Plasma Membrane Proton Pump in Plants

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In plants, the plasma membrane proton pump (PM H^+-ATPase) regulates numerous transport-dependent processes such as growth, development, basic physiology, and adaptation to environmental conditions.

plasma membrane H+-ATPase | pleiotropy | multitasking | plant physiology

1. Growth

The PM H^+-ATPase is a key element of plant molecular mechanisms regulating cell elongation and growth. Auxins are one of the extensively studied phytohormones involved in a wide range of growth and developmental processes such as embryogenesis, organogenesis, and tropisms [1][2]. As growth regulators, auxins indirectly modulate PM H^+-ATPase activity and manipulate H^+ ion fluxes across the plasma membrane for regulating processes involved in cell elongation [3][4][5]. It has been established that auxin induces the extrusion of protons via PM H^+-ATPase activation leading to apoplast acidification (pH approximately 4,5–6) [6]. The decrease in apoplast pH triggers the activation of proteins that mediate cell wall loosening by acting on the polysaccharide network [7]. Additionally, PM H^+-ATPase activation induces plasma membrane hyperpolarization and triggers K^+ ion influx leading to water uptake and cell turgor increase [8][9]. Both cell wall loosening and turgor increase are crucial factors for cell growth and are part of the Acid Growth Theory [3][6][8][10].

In shoots, the mechanism of growth promotion upon auxin perception is in agreement with the Acid Growth Theory. Takahashi et al. [3] proved that auxin application onto Arabidopsis hypocotyl segments increased AHA2 Thr-947 phosphorylation and 14-3-3 binding leading to its activation. It has been postulated that PM H^+-ATPase activation is induced via the TIR1/AFB pathway of auxin nuclear signaling, which is based on the de-repression of auxin-induced genes at the transcription level [11]. The expression of several members of the SMALL AUXIN UP mRNA (SAUR) gene family is strongly and rapidly induced by auxin perception [12]. Additionally, it has been established that SAUR19-24 subfamily proteins are engaged in promoting cell expansion [13]. Further studies revealed that SAUR9, -10, -19, -40, and -72 negatively regulate the PP2C.D subfamily of 2C protein phosphatases to modulate PM H^+-ATPase activity. PP2C.D proteins decrease PM H^+-ATPase activity by indirect regulation of penultimate Thr residue dephosphorylation and, therefore, 14-3-3 proteins binding inhibition. Thus, SAUR proteins, by inactivation of PP2C-family phosphatases, promote an increase in PM proton pump activity, leading to hypocotyl cell growth via an acid growth mechanism [14].

Increased ATP hydrolytic activity was observed in auxin-treated roots. Furthermore, analysis of phospho-proteomics data revealed that Thr-947 in AHA2 was highly phosphorylated in roots treated with auxin [4]. Recent

studies have described transmembrane kinases (TMKs) interacting with PM proton pumps as components of the auxin signaling pathway. Auxin-triggered TMKs activation leads to phosphorylation of the penultimate Thr residue and activation of PM H^+-ATPases [4][15]. Notably, TMK1-triggered apoplast acidification is increased at lower auxin levels [4].

2. Stomata Opening

Stomata opening and closing regulations are vital in terms of gas exchange and the maintenance of photosynthetic processes. Stomata opening is triggered by PM H^+-ATPase activation, leading to plasma membrane hyperpolarisation, massive ion influx, and, therefore, guard cell swelling [16]. Studies have shown that PM proton pump activation in guard cells is induced by the perception of blue light by protein kinases acting as blue light receptors expressed in guard cells, named phototropins (phot1 and phot2) [17][18]. Notably, it was observed that red light also induces penultimate Thr residue and activates PM H^+-ATPase in whole leaves via photosynthesis-dependent mechanisms [19].

Blue light perception by phototropins induces its activation via autophosphorylation and initiates a signaling pathway that leads to stomatal opening [18][20]. Auto-activated phototropins directly phosphorylate BLUE LIGHT SIGNALING1 (BLUS1) protein kinase, which indirectly passes the signal to type 1 protein phosphatase (PP1) [21]. PP1 and its regulatory subunit PRSL1 mediate the light signal to PM H^+-ATPase via yet unknown mechanism that triggers PM proton pump phosphorylation at the penultimate Thr residue [22][23][24][25]. Recently, it was revealed that membrane-localized type 2C protein phosphatase clade D (PP2C.D) members mediate the dephosphorylation of penultimate Thr residue in guard cells AHA2. The *pp2c.d6/9* double mutant exhibited an open stomata phenotype in the dark and delayed AHA2 dephosphorylation in guard cells after blue light illumination. Additionally, in plants overexpressing *PP2C.D9,* the stomatal opening is inhibited after light illumination [26]. However, the mechanism leading to PP2C.D proteins inhibition and PM H^+-ATPase activation upon blue light perception is still unknown.

3. Mineral Uptake

The essential mineral elements required for plant growth and development are taken up from the soil by roots and then transported to upper organs via the vascular system. The uptake of various charged compounds is facilitated by specific transporters and channels. Many of those transporters are H^+ symporters activated by extracellular acidification, therefore, require PMF generated by PM H^+-ATPase to maintain their transport activity [27][28]. Here the researchers present a few examples of PMF-coupled uptake of mineral compounds.

Nitrogen is taken up from the soil in inorganic forms, such as nitrate (NO_3^-) and ammonium (NH_4^+), as well as organic compounds (urea, amino acids, short peptides) [29]. NO_3^- uptake in roots is mediated by transporters belonging to NPF (NRT1/PTR) and NRT2 families [29][30][31]. It has been revealed by electrophysiological studies that nitrate uptake by roots is an active process that occurs via the $2H^+/NO_3^-$ symport mechanism [30][32]. Additionally, it has been described that the ammonium transporter AMT1, localized in the plasma membrane of root

cells, functions as an $NH4^+/H^+$ symporter [33]. Corresponding to its $NH4^+/H^+$ symporter activity, PM H^+-ATPase-mediated apoplast acidification leads to stimulation of AMT1-mediated $NH4^+$ transport [33][34].

Phosphorus can be absorbed by root cells in two inorganic forms–$H_2PO_4^-$ and HPO_4^{2-} via transporters belonging to the phosphate transporters family (PHT). PHT1 subfamily members of phosphate transporters are localized in the plasma membrane and mediate inorganic phosphate uptake from the soil via the H^+ symport mechanism [28]. The stoichiometry of $H_2PO_4^-$ and HPO_4^{2-} symport across plasma membrane has been described as $2H^+/1H_2PO_4^-$ or $4H^+/1H_2PO_4^-$ and $3H^+/1HPO_4^{2-}$ [35][36].

Potassium ions are transported into root cells via two K^+ electrochemical gradient-dependent transport systems characterized by their K^+ affinity level [37]. A low-affinity K^+ transport system mediated by the K^+ inward-rectifying channel of the Shaker family AKT1 is dominant upon high levels of K^+ external concentration and facilitates the passive influx of potassium ions [38]. Members of the KUP/HAK/KT transporter family, mainly HAK1 (in barley, rice, and pepper) and HAK5 (in Arabidopsis and tomato) are involved in high-affinity K^+ uptake systems in roots in low external K^+ concentration [39][40]. Electrophysiological studies have confirmed that in higher plants, high-affinity K^+ uptake is mediated by the H^+/K^+ symport mechanism [41][42]. Additionally, it was observed that extracellular acidification mediated by PM H^+-ATPase increased KUP/HAK/KT transport activity [42][43]. Thus, it has been concluded that K^+ uptake in low K^+ external concentration is an active process driven by plasma membrane potential created by PM H^+-ATPase via H^+/K^+ symporters [44].

In higher plants, SULTR1 and SULTR2, which belong to the SULTR family, are the major transporters responsible for sulfur (SO_4^{2-}) absorption from the soil [45][46][47]. The SO_4^{2-} uptake occurs against the electrochemical gradient of the plasma membrane and thus is an energy-driven process requiring PM H^+-ATPase activity [48]. The SULTR1 and -2 transporters localized in the plasma membrane couple sulfate influx with co-transport of protons and therefore function as H^+/SO_4^{2-} co-transporters. Additionally, SULTR1 and -2 transporter activity are increased in lower external pH, which is in agreement with the consensus that the proton gradient is the driving force for SO_4^{2-} uptake in plants [49].

4. Cytosolic pH Homeostasis

The cytoplasmic pH ($pH_{cyt.}$) of plant cells is strictly regulated and stabilized in the narrow range of 7.1–7.5 pH [50]. $pH_{cyt.}$ stabilization can be achieved via PM H^+-ATPase activity which converts chemical energy stored in ATP into an electrochemical H^+ gradient across the plasma membrane to fuel further secondary transport of charged compounds. It has been proven that secondary cation/H^+ antiporters belonging to NHX (Na^+/H^+ exchanger) and CHX (cation/H^+ exchanger) families play a major role in establishing $pH_{cyt.}$ in normal as well as stress conditions [50][51][52]. Moreover, intercellular metabolic processes, either consuming or producing H^+, are a vital part of a cytosolic pH stabilization mechanism, as they prevent alkalinization of the cytosol during H^+ efflux [53][54]. Studies have shown that PM H^+-ATPase activity is sensitive to changes in the pH of cytoplasm and achieves its maximum at approximately 6.5–6.8 pH [55][56]. Additionally, the generation of H^+ ions during metabolic processes provides PM H^+-ATPase with its substrate during long-term H^+ fluxes [57].

5. Adaptation to Salt Stress

One of the major environmental abiotic stress factors that globally affects plant growth and physiology is highly saline soil [58]. High concentration of salt in the soil causes the accumulation of toxic levels of Na^+ in plant cells, leading to osmotic stress and disruption of cellular ion homeostasis [59]. To overcome salt stress, plants have evolved a wide range of adaptation mechanisms by perceiving high Na^+ concentrations and changes in osmotic pressure [60][61]. Among various molecular pathways initiated during salt stress, PM H^+-ATPase is one of the key elements in osmoregulation and ion homeostasis maintenance [62]. Studies have shown that PM H^+-ATPase is upregulated at the transcriptional and posttranslational levels in high salinity conditions [63][64][65]. It has been established that maintaining an optimal intercellular K^+/Na^+ ratio is essential for salt stress tolerance in plants [66][67][68]. To sustain a low Na^+ level in the cytoplasm in a highly saline environment, Na^+ extrusion to the apoplast is mediated by Na^+/H^+ antiporter identified as SOS1 (Salt Overly Sensitive 1) [69]. The SOS1 transporter is a part of the SOS regulatory pathway activated under salt stress conditions [70][71][72]. The SOS1 Na^+/H^+ antiporter requires an electrochemical proton gradient generated by PM H^+-ATPase for the extrusion of Na^+ against its electrochemical gradient [62]. Additionally, it has been observed that the plasma membrane is depolarized under saline conditions due to massive Na^+ influx [73]. The most severe effect of PM depolarization for ion homeostasis in plant cells is drastic K^+ efflux via outward-rectifying depolarization-activated (GORK) channels [73][74]. It was reported that plant varieties with intrinsically higher activity of PM H^+-ATPase displayed lower NaCl-induced K^+ efflux due to more negative resting plasma membrane potential [74].

6. Adaptation to Drought Stress

Plant response to drought conditions involves mechanisms that lead to stomata closure during the daytime to minimize water loss due to transpiration [16]. Studies have shown that the abscisic acid (ABA) phytohormone signaling pathway regulates PM H^+-ATPase activity in guard cells under drought stress [75][76]. ABA-mediated stomata closure defense mechanism upon drought sensing relies on plasma membrane depolarization due to activation of anion efflux channels, which leads to K^+ efflux from guard cells via K^+ outward-rectifying channel. Additionally, ABA inhibits the activity of K^+ inward-rectifying channels [77]. Taken altogether, sustained K^+ efflux from guard cells leads to the loss of guard cell turgor and stomata closing [78]. To maintain plasma membrane depolarization and stomata closing under drought conditions, ABA negatively regulates PM H^+-ATPase activity in guard cells by dephosphorylation of penultimate Thr residue [79][80]. It has been suggested that PM H^+-ATPase inactivation in ABA-mediated pathway stomata closure is triggered by members of the SnRK2 kinase family [79][80][81].

7. Heavy Metals and Temperature Stresses—Poststress Responses for Homeostasis Maintenance

Some heavy metals, such as Zn and Cu, are essential elements for plant growth, while others, such as Cd, are not. Nevertheless, they are toxic to plants at high concentrations. Damage to the cell membrane system, especially the

plasma membrane, is one of the primary events of heavy metal toxicity in plants. Disruption of membrane integrity is thought to be due to complex interactions between heavy metals and the functional groups of membranes. It is well known that metal ions are easily bound to both the sulfhydryl groups of proteins and the hydroxyl groups of phospholipids [82]. They can also replace calcium ions at essential sites on the cell membranes [83]. All these events result in an increase in non-specific membrane permeability and a parallel decrease in specific transport activities that disrupt ionic homeostasis and, subsequently, the activities of many enzymes crucial for basic cell metabolism.

The effect of metals on PM H^+-ATPase activity depends on the time of exposure of plants to heavy metals, the type and concentration of heavy metals, and plant species. In cucumber seedling roots, a two-h treatment with Cd or Cu (10 and 100 μM) inhibited PM H^+-ATPase activity [84]. Kennedy and Gonsalves [85], Fodor et al. [86], and Burzyński and Kolano [87] observed a similar inhibitory effect of short-term treatment with Cd or Cu on the activity of proton pumps in the roots of different plants. However, a longer treatment time (six days) of plants (cucumber) with heavy metals (Cd and Cu) led to increased activity of the enzyme [88]. Moreover, the expression of a few isogenes encoding PM H^+-ATPase in Cd-treated cucumber seedlings was upregulated. In contrast, alteration of PM proton pump activity in cucumber roots stressed with Cu did not appear to affect gene expression levels [88]. Similarly, in rice treated for five or ten days with Cd, increased proton pump activity has been observed [89]. Hippler et al. [90] found that treating plants with copper for 72 h inhibited *AHA2* expression in *Arabidopsis thaliana* but had no effect on *AHA1* and *AHA5*. *AHA2* is the predominant proton pump in the roots and is upregulated after nitrate supply [91]. In contrast, treatment of the same plants with Cu for a longer period (15 days) did not inhibit *AHA2* gene expression [90].

When plants are exposed to heavy metal stress for a long time, they must replenish lost nutrients and remove toxic excess heavy metals from the cytosol to survive. Maintaining the active transport of ions and organic compounds across the PM requires the increased generation of a proton gradient by PM H^+-ATPase. Besides using the proton gradient to replenish the loss of essential substances in repair processes, the more important to plants is to remove excess toxic ions from the cytoplasm to the outside of cells. In plants, transporters of the cation diffusion facilitator (CDF) family appear to mediate the cytoplasmic efflux of heavy-metal cations. CDF family proteins are membrane-divalent cation transporters that transfer metal ions out of the cytoplasm into the extracellular space or vacuoles, and they act as metal^{2+}/H^+ antiporters [92]. These proteins are known as metal-tolerance proteins (MTPs). In this family, at least one MTP8 could participate in the efflux of Cd and Cu from the cytosol, as it has been shown that *MTP8* is upregulated in response to Cd and Cu in the roots of *Arabidopsis halleri* [93]. The mechanism of Cd detoxification that relies on Cd^{2+}/H^+ antiport activity in plant plasma membranes has been previously reported [87].

Similar to what has been observed regarding heavy metal stress, low- and high-temperature conditions disrupt plasma membrane integrity. The biophysical lipid properties of the plasma membrane contribute to its sensitivity to temperature changes [94]. Both low and high temperatures modulate plasma membrane fluidity [95]. During exposure to low-temperature conditions, the proportion of unsaturated fatty acids in the plasma membrane increases, leading to rigidification. Conversely, high temperatures contribute to membrane fluidization [95]. Alterations in plasma membrane fluidity may affect the activity of proteins localized within their structure [96]. Additionally, electrolyte leakage caused by increased plasma membrane permeability was observed under both

low- and high-temperature conditions [96][97][98]. Therefore, plants exposed to non-optimal temperature conditions survive, activate repair mechanisms that restore ionic homeostasis and replenish the loss of essential compounds [95][99][100]. PM H^+-ATPase is the key enzyme in maintaining ion homeostasis in plants, considering its role in generating protonmotive force and thus energizing the secondary transport of numerous chemical compounds, as described in previous sections. Therefore, multiple studies have investigated the expression and activity patterns of PM H^+-ATPase in plants exposed to temperature-stress conditions.

In cucumber seedlings, decreased hydrolytic and transporting activity of PM H^+-ATPase was observed in plants transferred to low temperatures (10 °C) for two and three days, compared to seedlings grown under control conditions (25 °C) [101]. Notably, the expression of the most abundant PM H^+-ATPase isogenes in the roots of cucumber grown for three days at 10 °C was lower than that in the control plants. However, a significant increase in the activity of cucumber PM H^+-ATPase was detected in plants grown for five, six, and seven days at 10 °C, as well as in plants grown at 10 °C for three days and then transferred to 25 °C for the following three–four days (post-stress plants). Additionally, the analysis of PM H^+-ATPase cucumber isoform transcripts showed that after six days at 10 °C, the expression of a few *CsHA* genes was significantly increased [101]. Parallel analysis was conducted on Arabidopsis seedlings grown under low-temperature conditions (4 °C) [96]. A strong decrease in the activity of PM H^+-ATPase in Arabidopsis seedlings after six hours of exposure to 4 °C was observed compared with the control group. The activity of PM H^+-ATPase in Arabidopsis seedlings recovered after 12 h of exposure to low-temperature conditions. Notably, 18 h of exposure to 4 °C caused a significant (approximately four-fold) induction of PM H^+-ATPase activity in comparison to seedlings in the control group. Exposure of Arabidopsis seedlings to low temperatures for 12 h resulted in a substantial increase in *AHA1* and *AHA2* transcription. Remarkably, after 18 h of cold stress, the expression of *AHA1* and *AHA2* was up to 100-fold higher than that in the control plants [96].

Data regarding PM H^+-ATPase activity and expression patterns under high-temperature conditions are currently limited. However, a study of cucumber seedlings exposed to heat shock (HS) showed stimulation of PM H^+-ATPase transport and hydrolytic activity after two hours of incubation at 48 °C, compared to plants grown under control conditions (25/22 °C). Corresponding results were obtained for cucumber seedlings transferred to control conditions for 24 h after 2 h of exposure to HS (post-stress plants). Real-time PCR revealed an increase in the expression levels of *CsHA4* and *CsHA8* in post-stress plants compared to control plants [102].

These studies presumably indicate that PM H^+-ATPase initially undergoes inactivation during temperature stress due to a plasma membrane integrity disorder. However, its activity is subsequently restored. The recovery of PM H^+-ATPase activity during ongoing temperature stress could be a part of the plant adaptation mechanisms to unfavorable temperature conditions. The increased expression levels of the most abundant isoforms of PM H^+-ATPase in plants treated with high or low temperatures indicate the participation of transcriptional pathways in the adaptation process. Additionally, studies have shown that PM H^+-ATPase is highly activated during the post-stress stage. Taken together, these observations imply that PM H^+-ATPase is a crucial element for ionic homeostasis restoration as well as a vital factor for replenishing the loss of vital compounds due to an increase in PM

permeability during temperature stress. Nonetheless, further studies on PM H^+-ATPase activity and regulation of expression under temperature stress are required.

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