学術講演会

MEMBRANE PERTURBATIONS AFFECT THE EXPRESSION, CELLULAR LOCALIZATION AND FUNCTION OF CHAPERONE PROTEINS

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場所：理学部3号館**11番教室**

Cells that have been preexposed to low or high “priming” temperature treatment can acquire a transient resistance against the killing effect of a subsequent temperature stress. It is suggested, that specific membrane lipids and stress proteins play a fundamental role in these rapid acclimation processes. Our major goal is to understand the mechanisms, by which temperature stress is detected, quantified and transduced to the transcriptional apparatus.

Our studies on the unicellular cyanobacterium, *Synechocystis* PCC6803 strongly supported that stress proteins have moonlighting functions under stress conditions. They can not only assume the functions of assisting the protein folding, but concomitantly are able to stabilize membranes under heat stress.

Our results suggest, that cellular membranes are ideal location for primary heat stress sensors.

Cellular stress management is of great importance to our understanding of how cells respond and adapt to various changes in their environment especially during pathophysiological conditions. The classic heat shock (stress) response (HSR) was originally attributed to protein denaturation. However, induction of the heat shock protein (HSP) occurs in many circumstances where no protein denaturation is observed. The "Membrane Sensor Hypothesis" supported by our studies predict that the level and ratio of HSPs is affected by the alterations of the plasma membrane. Chemical compounds that modify the membrane ultrastructure are unique drug candidates because they may regulate HSP expression in diseased cells, without significantly affecting healthy cells.

ご来聴を歓迎いたします

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