

EXPRESSION AND REGULATION OF A NOVEL STRESS PROTEIN, p66, INDUCED BY SALT STRESS

Kimiko Murakami-Murofushi

Department of Biology, Faculty of Science, Ochanomizu University
Ohtsuka 2-1-1, Bunkyo-ku, Tokyo 112, Japan

SUMMARY

Under high salt conditions, haploid myxoamoebae of a true slime mold, *Physarum polycephalum*, retracted their pseudopodia and changed the shape into disk-like form, and then differentiated into their dormant form, myxocysts. These morphological changes were associated with an apparent actin re-arrangement in the cell. A 66k-protein, p66, was induced prominently under stress conditions, and it was co-localized with actin filaments.

To know the biological function of p66, cDNA cloning was performed, and the effect of over-expression of p66 was observed. We used *Dictyostereum discoideum* instead of *Physarum polycephalum*, because of the lack of good expression vectors for *Physarum* cells. The cells that expressed about 40-times much p66 showed multinuclear phenotype and it suggests the involvement of p66 in the cytokinesis. But, the correlation of this function and stress response has not yet been clarified.

Salt stress and heat stress induced a rapid production of a certain glycolipid in the *Physarum* myxoamoebae. Structural studies of the purified glycolipid were done and this lipid was determined to be a poriferasterol monoglucoside. This substance was previously reported by us to be expressed during the differentiation of *Physarum* cells from myxoamoebae into plasmodia.

And in response to salt and heat stresses, calcium-dependent protein kinase activation was occurred. This enzyme activity was inhibited by staurosporine which is known as a potent inhibitor of tyrosine-type and serine/threonine-type protein kinases.

To clarify the early events in response to stresses, the biological significance of a glycosylation of membrane sterol and an activation of Ca²⁺-dependent protein kinase in the induction process of p66 should be studied, and these efforts may give valuable clues to resolve the molecular mechanisms of stress response of the cell.