埼玉大学　分子生物学科・環境科学研究センター共催セミナー
JSPS日本学術振興会二国間交流事業共同研究（H23/24）

日時：　2012年2月1日（水）15:00-16:00
場所：　理学部3号館・2階・11番教室

講師）　Attila Glatz博士
（ハンガリー科学アカデミー）

演題）　Membrane-associated gardedams: from cyanobacteria to fission yeast

- 概要 -

Molecular chaperones are known to interact with damaged proteins preventing their aggregation and promoting their folding. Using *Synechocystis* PCC6803 as a model, we were able to show that the chaperone genes (especially *hsp17*) is induced under isothermal conditions when cells were treated with membrane perturbing agents. This led to the “membrane as sensor” idea supposing that not only unfolded proteins, but changes in membrane’s physical state might induce the overexpression of chaperones. Moreover, Hsp17 is able to bind and protect membranes upon heat stress. Other chaperones (eg. GroESL) can also associate to membranes while retaining their intact “foldase” activity. Recently, we have started to study the chaperone systems of a “micromammal model” *Shizosaccharomyces pombe*. We could demonstrate that the two alpha-crystallin type-HSPs of the fission yeast are differently induced upon heat stress. In addition, both recombinant sHSPs tested are able to bind to model membranes made of *S. pombe* lipids and preferentially to lipid membranes derived from low temperature adapted cells, indicating that sHSP-membrane interactions might have stress protecting role in higher Eukaryotes as well.

問合せ：　理学部分子生物学科　仲本　準　内線4318

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講師）Imre Gombos博士
（ハンガリー科学アカデミー）
演題）Membrane as a heat sensor: Membrane imaging from heterogeneous cell populations to single molecules

-概要 –

The heat shock response (HSR) is one of the most ancient and evolutionarily conserved protective mechanisms found in nature. We demonstrated that not only heat induced protein denaturation can initiate HSR but cellular membranes are also act as sensitive thermal sensors even for mild heat stress. The present knowledge of gene expression and cellular responses is known to derive from analyses of heterogeneous populations. Although this approach provides useful insights into average population responses, they do not furnish information on individual cells or subpopulations. **In my presentation**: **1)** I will characterize the individual variability in the stress response of genetically homogeneous cell population with ultrasensitive high content imaging. More specifically, to link the population heterogeneity of the heat shock response and membrane structure (raft organization and dynamics) in mammalian cell cultures. The identification of specific changes in membrane domain structure leading to selective refinement of heat shock proteins in a heterogeneous cell population could help us to understand why a small subpopulation of cells could determine the outcome of important disease states. **2)** As an exploitation of the above principles, I will introduce the mode of action of a small molecule heat shock protein (HSP) co-inducer. I will discuss *in vitro* molecular dynamic simulation, experiments with lipid monolayers and *in vivo* ultrasensitive fluorescence microscopy, which showed that BGP-15 alters the organization of cholesterol-rich membrane domains. Imaging of nanoscopic long-lived platforms demonstrates that BGP-15 prevents the transient structural disintegration of rafts induced by fever-type heat stress and able to remodel cholesterol-enriched lipid platforms. These data indicate that BGP-15 has the potential to become a new class of pharmaceuticals for use in ‘membrane-lipid therapy’.

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