbitol, mannitol, KCl, or polyethyleneglycol [16-18]. After maize roots have been treated with sorbitol or KCl for several hours, cell elongation and the normal

arrangement of CMT arrays in the root cells are disturbed. Sorbitol treatments cause the rapid appearance of oval to circular holes in the CMT arrays. In KCl-treated roots, the CMTs reorient oblique to the longitudinal axis [16]. CMT destruction by hyperosmotic stress induced by mannitol is observed in the root-tip cells of *Triticum turgidum* [17]. Differences in CMT orientation (i.e. the angular distribution of MTs) are observed between well-watered and water-stressed maize roots [19]. In moss protonemal cells, the fine structure of CMTs becomes predominantly arranged in an orientation parallel to the long axis of the control cells [20]. Remarkable, however, is the observation that the fine CMT structure disappears and thicker cables form under drought stress. When the cells are returned to rehydration conditions, the fine CMT arrays reappear [20]. Denser CMT arrays in polyethyleneglycol-treated maize root cells than in control cells have also been observed [18]. These results suggest that different effects on MT organization are triggered under different water-stressed conditions or in different species. Thus, the MTs may be associated with a complex regulatory mechanism network that is activated in response to drought stress. To date, at least three MT regulatory mechanisms that are activated in response to drought stress have been reported, namely regulation of stomatal movement, orientation of cell wall components, and accumulation of ABA.

Partial or complete stomatal closure is an important plant stress symptom induced by drought [21]. Stomatal closure is associated with reductions in transpiration thereby enhancing the plant's drought resistant. It has been demonstrated that the stomatal aperture is regulated by changes in cytoskeletal organization [22]. Fukuda and colleagues report that MT inhibitors suppress the stomatal opening in response to white light in *Vicia faba* L., suggesting that MT organization is required for stomatal opening [23]. Studies using green fluorescent protein::MT binding domain (GFP::MBD) have provided compelling evidence for the utilization of MTs in stomatal opening [22]. Additionally, Huang and colleagues have shown that anti-MT drug treatment prevented opening of guard cells of *V. faba* induced by indole-3-acetic acid [24]. More recently, it has been demonstrated that a microtubule-associated protein (MAP), AtMPB2C, has an effect on stomatal patterning in response to drought stress. The authors found that AtMPB2C interferes with assemblies of CMTs and regulates stomatal development, and that plants overexpressing AtMPB2C tolerate severe drought stress significantly better than wild-type plants. They suggest that overexpression of AtMPB2C alters the CMT organization associated with changes in stomatal patterning, thereby increasing resistance to drought stress [8].

Plant cell osmotic adjustment is also a primary response to water deficit, which improves cell water retention and turgor maintenance [21]. Iwata and colleagues point out that MT orientation induced by osmotic stresses is correlated with turgor pressure in *Spirogyra* cells [25]. Cell turgor pressure can be regulated by plant cell walls, which consist of cellulose microfibrils [26]. Furthermore, a number of studies have reported correlations between the orientation of cellulose microfibrils and CMTs [26, 27]. Thus, CMT arrays may play a role by affecting formation of cell walls and turgor pressure under drought stress. Recent research indicates that CMT organization can drive cellulose microfibril arrays by guiding the trajectories of active cellulose synthase (CESA) in the plasma membrane, and that the association of CESA compartments with CMTs is greatly increased during osmotic stress [28]. Another report has confirmed that CMTs not only guide the trajectories of CESA complexes (CSCs), but also that osmotic stress can affect internalization of CSCs in MT-associated CESA compartments [29]. These results suggest that MTs enhance the effect on cellulose microfibril organization under drought stress, which probably influences the formation of cell walls and turgor pressure to adjust the plant's tolerance ability.

It is well known that changes in ABA levels induced by water deficits play an important role in plant drought tolerance [21]. MTs interact with integrin-like proteins that have been shown to be involved in cell wall–plasma membrane interactions and osmotic stress-induced ABA accumulation [30]. Evidence from studies using a pharmacological approach suggests that changes in MT dynamics, including both polymerization and depolymerization, result in a significant increase in ABA accumulation under osmotic stress [18]. Although it is not clear how MTs regulate ABA biosynthesis under osmotic stress, the stimulation of ABA biosynthesis may occur through alterations in MT dynamics. In addition, transcription of a microtubule-associated protein, MAP18, which plays a role in regulating directional cell growth and CMT organization by destabilizing MTs and which has also been identified as a hydrophilic Ca^{2+}-binding protein, is found to be upregulated under cold and drought stresses. These data provide additional evidence that MAPs may be involved in the response to drought and cold stresses by regulating cell growth or via a Ca^{2+}-mediated regulatory mechanism [5, 31].

MICROTUBULES AND LOW TEMPERATURE

Low temperature stress comprises cold (also termed chilling) and frost (also termed freezing) damage [32, 33]. Irreversible injury caused by chilling temperatures is relatively common in crops such as rice, maize, cotton, cucumber, and tobacco [32]. Consequently, plant cold tolerance has been the subject of intense research, and the regulatory mechanism of MTs in response to low temperature is currently an intensely studied topic. Depolymerization of MTs is often observed under freezing stress [6, 34, 35]. Furthermore, cold-induced stability of MTs is closely correlated with the general cold hardiness of plant species

[6]. The stronger the general cold hardiness of a plant, the more stable is its cold-induced MT organization [6, 34]. In addition, Rikin and colleagues have demonstrated that chilling damage in cotton is accompanied by destruction of the MT network and is significantly accelerated and enhanced by anti-MT drugs [36]. Conversely, pretreatment with ABA can prevent chilling damage and MT destruction. Thus, ABA counteracts the sensitizing effect of anti-MT drugs in the response to cold shock, indicating that cold-stable MTs are closely correlated with ABA signaling pathways under chilling stress.

Plants have differently mediated mechanisms that help them to adapt to frost stress [32]. MTs are not directly related to the targets and mechanisms of plant frost tolerance; however, they do play a role in influencing cold acclimation. Cold acclimation, which is defined as a prolonged exposure to low, but above zero, temperatures, has long been known to enhance the subsequent tolerance of plants to frost stress [32]. This process has been extensively studied, and significant progress has been made in elucidating the MTs involved. This process is impaired when MT disassembly is suppressed by paclitaxel [37, 38]. On the other hand, upon completion of cold acclimation the disassembly of MTs in response to a freezing shock is prevented, indicating that disassembly is a transient event that might be related to signal sensing [6, 35]. Mazars and colleagues report that the disruption of the MT network by various MT-interacting drugs increase $[Ca^{2+}]_{cyt}$ in cold-shocked *Nicotiana plumbaginifolia* protoplasts, whereas the simultaneous disruption of MT and microfilament networks led to a dramatic increase in the Ca^{2+} response to cold shock. The authors suggest that the organization of MTs have a profound influence on the Ca^{2+} response under chilling damage. Under low temperatures, the activity of BN115 is strongly induced at the transcriptional and translational levels as monitored by the accumulation of BN115 transcripts and by histochemical assay of glucuronidase activity [39]. However, cold activation of BN115 is inhibited by benzyl alcohol, taxol, and Gd^{3+} or ruthenium red, demonstrating that cold activation of BN115 requires membrane rigidification, cytoskeleton reorganization, and Ca^{2+} influx. They suggest that cytoskeleton rearrangements may mediate cold signal transduction from the rigidified membrane to the Ca^{2+} channels, thereby activating the expression of stress proteins [39]. In addition, MT stability increases with ABA treatment during the initial stages of plant cold acclimation [40]. Abdrakhamanova and colleagues also observed that cold acclimation proceeds faster in freezing-tolerant cultivars than in freezing-sensitive cultivars. They also show that early, transient MT disassembly is sufficient for an efficient induction of acclimation, whereas the expression of α-tubulin in the TUA1/2 type is downregulated in the freezing-resistant cultivars during cold acclimation [6]. These results suggest that cold acclimation may be triggered by MT dynamics and the activity of MAPs. Thus, MTs may function as "thermometers" to modulate the induction of cold acclimation [6]. Most recently, studies on the response of interphase and mitotic MT arrays in the root meristem cells of spring and winter cultivars of wheat (*Triticum aestivum* L.) to cold stress and acclimation to cold have indicated that, in general, interphase MTs are more resistant to cold than mitotic MT arrays in both cultivars. These observations suggest that CMTs play a more important role in plants under cold stress [41].

Although the identity of the MAPs that regulate MT organization in response to low temperature has not been clearly established, AtMAP65-2, a member of the AtMAP65 family, could play a crucial role in the low temperature response. Li and colleagues report that the region of AtMAP65-2 that encompasses amino acids 495-578, which forms a flexible extended loop, plays an important role in the stabilization of MTs. Bacterially expressed AtMAP65-2 fusion proteins have been shown to induce the formation of large MT bundles and markedly stabilize low temperature-treated MTs *in vitro*. Analysis of suspension-cultured *Arabidopsis* cells that express the AtMAP65-2-GFP fusion protein has also shown that CMTs in AtMAP65-2-GFP are more resistant to ice treatment *in vivo* [42]. These results demonstrate that AtMAP65-2 strongly stabilizes MTs at low temperature and may play a vital role in plant cold tolerance.

MICROTUBULES AND ABIOTIC STRESS SIGNALS

A generic stress signal transduction pathway starts with signal perception followed by the generation of second messengers and activity of cell-adaptive responses to abiotic stresses [10, 11]. It is of fundamental importance to understand the mechanisms by which plants perceive environmental stimuli and transmit signals to the cellular machinery. Although there have been significant advances in research on the adaptive mechanisms associated with the responses to high salinity, drought, and low temperature, there are still many facets of the signal transduction pathways that await elucidation. In particular, no sensor has been confirmed for any of the three aforementioned stresses [10, 11].

From the perspective of a single cell, an environmental signal is a packet of information transmitted from cell wall to plasma membrane to protoplasm. Because the CMTs underlying the plasma membrane in plant cells constitute a continuum with the cell wall and plasma membrane, it has been assumed for over 10 years that these CMTs may be responsible for perceiving environmental signals [1, 43, 44]. Recent advances have led to the discovery that CMT rearrangements mediate cell responses to certain environmental signals; for example, gravity, wounding, light, and biotic stresses [43]. Thus, changes in CMT arrays are often associated with the reg-

ulatory mechanisms of abiotic stress signaling pathways. To date, CMTs have been shown to be involved in at least three signaling pathways, namely, Ca^{2+} signaling, ABA biosynthesis, and the formation of plant cell walls.

The Ca^{2+} signal generated by transient Ca^{2+} influx into the cell cytoplasm can be induced under salinity, drought, and cold stresses [10, 11]. Therefore, the channels responsible for this Ca^{2+} influx may represent one type of sensor for these stresses [10]. The results of several studies have shown that CMTs may be involved in regulating the activity of Ca^{2+} channels under normal conditions [45, 46]. Some studies have found that under abiotic stress disruption of the CMT network is an important upstream event of Ca^{2+} signaling. For example, the disruption of the MT network by various MT-interacting drugs increases cytosolic Ca^{2+} in cold-shocked *N. plumbaginifolia* protoplasts [44]. Sangwan and colleagues report that cold activation of BN115 requires membrane rigidification, cytoskeleton reorganization, and Ca^{2+} influx. They proposed that in response to the changes, the cytoskeleton acts as a scaffold that transduces physical forces into biochemical signals. It is likely that cytoskeleton rearrangements may mediate the transduction of the cold signal from the rigidified membrane to the Ca^{2+} channels [39]. In addition, disruption of MTs can ensure the efficient functioning of cold-activated Ca^{2+} channels, thereby triggering the signal pathway, and culminating in cold acclimation [6]. By using yellow cameleon (YC 3.6) as a calcium reporter, notable increases in $[Ca^{2+}]_{cyt}$ after initial salt stress-induced depolymerization of the CMTs is found, and these increases are inhibited when paclitaxel is added [12]. These results suggest that it is likely that a Ca^{2+} influx follows the changes in MT array responsible for regulating Ca^{2+} channels under abiotic stresses.

ABA is a major phytohormone that can enhance plant tolerance under abiotic stresses. Rapid biosynthesis and significant accumulation of ABA in plant cells is an early and more obvious physiological event in the adaptive responses to abiotic stresses [10, 11]. It has also been shown that ABA can affect the organization of MTs under normal conditions [47, 48]. Under abiotic stress (chilling temperature) a clear destruction of MTs occurs, and anti-MT drug treatment significantly accelerates and enhances the chilling damage in cotton, whereas pretreatment with ABA can prevent chilling damage and MT destruction. Evidence for an ABA-induced decrease in the generation of MT depolymerization at the initial stages of plant cold acclimation has also been presented [36, 40]. These findings suggest that ABA enhances plant cold tolerance partly through affecting MT organization. Most interestingly, MTs that interact with integrin-like proteins have been shown to be involved in cell wall–plasma membrane interaction and osmotic stress-induced ABA accumulation [30]. Pharmacological approaches provide further evidence that disruption and stabilization of MTs significantly stimulate ABA accumulation in maize root cells, suggesting that MT dynamics may be involved in mediating osmotic stress-induced ABA biosynthesis [18].

The cell wall is the first structure of a plant cell to perceive environmental stimuli; accordingly, the cell wall must contain elements responsible for the response mechanisms. During primary wall formation, CMTs establish transverse organization during the early stages of cell elongation to direct the orientation of cellulose microfibril deposition, aligning the CESA complexes [26, 27, 49]. However, some recent studies have established that the alignment of CMTs and cellulose microfibrils is not always coupled during cell elongation [50, 51]. This suggests that cellulose microfibrils have the ability to largely self-align in the absence of the pre-stage orientation of CMTs. Therefore, the CMTs may only be required for directing cellulose into an orientation, for example, in response to various environmental stimuli. Recent research on plant drought tolerance has demonstrated that CMT organization can drive cellulose microfibril arrays by guiding the trajectories of active CESA in the plasma membrane, and that the association of CESA compartments with CMTs is greatly increased during osmotic stress [28]. Another report has confirmed that CMTs not only guide the trajectories of CSCs but also that osmotic stress can affect internalization of CSCs in MT-associated CESA compartments [29]. Therefore, CMTs may constitute a target in the plant cell wall—a complex sensory panel for transmitting extracellular information to the intracellular signaling network—under abiotic stresses.

In addition, reactive oxygen species (ROS), as signal molecules induced by abiotic stresses in plants, have been shown to cause fragmentation of MTs *in vitro* and mitotic arrest in tobacco BY-2 cells [52, 53]. Phospholipase D, which is involved in ABA- and ROS-mediated processes as part of the plant response to abiotic stresses, has been considered a putative linker between CMTs and the plasma membrane [54-56]. The close relationship between these signal molecules and CMTs further suggests that CMTs may be involved in the responses to signal transduction pathways under abiotic stresses.

In summary, the accumulated data point to the existence of highly dynamic MT arrays and the activation of complex MT regulatory networks in response to abiotic stresses. The important role played by MTs in mediating the plant cell response to abiotic stresses is particularly emphasized (Scheme). It is very clear that remodeling of CMTs, which constitute a continuum between cell wall and plasma membrane as the headstream of signal transduction, is an integral element in the signaling cascades that significantly enhance plant adaptation to abiotic stress. Although the mechanisms by which CMTs regulate abiotic stress signaling pathways and the identity of the MAPs involved in these mechanisms remain to be elucidated, we believe that accumulating evidence over the next few years will provide further support for the close relationship between MTs and the

adaptation of plants to abiotic stress, and in particular the process of signal perception by CMTs.

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