Cellular Mechanisms of Salinity Tolerance

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Salinity is a significant limiting factor to agricultural productivity, impacting about $9 \times 10^6$ ha of the land surface on the earth, an area about 3 times greater than all of the land that is presently irrigated (17, 18). Reduced productivity occurs as a result of decreased yields on land that is presently cultivated (about one-third of all irrigated land is considered to be affected by salt (18, 45)), as well as due to the restriction of significant agricultural expansion into areas that presently are not cultivated. In the United States, salinity is a major limiting factor to agricultural productivity, and as the quality of irrigation water continues to decline this problem will become more acute (1, 56). About 1.8 million ha of land are salt-affected in California (56), the major agricultural state in the nation. Annual losses to crop production in the salt-affected areas, including the Imperial, Coachella, and San Joaquin valleys, are substantial and are increasing at a significant rate each year (56).

Problems of salinity may be addressed using technological and/or biological approaches (18, 61, 68). Technological approaches to cope with salinity include advances in water and soil management (61), irrigation methodology (61), and perhaps desalinization (21). Biological approaches include the identification of halophytes that are potential crop plants and, if necessary, the introgression of more desirable horticultural or agronomic traits into them (57); the introgression of the genetic background from saline-tolerant wild species into cultivated plants (56, 67, 68, 78, 79). These effects can result in tolerance of plants to moderate or low levels of salinity (2, 51, 52). Today, great effort is being directed towards the development of salt-tolerant crop genotypes through the use of plant breeding strategies involving the introgression of the genetic background from saline-tolerant wild species into cultivated plants (56, 67, 68, 78, 79).

The detrimental effects of salinity are due to the influence of ions on the water activity of the external solution, which affects the water status of the plant, and/or to the direct effects of the ions on the physiological and biochemical functions of the cell (14, 20, 22, 85). These effects can result in turgor reduction, inhibition of membrane function or enzyme activity (20, 22, 85), inhibition of photosynthesis (66, 81), induction of ion deficiency due to inadequate transport/selectivity mechanisms (34), or increased use of metabolic energy for nongrowth processes involved in the maintenance of tolerance (62, 89).

Tolerance mechanisms used by plants to adapt to salinity can be separated into those that allow the growing cells of the plant to avoid high ion concentrations and those that permit the cells to cope with high ion concentrations upon exposure to salt (20, 22, 85). Salt avoidance mechanisms involve exclusion at the root (22, 40), absorption by xylem parenchyma cells (40), xylem-phloem exchange systems (20, 22, 40, 85), distribution of ion gradients between nongrowing and growing portions of the plant (85), and, in the case of halophytes, sequestration of ions into salt glands or trichomes (20). In general, exclusion mechanisms are effective at low to moderate levels of salinity, while ion accumulation is the primary mechanism used by halophytes at high salt levels, presumably in conjunction with the capacity to compartmentalize ions in low to moderate levels of salinity, while ion accumulation is the primary mechanism used by halophytes at high salt levels, presumably in conjunction with the capacity to compartmentalize ions in

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In recent years, cell lines have been isolated that exhibit enhanced tolerances to salinity (4, 6, 10, 12, 15, 24, 31, 32, 54, 60, 64, 83, 84, 88). In addition, salinity tolerance of plants that is based on cellular mechanisms is often exhibited by cell suspensions of these plants (58, 71, 78, 82). Salt-tolerant cell lines that are stable in the absence of stress can be obtained, and salinity tolerance exhibited by cells in vitro has been shown to be transmitted to regenerated plants (9, 54, 88). The fidelity of transmission of salinity tolerance from isolated cells to regenerated plants is dependent on several factors and only recently have we been able to develop an understanding of the complexity of the relationship between salt tolerance at the cell and at the whole-plant level (9).

Fig. 1. Average volume of tobacco cells adapted to 0, 10, 14, 20, 25, 30, 35, 40, and 42.5 g·liter−1 NaCl at stationary phase of growth.

Table 1. Area of fully expanded leaves of tobacco (Nicotiana tabacum L. ‘Wisconsin 38’) plants. Shown is the average area ± se of five leaves from each of five different plants except where noted.

<table>
<thead>
<tr>
<th>Plant type</th>
<th>Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wisconsin 38 (W 38) seed-derived</td>
<td>493 ± 26.5</td>
</tr>
<tr>
<td>Regenerated from unadapted cells</td>
<td>423 ± 20.9</td>
</tr>
<tr>
<td>Regenerated from cells adapted to 25 g·liter−1 NaCl (S-25)</td>
<td>63 ± 9.4</td>
</tr>
<tr>
<td>Regenerated from cells adapted to 30 g·(100 ml)−1 PEG</td>
<td>47 ± 9.4</td>
</tr>
<tr>
<td>(S-25 regenerated × W 38 seed-derived) sel²</td>
<td>546 ± 44.6</td>
</tr>
<tr>
<td>(S-25 regenerated × W 38 seed-derived) sel²</td>
<td>107 ± 9.2</td>
</tr>
</tbody>
</table>

* Single plant.
Table 2. Tobacco cell lines growing in medium without NaCl (S-0) or in media with 10 (S-10), 14 (S-14), 20 (S-20), 25 (S-25), 35 (S-35), 40 (S-40), and 45 (S-45) g-liter\(^{-1}\) NaCl. Concentrations of NaCl (g-liter\(^{-1}\), mM) and water potential (\(\psi_w\)) values are for media prior to the inoculation of cells. Listed are the number of cell generations that the respective cell lines have been maintained at that level of NaCl.

<table>
<thead>
<tr>
<th>Cell line</th>
<th>NaCl (g-liter(^{-1}))</th>
<th>NaCl (mM)</th>
<th>(\psi_w) (bar)</th>
<th>Cell generations</th>
</tr>
</thead>
<tbody>
<tr>
<td>S-0</td>
<td>0</td>
<td>0</td>
<td>-6</td>
<td>&gt;150</td>
</tr>
<tr>
<td>S-10</td>
<td>10</td>
<td>171</td>
<td>-14</td>
<td>&gt;150</td>
</tr>
<tr>
<td>S-14</td>
<td>14</td>
<td>240</td>
<td>-17</td>
<td>&gt;150</td>
</tr>
<tr>
<td>S-20</td>
<td>20</td>
<td>342</td>
<td>-23</td>
<td>&gt;150</td>
</tr>
<tr>
<td>S-25</td>
<td>25</td>
<td>428</td>
<td>-27</td>
<td>&gt;150</td>
</tr>
<tr>
<td>S-30</td>
<td>30</td>
<td>513</td>
<td>-31</td>
<td>&gt;100</td>
</tr>
<tr>
<td>S-35</td>
<td>35</td>
<td>590</td>
<td>-35</td>
<td>60</td>
</tr>
<tr>
<td>S-40</td>
<td>40</td>
<td>685</td>
<td>-39</td>
<td>20</td>
</tr>
<tr>
<td>S-45</td>
<td>45</td>
<td>770</td>
<td>-43</td>
<td>5</td>
</tr>
</tbody>
</table>

Fig. 2. Scanning electron micrographs of cross sections of leaves of tobacco (*Nicotiana tabacum* L. 'Wisconsin 38') plants (A) regenerated from unadapted cells, (B) obtained from seed of 'Wisconsin 38', (C) regenerated from cells adapted to 25 g-liter\(^{-1}\) NaCl, and (D) regenerated from cells adapted to 30 g-(100 ml)\(^{-1}\) polyethylene glycol.

levels of adaptation, tolerance of the cells through the culture growth cycle did not change (6).

Adaptation to NaCl was accelerated by the exogenous addition of Ca\(^{2+}\) (increased above the basal 3 mM level), proline, or abscisic acid (ABA, ref. 39) into the medium. The effect of ABA is apparently specific to osmotic stress induced by ions. ABA promotion of growth was observed if other salts were used to impose the stress; however, no ABA effect was observed if similar water deficits were imposed by sucrose, sorbitol, mannitol, or polyethylene glycol (39). ABA only enhanced the growth of cells that underwent a significant turgor reduction. Growth of unadapted cells in the absence of salt or salt-adapted cells in the level of NaCl to which they were adapted was not stimulated by ABA treatments (39).

Both maximum fresh and dry weight gain of the cell cultures adapted to NaCl could be enhanced by increasing the sucrose (30) or Ca\(^{2+}\) concentrations in the medium above the standard 87 mM and 3 mM, respectively. Fresh and dry weight gain that resulted from higher sugar levels was due to an increase in cell division and not to an increase in cell expansion. The promotion of growth fa-

Fig. 3. Fresh weight gain, in grams per 25 ml of culture medium, of unadapted tobacco cells in media (A) without and (B) with 10 g-liter\(^{-1}\) NaCl after inoculation at different fresh weight densities, in milligrams per milliliters of culture medium: 2, ●; 4, ○; 8, ▲; 16, △; and 24, ■.

cilitated by Ca\(^{2+}\) may be due to the maintenance of adequate Ca\(^{2+}\) binding to the plasma membrane (11, 37) preserving membrane function, i.e., facilitating Na\(^{+}\) discrimination and prevention of K\(^{+}\) leakage (11, 16, 37, 41).
Stability of the salt tolerance phenotype

From the literature it is difficult to conclude if in vitro selection for salt tolerance will result in the isolation of cells that express a stable phenotype (4, 6, 10, 12, 15, 24, 31, 32, 54, 60, 64, 83, 84, 88). Whether the salt tolerance exhibited by cell populations is due to physiological adaptations (47) or to enrichment of the cell population for preexisting, more-tolerant cells remains uncertain (9, 47). Tobacco cells exposed to moderate levels of NaCl (171 mM) failed to express tolerance in a stable manner when NaCl was absent (9). However, cells growing in high levels of NaCl (428 mM) retained about 40% of the tolerance they exhibited in the presence of NaCl when grown in the absence of the salt.

The unstable tolerance of cells adapted to 171 mM NaCl is indicative that apparently only physiological adaptations occurred despite the considerable variability for salt tolerance among cell clones in the unadapted cell population (9). Even though physiological adaptations appeared responsible for about 60% of the tolerance exhibited by cells grown in 428 mM NaCl, enrichment of the population for more tolerant cell types did occur when the stress exposure level was this high. Perhaps this population enrichment occurred as a result of selection for stable variants (25, 47) or was due to an environmentally driven stable genomic change (48, 70, 87) that was either specific for salt tolerance or occurred as a result of selection among a population of cells that exhibited an increased mutation frequency.

Certain clones, obtained from the cell population adapted to 171 mM NaCl and maintained in NaCl, exhibited levels of tolerance equivalent to clones obtained from the cell population adapted to 428 mM NaCl and maintained in the absence of NaCl (9). Thus, although the same degree of tolerance that occurs by stable expression of the tolerant phenotype can be achieved by physiological adaptations, an advantage is conveyed to cells that express the tolerant phenotype in a stable manner, which results in their enrichment in the cell population. Perhaps this advantage is linked to an inherently slower growth rate that these cells exhibit even in the absence of NaCl.

Based on our methodology, it appears that selection of salt-tolerant cells that exhibit a stable phenotype occurs more as a result of the degree of selection pressure to which the cells are exposed rather than the length of time to which the cells are exposed to the selection pressure (9). However, this conclusion is by no means unequivocal. In addition, other selection protocols appear to have led to contrasting results (54, 83).

Regeneration of salt-tolerant plants

Plants regenerated from salt-adapted cells growing in high levels of NaCl (428 mM) were markedly dissimilar from plants regenerated from the unadapted cell population or grown from seed (9). Plants regenerated from unadapted cells exhibited a high degree of morphological variability, reportedly attributable to some degree to aneuploidy that occurs in culture after various periods of time (47). However, the plants regenerated from cells adapted to 428 mM NaCl were morphologically similar, yet quite different from the normal ‘Wisconsin 38’ phenotype or any of the various other phenotypes observed in the more than 200 plants regenerated from unadapted cells. Major differences included reduced growth rates, reduced leaf area, increased leaf succulence, short internodes, male sterility, vivipary, and hexaploidy or aneuploidy close to the hexaploid genomic complement. Interestingly, some of these characteristics are typical of plants exposed to saline environments (43, 44, 74), despite the fact that these plants were regenerated in the absence of salt and had not been exposed to NaCl.

Shoots regenerated from salt-adapted cells were more able to survive exposure to extremely high levels of NaCl than shoots regenerated from the unadapted cells (9). However, shoots regenerated from salt-adapted cells and exposed to NaCl did not exhibit greater absolute growth in the presence of salt than did shoots regenerated from unadapted cells. Thus, it appears that exposure of the cells to high levels of NaCl results in genetic changes that allow plants regenerated from these cells to better survive the salt, but does not favor active growth of the plants either in the presence or absence of the stress. This finding is consistent with the view that osmotic adjustment is a survival mechanism rather than a mechanism that allows enhanced growth rates in the presence of stress. Although genetic changes that lead to a reduction in plant growth could enhance survival under drought conditions, this adaptation would appear less necessary under conditions of salinity (6). It is not clear if reduced growth under salinity stress is a requisite for increased tolerance. However, if it is, then this would have considerable impact on strategies to cultivate saline soils.

Water relations characteristics of salt-adapted cells and cells adapting to NaCl

Cells exposed to 171 mM NaCl either lost viability or underwent plasmolysis in order to establish water potential equilibrium (Fig. 4). Thus, the initial decrease in fresh weight observed upon immersion into NaCl medium was due both to plasmolysis as well as a loss of cell viability (Fig. 4). The half-time of $^{36}$Cl$^{-}$ efflux from these cells was considerably shorter, indicating greater membrane permeability for Cl$^{-}$ and perhaps reflecting general membrane leakiness. However, sufficient membrane integrity was maintained that plasmolysis occurred.

For the viable cells, osmotic adjustment commenced rapidly, as detected initially by changes in the percentage of plasmolyzed cells and later by measurement of osmotic potential by plasmometry (Fig. 4). Growth commenced after the initiation of osmotic adjustment and the maximum fresh weight gain and stationary phase water relations were similar to those of cells adapted to 171 mM.

Over a range of external water potentials from -6 to -27 bar,
The salt-adapted cells exhibited significantly reduced osmotic potentials (6). The osmotic potential was related to the external water potential as a logarithmic function; thus, cells exhibited higher average turgors at higher levels of adaptation.

Water relations of cells in culture appeared to be characteristic of cells in the plant as they make the transition from being meristematic to being highly differentiated. After inoculation, the cells began osmotic adjustment, which continued during the initial stages of the growth cycle, a time that coincided with the period of rapid cell division (ref. 6 and Figs. 4 and 5). During this phase of growth, the cells underwent only limited and predominantly isodiametric cell expansion and individual cells in the population became smaller with larger cytoplasm to vacuole ratios. Subsequent growth was due primarily to directional cell expansion (77), and much of the turgor that accrued as a result of the osmotic adjustment (which occurred during the rapid cell division phase) was dissipated (6). The extent of osmotic adjustment during initial stages of the growth cycle and the amount of turgor used during cell expansion increased with the level of adaptation. These data indicate that turgor maintenance is not restricting the expansion of the salt-adapted cells and suggest that major changes in the extensibility properties of the wall and/or turgor are either a part of this adaptation process or a consequence of adaptation.

Osmotic adjustment and solute accumulation

Osmotic adjustment of salt-adapted cells was due in large part to accumulation of Na⁺ and Cl⁻, which occurred against a concentration gradient (5). Na⁺ and Cl⁻ accounted for about 30% of the osmotic adjustment that was observed and 65% of the total solutes that were measured in cells adapted to 428 mM NaCl. Physiologically adequate intracellular levels of K⁺ appeared to be maintained regardless of the external Na⁺ level (5).

Numerous organic solutes accumulated to significant levels in salt-adapted cells (5). Levels of sugars, free amino acids, and proline increased with the level of adaptation. Proline accumulation was correlated with osmotic potential both in the presence or absence of osmotic stress, suggesting that proline accumulation does not initiate salinity adaptation but may itself be triggered to occur as a result of the initiation of other responses to salinity stress.

However, this does not preclude a significant role for proline in salinity tolerance. Levels of quaternary ammonium compounds and organic acids were not appreciably different among the cell lines. Although the levels of any individual organic compound did not increase to high enough levels in the whole cell to suggest their significant involvement in whole cell osmotic adjustment, little is known about the compartmentation of these solutes. Should these solutes be compartmentalized in the cytoplasm, their intracellular contribution to osmotic adjustment could be very significant. In addition, compounds such as proline and quaternary ammonium compounds are thought to have other functions in salinity adaptation (i.e., maintenance of hydration shells around proteins despite the water activity of the cell sap), which may not require their presence in concentrations as high as those necessary for osmotic adjustment (26, 59, 65).

The contribution of Na⁺ to dry weight was constant at all levels of adaptation (5), indicating that the intracellular concentrations of Na⁺ at different external NaCl levels were related to the reduction in cell volume that occurred with increased salt adaptation (Fig. 1). Such a mechanism might have important implications, since this adaptation should result in an increased membrane area to volume ratio of these cells compared to unadapted cells, which could facilitate ion transport and maintenance of ion compartmentation in salt-adapted cells.

During the process of adaptation, the period of osmotic adjustment after plasmolysis (Fig. 4) was mediated through the accumulation of Na⁺, Cl⁻, sugars, amino acids, and proline. A transient increase in the levels of organic acids occurred upon imposition of the stress; however, no difference was observed after adaptation had occurred. ABA appeared to stimulate the rate of osmotic adjustment in the presence of NaCl primarily through effects on sugar and proline accumulation.

Gene product differences associated with salt adaptation

NaDodSO₄-polyacrylamide gel electrophoretic analysis of unadapted and salt-adapted cells indicated a number of differences in the polypeptide profile (69). Several polypeptides increased with the level of adaptation, with one polypeptide of 26 kDa representing a significant portion of the NaDodSO₄-soluble polypeptides in cells adapted to 428 mM NaCl. Despite the fact that overall protein synthesis was inhibited in cells growing in media with NaCl, the prev-
alance of synthesis of the 26-kDa polypeptide increased with the level of adaptation. The 26-kDa polypeptide was present also in cells adapted to polyethylene glycol (PEG)-induced water stress (23); thus, its occurrence appears to be associated with osmotic stress adaptation in general.

Partial digestion of newly synthesized polypeptides measured by pulse labeling with $^{35}$S indicated that a polypeptide that migrated at 26 kDa was synthesized in unadapted cells, but only at the later stages of the growth cycle (69). In contrast, the salt-adapted cells synthesized a 26-kDa polypeptide at the onset of rapid cell growth, particularly at high levels of salinity. The 26-kDa polypeptide from unadapted and salt-adapted cells were similar by immunological reactivity and by protease digestion fragments, but did not have similar isoelectric points. However, the 26-kDa polypeptide did not accumulate in unadapted cells, whereas this polypeptide constituted about 10% of total cellular protein present in cells adapted to 428 mM NaCl (69).

In vitro translation of poly(A)$^+$ RNAs from unadapted and salt-adapted cells resulted in similar translation products. However, poly(A)$^+$ RNAs from salt-adapted cells showed higher levels of 26-kDa polypeptide synthesis than poly(A)$^+$ RNAs from unadapted cells, suggesting either a greater abundance of the 26-kDa polypeptide message or greater efficiency of translation of this message (9). In vitro translation of the 26-kDa polypeptide mRNA from the salt-adapted cells was less affected by NaCl than was the translation of the 26-kDa polypeptide mRNA from the unadapted cells. The level of mRNA for the 26-kDa polypeptide appeared to be induced by ABA in the absence of osmotic stress. The in vitro translation of the ABA-induced poly(A)$^+$ RNAs was less inhibited by NaCl than the translation of those from unadapted cells.

The 26-kDa polypeptide was present in both a buffer-soluble fraction as well as a fraction that was extracted with nonionic detergents from the buffer-insoluble material, suggesting that the polypeptide may exist in two forms. Partial sequence determination has indicated that the 26-kDa polypeptide possesses a highly hydrophobic region, representing about 25% of the total protein.

Expression of the 26-kDa polypeptide gene(s) appears to be altered permanently after adaptation to salt has taken place. Enhanced accumulation of the polypeptide occurred in cells returned to medium without salt and in suspension cells obtained from plants regenerated from adapted cells.

**SUMMARY**

Isolated cell populations of a glycophytic species can be manipulated in vitro to behave like cells of a halophytic species (6). Cells of a glycophytic plant, after selection for growth in the presence of NaCl, gain dry weight to an equivalent or greater extent in the presence of NaCl than in its absence. This halophytic behavior is expressed when cells are maintained in the presence of salt and the extent of expression is dependent on the level of NaCl to which the cells are adapted. In the absence of salt, stability of the tolerance phenotype varies significantly, depending apparently on the level of NaCl to which the cells are adapted. At low to moderate NaCl levels, tolerance of the cells is due entirely to unstable physiological adaptations, while, at higher levels of NaCl, enrichment of the cell population for stable, tolerant cell variants also occurs.

Correlated with an increase in level of salt adaptation of the cells was a decrease in cell volume, suggesting that reduced cell expansion and/or cell volume are adaptive features in salinity tolerance. Reduced cell expansion was not a result of lack of turgor maintenance or the lack of C availability, as the cell turgor increased logarithmically with decreased external water potential (6) and increased C availability did not result in increased cell expansion (30). The relationship between cell turgor and volume at various levels of adaptation is indicative of significant changes that directly or indirectly affect the turgor threshold and/or extensibility properties of the cell (33, 77). Similar growth inhibition, despite turgor maintenance, has been observed after exposure to osmotic stress (13, 33, 46, 49, 50, 80) and have been related to changes in the cell wall properties (8, 35). The decreased capability of adapted cells to expand is reflected not only in their smaller average cell volume (Fig. 1), but also in a dramatically reduced fresh weight growth rate (6). Average growth rate was greatly reduced as the cells became adapted to NaCl and the osmotic potential decreased (Fig. 6). Growth rate continued to decrease as osmotic potential declined after adaptation to higher levels of NaCl.

Osmotic adjustment in the cell lines occurred as a result of the accumulation of Na$^+$ and Cl$^-$ with maintenance of adequate intracellular levels of K$^+$ and the accumulation of several organic solutes. The maintenance of adequate intracellular K$^+$ levels is indicative that K$^+$ deficiency is not responsible for the inhibition of cell expansion, and may indicate that membrane K$^+$/Na$^+$ selectivity adaptations have occurred in the salt-adapted cells to facilitate K$^+$ uptake (11, 34).

It is likely that Na$^+$ and Cl$^-$, which are accumulated intracellularly in salt-adapted cells, are compartmentalized to a great extent in the vacuole and that the organic compounds serve as compatible solutes in the cytoplasm (5, 17, 20, 22, 43, 85). The capability to accumulate and compartmentalize Na$^+$ and Cl$^-$ intracellularly might be indicative of membrane adaptations that have occurred in the cells that facilitate transport of ions and preserve selective membrane permeability. Although major differences in the activities of enzymes between halophytes and glycophytes in the presence of salt are not always discernable (22, 85, 86), it is clear from our results (i.e., in vitro translation) and those of others that the cytoplasmic metabolism of adapted cells may be more functional under conditions of salinity (9, 20). The capacity to sustain metabolic function in the presence of low to moderate levels of salinity could reduce the electrochemical gradient between the cytoplasm and vacuole necessary to maintain ion compartmentation and greatly reduce the energy necessary to facilitate such gradients.

Reduced cell volume may represent a significant adaptation contributing to salinity tolerance. The small salt-adapted cells may have a reduced demand for catabolic metabolism necessary for growth than the large unadapted cells, and perhaps more energy would be available for salt tolerance-maintenance processes (27, 61, 63, 89). The ratio of total dry weight production per gram of C consumed increased with the level of adaptation (6, 30) and the percentage of contribution of salts to the dry weight was higher in the salt-adapted cells, which may indicate that organic dry weight gain efficiency (grams of organic dry weight gain per gram of C consumed) is not the same. In addition, as the level of adaptation increased, the portion of the organic dry weight contributed by osmotic solutes was substantially increased (6). At this time, we are uncertain as to whether this different partitioning of C for salinity tolerance reflects any differences in maintenance coefficients or growth yield efficiencies (36, 62, 72, 73).

Reduced cell volume may also be advantageous since solute accumulation necessary for osmotic adjustment of a small cell would be energetically less costly relative to that of a large cell. This appears to be the case for salt-adapted cells, since the percentage of Na$^+$ and Cl$^-$ per dry weight is constant (5). In addition, the membrane surface to volume ratio of the salt-adapted cells is markedly different, which may help facilitate the transport of solutes into the various intracellular compartments. Restricted cell expansion associated with osmotic adjustment has been suggested as an adaptive mechanism to counteract reduced rates of solutes uptake (49, 80).

It is clear that as cells become tolerant to NaCl, numerous physiological and biochemical mechanisms function as significant adaptive processes in salinity tolerance. This conclusion is substantiated by the fact that numerous gene products increased as cells adapted to NaCl (69). It is not surprising that a number of these gene products are involved in salinity adaptation nor is it possible to assign precise functions to them. However, it is possible to speculate on some functions for these proteins based on the hormonal and biochemical processes that have been identified as being involved in salinity tolerance of these cells (e.g., osmotic adjustment, ion transport, and proline biosynthesis).
Literature Cited


