

Molecular Biology of Plant Tolerant to Saline Soil

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SUMMARY

As we all know that Agriculture is totally dependent on climate. So a variety of Abiotic Stresses cause a serious crop losses, thus limiting the agricultural productivity worldwide. By 2025, 30% of crop production will be at risk due to the declining water availability. World Bank projects that the climate change will depress crop yields by 20% or more by the year 2050. Efforts have been made by Plant breeder in developing abiotic stress resistant crop plants but alone are not sufficient enough. Thus the role of Genetic engineering in combination with plant breeding in crop improvement has become of great importance in assuring world future food security.

INTRODUCTION

Soil salinity is the total salt content in the soil. When there is high concentration of soluble salts, high enough to affect the plant growth is called saline soil. The process of increasing the salt content is known as salinization. Unfortunately the salinization process in agricultural fields will decrease the suitable land for cultivation by 30% within the next 25 years, and up to 50% by the year 2050. Therefore, developments of salt tolerant crops can be the best and most practical way to produce enough food. In the course of evolution, plants have developed several protecting mechanism (avoidance and tolerance) so as to adapt to salt stress. Understanding the cellular basis of salt stress tolerance mechanisms is necessary for breeding and genetic engineering of salt tolerance in crops. Tolerance mechanisms mainly are applicable to practical manipulations.

Source of Saline Soil

1. Irrigation water

2. Type of soil

- From reclamation a management point of view, the salt affected soil in India are broadly placed into 2 categories- alkali soil a saline soil.
- The alkali soil having high soil pH (up to 10.8), high exchangeable sodium percent (ESP) up to 90, low organic carbon, poor infiltration and poor fertility status.
- On the other hand, the saline soils have higher electrical conductivity ($>4\text{dS/m}$), low ESP ($<15\%$) and low pH <8.5 .

Effects on the Plants

Osmotic or Water Deficit Effect of Salinity –

The presence of salt in the soil solution reduces the ability of the plant to take up water and this leads to reduction in the growth of the plant which is called osmotic or water deficit effect of salinity. It reduce the leaf growth, decrease stomatal conductance and photosynthesis. It leads to the defect in the formation of new leaves.

Salt-Specific or Ion-Excess Effect of Salinity –

If the excessive amount of salt enter the plant in the transpiration stream, there will be injury to cells in transpiring leaves and this may cause further reduction in growth which is called salt-specific or ion excess effect of salinity. It cause increase in the senescence of the leaf and in severe condition, death of the plant.

Molecular Mechanism

Under non-saline conditions, nutritional Na^+ uptake is mediated by dedicated ion transporters of the HKT family. When Na^+ concentration in the surrounding environment increases, Na^+ is thought to enter the plant symplast through plasma-membrane non-selective cation channels (NSCCs), whose functional molecular identities are still uncertain, or Na^+ may also passively enter via anatomical ‘leaks’ in the root endodermis. Three major processes have long been considered to participate in protection against high cytosolic Na^+ : (1) minimize Na^+ entry into cells; (2) maximize compartmentation of Na^+ into the vacuole; and (3) increase efflux of Na^+ out of the cell. Of these, an efficient Na^+ extrusion mechanism has been demonstrated to be a key homeostatic process for keeping a low concentration of cytosolic Na^+ and maintaining a dynamic equilibrium of essential ions such

as K^+ and Ca^{2+} . In the 1990s, a forward genetic approach was used to dissect the molecular mechanisms regulating salt tolerance. Isolation and characterization of several mutants showing root growth hypersensitivity under salt stress led to the identification of SOS3, SOS2, and SOS1, the key components of the SOS signaling pathway involved in Na^+ extrusion. It has been recognized for many decades that excess Ca^{2+} can 'protect' plants from Na^+ toxicity. There is compelling evidence gathered over the years that, after perception of salt stress, a Ca^{2+} spike generated in cytoplasm of root cells activates the SOS signal transduction cascade to protect the cells from damage due to excessive ion. SOS3 encodes a myristoylated calcium-binding protein that appears to function as a primary calcium sensor to perceive the increase in cytosolic Ca^{2+} triggered by excess Na^+ that has entered the cytoplasm. Upon binding with Ca^{2+} , SOS3 is able to interact with and activate the serine/threonine protein kinase SOS2, which belongs to the SnRK3 family of protein kinases (sucrose non-fermenting-1-related protein kinase-3). More recently, SOS3-like Calcium Binding Protein 8 (SCaBP8, also known as Calcineurin B-like CBL10) has been shown to be an alternative regulator of SOS2 activity that functions primarily in the shoot of Arabidopsis, whereas SOS3 is more prominent in roots. SOS2 phosphorylates SCaBP8 and this event stabilizes the protein complex. SOS3-SOS2 or SCaBP8-SOS2 interactions recruit SOS2 to the plasma-membrane leading to activation of the downstream target SOS1, a Na^+/H^+ antiporter. This causes subsequent extrusion of excessive Na^+ from the cytosol which leads to the tolerance of the plants under saline condition. Also, SOS1 is a target of the phospholipase D (PLD) signaling pathway in ion sensing and dynamic equilibrium adjustment under salt stress. Exposure to salt stress causes an increase in enzyme activity of PLD α 1 in Arabidopsis, resulting in rapid and transient accumulation of the lipid second messenger phosphatidic acid (PA). PA in turn activates Mitogen-Activated Protein Kinase 6 (MPK6), which can directly phosphorylate SOS1 and leads to the tolerance mechanism. Another mechanism is by changing the architecture of the root system. SOS signaling pathway plays a role in the plastic development of root hairs under salt stress. In response to NaCl stress, primary root elongation is inhibited whereas the effect on lateral root (LR) formation seems to vary depending on the severity of the stress, with high concentrations of NaCl inhibiting LR formation, whereas lower concentrations of salt stimulate LR proliferation in an auxin-dependent manner. Molecular and physiological analyses revealed that SOS3 is required for auxin biosynthesis, polar movement to stressed roots, and for the formation and maintenance of an auxin gradient. So with the help of SOS3 gene, this is being transported to the outside with the help of PINs which lead to the lateral root formation which ultimately give tolerance mechanism of the plant.

Besides this, ROS are very reactive and interact with cellular molecules and metabolites resulting in several destructive processes and cellular damage. At the time of salinity stress and consequently osmotic stress, CO_2 availability for photosynthetic carbon assimilation because of stomatal closure, is limited. This even leads to over reduction of electron in the photosynthetic transport chain, so causing the production of ROS, in that respect causing high accumulation of superoxide in chloroplast and finally photoinhibition and photooxidation damage. Plants can get rid of superoxide with the help of superoxide dismutase (SOD), which catalyzes the superoxide into hydrogen peroxide (H_2O_2) and oxygen, and also SOD is important in hindering the reduction of metal ions and hydroxyl radical synthesis in salinity condition poisonous levels of sodium and also inadequate amount of K for enzymatic reactions and osmotic adjustment is happened. Plants by synthesis and accumulating of various compatible osmolytes in cytosol, decreasing osmotic potential to keep water absorption from saline soil solutions and limit salt absorption. The DREB1A transcription factor is particularly to DRE elements and induces the stress tolerance gene expression. Over expression of genes encoding these proteins can induce high gene expression leading to stress tolerance, but reduced growth is inevitable event even in the condition without stress.

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