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DISSERTATION SUMMARY

Small heat-shock proteins can stabilize heat-stressed membranes

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Small heat-shock proteins (sHSPs) are reported to protect (substrate) proteins against irreversible aggregation (van Montfort et al. 2002). However, growing evidence suggests that sHSPs are also involved in the maintenance of membrane integrity during stress, ageing or pathophysiological conditions (Török et al. 2001; Narberhaus 2002).

Our results indicate that sHSPs bind to lipids stabilizing the bilayer liquid-crystalline state affecting both the polar headgroup region and the hydrophobic core (Tsvetkova et al. 2002). Besides, the nature of sHSPs-membrane interactions largely depends on the lipid composition. Specific protein-lipid interactions seem to be operating in membrane reorganization paralleled with increased resistance to stresses (*e.g.* heat, oxidative, etc.).

Photosynthesis is one of the most heat sensitive cellular function in photosynthetic organisms. It is clearly emerging that cell membranes, when exposed to an abrupt increase in temperature, undergo an immediate reorganization through changes in the membrane physical structure (Horváth et al. 1998). In cyanobacteria membrane microdomain reorganization is paralleled with the appearance of highly saturated monoglycosyl diacylglycerol and the association of heat shock protein with the thylakoid.

Non-bilayer forming lipids (MGLDG, MGDG) are of striking importance in HSP17 interaction which shows a remarkable fluidity dependence underscoring the relevance of membrane hydrophobic interior in a protein-induced membrane reorganization.

By assuming that lipid phase of membranes act as cellular thermometer (Vigh et al. 1998) membrane association of sHSPs may constitute a feedback regulation of the expression of stress genes.

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