

Review Article

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Organelle Dynamics during Salinity-Induced Programmed Cell Death in Plants

Programmed cell death (PCD) is a complex mechanism induced by environmental stress (ePCD) or developmental processes (dPCD). ePCD can be triggered by various types of unfavorable abiotic conditions, including salinity stress. Exposure to high concentrations of salt not only alters plant morphological characteristics but also severely affects biochemical and molecular processes. Furthermore, plants activate numerous defense mechanisms that operate in different organelles within the plant cell. These mechanisms involve ionic, osmotic, and oxidative damage affecting the plasma membrane, mitochondria, chloroplasts, vacuoles, and nucleus. A major disturbance in the plasma membrane is characterized by impaired transport processes, ROS accumulation, and Ca^{2+} influx. Mitochondrial dysfunction is closely associated with excessive ROS accumulation, activation of protective pathways such as AOX, UCPs, and the GABA shunt, and cytochrome c release. Moreover, the photosynthetic rate is substantially reduced in association with a decline in chlorophyll content. Vacuoles integrate osmotic and ionic signals by sequestering Na^+ through NHX antiporters and V-ATPase activity; however, when this system collapses, Ca^{2+} signals converge on the nucleus. Furthermore, the overproduction of ROS acts as a signaling molecule in the activation of EXECUTER proteins involved in chloroplast–nucleus communication. Salinization activates osmotic, ionic, and oxidative signaling pathways that ultimately result in chromatin degradation. Altogether, these interconnected processes contribute to the induction of PCD.

Keywords: Programmed cell death, salinity stress, reactive oxygen species, nitric oxide, phytohormones, enzyme.

Introduction

Programmed cell death (PCD) is a genetically regulated series of events resulting in cell death [1]. It eliminates unnecessarily damaged cells across all developmental stages, starting from germination to the plant's death [2]. PCD is classified into two distinct groups based on the mode of induction, triggered by environmental stress (ePCD) and triggered by developmental processes (dPCD) [3]. dPCD is involved in the latest stage of cell differentiation in various parts of the plant, controlling the formation of new cells; moreover, it may occur as a result of cell-to-cell signaling, and it is the final point of senescence in all plant tissues [1]. On the other hand, ePCD is induced by abiotic (drought, salinity, temperature, heavy metals, etc.) or biotic (pathogens, parasites, insects, etc.) stresses. In both types, PCD is a crucial step in plant growth and development, helping to control and respond to the internal or external stimuli in plants.

Among other defense mechanisms in a plant, PCD plays a key role during stress conditions, but it acts differently compared with other responses. In most cases, the defense systems have interplay with reactive oxygen species (ROS), which, in the case of PCD, is a main regulatory factor. However, during pPCD, damaged or highly affected cells by stress conditions are purposely killed in order not to affect the other living cells. So the plant can maintain a stable and acquired number of healthy living cells [4].

Review

Firstly, the term PCD was used by Lockshin and Williams (1964) and since then, many studies on the role in plant growth and development have been carried on. Several studies indicate that signaling pathways regulating PCD in both animals and plants are similar. PCD in plants is not only a developmental process, but also a significant response mechanism under salt stress. Salinity triggers ion imbalance, osmotic stress and oxidative damage, which can activate PCD pathways in plant cells [5]. Under salt stress,

overaccumulation of ROS becomes a “secondary stress” that impairs cellular function in organelles like chloroplasts, mitochondria, peroxisomes and the apoplast are principal sources of ROS under such conditions [6]. ROS accumulation under salinity not only damages molecular component, but also serves as a signaling cascade to initiate PCD. In salt-stressed cells, NADPH oxidases and mitochondrial respiration are activated, further enhancing ROS levels that lead to cell death [7]. NO acts as a redox-active signaling molecule, promoting survival at low concentrations but contributing to PCD at high levels, particularly when ROS are elevated. Crosstalk between NO and ROS regulates redox balance and forms peroxynitrite (ONOO⁻), a potent oxidant that damages membranes, proteins, and nucleic acids, triggering PCD (a phenomenon called nitrosative burst) [8, 9]. Ionic imbalance, including high Na⁺ influx and K⁺ efflux, also contributes to PCD activation under salinity [7]. Calcium signaling is another critical regulator of salt-induced PCD, as elevated cytosolic Ca²⁺ activates downstream cascades, including ROS-producing enzymes like vacuolar processing enzymes (VPEs) and other cell death effectors [10]. Phytohormones coordinate stress responses and PCD: salicylic acid (SA) enhances antioxidant activity and reduces ROS-mediated cell death [11], while ethylene provides protection, as shown by higher salt-induced death in ethylene-insensitive *Arabidopsis* mutants (*ein2-5*, *ein3-1*) [12]. Jasmonic (JA) and gibberellins (GA) interact with SA pathways, redox balance and gene expression, influencing cell survival of PCD under salinity [13].

During PCD, various organelles play specific but interconnected roles. For example, the plasma membrane (PM) acts as the primary sensor of environmental stress, detecting ionic, osmotic and oxidative changes and initiating signaling cascades [21]. The mitochondria serve as one of the early stress sensors by releasing signaling factors, including the release of cytochrome c, which initiates further downstream cell death events [14]. Also, nuclear changes include chromatin condensation and nuclear degradation, triggered downstream of vacuole rupture during tracheary element differentiation and PCD in *Zinnia* plant [15]. Moreover, the vacuole undergoes tonoplast premeabilisation or rupture to release hydrolyses into cytoplasmic contents [16]. Finally, chloroplasts participate in ROS generation under stress and may experience thylakoid disorganization [17]. Altogether, these events are intertwined, forming cross-talk circuits that push the cell toward irreversible death.

Under salinity stress, PCD is a tightly regulated process in which stress signals like ROS, NO, Ca²⁺, and phytohormones activate proteolytic enzymes and trigger organelle responses. Excessive salinity alters ions as well as redox balance, leading to structural and biochemical alterations in PM, mitochondria, chloroplasts, vacuoles and nuclei. These organelles communicate through ROS as well as calcium signaling, ensuring coordinated execution of cell death. Understanding their cross-talk during salinity stress gives valuable information about plant adaptations and the regulation of PCD during stress. In many studies, researchers described this mechanism mostly in animals; however, there are limited sources about plant PCD induced by salinity. Moreover, numerous amount of discoveries were made on the separate organelles, whereas few reviews summarise that knowledge about the relationship between plant organelles and PCD. Hence, in the following review, we will focus on detailed biochemical changes occurring in these organelles and their interconnections during salinity stress that lead to PCD.

1. Salinity stress

One of the most widespread stress factors affecting plants is salinization. It is estimated that by 2050, 50 % of cultivable land will be impacted by salinity stress [18]. It is described as a high concentration of sodium (Na⁺), chloride (Cl⁻), potassium (K⁺), and sulfate (SO₄²⁻) in a soil, which inhibits mineral uptake by roots. Further, these ions can subsequently be dissolved in water and transported to plant cells [18]. According to the type of salinity-inducing elements and pH, salinization is classified into two distinct groups: neutral and alkaline salinity. Neutral salinity is mainly induced by NaCl at pH = 6.998, and alkaline one is caused by sodium carbonate (Na₂CO₃) or potassium carbonate (K₂CO₃), where pH > 10.5 [19].

Salt stress conditions not only affect plant morphology but also exert profound effects on biochemical and molecular processes. Therefore, it affects many organelles within a plant cell, including the PM, mitochondria, vacuole, chloroplast and nucleus. Moreover, the homeostatic disturbance during high salinity exposure is classified into two stages known as primary (decline in plant growth and mineral absorption) and secondary (damage of biomolecules by ROS) [20].

2. Plasma Membrane (PM) dynamics under salinity stress

Plasma membrane (PM) serves as a vital interface between the plant cell and its environment, exhibiting high dynamism and changing structural and functional features in response to environmental

stresses [21]. Under salinity, the PM becomes the primary sensor of ionic, osmotic and oxidative stresses, which act together and directly affect the behavior of PM channels [22]. The ionic aspect of salinity arises from toxic effects of specific ions, such as Na^+ and Cl^- , and from nutrient disruptions, while high salt concentrations lower water potential, causing water deficit or osmotic stress [23]. In addition, salt stress promoted the generation of ROS, including singlet oxygen, superoxide anion, hydrogen peroxide and hydroxyl radicals, causing oxidative stress in many plants [24].

Figure 1 indicates that PM-associated stress sensors, such as receptor-like kinases (RLKs) [25], glycosyl inositol phosphoryl ceramides (GIPCs) [26], and other membrane proteins, first detect high external salts. These sensors trigger downstream signaling pathways, notably the SOS (Salt Overly Sensitive) pathway, which attempts to regulate Na^+ extrusion and maintain ionic homeostasis [25–28].

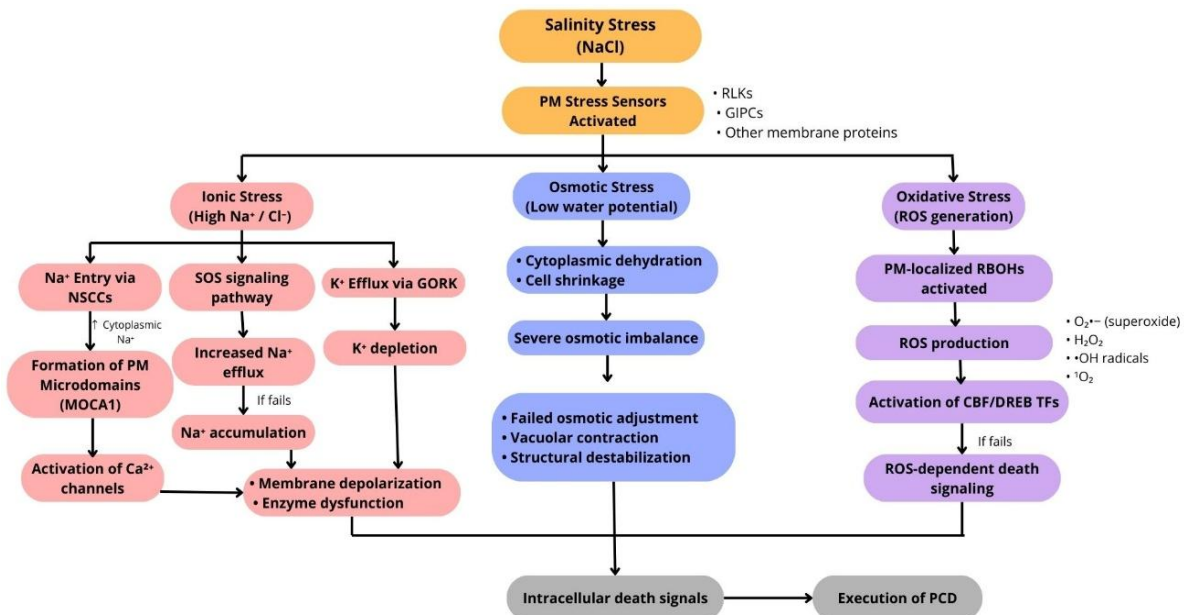


Figure 1. Plasma membrane function during salinity stress, which leads to PCD.
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For ionic stress, Na^+ enters the cytoplasm through non-selective cation channels (NSCCs) like cyclic nucleotide-gated channels (CNGCs) [29] and glutamate-activated channels (GLRs) [30], while K^+ efflux occurs via outward-rectifying K^+ channels (GORK) [31]. High cytoplasmic Na^+ leads to the formation of PM microdomains (MOCA1), which in turn activate Ca^{2+} channels, resulting in a significant influx of Ca^{2+} into the cytoplasm. If the PM transporter, like SOS1, that plays a critical role in controlling the NHX antiporters in the vacuole, becomes overwhelmed [32], then cytoplasmic Na^+ accumulation and K^+ depletion destabilize membrane potential and disrupt enzyme activity, generating strong intracellular death signals that contribute to PCD initiation [33].

Osmotic stress caused by high salt reduces water potential [34], leading to cytoplasmic dehydration and cell shrinkage. PM sensors, including RLKs and GIPCs, detect these changes, and the elevated Na^+ levels further trigger Ca^{2+} influx through PM channels [25, 26]. Under extreme stress, osmotic adjustment fails, resulting in vacuolar contraction and structural destabilization that amplify the intracellular death signal, further driving PCD [35].

Meanwhile, the PM acts as a major site for ROS production via NADPH oxidases (RBOH) [36]. ROS such as superoxide anion and hydrogen peroxide accumulate in the cytoplasm and apoplast, overwhelming detoxification mechanisms [37–39]. ROS production at the PM, combined with ionic and osmotic imbalances, acts as a death signal, activating downstream transcription factors (e.g. CBF/DREB) [40–42] and organelle-mediated PCD pathways, including mitochondrial dysfunction, vacuolar collapse and caspase-like protease activation [33].

Thus, under severe salinity stress, the PM integrates ionic, osmotic and oxidative stress signals, but rather than protecting the cell, its dysregulated responses converge to trigger PCD.

3. Mitochondrial Dynamics Under Salinity Stress

Mitochondria show a distinct and highly sensitive response to salinity stress because of their central roles in energy production, signaling and carbon metabolism [43]. Salt stress disturbs the normal balance of mitochondrial fission and fusion, leading to swelling, fragmentation and a decrease in membrane potential [44]. As ion homeostasis and osmotic balance shift under high salt, mitochondrial compartments sense cytosolic Na^+ accumulation and initiate early adaptive responses [45]. One of the earliest changes is a decline in the inner membrane potential ($\Delta\Psi_m$) due to the opening of permeability transition pores (PTPs) [46], as illustrated in Figure 2.

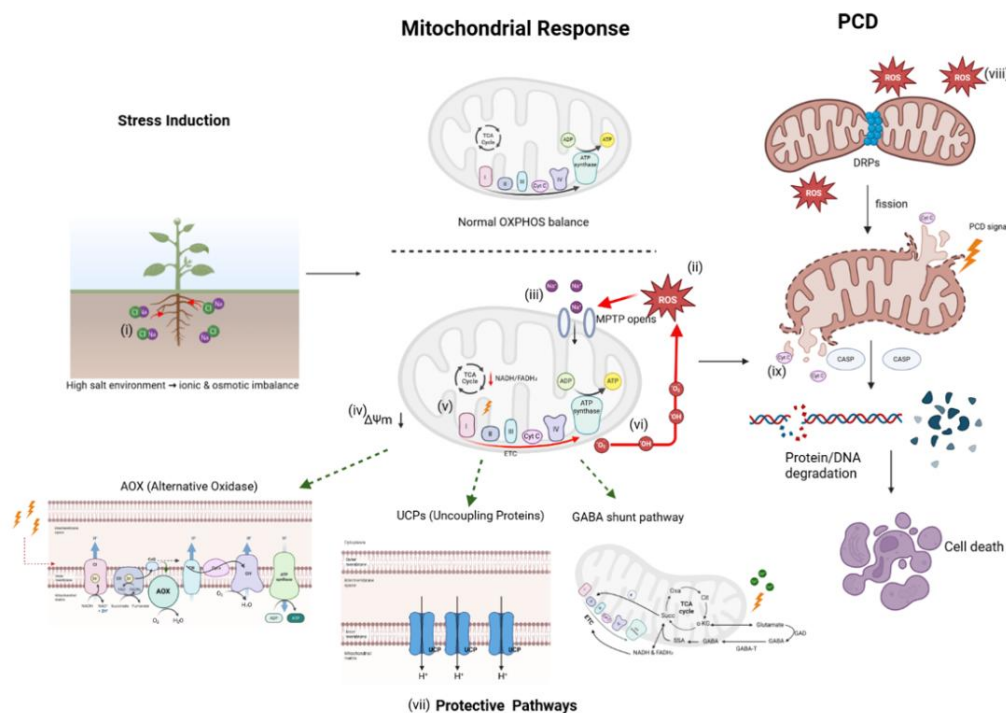


Figure 2. Mitochondrial response during salinity stress exposure.
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Mitochondrial oxidative phosphorylation (OXPHOS) depends on the electron transport chain (Complexes I-IV) and ATP synthase (Complex V) to generate ATP [47]. Complexes I, III and IV pump protons into the intermembrane space, creating an electrochemical gradient that drives ATP synthesis [48]. Under normal conditions, proton pumping and ATP production remain balanced, ensuring stable $\Delta\Psi_m$. However, salinity disrupts this balance, causing excess proton buildup, over-reduction of the ETC, and impaired ATP synthesis [49].

Salt stress affects the tricarboxylic acid (TCA) cycle, reducing the activity of key enzymes such as citrate synthase and isocitrate dehydrogenase [50]. This limits the supply of NADH and FADH_2 to the ETC, weakening OXPHOS and promoting electron leakage, which increases mitochondrial ROS production [6].

To restore homeostasis under salt stress, mitochondria activate Ca^{2+} transporters and bypass systems such as AOX and UCPs, which reduce redox pressure and help stabilize $\Delta\Psi_m$ [49, 51]. AOX maintains NADH oxidation by bypassing Complexes III and IV, while UCPs mildly dissipate the proton gradient to limit ROS without fully stopping ATP production [52; 51]. Antioxidant defenses, including MnSOD and enzymes of the ascorbate-glutathione cycle, further detoxify ROS and support redox balance [51].

Salt stress also induced the GABA shunt, where glutamate is converted to GABA and then to succinate, supplying the TCA cycle and reducing ROS when photosynthesis and growth decline [53, 54].

Under severe salinity, excess ROS act as damage signals, activating DRP3A/DRP2B and promoting mitochondrial fragmentation [55, 56]. ROS also trigger mPTP opening, causing $\Delta\Psi_m$ collapse and releasing cytochrome c, which activates caspase-like proteases and initiates irreversible PCD [57, 58]. When AOX, UCPs, antioxidant systems and the GABA shunt can no longer rebalance energy and redox status, ATP depletion worsens mitochondrial failure [43], driving the cell toward programmed death.

4. Chloroplast During Salinity Stress

Chloroplast is highly affected during exposure to high saline conditions. Many vital processes, which take place in chloroplasts, are declined, including photosynthesis. Those effects can be subdivided as osmotic and ionic stresses [59]. Osmotic stress is triggered by high salt concentration in soil, which disturbs the water absorption by roots, resulting in water deficit, and ionic stress is described as mechanical damage by an extreme amount of salt in transpiring leaf cells [60]. Munns proposed the idea of the biphasic model of plant response to salinity stress in 1993. Where he described the first phase as not salt-specific growth retardation due to water deficiency, while the second salt-specific phase is related to leaf death associated with rapid accumulation of salt in cytoplasm and cell walls [61]. Furthermore, it leads to stomatal closure, reduced chlorophyll content, decreased CO₂ concentration, and a decline in photosynthesis [62].

Exposure of plants to salinity stress leads to a decline in CO₂ assimilation, causing an over-reduction of photosystem II (PSII). This imbalance results in the formation of ROS, particularly singlet oxygen (¹O₂) [63]. In photosynthetically active leaves, chloroplasts serve as primary sites of ROS generation [64]. The major ROS species formed in chloroplasts include singlet oxygen (¹O₂), superoxide radicals (O₂⁻), hydrogen peroxide (H₂O₂) and hydroxyl radicals (•OH) [65] (Tab. 1). Because radical forms of ROS are difficult to detect directly, most studies focus on measuring non-radical H₂O₂ accumulation as an indicator of oxidative stress under saline conditions [63].

Table 1

The effect of different concentrations of salinity on different plant species

	Plant species	Salt concentration (NaCl)	Chloroplast content	Photosynthetic efficiency	PCD marker	References
1.	A Korean wheat cultivar cv. Keumgang (<i>Triticum aestivum</i> L.)	150 mM for 1, 2, or 3 days	The levels of Chl a and b decreased to 79 % of the control at 1 day, 59 % at 2 days, and 53 % at 3 days	Photosynthetic rate decreased to 79 % at 1 day, 59 % at 2 days, and 53 % at 3 days	Proline contents rose to 102 % of the untreated control at 1 day, 104 % at 2 days, and 109 % at 3 days	[66]
2.	Barley (<i>Hordeum vulgare</i> L.)	100 and 200 mM	El Arich, Giza2000, Giza127 lose, respectively 33 %, 36 % and 43 % of their total chlorophyll content	A significant decrease only in Giza127 at 200mM NaCl	An increase in MDA content in almost all barley genotypes	[67]
3.	(<i>Vicia faba</i> L.)	60, 120, 240 mM	Chl a content decreased, reaching its lowest content, 0.270 and 0.426 mg/g fresh weight, at 240 mM	Due to the decline in photosynthetic pigments, decrease in photosynthetic rate as well	Decline in protein content	[68]
4.	Barley (<i>Hordeum vulgare</i> L.)	200 mM	Chl a content from 14 % to 41 %, chl b contents were reduced from 12 %, to 46.8 % and similarly, total chlorophyll contents were decreased from 10.3 % to 50.3 %	The efficiency of electron donation to PSI (Fv/Fo) and maximum quantum yield of PSII (Fv/Fm) was significantly reduced	Enhanced dissipation energy flux per reaction center (DIO/RC) in thylakoid membrane.	[69]
5.	Oat (<i>Avena sativa</i> L.)	48, 72, 96, 120, 144 mmolL ⁻¹	At 96 mmolL ⁻¹ NaCl, the content of chlorophyll a decreased to 0.86 times, while the content of chlorophyll b decreased to 0.7 times compared with the controls	Decline in photosynthetic rate due to the decrease in chloroplast content	The proline content in shoots was increased by 3.26 times under NaCl stress, and the proline content in roots by 3.41 times	[70]

5. Vacuole During Salinity Stress

Under saline conditions, plants rapidly experience osmotic, ionic and oxidative stress, while excess Na^+ and Cl^- disrupt cytosolic ion homeostasis, enzyme activation, biomolecule synthesis and hormonal imbalance [71]. Excess accumulation of these ions provokes the activation of vacuolar transport systems to sequester toxic ions into the vacuoles and maintain a favorable K^+/Na^+ ratio [72]. Vacuolar transport systems include Na^+/H^+ antiporters, V-ATPase, V-PPase, Ca^{2+} -ATPase, aquaporin, ABC transporters and etc. [73]. At the same time, some adjustments of the morphological and functional structure of the vacuole, such as vacuole enlargement (swelling), tonoplast remodeling, changes in cytosolic pH as a response to osmotic imbalance [74], and thereby protecting the cytoplasm from ion toxicity. These dynamic changes in vacuolar volume, tonoplast structure and internal environment, altogether contribute to the maintenance of turgor pressure [3] and support osmotic adjustment [67] during the early stages of salt stress, thus protecting the cytoplasm from rapid ionic stress damage.

However, failure to sequester excess Na into the vacuole causes severe cytosolic stress [75]. High salt levels induce ROS production, leading to oxidative damage of tonoplast lipids and proteins [76]. Under these conditions, cells shift to an energy-saving metabolic state, degrading stored reserves and nonessential components to supply metabolites for energy production and essential biosynthesis [77]. Elevated cytosolic Ca^{2+} further activated stress-signaling pathways that weaken vacuolar integrity. Early stress hormones such as salicylic acid (SA), gibberellin (GA) and ethylene trigger Ca^{2+} signaling and play critical roles in plant PCD [3; 10]. Disruption of the vacuolar Ca pumps ACA4 and ACA11 increases apoptosis-like cell death through an SA-dependent pathway [10], highlighting their role in linking vacuolar Ca^{2+} signaling to PCD onset [78]. Stress-induced autophagy often protects cells by restoring energy balance and suppressing PCD [79–81]. However, unknown signals can redirect autophagy into a pathway that promotes vacuole-dependent PCD [83, 82], characterized by tonoplast permeabilization and rupture, which releases hydrolases and drives cytoplasmic degradation [84, 77]. Vacuolar processing enzymes (VPEs) and other proteases can also initiate hypersensitive-response (HR) PCD under abiotic and biotic stress, and peroxisomal catalase removal via pexophagy may be required for HR activation [83, 86, 85]. Before tonoplast rupture, vacuoles show increased lytic activity as autophagy-derived cargo and stress-induced proteases accumulate [77]. Activation of VPE and related proteases triggers membrane collapse and release of acidic contents—a hallmark of vacuole-mediated PCD [85]. Disrupted coordination between autophagy and osmotic adjustment may contribute to tonoplast rupture [87]. Once the membrane collapses, hydrolases degrade the protoplast and later the cell wall. Notably, under severe energy deficiency, autophagy itself can act as a direct inducer of PCD [88].

6. Nucleus Under Salinity Stress

A number of processes are affected by salt exposure, including cell division. Cell division is a primary process determining the cell shape and its further development. High salinity conditions can alter symmetry and asymmetry ratio, meaning the distribution of cytoplasmic components between daughter cells during division [89]. Moreover, it severely disrupts the work of the cytoskeleton. Salt stress induced significant disturbances in the interactions among cytoskeletal components. It affects both ion transport regulation and the biosynthesis and regulation of various membrane and non-membrane proteins, including transporters and enzymes [90]. These can happen in individual cells. In some cases, cytoskeletal damage becomes irreversible, as components may form aberrant structures, including crystalline-like aggregates, which cannot be properly assembled and persist in daughter cells after division [91]. Additionally, earlier it was reported that the chloroplast proteins EXECUTER1 and EXECUTER2 transfer oxidative signals from the plastid to the nucleus [92]. Overaccumulation of ROS species in the nucleus resulted in cell death.

Another type of PCD regulation involved iron homeostasis. Iron is a crucial element that plays a key role in development, photosynthesis, chlorophyll biosynthesis and DNA synthesis through the formation of Fe-S clusters and heme groups [93]. Both ROS and NO can influence ion homeostasis in plant cells. Iron readily reacts with ROS to generate highly reactive and toxic species; for example, during the Fenton reaction, Fe^{2+} reacts with hydrogen peroxide (H_2O_2) to produce hydroxyl radicals ($\bullet\text{OH}$), which can damage nucleic acids, proteins and lipids. NO is also a key element in maintaining iron balance; it helps sequester iron and reduce oxidative damage [94]. Furthermore, NO functions as a signaling molecule during ferritin synthesis, contributing to iron storage and cellular protection [93].

7. Interconnection of Cellular Organelles under Salinity Stress

Under salinity stress, plant organelles do not act independently; instead, they form an integrated signaling network in which ionic, osmotic and oxidative cues propagate rapidly between the plasma membrane (PM), mitochondria, chloroplasts, vacuole and nucleus. As shown in Figure 3, high Na^+ and Cl^- levels first disturb PM transport systems, driving Ca^{2+} influx and ROS accumulation that serve as early signals transmitted to internal organelles [22, 25, 36]. Mitochondria respond by altering $\Delta\Psi_m$, increasing ROS production and activating protective pathways such as AOX, UCPs and the GABA shunt, but excessive stress leads to mPTP opening and cytochrome c release, linking mitochondrial dysfunction directly to PCD initiation [46, 51, 57]. Chloroplasts similarly generate large amounts of ROS due to PSII over-reduction, and these oxidative signals are relayed to the nucleus via chloroplast-nuclear communication pathways such as EXECUTER proteins, reinforcing stress-induced transcriptional reprogramming and DNA degradation [63, 92]. Vacuoles integrate osmotic and ionic signals by sequestering Na^+ through NHX antiporters and V-ATPase activity, but when this system collapses and Ca^{2+} converge on the nucleus, causing chromatin condensation and fragmentation, ultimately sealing the commitment to PCD [89]. Together, these interconnected organelles create a coordinated stress-response circuit in which membrane transport, ROS signaling, Ca^{2+} fluxes and protease activation collectively determine whether the cell adapts or undergoes PCD.

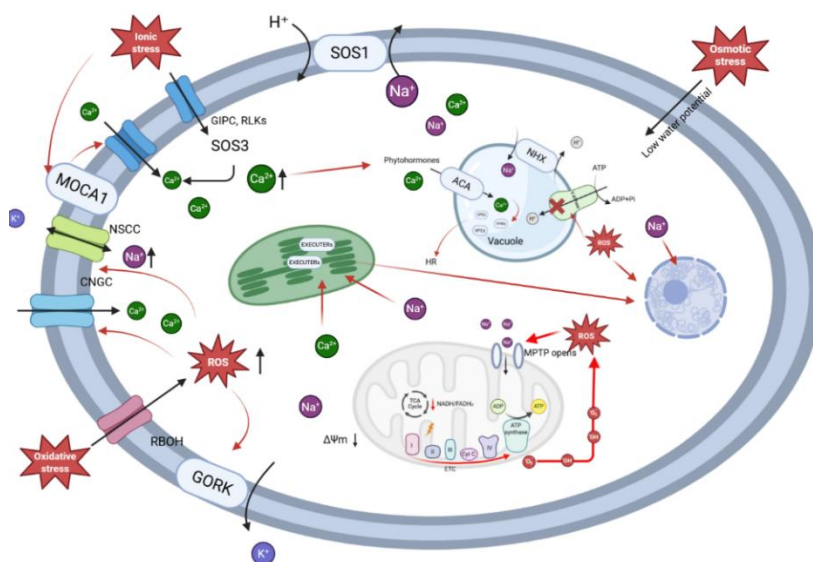


Figure 3. Interconnection of cellular organelles during salinity stress leading to PCD.
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Future perspectives

Although significant progress has been made in understanding how individual organelles respond to salinity, their coordinated behavior during PCD remains insufficiently explored. Future research should focus on identifying the precise molecular signals, such as ROS, NO, Ca fluxes and phytohormone crosstalk that mediate communication between mitochondria, chloroplasts, vacuoles, the plasma membrane and nucleus under salt stress. Advancing imaging and omics technologies could help visualize real-time organelle interactions and uncover early biomarkers predicting the shift from stress tolerance to PCD. Moreover, engineering key regulators such as SOS transporters, vacuolar pumps, antioxidant systems, mitochondrial metabolic pathways (AOX, UCPs, GABA shunt), and chloroplast ROS-detoxifying components may provide new strategies for developing salt-tolerant crops. Integrating these findings will be essential for constructing comprehensive models of organelle-driven PCD and improving plant resilience in increasingly saline environments.

Conclusion

This review discussed the effect of high salinity exposure on the five main organelles in a plant cell, resulting in PCD. Over time, plants have developed a defensive system in order to protect themselves from high salinity. Indeed, although plants do not possess an equivalent to the animal adaptive immune system,

they deploy a number of defense mechanisms, and the severity of plant stress is tightly correlated with plant species, salt concentration and exposure longevity. The ratio between these two (plant defense system and severity of salinity) determines whether the plant will tolerate or activate PCD mechanisms. Hence, it is crucial to investigate the defensive mechanism and their interconnection in order to develop new strategies for producing plant species tolerant to salinity.

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Conflict of Interest

The authors declare no conflict of interest.

Author Contributions

The manuscript was written through contributions of all authors. All authors have given approval to the final version of the manuscript. CRediT: **Kumargazy K.D., Satu A.S.** — investigation, data curation, writing — original draft; **Temirkhanov A.Zh, Nurbekova Zh.A., Khusanov T.** — writing — review & editing; **Nurbekova Zh.A.** — supervision; **Temirkhanov A.Zh.** — project administration, funding acquisition.

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Өсімдіктердегі тұздылықтан туындаған бағдарламаланған жасуша өлімі кезіндегі органелла динамикасы

PCD (бағдарламаланған жасуша өлімі) — қоршаған орта стрестерінен (ePCD) немесе даму процестерінен (dPCD) туындаған күрделі механизм. ePCD әртүрлі қолайсыз абиотикалық жағдайлардан, соның ішінде тұздану стресінен туындауы мүмкін. Тұздың жоғары концентрациясының әсері өсімдіктердің морфологиялық ерекшеліктерін төмендетіп қана қоймай, сонымен қатар биохимиялық және молекулалық процестерге әсер етеді. Өсімдік әртүрлі органеллаларда болатын көптеген қорғаныс механизмдерін белсендіреді. Оған плазмалық мембранадағы, митохондриядағы, хлоропласттардағы, вакуольдегі және ядродағы иондық, осмотық және тотығу стрестері жатады. Плазмалық мембрананың негізгі бұзылуы тасымалдау жүйесінің төмендеуімен, ROS жинақталуымен және Ca^{2+} ағынымен сипатталады. Митохондриялық дисфункция ROS-тың жоғары жинақталуымен, AOX, UCP, GABA және цитохром с-ның бөлінуі сияқты қорғаныс жолдарының белсендірілуімен байланысты. Сонымен қатар фотосинтез жылдамдығы хлорофилл мөлшерінің төмендеуіне байланысты айтарлықтай төмендейді. Вакуольдер NHX антипорттерлері және V-ATPase белсенділігі арқылы Na^+ секвестрлейді және осмотық, иондық сигналдарды біріктіреді, бірақ бұл жүйе ыдырағанда Ca^{2+} ядроға жинақталады. ROS-тың артық мөлшерде генерациясы хлоропласт-ядролық байланыстағы EXECUTER ақуызы үшін сигналдық молекула ретінде әрекет етеді. Тұздану хроматиннің ыдырауына әкелетін осмотық, иондық және тотығу стресс сигналдарды белсендіреді. Қорытындылай келе, бұл өзара байланысты процестер PCD-ге әкеледі.

Кілт сөздер: бағдарламаланған жасуша өлімі, тұз стресі, реактивті оттегі түрлері, азот оксиді, фитогормондар, ферменттер.

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Динамика органелл при запрограммированной клеточной смерти растений, вызванной засолением

PCD (запрограммированная гибель клеток) — это сложный механизм, вызываемый экологическим стрессом (ePCD) или процессами развития (dPCD). ePCD может быть вызван различными типами неблагоприятных абиотических условий, включая солевой стресс. Воздействие высоких концентраций соли не только ухудшает морфологические особенности растений, но и серьезно влияет на биохимические и молекулярные процессы. Кроме того, растения активизируют многочисленные защитные механизмы, которые реализуются в различных органеллах растительной клетки. Они включают в себя ионное, осмотическое и окислительное повреждение плазматической мембраны, митохондрий, хлоропластов, вакуолей и ядер. Основные нарушения в плазматической мембране характеризуются снижением транспортной системы, накоплением АФК и притоком Ca^{2+} . Митохондриальная дисфункция

связана с высоким накоплением АФК, активацией защитных путей, таких как АОХ, УСР, ГАВА, и высвобождением цитохрома с. Вакуоли интегрируют осмотические и ионные сигналы, изолируя Na^+ посредством антипортеров NHX и активности V-АТФазы, но когда эта система разрушается, Ca^{2+} концентрируется в ядре. Кроме того, скорость фотосинтеза значительно снижается из-за снижения содержания хлорофилла. Более того, избыточное производство АФК действует как сигнальная молекула для белков EXECUTER, обеспечивающих взаимодействие хлоропластов с ядрами. Засоление активирует осмотические, ионные и окислительные сигналы, которые приводят к деградации хроматина. В совокупности эти взаимосвязанные процессы приводят к образованию PCD.

Ключевые слова: запрограммированная гибель клеток, солевой стресс, активные формы кислорода, оксид азота, фитогормоны, ферменты.

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