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Mechanisms Involved in Salt Tolerance of Plants

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INTRODUCTION

A large part (about 70%) of the surface of the earth is covered by oceans that comprise a salt solution with an osmotic potential of about −2.0 MP, derived primarily from sodium and chloride—about 0.5 and 0.6 M, respectively [1]. It is further estimated that a third of the world's irrigated land has been salinized to various degrees. This salinization results from an accumulation of salts dissolved in the irrigation water. Many wild as well as cultivated plants have thus to deal with saline environments.

A saline environment imposes two principal kinds of stress on plants: an osmotic stress and a toxicity stress.

OSMOTIC STRESS

The water potential of plant cells generally equilibrates with that of their environment. The water relations of plant cells and their environment are given by Equation (1) [2]:

$$\Psi_w^o = \Psi_w^i = \Psi_\pi^i + \Psi_p \tag{1}$$

where Ψ_w = water potential, Ψ_π = osmotic (or solute) potential, Ψ_p = turgor, o = outside and i = inside. The water potential of the saline environment, Ψ_w^o, is primarily determined by its salt concentration (Ψ_π). Exposure of wall-encased plant cells to the low Ψ_w^o of a saline environment results in equilibration of Ψ_w, by cell-water loss and an accompanying decreases of Ψ_π^i and turgor (Ψ_p), according to Equation 1. In wall-less cells, such as those of some microalgae, turgor is almost nonexistent and $\Psi_w^i = \Psi_\pi^i$. In such cells, the lowering of Ψ_w^o, the consequent water loss, and the decrease of Ψ_w^i, are accompanied by a decrease of Ψ_w^i and of cell volume.

Turgor is a prerequisite for plant cell expansion and growth. A simplified description of the growth in relation to turgor is given in Equation (2) [3]:

$$G = m (\Psi_p - y) \quad (2)$$

where G = growth rate, m = plasticity of cell walls, and y = threshold turgor for cell enlargement. In a saline environment, growth should, hence, cease if turgor is not regulated. Salt-resistant plants are able to regulate their turgor within the range of their salt resistance, or they are able to adjust cell-wall plasticity and threshold values.

Turgor Regulation

Bisson and Gutknecht [4] described the sequence of events occurring in plant cells on external salinization and decrease of Ψ_w^o (Fig. 1): Water exits from the cell, turgor decreases, and water potentials equilibrate. The turgor decrease is sensed by a ''turgor sensor,'' apparently in the plasma membrane. The sensor emits an ''error signal'' that is transduced to the activation of some biochemical processes, such as increased solute accumulation or synthesis. Changes in the physical tension of the cytoskeleton during water stress might be involved in triggering the responses [5]. Enhanced accumulation and synthesis results in an increase of the amount of solutes in the cell, a transient decrease of Ψ_π^i and Ψ_w^i water influx, and eventually recovery of the original (regular) turgor pressure. During the recovery phase, Ψ_w^i and Ψ_π^i do not change, but the amount of solutes in the cell and turgor increase concurrently. In wall-less cells, a similar sequence of events regulates volume instead of turgor.

Some initial error signals resulting from turgor decrease have been investigated. In the salt-resistant Characean *Lamprothamnium* [6], a hypertonic salt shock induced a hyperpolarization of the plasma membrane potential. Concordantly, in red beet tissue slices and some plant roots, a nonplasmolysing hypertonic DASW, (dilute artificial sea water −0.5 MPa) shock induced an enhancement of plasma membrane (PM) adenosine triphosphatase (ATPase) activity; in response to

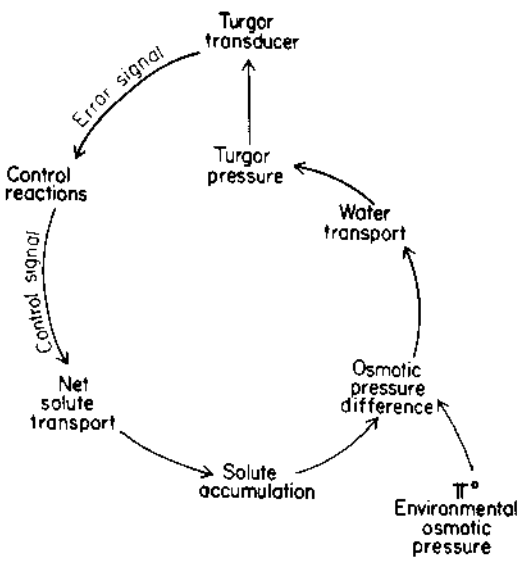

FIGURE 1 Basic elements of turgor regulation system based on solute and water transport. Input of system is random fluctuations in environmental water potential and output is turgor pressure. (From, Ref. 4.)

a similar (−0.5 Mpa) mannitol shock, enhanced K^+ uptake could be measured as well. The DASW shock also induced an increase of the inositol-1,4,5-trisphosphate (1,4,5-IP_3) content in the cells [7] (Table 1), a decrease in PM phosphatidylinositol-4,5-bisphosphate ($PtdInsP_2$) (Table 1), and phosphorylation of some PM membrane proteins [7] (Table 1). The effects of a DASW shock on ATPase activity, 1,4,5-IP_3 and $PtdInsP_2$ were observed 1 min after shock application and before enhancement of protein phosphorylation was evident [8] (Table 1). This sequence of events implied that protein phosphorylation was not a prerequisite for DASW-induced enhancement of ATPase activity. All the cited effects of DASW were inhibited by neomycin, an inhibitor phospholipid interconversion and hydrolysis in animals [9] and plants [10]. These cited DASW effects could be induced by secondary butanol in the absence of a DASW shock (Table 1); the latter compound artificially activates G-proteins [11]. These results indicated that the initial, turgor loss–induced, *error* signal involves G-proteins and the phosphoinositide cascade [12]. Changes in PM phosphoinositide composition may activate the PM ATPase [9,12,13]. Protein phosphorylation may be involved in subsequent activation of processes responsible for long-term turgor regulation, such as synthesis of osmoprotective compounds. For example, osmotic stress increased the phosphorylation of spinach leaf sucrosephosphate synthase, catylized by a Ca^{2+}-dependent protein kinase [14]. In yeast the protein phosphatase calcineurin was essential for salt tolerance. The latter data indicated that NaCl adaptation in yeast depended on signal transduction involving Ca^{2+} and protein phosphorylation/dephosphorylation.

Calcium ions also seem to be involved as a second messenger in transduction of the error signal in the unicellular, wall-less alga *Poterioochromonas*. In response to an osmotic shock, this alga regulates volume first by enhanced K^+ uptake and later by isofloridozide synthesis. The synthesis depends on Ca^{2+}-mediated activation of the enzyme isofloridozidephosphate synthase [15]. Volume regulation was not hinged on the presence of external Ca^{2+}. The Ca^{2+} needed for activation of isofloridozide synthesis should, hence, have originated from an internal compartment, apparently the vacuole. Calcium release from the vacuoles of plant cells is induced by elevation of cytosolic 1,4,5 -IP_3 [16]. Increased cytosolic Ca^{2+} concentration seems to induce the release of a membrane-bound protease in *Poterioochromonas* cells. The protease, in turn, activates isofloridosidephosphate synthase [15].

Joset et al. [17] distinguish between immediate responses to salt stress, such as those cited above and long-term adaptations that are protein synthesis dependent. The latter kind of adaptations reported for higher plants include synthesis of neutral organic compounds; induction of salt stress–associated proteins, such as osmotin [18] and gluthatione peroxidase [19]; and upregulation of PM [20] and tonoplast [21] H^+-ATPases. Some of the stress-inducible genes that encode proteins, such as Δ^1-pyrroline 5-carboxylate synthetase, a key enzyme for proline biosynthesis, were overexpressed in transgenic plants to produce a salt-tolerant phenotype of the plants [22]; the latter results indicated that the gene products really function in stress tolerance.

Genes induced during water- and salt-stress conditions are thought to function not only in protecting cells by the production of important metabolic proteins but also in the regulation of genes for factors involved in the signal transduction cascades of the stress response [23]. The latter include such factors as protein kinases and phosphlipase C [5,24].

Solutes Employed for Turgor Regulation in Plants

Various organic solutes, as well as mineral ions, in particular Na^+, K^+, and Cl^-, are accumulated in plants during turgor or volume regulation. Some halophytes, the native flora of saline environments [25], adjust their solute content mainly with inorganic ions. *Suaeda maritima* plants grown in 370 mM NaCl (−1.76 MPa) maintained the Ψ_π of their leaves at −2.5 MPa and NaCl accounted for 93% of the accumulated salt [26]. In other plants, such as the marine alga (*Porphyra purpurea* L.) [27], sodium is excluded or excreted, and KCl is the major solute accumulated for turgor regulation. Potassium chloride also comprises most of the solute accumulated in the extremely halophylic bacteria *Halobacterium halobium* grown in 3 M NaCl, whereas Na^+ is excreted and maintained at a low

TABLE 1 Initial Responses in Plasma Membranes of Aged Red Beet Slices to a Dilute Artificial Sea Water (−0.52 MPa) Shock

DASW shock	ATPase: μmol (h mg protein)$^{-1}$	1,4,5-IP_3 pmol (g FW)$^{-1}$	$PtdInsP_2$: % of ^{32}P-labeled phosphoinositides	20-kDa poly-peptide phosphorylation
None	98 ± 6.7[a]	9.1 ± 0.9[a]	1.36 ± 0.16[a]	100 ± 4.2[a]
1 min	149 ± 3.0[c]	18.9 ± 1.6[c]	0.62 ± 0.07[c]	75 ± 4.2[a]
2 min	155 ± 3.3[c]	19.2 ± 1.8[c]	0.74 ± 0.06[c]	325 ± 29[c]
None, neomycin	110 ± 7.1[a]	5.2 ± 0.4[a]	1.32 ± 0.1[a]	
2 min + neomycin	102 ± 6.8[a]	7.8 ± 0.6[a]	0.99 ± 0.04[a]	112 ± 5.2[a]
None, 0.8% secondary-butanol	180 ± 7.2[c]	17 ± 1.0[c]	0.58 ± 13[c]	

PM ATPase activity; 1,4,5-IP_3 (inositol-1,4,5-trisphosphate) content; PM $PtdInsP_2$ (phosphatidylinositol Bisphosphate) content; and phosphorylation of 20-kDa PM polypeptide. Relative Density of SDS-PAGE autoradiographs. Mean ± SE. Different superscripts in each column indicate significant differences at $P < .01$.

internal concentration [28]. In other plants, a larger part of the solutes comprise organic compounds. Thus, in mature leaves of *Thinopyrum bessarabicum*, a salt-tolerant perennial grass [29], K^+ and Na^+ salts accounted for only 50–60% of the sap Ψ_π in both control and salt-treated plants. In control plants, the K^+/Na^+ ratio was 60, and it changed to 1.0 in plants treated with 0.37 mM NaCl in the medium. A survey of salt marsh plants [30] showed low K^+/Na^+ ratios in dicotyledonous halophytes and high ratios for monocotyledons. The range of K^+/Na^+ ratios for dicotyledons was 0.06–1.19 with a mean of 0.38 ± 0.3, and for monocotyledons, it was 0.27–14.2 with a mean of 2.4 ± 0.6.

Neutral organic solutes make major contributions to turgor regulation in unicellular, slightly vacuolated algae [31]. A large part of the biomass of plants would have to be diverted to turgor regulation if organic solutes were the main compound employed for this in highly vacuolated plant cells. Greenway [32] calculated that for adaptation to 100 mM external NaCl with hexoses, 20–30% of the total biomass would be needed. Raven [33] analyzed the cost benefit of turgor regulation with different solutes. These calculations show that 2–4 mol photons of light energy are needed for the accumulation of 1 osmol KCl or NaCl, whereas 68–78 mol photons are needed for the synthesis of 1 osmol sorbitol or mannitol, 70–93 mol photons for 1 osmol proline, and 78–101 mol photons for 1 osmol glycinebetain. The exact amount of mol photons needed in each case depends on whether the solutes are accumulated in the roots or shoots, and for proline and glycinebetain, also on the N source—NH_4^+ or NO_3^-.

Energy inexpensive turgor regulation with mineral ions, seems to be limited by the inhibitory effects of high salt concentrations on various metabolic processes in the cytoplasm. Hence, adjustment to low Ψ_w^o with mineral salts is limited in the cytoplasm and largely confined to the vacuoles. Slightly vacuolated organisms, such as *Chlorella, Ochromonas*, and *Dunaliella*, have to use compatible organic compounds for a large part of the adjustment. The same seems to be true for the cytoplasmic compartment of vacuolated cells.

Cytoplasmic Compartmentation of Organic Solutes

Various lines of evidence indicate that, in response to salt stress, organic solute accumulation in vacuolated plant cells is primarily restricted to the cytoplasmic compartment (cytosol and cytoplasmic organells). As the cytoplasm constitutes only 5–10% of the osmotic volume [34] of vacuolated cells [35], relatively small amounts of solute can account for the adjustment therein to high external salt concentrations.

Cytoplasmic confinement of digeneaside (2-D-glyceric acid α-D-mannopyranoside) accumulated under saline conditions is indicated for the marine red alga *Griffithia monilis* L. [36]. Digeneaside concentration decreased in the cells of this alga with cell size and concomittant vacuolization. The digeneaside/chlorophyll *a* ratio of the cells however did not change (Table 2). These relations indicated that digeneaside accumulation was restricted to the cytoplasm that also contains the chlorophyll. Confinement of organic solutes to the cytoplasm was also shown for *Mesembryanthemum crystallinum*. Exposure of this plant to 0.4 M NaCl was accompanied by pinitol (1-D-3-O-methyl-

TABLE 2 Variation of Digeneaside Concentration with Size of *Griffithia monilis* Cells[a]

Cell size	Digeneaside (μmol g^{-1} FW)	Chlorophyll a (mg g^{-1} FW)	Digeneaside (μmol g^{-1}/Chlorophylla *a*)
Large	1.97	0.097	20.3
Small	5.87	0.280	20.9

[a] Large cells were those with about 50% >2 mm; small cells were those with few >2 mm.
Source: From Ref. 36.

chiro-inositol) accumulation in the leaves to 10–14 mmol (kg frwt)$^{-1}$ [37]. Leaf-cell protoplasts, chloroplasts, and vacuoles were separated and analyzed. Calculations indicated a pinitol concentration of 230 mM in the chloroplasts and of 100 mM in the cytosol; none was detected in the vacuoles.

Transmission electron microscopy and x-ray microanalysis were employed by Hall et al. [38] to localize glycinebetaine in shoot cells of *Suaeda maritima*. Glycinebetaine was shown to be accumulated under saline conditions and to be restricted to the cytoplasm (Fig. 2).

Adjustment of Cell Wall Characteristics

Equation (2) [$G = m(\Psi_p - y)$] shows that the growth rate (G) of plant cells depends on cell wall plasticity (m) and on the turgor above a threshold value (y). Hence, in order to maintain growth under saline conditions, plants may either increase the amounts of solutes in the cells and regulate turgor or adjust plasticity and/or threshold turgor. Adjustment of threshold turgor can indeed be considered as regulation of the effective turgor ($\Psi_p - y$). Plasticity and threshold turgor are both cell wall characteristics.

Munns et al. [39] found only partial turgor regulation in the unicellular microalga *Chlorella emersonii* L. when exposed to low external Ψ_w. However, growth decreased much less than turgor (Table 3). They found a large decrease in the volumetric elastic modulus ε of the cells. This modulus is the relation between turgor change ($\Delta\Psi_p$) and relative volume change (ΔV) during variations in water content of plant cells ($\varepsilon = \Delta\Psi_p \times V_{initial}/\Delta V$). The decrease of ε was not related to a decrease in wall thickness; the latter, indeed, increased with water stress. The investigators concluded that the decrease of ε indicates a change of cell wall properties that also effects plasticity and threshold turgor. The latter changes would explain the relatively small effect of turgor decrease on the growth rate of *Chlorella emersoni*.

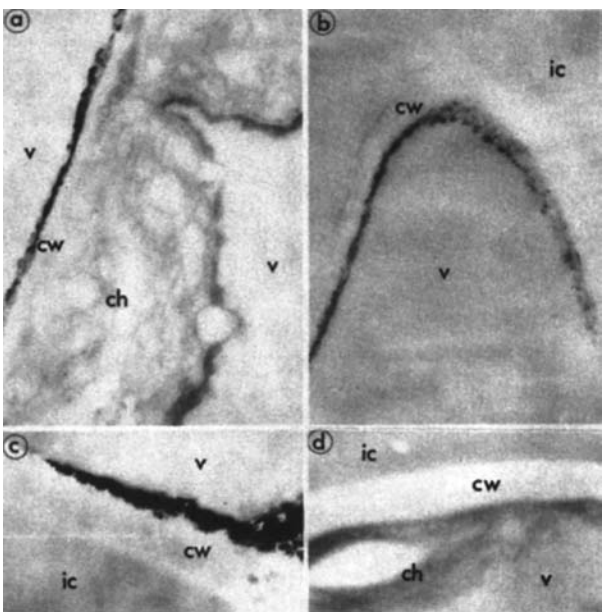

Figure 2 Electron micrograph of *Suaeda maritima* cells freeze-substituted in the presence of iodoplatimate stain **a, b**. Grown in the presence of 1 % NaCl showing dense betaine deposits in the cytoplasm and no staining in the vacuole **a,** ×25,500, **b,** ×38,000. **c**. Grown in the presence of 3% NaCl showing dense cytoplasmic deposits ×30,000. **d**. Grown on tap water showing no staining ×25,500. (From, Ref. 38.)

TABLE 3 Relative Growth Rate (RGR), Turgor, and Volumetric Elastic Modulus (VEM) of *Chlorella emersonii* Grown for 6–10 Days at Various NaCl Concentrations

Growth medium		RGR (% of rate	Turgor	VEM
NaCl (mM)	Ψ_π (MPa)	at 0.08 MPa	(MPa ± SE[a])	MPa ± SE
1	0.08	100	0.54 ± 0.18	8.5 ± 1.7
200	1.02	90	0.16 ± 0.009	1.4 ± 0.7
300	1.64	55–70	0.012 ± 0.023	0.9 ± 0.6

[a] Standard error of the mean.
Source: From Ref. 23.

SALT TOXICITY

Sodium chloride is the most important constituent of saline environments. The accumulation of NaCl by plant cells for turgor regulation is limited by the toxicity of a high salt concentration. Such cytoplasmic Na^+ toxicity is ubiquitous in all eucaryotes and bacteria. Even the ancient halophilic *Halobacteria* [40] accumulate K^+ and Cl^- to concentrations of several mols L^{-1}, but not Na^+. The accumulated K^+ and Cl^- ions are located in the cytoplasm of these bacteria and the enzymes are adapted to the high KCl concentration. Enzymes extracted from salt-adapted halophytes are NaCl sensitive. These enzymes are severely inhibited in vitro at salt concentrations similar to those that are optimal in the medium for growth of these plants [41,42]. The in vitro salt sensitivity of amino acid incorporation into proteins by microsomes from salt-adapted halophytes (Fig. 3) did indeed not differ from that of microsomes obtained from glycophytes [43].

The specific harmful effect of NaCl, in addition to its osmotic effect, was elegantly demonstrated by Cramer et al. [44]. They monitored the growth of maize roots in the presence of mannitol

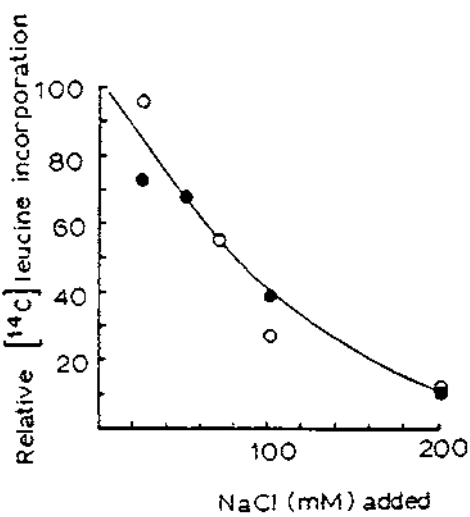

FIGURE 3 The effect of NaCl on the incorporation of leucine into protein by microsomal fractions prepared from *Suaeda* grown in the presence (open circles) and absence (closed circles) of salt. (From, Ref. 43.)

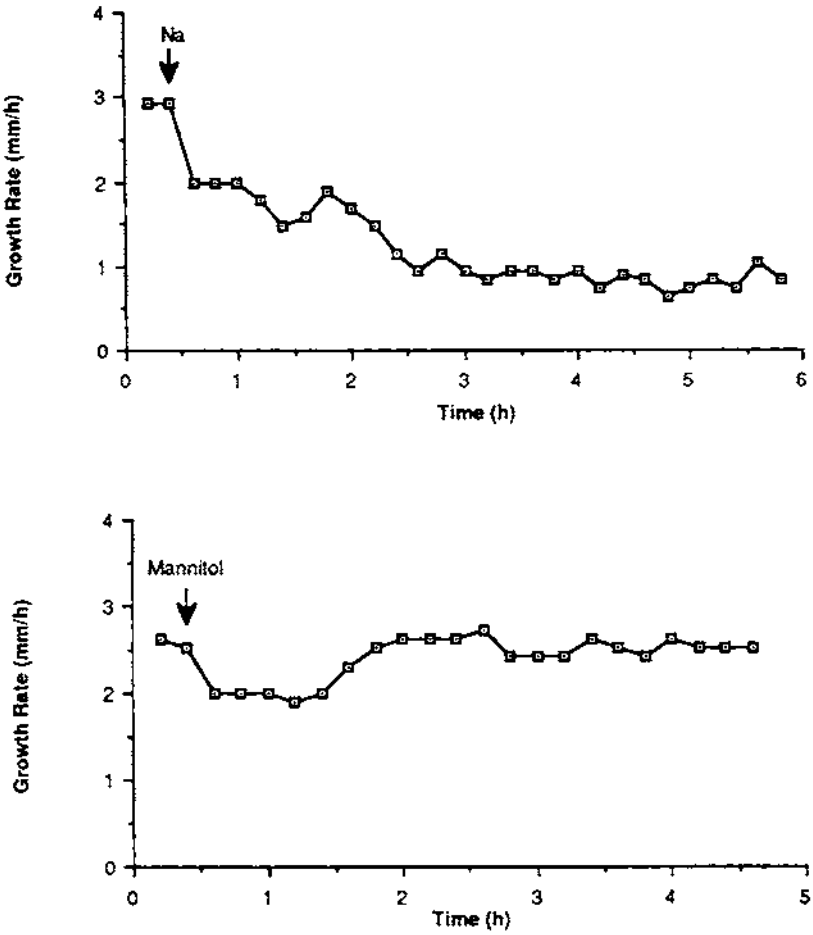

FIGURE 4 The effects of NaCl (above) and mannitol (below) on root elongation over time. At the time indicated by the arrow, 75 mM NaCl or 138 mM (isotonic) mannitol were added. (From, Ref. 44.)

and isotonic NaCl (Fig. 4). In mannitol, an initial decrease of growth rate occurred followed by gradual recovery. In NaCl, the growth rate declined to 20% of that before salt addition and did not recover.

Plant Strategies for Sodium Avoidance

Plants have apparently evolved two principal strategies for avoiding high sodium concentrations in the cytoplasm: compartmentation and exclusion.

Sodium Compartmentation and Compatible Solutes

Many halophytes regulate turgor by NaCl accumulation to a concentration higher than that in the saline medium. Numerous essential enzymes are severely inhibited in vitro at such Na^+ concentrations. Flowers et al. [45] compiled a list of enzymes that are 50% inhibited when exposed in vitro

to the salt concentration found in their source tissue. Wyn Jones et al. [46] suggested compartmentation of salts in plant cells. Thus, in plants, such as the halophilic grass *Distichis spicata* L. [47] that accumulate large amounts of sodium salts in their cells, these salts seem to be occluded in the vacuole, where they serve for turgor regulation. Organic solutes that are compatible with enzyme function apparently have a large share in turgor regulation in the cytoplasmic compartment of the plant cells.

Compatible osmolytes found in higher plants comprise a relatively small number of low molecular weight organic compounds, mainly proline [47–55], glycinebetaine [29,38,51,55–57], some sugars [29,58–60], polyols [37,60], and malate [60]. A larger variety of such compounds is found in lower plants [31,47]. Compatible solutes are supposed to provide an environment that is compatible with macromolecular structure and function [61]. It was proposed that these solutes are preferentially excluded from the surface of proteins and their immediate hydration sphere. Thus, the addition of these solutes to a protein suspension creates a thermodynamically unfavorable situation, since the chemical potentials of both the protein and the additive are increased. This situation stabilizes the native conformation of the proteins, because denaturation would lead to a greater contact surface between the protein and the solvent, thus augmenting the unfavorable effect [62]. Steward and Lee [50] demonstrated the compatibility of proline with glutamate dehydrogenase extracted from the halophyte *Triglochin maritima*. The enzyme was not inhibited in vitro by proline up to a concentration of 0.6 M. Similar results were obtained for barley leaf malate dehydrogenase and barley-embryo pyruvate kinase [63]. These enzymes were not inhibited in vitro by up to 0.5 M glycinebetaine. In addition, glycinebetaine, and to a lesser extent dimethylglycine, partially restored malate dehydrogenase activity in the presence of NaCl. The enzyme was 70% inhibited in the presence of 0.3 M NaCl alone. The inhibition decreased linearly with addition of glycinebetaine to 50% at 0.5 M glycinebetaine (Fig. 5).

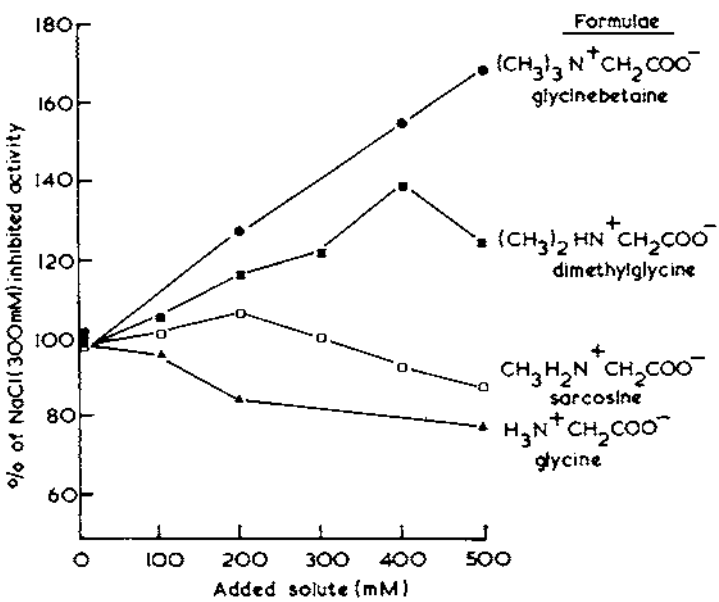

FIGURE 5 Comparative effects of successively methylated derivatives of glycine on inhibition of barley leaf malate dehydrogenase (decarboxylating) by 0.3 M NaCl. Activity was 70% inhibited by 0.3 M NaCl alone. (From, Ref. 63.)

Mechanisms of Sodium Compartmentation

Various lines of evidence show that Na^+ is occluded in the cell vacuoles of many plants, particularly in halophytes, and is excluded from the cytoplasm of all plants. Indirect evidence for such compartmentation comes from measurements of longitudinal profiles of Na^+ and K^+ concentrations in roots. In such experiments with *Hordeum distichum* grown in the presence of 1 M NaCl [64], Na^+ concentration in meristemic, nonvacuolated, cells at the root tip was 10 mM. Sodium concentration increased rapidly with distance from the root tip and with cell vacuolization to 65 mM at 2 mm from the tip. Potassium concentration changed in the opposite direction; that is, it decreased with distance from the root tip. Comparable results were obtained for *Atriplex hortensis* and *Plantago maritima* roots [65].

More direct evidence for compartmentation was obtained with electron probe x-ray microanalysis. Harvey et al. [66] examined compartmentation of the major mineral ions in leaf cells of *Suaeda maritima* grown in the presence of 350 mM NaCl (Table 4). They found a large accumulation of Na^+ and Cl^- in the vacuoles and relatively low concentrations in the cytoplasm; the K^+ concentration was similar in both compartments. The data for glycinebetaine presented in Table 4 were taken by the authors from their earlier work, where the concentration [67] and cytoplasmic localization of this solute [38] were established. Glycinebetaine accounted for more than 75% of the osmolality of the cytoplasm. Hijibagheri and Flowers [68] found similar Na^+ compartmentation in the roots of *S. maritima* 118 mM in the cytoplasm and 432 mM in the vacuoles.

Mechanisms of Na^+ Transport

Sodium transport from the environment into the cytoplasm of plant cells is a passive process. It depends on the electrochemical-potential gradient of Na^+ and the presence of Na-permeable channels in the plasma membrane. In principle, Na^+ could accumulate in the cytoplasm to a few hundred times of its concentration in the environment. For steady-state conditions and 30°C, the relation is $E_M/60 = \log [Na^+]^\circ/[Na^+]^i$, where E_M = membrane potential [69]. Thus, at an E_M of -120 mV (cytoplasm negative), Na^+ could accumulate in the cytoplasm to 100 times the external concentration. Such accumulation is prevented in salt-tolerant plants by control of influx (channel gating) and/or by active export from the cytoplasm to the vacuoles and also back to the environment.

Active sodium transport in plant cells is performed by Na^+/H^+ antiport [70] that is ordinarily driven by an ATPase-activity derived protonmotive force [71]. Such antiport has been documented at plasma membranes and tonoplasts of some plants [72]. In yeast, gene amplification at a locus encoding a putative Na^+/H^+ antiporter conferred Na^+ tolerance [73].

A survey of 16 crop plants [72], however, showed that the presence of a Na^+/H^+ antiporter is not ubiquitous in plants. It could not be demonstrated in 10 of the 16 surveyed plants, including *Zea mays*, *Phaseolus vulgaris*, and *Gossipium hirsutum*. In *Chara longifolia*, a salt-tolerant charophyte, Na^+/H^+ antiport at the PM was induced by 24 h preculture in artificial sea water [74].

The presence of a Na^+/H^+ antiporter would be expected in the tonoplasts of plant cells that

TABLE 4 Compartmentation of Na^+, K^+, Cl^-, and Glycinebetaine in *Suaeda maritima* Leaf Cells

	Concentration (mM)	
Solute	cytoplasm	vacuole
Na^+	109	565
K^+	16	24
Cl^-	830	388
Glycinebetain	830	—

Source: From Refs. 38,66,67.

tolerate Na^+ by its excretion to and occlusion in the vacuoles. Plants that have not conserved this antiporter during their phylogenesis should have to regulate cytoplasmic Na^+ concentration by Na^+ exclusion.

Ion Channels and Sodium Exclusion

The sodium permeability of biological membranes is 10^2–10^6 times higher than that of artificial phospholipid bilayers [75]. This permeability is facilitated by intrinsic proteins that constitute ion channels in the phospholipid bilayer [76]. Sodium-specific channels have hitherto not been demonstrated in the plasma membranes of plant cells. Sodium apparently moves through a general cation channel with different permeabilities for the various ions [77]. Calculations for cells of the Characean alga (*Nitella obtusa* L.) [78] indicated that the measured permeability and density of such channels could quantitatively account for Na^+ influx in salt-stressed cells. Regulation of gating and selectivity of such channels seem to be responsible for sodium exclusion in many salt tolerant crop plants. The presence of K^+ and in particular Ca^{2+} ions has been shown to decrease Na^+ influx to plant cells (Fig. 6) [79–85], and consequently to decrease Na^+ damage [80] and yield reduction [83,84].

The existence of two kinds of channels that allow Na^+ permeation has been reported for the plasma membrane of plant cells. One is an inward rectified channel (closes on membrane depolarization) with P_K/P_{Na} (K^+/Na^+ permeability ratio) of 5–10 [86] and an outward rectified one (opens on depolarization) with P_K/P_{Na} of 20–60 [74]. The latter channel may serve as a possible route for Na^+ entry and K^+ loss under high salt conditions [87]. Schachtman et al. [77] suggested that depolarization opens the outward rectified channel allowing Na^+ influx and K^+ efflux under saline conditions and increasing conductivity. Indeed, Katsuhara and Tazawa [82] showed that 0.1 M NaCl depolarized the plasma membrane of *N. obtusa*, increased its electrical conductivity (EC), increased Na^+ content of the cells, and decreased their K^+ content.

Regulation of the inward rectified cation chanel seems to be involved in salt adaptation [87]. Adaptation of tobacco cells to 50 or 100 mM NaCl resulted in an about twofold reduction of the PM outward rectifying cation-channel permeability. Such reduction in the permeability to K^+ and Na^+ of the PM cation channels, caused by adaptation to salt stress, would decrease the entry of

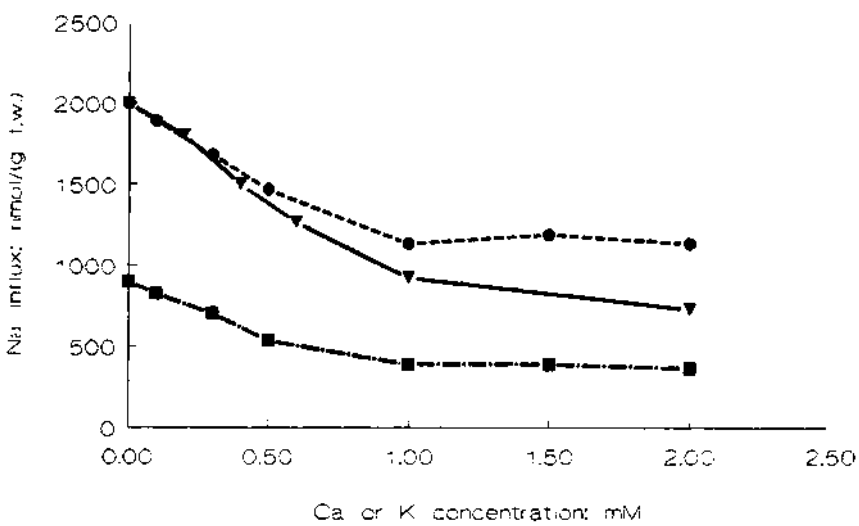

FIGURE 6 Effects of Ca^{2+} (triangles), K^+ (circles), and K^+ in the presence of 10 mM $CaSO_4$ (squares) on Na^+ influx from 10 mM NaCl for 30 min into corn root segments. (Compiled from Ref. 81.)

Na^+ ions into cells and the leakage of K^+ ions out of cells under high salt conditions. The latter study revealed no significant difference between NaCl–adapted and NaCl–unadapted cells in the K^+/Na^+ permeability ratio (P_K/P_{Na}). Similar results were reported for the P_K/P_{Na} of the outward rectfying PM channels of root cells from a NaCl-tolerant and a NaCl-sensitve species of wheat [80]. The investigators concluded that salt-induced reduction of conductivity should be ascribed to a reduction in the frequency of channel opening and/or in the number of channels. A different situation was reported for yeast (see Ref. 88 and references therein). Yeast cells absorb Na^+ by the K^+ uptake system, and the ratio between K^+ and Na^+ K_M values (affinities; low K_M = high affinity) varies depending on the growth conditions. When this system was in the low-affinity state, the ratio between K_M values for Na^+ and K^+ was approximately 15; in the high-affinity state, this ratio increased to 300. Under Na^+ stress, the uptake system converted to the high-affinity system, thus increasing the discrimination between K^+ and Na^+. *TRK1* is *a gene* required for the expression of the high-K– affinity mode of transport. The salt tolerance of a yeast strain carrying a disruption in *TRK1* was 125 mM NaCl, whereas that of the wild type was 400 mM.

Membrane potential–dependent Na^+ influx to corn root was abolished in the presence of K^+ [81] and Ca^{2+} [82]. These cations thus seem to prevent Na^+ movement across the inward rectified channel.

Katsuhara and Tazawa [82] investigated the effect of Ca^{2+} on the salt tolerance of *N. obtusa*. They showed that Ca^{2+} inhibits the Na^+-induced depolarization of the plasma membrane, its increase in electrical conductivity, the increase of Na^+ content of the cells, and the decrease of their K^+ content. Investigations by Hoffmann et al. [89] with *Chara* showed that addition of Ca^{2+} drastically decreased P_{Na} and, hence, Na^+ fluxes at all concentrations.

The sites of Na^+ action and its prevention by Ca^{2+} as well as the sequence of these events are still not clear. Cramer et al. [90] speculated that displacement of Ca^{2+} by Na^+ from the surface of the plasma membrane may be the primary event, and that this is prevented by increased external Ca^{2+} concentration. The investigations further suggested that the opening of K^+ channels and K^+ leakage may either be a direct result of Ca^{2+} displacement from membrane surfaces or from membrane depolarization and a rise of intracellular Ca^{2+}. Either way, potassium leakage should probably be preceded by a change in the direction of the electrochemical K^+ gradient. Such a change would be induced by membrane depolarization, and it should also open the outward rectified K^+ channel.

Evidence for a possible intracellular action of Ca^{2+} is provided by Lynch et al. [91] for maize root protoplasts showing an increase of cytosolic Ca^{2+} concentration in the presence of external 120—150 mM NaCl. The investigations proposed that this Ca^{2+} originated from an internal compartment. However, the possibility that Ca^{2+} may have permeated from the outside, where the Ca^{2+} concentration was 0.1 mM, can not be excluded. Membrane depolarization has been shown to increase Ca^{2+} influx [92], apparently due to Ca^{2+} channel opening [93].

Sodium-induced membrane depolarization may indeed, be activated by Ca^{2+} displacement from membrane surfaces [90,94], or alternatively by Na^+ influx and increased cytoplasmic Na^+ concentration. In *N. obtusa* cells, the protective effect of externally supplied Ca^{2+} depended on the concurrent intracellular presence of ATP or ADP [95]. The presence of the adenine nucleotides decreased the opening frequency of a Na^+-permeable channel [78]. The data for *N. obtusa* [95] further indicate that Ca^{2+} does partially prevent Na^+-induced membrane depolarization (Fig. 7). In the absence of Ca^{2+}, externally supplied Na^+induced a complete depolarization of the plasma membrane. In the presence of Ca^{2+}, only partial and transient depolarization was induced by Na^+; E_M then recovered and receded to -116 mV instead of -131 mV in the absence of Na^+. A transient depolarization induced by Na^+ influx could cause Ca^{2+} channel opening and Ca^{2+} influx. Elevated cytosolic Ca^{2+} concentration may then regulate Na^+ permeability in concert with adenine nucleotides and prevent further Na^+-dependent malfunction of the cells.

Effect of Salinity on Potassium Content

The deleterious effects of salt, reported for *N. obtusa*, included excess Na^+ accumulation as well as K^+ leakage [82]. Both are prevented by Ca^{2+}. Thus, the presence of Ca^{2+} seems to increase K^+/

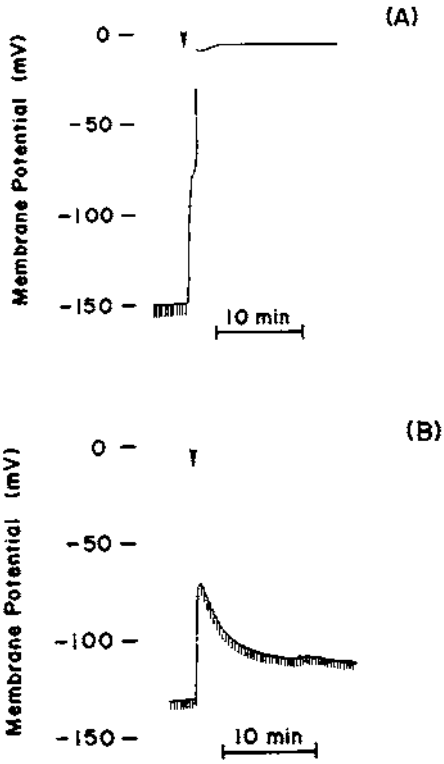

FIGURE 7 Changes in membrane potential of tonoplast free *Nitellopsis* cells perfused with a medium containing 1 mM ATP. Cells were first incubated in APW (artificial pond water) and then treated with APW + 0.1 M NaCl (A) or APW + 0.1 M NaCL + 10 mM $CaCl_2$ (B) at the time indicated. (From, Ref. 95.)

Na^+ selectivity [79] and to be necessary for the maintenance of an appropriate K^+ concentration in plant cells. The importance of Ca^{2+} for adequate K^+ absorption and growth under saline conditions was demonstrated in *Citrus* cell cultures grown on a range of NaCl concentrations in the presence of various $CaCl_2$ concentrations [84]. The growth rate of these cell cultures was related to their K^+ content. The capability of plants to maintain an adequate K^+ content under saline conditions is also enhanced by ample K^+ supply. Thus, salt-adapted *Sorghum* plants [96] were able to grow on 0.3 M NaCl in the presence of a full-strength Hoagland solution or half-strength Hoagland solution supplemented with K^+ to its concentration in full-strength Hoagland solution. The plants did not grow in 0.3 M NaCl with unsupplemented half-strength Hoagland solution.

The response of K^+ content in different plants to external Na^+ increments is not uniform, as shown in Table 5. Many plants, in particular relatively salt-tolerant glycophytes such as *Atylosia sericea* and *Glycine max* cv. Lee, maintain K^+ content constant or even increase it in the presence of salt. More sensitive glycophytes fail to maintain K^+ content in the presence of a high salt concentration. Such decrease of K^+ content may indicate damage [97]. This is demonstrated by two *Atylosia* species [98] and two *G. max* cultivars [99] differing in salt tolerance. The tolerant plants, *A. sericea* and *G. max* cv. Lee, are capable of increasing leaf K^+ content in the presence of salt as well as excluding Na^+ more efficiently than the sensitive ones, *A. acutifolia* and *G. max* cv. Jackson (Table 5). On the other hand, halophytes such as *Suaeda maritima* and *Simondsia chinensis*, as well as tolerant glycophytes that accumulate Na^+ such as *Lycopersicum peruvium, Solanum pennellii* [100], and *Sorghum bicolor* (Table 5), decrease their K^+ content with increasing external salt concentration

TABLE 5 Effect of Na^+ Concentration in the Medium on Na^+ and K^+ Concentration in Some Plant Species and Cultivars

Species	Medium Na^+ (mM)	Concentration in plants		Units[a]	Plant organ	Reference
		Na^+	K^+			
Atylosia sericea (tolerant)	0	80	350	1	Leaf	98
	50	133	500			
A. acutifolia (sensitive)	0	40	350	1	Leaf	98
	50	850	115			
Chlorella emersonii	0	9	282	2	Cell	39
	335	21	342			
Glycine max cv. Jackson (sensitive)	0	50	600	1	Leaf	99
	100	650	700			
Glycine max cv. Lee (tolerant)	0	58	588	1	Leaf	99
	100	176	882			
Lycopersium peruvium	0	200	2500	1	Callus	100
	350	1800	2200			
Simondsia sinensis	0	150	550	1	Leaf	55
	600	1300	250			
Solanum pennellii	0	250	2200	1	Callus	100
	35	2700	700			
Sorghum biocolor	0	0.9	349	3	Leaf	50
	184	51	140			
Suaeda maritima	0	50	1600	1	Shoot	26
	340	5000	330			

[a] (1) mmol/kg DW; (2) mM; (3) nmol/kg FW.

without concomitant damage. This decrease seems to be related to the replacement of vacuolar K^+ with Na^+ [101]. The maintenance of adequate K^+ content under saline conditions seems to depend on selective K^+ uptake as well as selective K^+ and Na^+ compartmentation in the cells and distribution in the shoots.

Sodium Distribution in the Plant

Most plants, when grown in the presence of salt, accumulate some Na^+ in their roots even when it is excluded from the shoots. Collander [102] distinguished between Na^+ accumulator plants and nonaccumulators. The former plants, transport large amounts of Na^+ to their shoots, whereas the latter exclude Na^+ from their shoots and retain it in their roots. Dicotyledonous halophytes are the most prominent Na^+ accumulators, but some salt-resistant glycophytes, such as barley, also belong to that group. Generally, salt-sensitive plants, such as beans and corn, are the most prominent Na^+ excluders. Table 6 compares Na^+ distribution in corn and barley.

Sodium retention in the roots of bean (*Phaseolus vulgaris L.*) plants was shown to result from metabolic energy–dependent depletion of Na^+ in the ascending xylem sap and in roots as well as stems [103,104]. Derooted bean plants retained Na^+ at the base of the stem. Absorption from the xylem was Na^+ specific as compared with K^+ and Cl^-. Sodium depletion of the xylem sap is accomplished by stelar cells lining the xylem [105,106]; transfer cells also have been implicated in this process [107]. Sodium that is removed from the xylem is transferred to the phloem and retransported to the roots [108,109]. Preferential removal of Na^+ from the xylem sap and recirculation to the roots occurs also in petioles [110] and veins of mature leaves [111,112]. In the absence of an inward directed electrochemical Na^+ gradient in the roots, Na^+ leaks to the medium [109,113]; otherwise, it is recirculated.

Sodium recirculation is a mechanism for Na^+ exclusion from the shoots employed by relatively salt-sensitive plants. It breaks down at high salt concentrations [103,104]. Cell membranes of sodium nonaccumulators, such as beans and apparently many other crop plants, seem not to comprise a Na^+/H^+ antiporter at the tonoplast [72] and, hence, cannot excrete Na^+ from the cytoplasm to the vacuoles. Sodium influx to the root and xylem is passive uniport via channels and also possibly by apoplastic bypass flow [114]. The latter flow bypasses the Casparian strips of endodermal cell walls. It is suggested to occur at sites of secondary root emergence [115,116] or through the apical region of the roots [117]. Bypass flow seems to increase under conditions of stress damage. Under saline conditions, bypass flow contributed substantially to the total quantity of Na^+ reaching the xylem of rice plants [114].

The mechanism of selective Na^+ absorption from the xylem is still being explored. It is inhibited by anoxia and depends on energy metabolism [104,118]. It cannot be envisioned as simple

TABLE 6 Distribution of ^{22}Na in Corn and Barley Grown for 25 h in 0.2 m $MCaSO_4$ and 10 mM $^{22}NaCl$

	^{22}Na distribution (% of absorbed)	
Plant part	corn	barley
Roots[a]	98.1	65
Stem base, 0–30 cm	0.8	10
Stem base, 30–70 cm	0.6	5
Rest of stem and leaves	0.5	20
Total export from roots	1.9	35

[a] Washed in 10 mM $CaSO_4$.

Na^+/H^+ antiport, because stelar cell plasma membrane ATPases secrete protons into the xylem [119], and the proton gradient is in the wrong direction—as evidenced by the relative acidity of the xylem sap. Lacan and Duran [120] suggested that the absorption of Na^+ from the ascending sap is primarily accomplished by indirect K^+-Na^+ exchange; namely, reverse H^+/Na^+ antiport (against the proton gradient) linked to K^+/H^+ antiport (with the proton gradient) and anion-proton symport to the symplast of cells bordering the xylem. They hypothesize that the process is primarily driven by a proton gradient resulting from proton pumping into the xylem by adjacent cells. This proton gradient is then utilized for K^+ transport to the xylem by H^+/K^+ antiport and for H^+-anion symport. The latter proton movements decrease the cytosolic pH of stellar cells lining the xylem and facilitate H^+/Na^+ antiport and Na^+ depletion of the xylem. The assumption of indirect Na^+-K^+ exchange was supported by the absence of a fixed stoichiometry between K^+ and Na^+ transport [121] and by the pH sensitivity of the Na^+/K^+ exchange. Also, increased xylem K^+ concentration resulted in decreased K^+ extrusion but not in decreased Na^+ uptake. The investigators do not provide direct evidence for reversed H^+/Na^+ antiport. Indeed, the proposed indirect Na^+/K^+ exchange could be sustained by K^+/H^+ antiport, as suggested, and electrophoretic Na^+ transport via cation channels (uniport). Such Na^+ transport would depend on the negative E_M of the living cells sorrounding the xylem, and hence on proton pumping as suggested in Lacan and Durand's [120] model. A previous proposal for reversed H^+/Na^+ antiport [122], cited by Lacan and Duran [120], concerns cells acidified by propionic acid.

Sodium recirculation has been found to contribute to salt resistance in many plants such as reed [123], the relatively salt tolerant soybean variety Lee [124], castor bean [125], trifoliate orange [126], *Trifolium alexandrinum* [127], *Atylosia albicans*, and *A. platicarpa* [98].

Chloride Toxicity

Chloride is the prevalent anion accompanying Na^+ and K^+, hence its concentration in vacuoles, as well as cytoplasm, is usually in the same range as the sum of Na^+ and K^+. This concurrence of Na^+ and Cl^- complicates the evaluation of Cl^--specific toxicity. Only a small number of experiments have been published that attempt to determine the direct toxicity of Cl^-, and their interpretation is not straightforward. Leopold and Willing [128] exposed soybean cotyledonary leaf slices to different salts and determined their effect on membrane integrity by measuring the subsequent leakage of organic solutes into water. They found a 28% increase of leakage when 133 mM Na_2SO_4 was replaced with near-isotonic (200 mM) NaCl. These results may be explained as a specific Cl^- toxicity, but Cl^- concentration was higher than that of SO_4^{2-}, and absorption as well as subsequent internal Cl^- concentration may have been much larger than that of SO_4^{2-}. In other experiments by Meiri et al. [129], 96 mM NaCl was less detrimental to the growth of bean plants than 72 mM (isotonic) Na_2SO_4.

Greenway and Munns [130] compared Na^+ and Cl^- contents in the leaves of seven salt-tolerant and salt-sensitive varieties or subspecies. In four of these plants, tolerance was related to lower contents of Na^+ as well as Cl^-. In two cases, there was little difference in concentration of either ion, or there was some increase in the concentration of both ions in the tolerant plants. In one case (avocado), a large decrease of Na^+ concentration was found in the tolerant variety but no difference in Cl^- concentration. In summary, these data do not indicate, that high Cl^- concentration in the leaves may have been related to sensitivity in any of the cases. A similar conclusion may be drawn from the comparison of Na^+ and Cl^- contents in salt-tolerant and salt-sensitive corn varieties [131] and *Atylosia* species [98]. In both cases, Na^+ and Cl^- were excluded from the leaves of the tolerant varieties and species, but exclusion was much more efficient for Na^+ than it was for Cl^-. Furthermore, in some salt-sensitive species, such as *Phaseolus coccineus* [107] and *P. vulgaris* [104,105], Na^+ was found to be excluded from the shoots but not Cl^-.

The growth rate of castor bean at different salinities [125] was not related to Cl^- content of the leaves but rather to Na^+ content. The growth rate was not affected by external NaCl up to 70 mM and decreased by about 80% at concentrations between 80 and 160 mM. Chloride

content of the leaves increased linearly with external NaCl concentration, whereas Na^+ was excluded from the leaves (up to 70 mM NaCl outside), and its leaf content was correlated with growth inhibition.

Although the cited experiments indicate that many salt-tolerant species can deal with higher Cl^- than Na^+ contents in the shoots, a greater Na^+ than Cl^- toxicity in the cytoplasm cannot be deduced. The apparently greater Cl^- than Na^+ tolerance may result from different capabilities for compartmentation of these ions in the vacuoles. All plants seem to be able to accumulate Cl^- in the vacuoles of their cells, whereas many are deficient in the Na^+/H^+ antiporter needed for Na^+ occlusion in the vacuoles [72].

SALT SECRETION

The transpiration stream continuously carries salts to plant shoots. Large amounts of salt should hence be delivered to the leaves of plants growing in a saline environment if the salts are not excluded from the shoots. Even in halophytes that accumulate Na^+ and Cl^- in their leaf cells, the amount of salt carried to the shoot is much in excess of that needed for turgor regulation. Secretion by special salt glands is one important mechanism for the removal of excess mineral ions from the leaves [132].

Structure of Salt Glands

The structural details of various kinds of plant salt glands (Fig. 8) were recently reviewed [133,134]. Based on their structure, three principal types of salt glands may be distinguished: two-celled glands of the grasses, multicellular glands of various dicotyledonous plants, and bladder hairs of the *Chenopodiacea*. The glands eliminate salts to the leaf surface, whereas bladder hairs eliminate them to the central vacuole of the bladder hair.

Some unifying principles in the structure of the different kinds of salt glands may be summarized. They all contain one or more subtending cells that are in apoplastic as well as symplastic continuum with both the adjacent mesophyll and the distal, secreting gland cells. These subtending cells are the basal cells in the two-celled glands, the innermost secretory cells in multicellular glands, and the stalk cells in bladders (see Fig. 8). The exterior walls of secretory cells in all salt glands are covered by a cuticle. The cuticle extends inward along the lateral walls of the external gland cells but not into the walls between the secretory and basal cells. In glands that excrete to the leaf surface, the cuticle is continuous with that of the epidermal cells and partially detached from the exterior walls of the secretory cells. The space formed between the detached cuticle and the walls forms a collecting compartment for the excreted solution. Small pores occur in the detached portion of the cuticle in all glands examined except those of *Aegiceras corniculatum* [135].

Pathway of Salts

As pointed out, structural investigations reveal the existence of an apoplastic continuum from the mesophyll to the subtending gland cells in all three types of glands. The availability of this route to solute transport was shown with the aid of La^{3+} [136,137]. This ion is able to move in the apoplast with the transpiration stream but is unable to penetrate into the symplast. The ion is visible as a precipitate in the electron microscope [138].

The existence of a symplastic continuum between the mesophyll and the gland cells suggests that symplastic flow can also occur. Cytochemical studies utilizing silver precipitates of Cl^- show the presence of Cl^- in plasmodesmata connecting the mesophyll and proximal gland cells of *Limonium* [139] and *Tamarix* [140]. Campbell and Thomson [140] concluded that salt moves to the salt glands apoplastically as well as symplastically, but the predominant route was probably the apoplast.

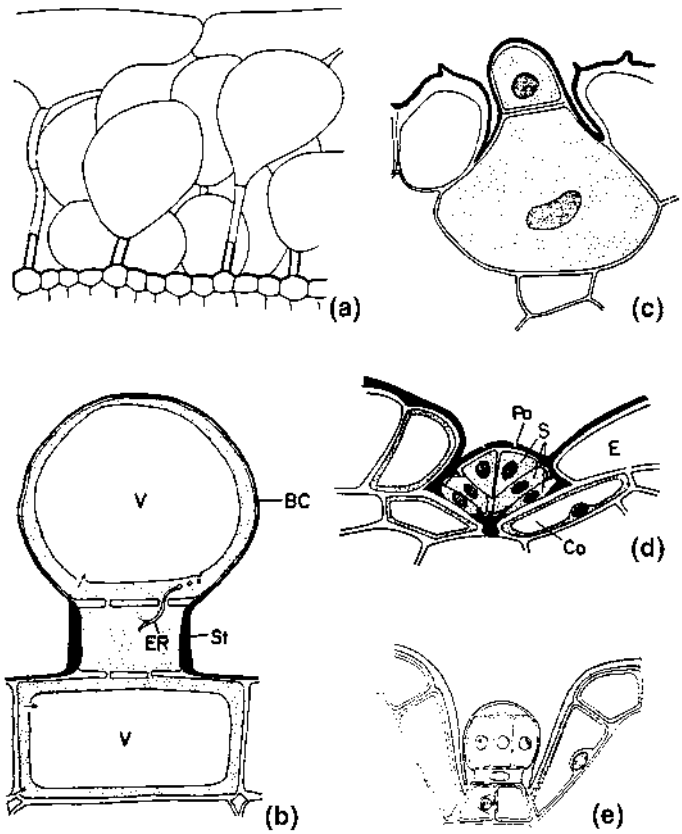

FIGURE 8 Salt glands (a,b) *Atriplex halimus*. L. (a) Epidermis and bladder hairs; the lateral walls of the lowest stalk cell are completely cutinized. (b) Diagram of a bladder hair showing possible routes of chloride transfer to the bladder cell and its vacuole. Arrows indicate active transport through membranes. One vesicle is seen fusing with the bladder tonoplast. BC, bladder cell; ER, endoplasmic reticulum; St, stalk cell; V, vacuole. (c) *Spartina townsendii*. H. and J. Groves. (d) *Tamarix aphylla*. Co, collecting cell; E, epidermal cell; Po, pore in the cuticle; S, secretory cell. (e) *Avicennia marina*. (From Ref. 134.)

Function of Salt Glands

The qualitative composition of salts secreted by glands was usually found to be similar to that of the native environment [141] or the culture solution [142]. However, the proportions and concentrations of the various ions are different. Selectivity, therefore, occurs at some site in the path from the roots to the glands. Different orders of mineral-ion selectivity have been reported for different plants [143–145].

Ionic concentration and Ψ_π of solution secreted by salt glands were found to be higher than those of the root medium or the challenging solution in experiments with excised leaves or leaf tissues [146–149]. Similarly, Mozafar and Goodin [150] found higher NaCl concentrations in the bladder hairs of *Atriplex* than in the medium. The salt concentration of the secreted solution was also found to be higher than that in the xylem sap [141,143]. These concentration gradients indicate the involvement of a metabolic energy–dependent process in secretion. This was explicitly demonstrated by Arisz et al. [146], who measured the effect of light and inhibitors of energy metabolism on

salt secretion by *Limonium* leaf disks. The requirement for a metabolic energy source [147,151,152] and the involvement of the PM H^+-ATPase [153] were confirmed by other investigators.

Thomson et al. [133] proposed two possible mechanisms for secretion by salt glands. One proposal assumes symplastic transport to the secreting cells and metabolic energy–dependent secretion of the respective ions to the collecting chamber or vacuole in bladder hairs. Water movement should follow this salt secretion and expand the collecting chamber. This expansion is supposed to open the cuticular pores and enable outflow of the solution. The second proposal assumes apoplastic flow of solution to the subtending gland cells and metabolic energy–dependent accumulation of the respective ions by the latter cells. The ions are then supposed to move down their electrochemical potential gradient to the secreting cells.

The passive permeation of an accumulated salt solution from secreting cells to the collecting chamber could be regarded as a special case of turgor downregulation as described for the charophyte *Lamprothamnium* [93,154–156]. In this series of publications, turgor downregulation, in response to a hypotonic shock, was shown to be accompanied by depolarization and increased EC (electrical conductivity) of the plasma membrane in the involved cells. The presence of Ca^{2+} in the medium was needed for EC increase but not for depolarization. The proposed sequence of events is water influx from the hypotonic medium and turgor elevation; membrane depolarization; Ca^{2+} influx, apparently consequent to opening of Ca^{2+} channels; increased PM conductivity; and ion efflux accompanied by water. In the special case of salt glands, the initial water influx and turgor elevation would be induced by salt accumulation in the subtending gland cells.

SALT ADAPTATION

Suspension cultures and calli of plant cells have been adapted to NaCl by stepwise transfer to increasing salt concentrations. With this procedure, cell lines evincing enhanced resistance to salt have been isolated from various plants [45,52,157–163]. Dry weight production of some of the adapted cell lines, in the presence of salt, was similar to that of the wild lines in the absence of salt (Fig. 9) [158,161,164,165]. Such adapted cell lines may retain their resistance for many generations even after growth in the absence of salt [157,161,162,164].

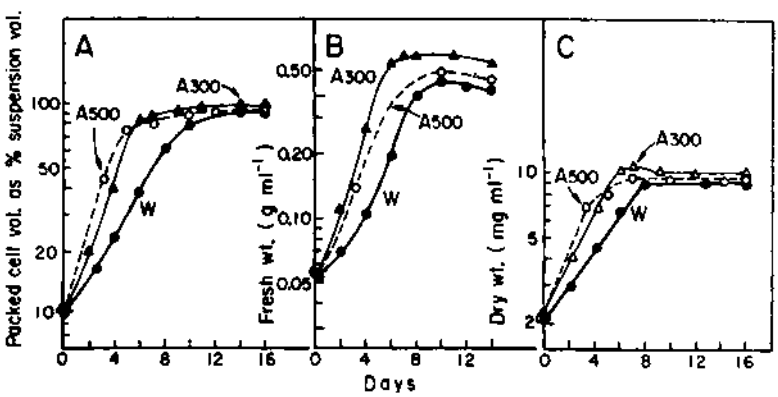

FIGURE 9 Growth curve (dry weight) for various NaCl adapted lines of *Nicotiana* cells growing in media to which they were adapted; also for wild-type cells growing in standard medium and in 0.3 M NaCl (closed circles), wild-type cells in standard medium and (open circles) in 0.3 M NaCl; (closed triangles), cells adapted to 0.3 M NaCl in 0.3 M NaCl; (open triangles) cells adapted to 0.4 M NaCl in 0.4 M NaCl; (open squares) cells adapted to 0.5 M NaCl in 0.5 M NaCL. Growth measured as packed cell volume (A); fresh weight (B); and dry weight (C) (From Ref. 164.)

Increased salt tolerance of salt-adapted cultured cells has rarely led to increased salt tolerance in normal regenerated plants [160,165,166]. Selected cultures, however, are systems where nearly isogenic cells differ, at least in theory, only in the desired tolerance trait [167]. Cell cultures and stress-adapted cell lines from such cultures provide a convenient tool for elucidating salt-resistance mechanisms at the cellular level.

Both of the strategies employed by intact salt-resistant plants can be found in salt-adapted cell lines. Thus, in the presence of salt, tolerant cell lines of *Citrus* [162] and potato [163] more efficiently excluded Na^+ and prevented the decrease of K^+ content than unadapted lines. In cultured *Citrus sinensis* cell lines, the most pronounced characteristic of adapted cells was indeed their capability for larger accumulation of K^+ [168]. A similar trait was reported for NaCl-selected alfalfa cell lines [160]. On the other hand, in tobacco cell lines, salt tolerance was associated with a decrease in K^+ content in concert with increasing salinity [159,169], and an increase of Na^+ [159,169] as well as Cl^- [169], as principle solutes for turgor regulation. Organic compounds also accumulated with salinity, in particular, proline [159,169] and sucrose [169]. Sodium and Cl^- were occluded in the vacuoles of adapted tobacco cells. In cells adapted to 428 mM NaCl, the vacuolar contents of Na^+ and Cl^- were 780 and 624 mM, respectively, whereas cytoplasmic concentrations were maintained at 96 mM [170].

Abscisic acid (ABA) accelerated the adaptation of cultured tobacco cells to high salt concentrations [171]. Abscisic acid, as well as exposure to salt, enhanced the synthesis of a number of proteins [172]. The most striking effect of both treatments on previously unadapted cells was induction of the synthesis of a cross-reactive 26-kDa protein. This protein appeared to be associated with adaptation. When induced by ABA, it was transient unless the cells were simultaneously exposed to salt. Salt-induced changes in the amounts of several proteins were also reported for salt adapted *Citrus* and tomato cell lines [167].

Salt adaptation was also accomplished with whole plants. Eight-day-old *Sorghum* seedlings could be adapted to high salinity by growth in 150 mM NaCl for 20 days [173]. At that time, NaCl could be increased to 300 mM without an effect on the relative growth rate and dry weight produced. The adaptative treatment (150 mM NaCl), however, decreased shoot dry weight production by about 70% as compared with unsalinized control plants. The salt adaptation of *Sorghum* plants was accompanied by an increased capability to exclude Na^+ [173] and an increase in phosphoenolpyruvate carboxylase activity [174]. Treatments with 40 mM ABA increased the growth of salt-treated *Sorghum* seedlings and inhibited the growth of the controls. Abscisic acid also accelerated the adaptation of *Sorghum* plants [174] similar to its effect on the salt adaptation of cultured tobacco cells [171]. The time needed for adaptation of *Sorghum* plants in the presence of 150 mM NaCl was decreased by ABA from 20 to 10 days [174].

CONCLUSIONS

Salt-resistant plants have to maintain growth in the presence of an osmotic stress and, concomitantly, avoid high salt concentration in their cytoplasm. Growth is primarily maintained by an increase of the amount of solutes in the cells and by subsequent turgor regulation. This mechanism may be supplemented by increased cell wall plasticity and decreased threshold turgor. The turgor decrease is sensed by a ''turgor sensor'' apparently in the plasma membrane. The sensor emits an ''error signal'' that is transduced to the activation of adaptive processes.

Salt toxicity is avoided by employing compatible solutes for osmotic cytoplasm adjustment and by confining salt, in particular Na^+, to the vacuoles. Some plants excrete Na^+ from the cytoplasm by active Na^+/H^+ antiport into the vacuole and also to the apoplast. The leaves of such plants may also contain salt glands. These glands accumulate excess salts and subsequently excrete it. This excretion may be explained as a special case of turgor downregulation. Other plants that apparently lack the Na^+/H^+ antiporter accumulate organic solutes and K^+ salts; they prevent Na^+ influx to the roots and its translocation to the more sensitive shoots. The latter is accomplished by selective Na^+ absorption from the ascending xylem sap and its recirculation to the roots via the phloem.

Sodium ions permeate into plant cells through outward rectified cation channels that apparently open in response to Na^+-induced depolarization. The presence of Ca^{2+} and K^+ enhances Na^+ exclusion by controlling channel selectivity. High potassium concentration in the medium also ensures its adequate supply to the plant in the presence of excess Na^+.

Some plant tissue cultures and intact plants can be adapted to salinity. The same strategies for maintaining growth employed by salt tolerant plants can be induced in response to adaptation.

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