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REVIEW ARTICLE

Comprehending the effect of Salinity Stress and Tolerance Mechanisms in Cyanobacteria: a review

Neha Gupta[1], Ankit Srivastava[1], Alka Bhardwaj, Satya Shila Singh, Arun Kumar Mishra*

Laboratory of Microbial Genetics, Department of Botany, Institute of Science, Banaras Hindu University, Varanasi-221005, U.P., India.

*Corresponding author email: akmishraau@hotmail.com; akmishraau@rediffmail.com (Arun K. Mishra)
[1]Authors have equally contributed and 2nd first authors
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Abstract

Abiotic stressors, including high and variable salt concentrations, significantly impede the growth and survival of cyanobacteria. Cyanobacteria have evolved to adapt diverse aquatic environments with varying salt concentrations. As the salt concentration increases, the cyanobacteria face challenges related to water availability and the maintenance of internal ion concentrations. To cope with these changes, cyanobacteria utilize the salt-out strategy to maintain internal ion homeostasis and accumulate organic compounds to effectively counter salt stress. However, the mechanisms underlying salt stress signalling and regulation in cyanobacteria remain unclear. This may be attributed to the complexity of salt stress, which is characterized by ionic, osmotic, and oxidative stresses, making it difficult to distinguish from other stress conditions. A comprehensive understanding of cyanobacterial salt acclimation will enhance our knowledge of salt sensing mechanisms and enable the better utilization of salt-tolerant cyanobacteria for bioenergy production. Additionally, this understanding will facilitate the application of stress-responsive genes to improve salt resistance in plants.

Keywords: Cyanobacteria; Salt-out; Compatible Solutes; Osmotic Stress; Ionic homeostasis

1. Introduction

The evolution of cyanobacteria approximately 3.5 billion years ago in the geological timescale marked a significant transition in the history of life as it initiated oxygenic photosynthesis. Later, through endosymbiosis, this ability was transferred to eukaryotes, leading to the emergence of eukaryotic algae (Shih, 2015). Genome analyses suggest a close relationship between present-day filamentous algae and cyanobacteria (Deusch et al., 2008). Cyanobacteria's remarkable ability to adapt to diverse environments has enabled them to occupy almost every ecological niche, ranging from fresh to marine environments, and cold to hot springs. Their cosmopolitan distribution, prokaryotic nature, and resemblance to higher plants make them an excellent candidate for understanding stress management.

Salinity is a critical abiotic stressor for cyanobacteria inhabiting aquatic and terrestrial ecosystems. Salinity generally refers to the total concentration and composition of dissolved inorganic ions, and thus can vary significantly. Increased salinity reduces water availability and simultaneously increases osmotic stress because the amount of salt is inversely related to the amount of water in a solution. To counter this, cyanobacteria maintain a constant cellular ionic and osmotic composition to sustain water uptake via osmosis and create the turgor pressure necessary for cell enlargement and growth, which is hyperosmotic relative to the surrounding medium. Hence, a change in external salt concentration and/or water availability can affect cellular metabolism and may pose challenges to cell survival, necessitating a rapid response to changing salt concentrations for cell survival (Kharwar et al., 2019).

The presence of high salt concentrations leads to two major problems: reduced water availability and increased ion concentration. The accumulation of inorganic ions in the cytoplasm at high concentrations may have toxic effects on cellular metabolism. In order to ensure proper water uptake via osmosis, the cytoplasmic concentration of osmotically active compounds must be higher than that of the surrounding medium (Ladas et al., 2000). To cope with these challenges, microorganisms develop two effective strategies for osmotic acclimation of the cytoplasm in response to changing salt concentrations: the "*salt-in-strategy*" and the "*salt-out-strategy*" (Galinski and Trüper, 1994; Hagemann, 2011). The "*salt-in-strategy*" is used by organisms that accumulate large numbers of inorganic ions in the cytoplasm to ensure water uptake and turgor pressure. The "*salt-out-strategy*" is used by organisms that keep their internal ion concentration low via accumulation of low organic compatible solutes and active export of inorganic ions. Most prokaryotes, such as cyanobacteria, and all eukaryotic microorganisms use the "*salt-out-strategy*" to acclimate to high or changing salt concentrations. This strategy involves the accumulation of compatible solutes that does not interfere with metabolism at high concentrations (Brown, 1976) and the active export of inorganic ions that steadily diffuse along their electrochemical gradients from the cytoplasm. This review primarily focuses on salt toxicity in cyanobacteria from different aspects and the tolerance mechanisms offered by cyanobacteria in physiological, biochemical, and molecular dimensions.

2. Impact of salinity stress on Cyanobacterial physiology and metabolism

2.1. Cyanobacterial strategies to combat osmotic and oxidative stress

In the presence of high salt concentrations, cyanobacteria are subjected to various physiological challenges, including impaired growth, photosynthesis, cellular homeostasis, and osmotic balance (Joset et al., 1996; Singh, 2002; Oren, 2007). The salinity-induced disturbances in cellular physiology may lead to the production of reactive oxygen species (ROS), changes in gene expression, and protein synthesis, which in turn may trigger programmed cell death (PCD) (Ding et al., 2012). It is noteworthy that osmotic stress is the primary effect of salinity, while oxidative stress tends to be a secondary outcome (Zhu, 2003). At the molecular level, the transporters that mediate the uptake and efflux of these solutes have been extensively studied in cyanobacteria. For example, the ABC transporter OpuA in *Synechococcus* sp. PCC 7942 is responsible for the uptake of the compatible solute glycine betaine, while the efflux of trehalose is mediated by the transporter TreT in *Synechocystis* sp. PCC 6803 (Los and Murata, 2004).

Oxidative stress, on the other hand, is caused by the accumulation of reactive oxygen species (ROS) in the cell, such as superoxide radicals, hydrogen peroxide, and hydroxyl radicals. These ROS can damage cellular components like proteins, lipids, and DNA, leading to oxidative damage and cell death (Kharwar et al., 2019). To protect against oxidative stress, cyanobacteria have developed a complex antioxidant defense system that involves enzymes like superoxide dismutase, catalase, and peroxidases (Lupínková and Komenda, 2004).

The expression and activity of these enzymes are regulated by various transcription factors and signalling pathways, such as the two-component system Hik34-Rre1 in *Synechocystis* sp. PCC 6803 and the redox-responsive regulator OxyR in *Synechococcus elongatus* PCC 7942 (Vidal et al., 2009; Latifi et al., 2009). In addition, some cyanobacteria also produce extracellular polysaccharides that can scavenge ROS and protect cells from oxidative damage (Wada et al., 2013). Comprehending these mechanisms can facilitate the development of novel approaches to enhance cyanobacterial proliferation and viability under high salinity circumstances, providing potential benefits for biotechnology, environmental restoration, and sustainable food and energy systems.

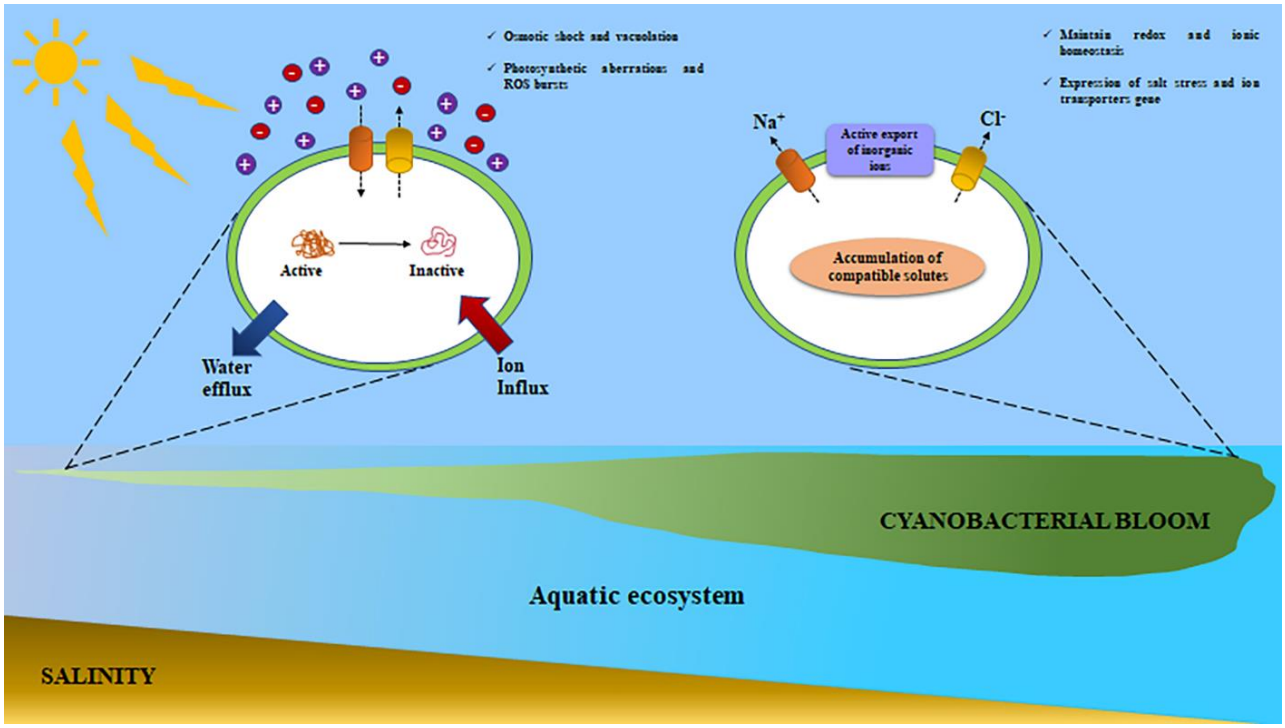

Fig.1. Aquatic ecosystem, salinity stress, Cynobacterial physiology and metabolism

2.2. Unravelling the mechanisms of salt stress-induced alterations in membrane lipids

Salt stress leads to desaturation of membrane lipids, which in turn alters the fluidity of the membrane (Davies et al., 1987). Salinity causes lipid peroxidation (Srivastava et al., 2005), which can be divided into three distinct steps: initiation, propagation, and termination. The initiation step is the rate-limiting step, and it involves the activation of molecular oxygen to produce reactive oxygen species (ROS), which can be accelerated under different stress conditions (Verma et al., 2018). Once initiated, the other two steps, propagation and termination, occur readily, ultimately leading to damage of the membrane (Frankel, 1985). Lipids have also been found to play a protective role against salt stress (Singh et al., 2002), as the fatty acids of the membrane become desaturated in response to salt stress. An increase in the unsaturation of fatty acids in membrane lipids has been reported to enhance the tolerance of the photosynthetic machinery of *Synechococcus* sp. to salt stress (Allakhverdiev et al., 2001). High salt concentration may also curtail the activities of potassium, sodium ion channels, and water channels. Altered membrane fluidity can also affect the activities of several membrane-bound enzymes. The impact of salt stress on membrane lipids in cyanobacteria is driven by intricate molecular mechanisms. One of the key changes in membrane lipids in response to salt stress is an increase in the proportion of glycolipids, such as monogalactosyldiacylglycerol (MGDG), which have been shown to increase in abundance in several cyanobacterial strains under high salt concentrations. MGDG can play a role in membrane stabilization by decreasing membrane fluidity, thus reducing the potential for ion leakage (Liu et al., 2017).

In addition to changes in lipid composition, cyanobacteria can also adjust their lipid biosynthesis pathways in response to salt stress. For example, the enzyme acyl-lipid desaturase (desA) catalyzes the conversion of saturated fatty acids to unsaturated fatty acids, which can enhance membrane fluidity and protect against oxidative stress (Singh et al., 2002; Tiwari et al., 2017). Cyanobacteria can also regulate the degradation and remodelling of membrane lipids under salt stress conditions. The enzyme phospholipase A2 (PLA2) has been shown to be involved in lipid remodelling in response to salt stress in cyanobacteria, facilitating the release of fatty acids from membrane phospholipids and allowing for the incorporation of more stable fatty acids (You et al., 2019). Furthermore, salt stress can also activate various signalling pathways that regulate the expression of genes involved in lipid metabolism. For example, the transcription factor SigE has been shown to regulate the expression of genes involved in lipid biosynthesis and degradation under salt stress in the cyanobacterium *Synechocystis* sp. PCC 6803 (Tyystjärvi et al., 2013). In conclusion, the effect of salt stress on membrane lipids in cyanobacteria involves various molecular mechanisms that are regulated at multiple levels. Understanding these mechanisms can provide insights into the adaptation of cyanobacteria to salt stress and help to develop strategies for improving their growth and survival in high salinity conditions.

2.3. *Disrupting the balance: unravelling the impact of salt stress on Cytosolic homeostasis*

The concentration and duration of salt stress have been found to affect the cytoplasm in *Synechococcus* PCC 6311, as reported by Lefort-Tran et al. (1988). This leads to significant changes in the internal cell organization, including a decrease in cytoplasm density, complete loss of cellular granules (glycogen and carboxysomes), and modification in the appearance of DNA. Salinity also hampers cell division, resulting in an increase in cell size, as reported by Ferjani et al. (2003). At the molecular level, salt stress significantly impacts the cytosol of cyanobacteria by causing changes in various biological mechanisms such as ion homeostasis, protein synthesis, and signalling pathways. These molecular processes are crucial for maintaining cytosolic integrity and functionality under salt stress conditions. One of the primary effects of salt stress is the disruption of ion balance, leading to an increase in intracellular sodium (Na^+) and chloride (Cl^-) ions. To counteract this, cyanobacteria employ several mechanisms to regulate ion homeostasis in the cytosol, such as the upregulation of transporters, such as Na^+/H^+ antiporters, that pump out excess sodium ions from the cytosol to the extracellular environment (Waditee et al., 2006). In addition, cyanobacteria can also synthesize and accumulate compatible solutes, such as glycine betaine and trehalose, which can act as osmoprotectants and maintain cytosolic water potential (Klähn and Hagemann, 2011). Furthermore, salt stress can also impact protein synthesis and turnover in the cytosol. The accumulation of misfolded or denatured proteins under salt stress can trigger the activation of molecular chaperones and proteases, such as Hsp70 and ClpP, that help refold or degrade these proteins to maintain cytosolic protein homeostasis (Parsell and Lindquist, 1993). Moreover, the effect of salt stress on the cytosol can also trigger various signalling pathways that regulate the expression of genes involved in stress response and adaptation. For example, the two-component system (TCS) Hik33-Rre1 has been shown to be involved in regulating gene expression in response to salt stress in the cyanobacterium *Synechocystis* sp. PCC 6803 (Li et al., 2012). Understanding these mechanisms can provide insights into the adaptation of cyanobacteria to salt stress and help to develop strategies for improving their growth and survival in high salinity conditions.

2.4. *Halting photosynthesis: understanding the impact of salt stress on photosynthetic machinery*

Photosynthesis in cyanobacteria mainly involves four multiprotein complexes embedded in thylakoid membranes present in the cytoplasm, namely, photosystem I (PS I), photosystem II (PS II), cytochrome b/f complex, and ATP synthase, which participate in photosynthetic electron transport. Plastoquinone (PQ) and plastocyanin (PC) act as mobile electron carriers, and PS II is responsible for the production of NADPH, while PS I mediate cyclic electron transfer and ATP generation (Thomas et al., 2001). Chlorophyll content varies depending on the nature, habitat, and morphology of cyanobacteria, and salinity-induced decrease or enhancement of chlorophyll content has been reported (Lu and Vonshak, 1999). Phycobiliproteins, which are responsible for light harvesting in cyanobacteria, are sensitive to salt stress, and carotenoid content increases under salt stress, providing protection to chlorophyll and exhibiting an antioxidative role (Chakraborty et al., 2021). The PS II reaction center, particularly its 34 kDa D1 protein, is the prime target of salt stress (Sudhir et al., 2005). Salt stress inhibits the fixation of CO_2, diminishes the regeneration of acceptors for the linear electron transport, and induces the generation of ROS, which subsequently may inhibit protein synthesis. Under salt stress, an alternate electron transfer route from PQ to cytochrome C553 via PC has been reported in cyanobacteria, which decreases PS II activity, while enhancing PS I activity and leads to an increase in the PS I/PS II ratio (Bhargava et al., 2008). Carbon assimilation is suppressed under salt stress (Moisander et al., 2002), and cyanobacteria have evolved a mechanism of carbon concentration to improve carboxylation by their relatively insufficient RuBisCO (Badger and Price, 2003). However, under carbon-limited or salt stress conditions, the oxygenase activity of RuBisCO prevails over the normal carboxylase, leading to the operation of the photorespiratory glycolate pathway (C2 cycle) (Sivakumar et al., 2000).

At the molecular level, the effect of salt stress on photosynthesis involves alterations in the expression of genes involved in photosynthesis. For instance, in the cyanobacterium *Synechocystis* sp. PCC 6803, the expression of psbA, the gene encoding the D1 protein of photosystem II (PSII), is downregulated under salt stress conditions (Jantaro et al., 2005). PSII is a critical component of the photosynthetic machinery that captures light energy and utilizes it to drive electron transport. The downregulation of psbA leads to a decrease in the number of functional PSII complexes, which impairs the electron transport chain and photosynthetic activity (Rochaix, 2013). In addition to these effects, salt stress can also cause oxidative stress, which can damage the photosynthetic machinery by generating reactive oxygen species (ROS). ROS can damage the pigment-protein complexes and disrupt electron transport, leading to decreased photosynthetic activity (Wilson et al., 2006). Cyanobacteria have evolved various molecular mechanisms to counteract the effects of oxidative stress, including the production of antioxidant enzymes such as superoxide dismutase (SOD) and catalase. To cope with the effects of salt stress on photosynthesis, cyanobacteria have also evolved various molecular mechanisms to regulate the expression of genes involved in photosynthesis, repair damaged photosynthetic membranes, and scavenge ROS (Latifi et al., 2009). For instance, under salt stress conditions, the expression of genes encoding carotenoids, which are important photoprotective pigments, is upregulated to prevent photo-oxidative damage. Additionally, the expression of genes encoding proteins involved in the repair of photosynthetic membranes, such as D1 protein turnover factors, is also upregulated under salt stress (Niyogi, 1999). Overall, salt stress exerts complex molecular mechanisms on photosynthesis in cyanobacteria, altering the structure and function of the photosynthetic machinery. In-depth understanding of these mechanisms is crucial for devising strategies to improve photosynthetic efficiency of cyanobacteria.

2.5. *Unravelling the effects of salt stress on respiration and metabolism*

Cyanobacteria employ cytoplasmic and thylakoid membranes for respiration and respiratory electron transfer. The distribution of respiratory electron transport capacity between these membranes varies depending on the growth conditions. Salt stress can induce respiration activity in one or both sites. Under salt stress, *Synechocystis* PCC 6803 shows an increase in respiration, without affecting electron transport through complex I and II, but it does increase complex IV activity (Van Thor et al., 2000). Sodium is an essential nutrient for cyanobacterial metabolism, playing a crucial role in nitrogen metabolism (Jeanjean et al., 1993). Na^+ concentration affects the stability of the cyanobacterial nitrogenase activity, which depends on it.

This may be due to the influence of Na^+ on the transport of certain cations, such as Ca^{+2}, which are essential for heterocyst differentiation, as well as anions, such as phosphate, amino acid, and sugars (Thiel et al., 1995). Studies showed that *Anabaena doliolum* experiences an enhancement in nitrogenase activity under salt stress, primarily to meet the requirement of the alternate electron source in the form of H_2. This H_2 is produced due to the activity of the nitrogenase-hydrogenase complex for PS I activity. On the other hand, high salinity inhibits the nitrogenase activity of *Anabaena azollae* (Rai et al., 2001) and *Nostoc muscorum* (Bhargava et al., 2006). The adverse effects of salt stress on nitrogen fixation are attributable to the ionic component rather than the osmotic component (Fernandes et al., 1993). Nitrogenase activity is more sensitive to salt stress than photosynthesis, mainly because fixed carbon is primarily used in the synthesis of osmoprotectants, which leads to less availability of carbon as an energy source for nitrogenase (Tel-Or et al., 1980; Reed et al., 1983).

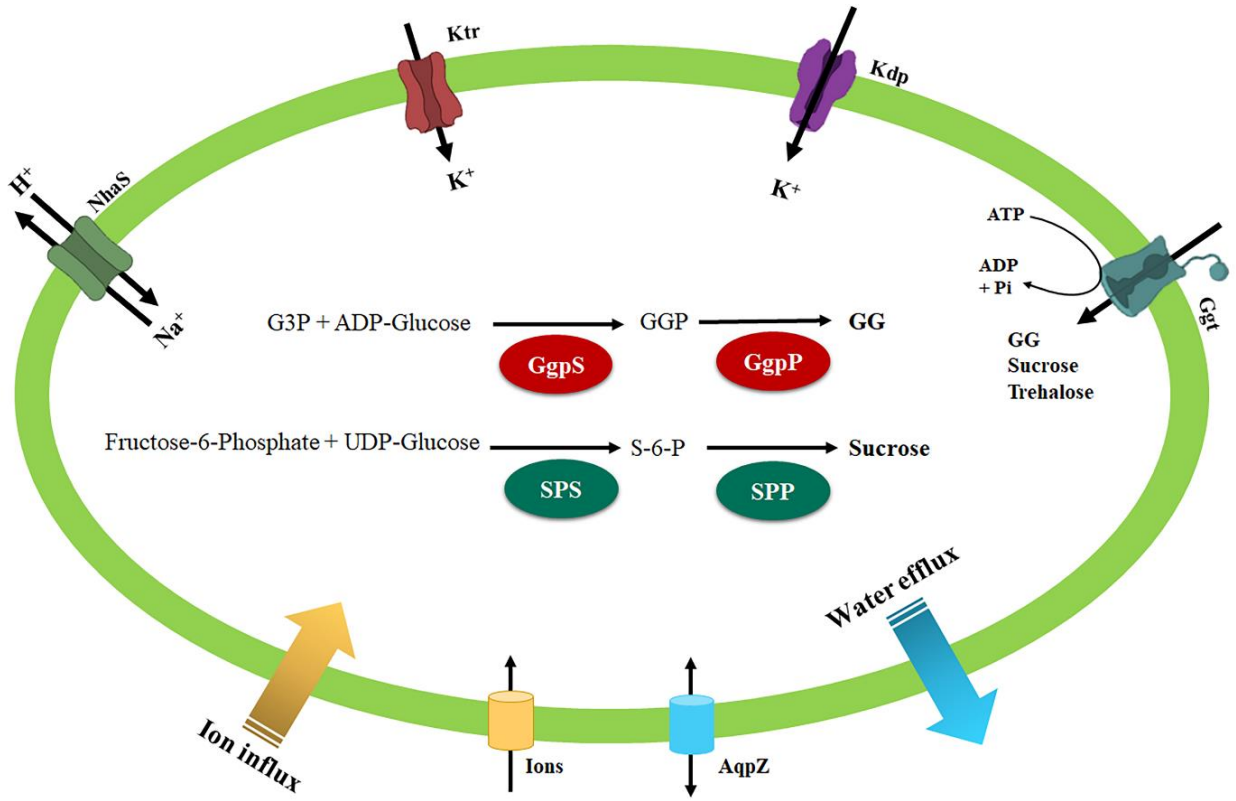

Fig. 2. A schematic representation elucidating the underlying mechanisms and proteins implicated in the acclimation of *Synechocystis* sp. PCC 6803, a moderately halotolerant cyanobacterium, to saline environments. The proteins that mediate water fluxes following exposure to both hyper- and hypo-osmotic stress are also depicted (NhaS: Sodium/Hydrogen Antiporter; Ktr: Potassium ion transporters; Kdp: Potassium ion P-type ATPase transporter; AqpZ: Aquaporin Z; Ggt: Glucosylglycerol (compatible solute) transporter; GgpS: Glucosylglycerol phosphate synthase; GgpP: Glucosylglycerol phosphate phosphatase; SPS: Sucrose phosphate synthase; SPP: Sucrose phosphate phosphatase).

3. Current understanding of salt stress signalling in Cyanobacteria

The primary signal sensed during salt stress and its signal transduction to changed gene expression is not well understood. In *E. coli*, two component systems have been identified that are responsible for salt stress-induced regulation of porin genes and high-affinity K^+ uptake system, Kdp (Mizuno and Mizushima, 1990; Jung and Altendorf, 2002). In *Synechocystis* sp. PCC 6803, five Hik/Rre pairs have been found to be involved in the upregulation of 38 genes 30 min after the addition of 0.5 M NaCl (Shoumskaya et al., 2005). However, many genes, including those essential for salt-acclimation processes, remained salt-induced in all two-component mutants tested. There is an overlap of salt signalling with the sensing of osmotic stress, as well as with many other stress treatments (Los et al., 2010). Sigma factors provide promoter specificity for RNA polymerases, and many sigma factors found in cyanobacteria execute specific tasks (Osanai et al., 2008). In recent studies, alternative sigma factors were found to function in the acclimation to multiple stresses. The group two sigma factors showed overlapping promoter recognition with that of the principal sigma factor and each other. The group three sigma factor SigF has been found to be more specifically involved in the reprogramming of gene expression after salt stress (Huckauf et al., 2000). However, it is still ambiguous whether SigF represents the missing regulator for induction of salt-stress-specific genes.

4. Inorganic ion homeostasis

4.1. Sodium ion homeostasis

Sodium (Na^+) is the predominant inorganic cation in saline environments, including oceans. Under hypersaline conditions, Na^+ enters cells through the electrochemical gradient. However, high cytoplasmic concentrations of Na^+ can be toxic to cells, and therefore, cyanobacteria grown under salt-stressed conditions exhibit lower internal Na^+ concentrations compared to the surrounding medium.

4.2. Potassium ion homeostasis

Potassium (K +) is an essential cation in the regulation of various physiological processes in living cells, including salt and turgor acclimation, pH regulation, enzyme activities, and gene expression (Ballal et al., 2007). In cyanobacteria, the K + uptake is facilitated by two main transporters, Kdp and Ktr (Fig. 1). Kdp is involved in the uptake of K + at low external concentrations, while Ktr is the primary importer of K + in cyanobacteria, and it is activated by the binding of Na + ions. The Ktr system is also dependent on Na + ions, and it remains unaffected during salt acclimation, except for ktrB, which is upregulated during salt shock. The Kdp system plays a crucial role in the uptake of K + ions under low external concentrations, whereas the Ktr system is the primary system that facilitates the transport of K + ions in the cytoplasm of cyanobacteria. The activation of the Ktr system is induced by the binding of Na + ions to the KtrB subunit, which triggers a conformational change, leading to an increase in K + uptake. The Ktr system is not affected by salt acclimation, as it continues to function normally during the adaptation to varying salinity levels (Checchetto et al., 2016). In contrast, the Kdp system is not essential for salt acclimation and is typically downregulated during high salt conditions. The regulation of K + uptake in cyanobacteria is a complex process that involves the interplay of several transport systems and regulatory pathways. The Ktr system, with its dependence on Na + ions, provides an important link between salt acclimation and K + uptake in cyanobacteria. The upregulation of ktrB during salt shock suggests that it plays a crucial role in the acclimation of cyanobacteria to high salt concentrations (Cui et al., 2020).

4.3. Chloride ion homeostasis

Cyanobacteria have evolved various mechanisms to cope with changes in salt concentrations, which are essential for their survival and growth. Chloride transport plays a critical role in this process, and understanding the molecular mechanisms underlying chloride transport in cyanobacteria is of great interest to researchers. Previous studies have suggested that cyanobacteria take up chloride under low-salt conditions by either exchanging it with sodium ions or actively transporting it using ATP (Bahmani et al., 2015; Hageman, 2011). However, the molecular mechanism involved in chloride export remains unclear. In recent years, two putative chloride transporters have been identified in *Synechocystis* sp. PCC 6803, a model cyanobacterium widely used for studying salt acclimation. The first is a chloride channel encoded by the *sll1864* gene, and the second is a protein named Slr0753. Studies have revealed that the expression of both the transporters are upregulated under high salt conditions, indicating their potential role in salt acclimation. Furthermore, a H^+-Cl^- transporter similar to that found in *E. coli* has also been identified in *Synechocystis* sp. PCC 6803, encoded by the *sll0855* gene. This transporter has been reported to be playing a role in pH regulation and chloride ion transport in cyanobacteria. Despite these recent findings, the characterization of chloride transport in cyanobacteria remains incomplete, and further research is needed to fully understand the molecular mechanisms involved (Nanatani et al., 2015; Garci´a-Domi´nguez et al., 2002). Future studies using genetic and biochemical approaches may help to elucidate the specific roles of these transporters in salt acclimation and other physiological processes. Such studies could provide insights into the development of novel strategies to improve salt tolerance in cyanobacteria and other organisms, with potential applications in biotechnology, agriculture, and environmental conservation.

5. Compatible solutes and their role in osmoregulation and growth of cyanobacteria

Compatible solutes are important for cyanobacteria to survive in various habitats with different salinities. They are small organic compounds that can accumulate in high amounts without affecting cellular metabolism (Rhodes and Hanson, 1993; Takabe et al., 1998; Kempf and Bremer, 1998). These solutes, including sugars, polyols, amino acids, and amino acid derivatives, help to balance the osmotic potential inside and outside the cell, maintaining turgor and promoting cellular growth (Brown et al., 1976). Compatible solutes work by changing the water structure around macromolecules, preserving their hydration shell, and preventing them from denaturing. Different cyanobacteria accumulate different compatible solutes depending on their natural habitat and salt tolerance level (Fig. 3). For example, sucrose is a primary compatible solute for freshwater strains (Blumwald et al., 1983; Erdmann, 1983), while glucosylglycerol is predominantly found in moderately salt-tolerant species (Kollmann et al., 1979; Hagemann, 2011). Extreme halophilic strains accumulate glycine betaine and glutamate betaine (Klähn et al., 2010), and some strains use other solutes such as trehalose or homoserine betaine (Pade et al., 2016). The primary compatible solute for ancient cyanobacteria was sucrose (Blank, 2013), and some groups lost the genes for sucrose metabolism over time. Sucrose biosynthesis in cyanobacteria involves two enzymatic steps, mediated by sucrose phosphate synthase (Sps) and sucrose phosphate phosphatase (Spp) (Fig. 1) (Hagmann and Marin 1999; Lunn et al., 2002). Sps catalyzes the conversion of UDP-glucose and fructose-6-phosphate into sucrose-6-phosphate, which is subsequently dephosphorylated by Spp, resulting in the release of sucrose. Cyanobacteria possess two structurally different forms of Sps, with one type having an inactive phosphohydrolase domain and relying on a separate Spp protein for the second reaction (Salerno and Curatti, 2003). Sucrose synthase (SuSy) can also synthesize sucrose through the reversible linkage of NDP-glucose with NDP-fructose. In cyanobacteria (Porchia et al., 1999), SuSy is primarily found in heterocystous nitrogen-fixing strains, where it catalyzes the cleavage of sucrose to produce carbon skeletons (Curatti et al., 2002; Kolman et al., 2015). The regulatory mechanisms involved in sucrose synthesis in cyanobacteria are not well understood. Salt stress has been found to lead to rapid accumulation of sucrose in *Synechocystis* sp. The enzyme Sps is the rate-limiting enzyme in sucrose synthesis, and modifications in Sps gene expression affect sucrose accumulation (Du et al., 2013). However, different cyanobacterial strains may have variable Sps activity (Hagmann and Marin, 1999). The expression of spsA gene is induced shortly after salt shock (Marin et al., 2004; Desplats et al., 2005), but the factors involved in its regulation are unknown. Rre 39 negatively regulates spsA gene expression, while *Nostoc* sp. PCC 7120 regulates sucrose synthesis through Rre OrrA (Ehirra et al., 2014). The *Synechocystis* sp. Rre is an orphan regulator, and the signalling cascade leading to transcription regulation remains unclear.

5.1. Regulation of glucosylglycerol synthesis in Cyanobacteria: biochemical and transcriptional mechanisms

Glucosylglycerol (GG) is a compatible solute found in over 60 different cyanobacteria, primarily marine strains (Hagemann, 2011). The discovery of GG in cyanobacteria was first reported by Kollmann et al. 1979 in *Agmenellum quadriplicatum* (now known as *Synechococcus PCC 7002*). However, some strains, such as picocyanobacteria *Prochlorococcus* spp., do not possess the gene for GG metabolism (Scanlan et al., 2009). In freshwater cyanobacteria, such as *Synechocystis* sp., GG synthesis has been reported (Reed and Stewart, 1983).

The synthesis of GG in *Synechocystis* sp. involves two important enzymes, GG phosphate synthase (GgpS) (Marin et al., 2002) and GG-phosphate phosphatase (GgpP) (Fig. 1) (Hagemann et al., 1997). GgpS is a glucosyltransferase that catalyzes the synthesis of GG 3-phosphate from ADP-glucose and glycerol-3-phosphate, which is subsequently dephosphorylated by GgpP (Hagemann and Erdmann, 1994). These enzymes are largely inactive and are activated upon salt shock. *Synechocystis* sp. activates the pathway of GG accumulation both biochemically and transcriptionally in response to salt stress. The cells already have a basal level of inactive GgpS protein (Hagemann and Erdmann, 1994), which is rapidly activated by a sudden increase in salinity. At low salinities, GgpS remains bound to nucleic acid via electrostatic binding to the negatively charged phosphate groups, rendering the enzyme inactive (Novak et al., 2011). However, at high salinity, the increase in inorganic ions within the cell disrupts this interaction, leading to GgpS release and activation. Biochemical activation of GgpS is initially due to the influx of ions following salt shock (Hagemann et al., 1994).

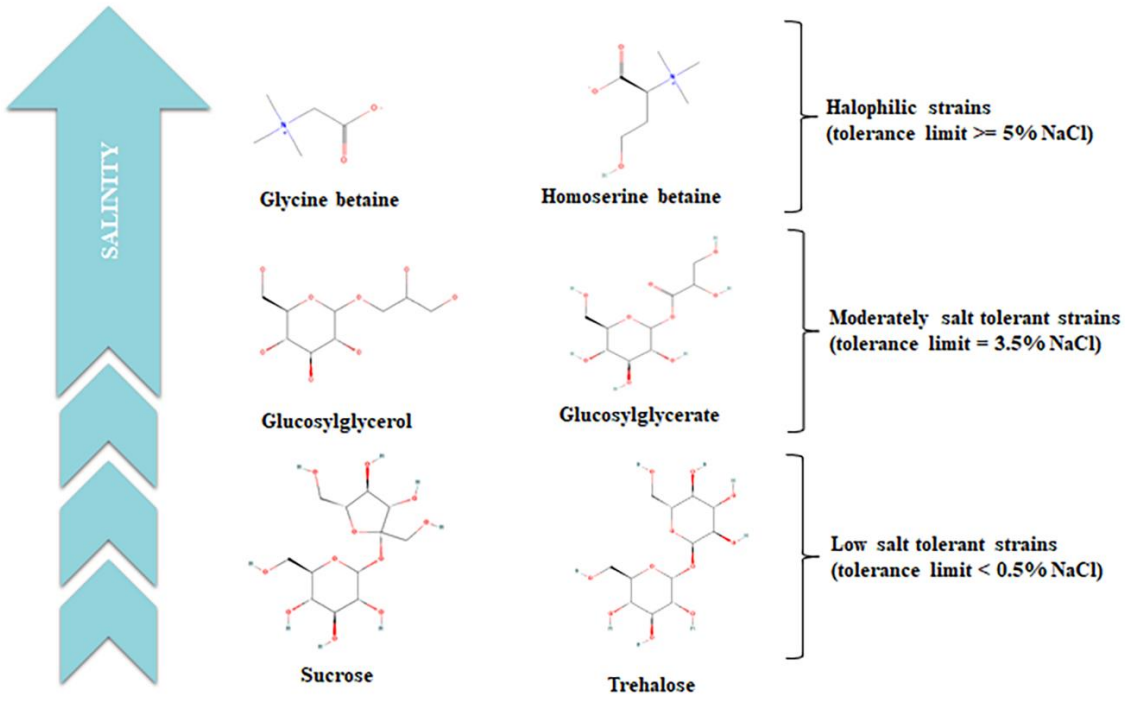

Fig. 3. Diversity in the compatible solute profiles of cyanobacteria under varying saline conditions.

In *Synechococcus* sp. PCC 7002, transcriptional induction of the ggps gene plays a more significant role in GG accumulation than biochemical activation (Engelbrecht et al., 1999). The ggps mRNA shows salt-dependent accumulation that roughly follows the kinetics of intracellular salt ion concentration in response to salt stress. The transcription of ggps mRNA involves the use of alternative sigma factors, with SigF playing a vital role in salt acclimation (Huckauf et al., 2000; Marin et al., 2002).

A transcription factor, LexA, negatively regulates GG metabolism in *Synechocystis* sp., with a lexA mutant strain showing increased ggps expression under low salt conditions (Kizawa et al., 2016). The ggps gene in *Synechocystis* sp. is also regulated post-transcriptionally via a small regulatory mRNA called iron stress activated RNA (IsaR1). This regulatory RNA adjusts the photosynthetic apparatus of various cyanobacteria in response to iron deficiency (Georg et al., 2017). IsaR1 targets ggps mRNA and diminishes GgpS protein synthesis, as well as de novo synthesis of GG, under iron deficiency when IsaR1 levels are high (Rübsam et al., 2018). The biological significance of this post-transcriptional regulation is of importance when fluctuations in salinity and iron availability occur, such as in estuary brackish water where an increase in salinity is accompanied by iron removal from river water (Boyle et al., 1977). Cyanobacteria have evolved various regulatory mechanisms to cope with the fluctuating environments they encounter in their natural habitats. The ability of cyanobacteria to accumulate different compatible solutes, such as GG, helps them to maintain cellular turgor and promote growth under different environmental conditions.

6. Conclusion and future perspectives

Cyanobacteria have developed efficient strategies for adapting to various salinity levels throughout their evolutionary history. These mechanisms involve the expulsion of toxic ions, such as Na^+ and Cl, as well as the synthesis of compatible solutes, which are fuelled by photosynthesis. Salt acclimation results in significant modifications in photosynthetic electron transport, leading to interference with the respiratory process. *Synechocystis* sp. PCC 6803 is an exemplary model for investigating salt acclimation, offering insights into physiological processes, molecular mechanisms, and the dynamic resolution of salt acclimation. Transcriptomic analysis has identified several genes implicated in salt acclimation, but additional research is necessary to uncover the precise molecular pathways triggered during salt stress. Functional characterization of hypothetical proteins associated with salt acclimation could reveal novel mechanisms. Understanding salt acclimation in cyanobacteria has significant academic, economic, and environmental ramifications. The effective use of cyanobacteria in bioenergy production and environmental remediation depends on mass culturing of suitable strains in saline media, and knowledge of salt acclimation is essential for optimizing product yield while preventing the formation of unintended by-products. Additionally, the development of salt-tolerant crop is crucial for addressing the issue of soil salinity-induced reduction in arable land, and cyanobacteria represent a novel tool for such breeding experiments.

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Authors' contributions

NG contributed in conceptualisation and manuscript writing; AS developed illustrations, figures and referencing; AB played role in manuscript writing and referencing; AKM contributed in conceptualisation, literature supports, manuscript editing and communication; SSS contributed in conceptualisation and manuscript editing.

Declaration of conflict of interests

All the authors have no potential conflict of interest.

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