A ROLE OF CTA4 ATPASE IN ER STRESS PROMOTED BY ALUMINUM IN FISSION YEAST

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Endoplasmic reticulum (ER) stress is caused by disturbances in the normal functions of the ER, which is characterized by the inability to fold properly the proteins. The cell incapacity to respond to ER stress culminates in cell death which, when occurs in neurons, contributes to the neurodegeneration. However, the mechanisms by which ER stress leads to cell death remain unknown. Aluminum is proposed to act as neurotoxic agent although precise mode of its action is unclear. Using the fission yeast *Schizosaccharomyces pombe* as a model to study Al toxicity, we showed that yeast growth in the presence of 0.5 mM $AlK(SO_4)_2$ resulted in 2.0-fold increase in BiP, known ER stress marker. In addition, cellular membranes exhibited an increase in the expression of ER cation Cta4 ATPase. We previously demonstrate that Cta4 ATPase is required for Ca^{2+} homeostasis and that cells lacking Cta4p are sensitive to ER stress. Consistent with this idea, we analyze $^{45}Ca^{+2}$ transport in total membranes isolated from *S. pombe* cells treated with aluminum. The exposure of cells to Al resulted in $^{45}Ca^{2+}$ ATP-dependent increase. The data indicate that Al promotes ER stress, which modulates the activity of calcium transporters, and suggest that Cta4 ATPase protect cells against ER stress.

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