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Heat shock response in *Synechocystis*

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Earlier we have shown, that changes in plasma membrane physical state may act as a primary low temperature sensor in *Synechocystis* (Vígh et al., 1993). Altering experimentally the molecular order of thylakoid membranes affected dramatically the temperature range over which genes of heat shock proteins (*groEL*, *cpn60*, *dnaK, hsp17)* are activated by heat evidencing that thylakoid membrane is an ideal location for primary heat stress sensors in cyanobacteria (Horváth et al., 1998). By using the desaturase deficient *Ole1*- mutant of *S. cerevisiae* supplemented with various desaturase constructions (Carratu et al., 1996**)** together with mammalian cells treated with various membrane perturbing agents further corroborated that the actual membrane physical sate is able to influence the threshold temperatures of heat shock gene activation.

Both the active protein-folding GroEL and HSP17 oligomers was shown to be able to associate with biomembranes and model lipid membranes, as well. Unlike GroEL, the lipid associated HSP17 revealed a highly altered protein refolding activity if tested in collaboration with DnaK/DnaJ/GrpE, GroEL/ES and ATP, in vitro. Binding of stress proteins to lipid matrix was shown to rigidify membranes (Török et al., 1997; Török et al., 2001) and thereby may rapidly stabilize them under heat stress before readjustment of lipid molecular species. In accordance with the "membrane sensor" hypothesis, increased membrane physical order caused by chaperone binding may also lead to a down-regulation of heat shock genes, simultaneously.

Our findings with *Synechocystis*, yeast and mammalian systems lend support to a model in which thermal stress is transduced into a cellular signal at the level of membranes (Vígh et al., 1998; Glatz et al., 1999). Investigations to explore the pathways for perception and transduction of temperature-stress signals are underway in our laboratory. Based on the recent discovery of "lipochaperones" we propose, that the specific and reversible interaction of some stress proteins with membrane lipids acts primarily as a powerful tool to rapidly regulate the membrane physical state (packing order, permeability and other attributes) and thereby preserve various membrane functions, especially under fluctuating stressed conditions.

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