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RESEARCH AREA

As a "central dogma" earlier it was suggested that stress-induced protein denaturation serves as a major stress-sensing machinery, which triggers the expression of the molecular chaperone heat shock proteins (HSPs). We have introduced a new but not exclusive cellular "membrane thermosensor" model, which predicts the existence of membrane-associated stress sensing and signaling mechanisms. It proposes that changes in the physical state and composition of lipid molecular species with the concomitant destabilization/reorganization of membrane microdomains ("rafts") can serve also as "molecular switches" to operate "cellular thermometers". Using mammalian cells and the fission yeast *(S.pombe)* as models we intend to elucidate the mechanism of membrane-associated stress sensors, signaling pathways and the interplay and networking of potential cellular stress survival strategies. Since HSPs play a fundamental role in the pathology of several human diseases, understanding the mechanism whereby mammalian cells can elicit a stress response may also be of paramount importance for the design of novel drug molecules.

TECHNIQUES AVAILABLE IN THE LAB

Classical biochemical and molecular biology methods. Membrane biophysics: spectroscopy, Langmuir monolayers, ultrasensitive fluorescence microscopy, single molecule tracking, image analysis. Lipidomic analysis: chromatographic and mass spectrometry techniques. Multidimensional data analysis, statistical methods.

SELECTED PUBLICATIONS

Escribá, P.V., Busquets, X., Inokuchi, J.I., Balogh, G., Török, Z., Horváth, I., Harwood, J.L., **Vigh, L.** (2015) Membrane lipid therapy: Modulation of the cell membrane composition and structure as a molecular base for drug discovery and new disease treatment. **Prog Lipid Res 59:** 38-53.

Nagy, E., Balogi, Z., Gombos, I., Akerfelt, M., Björkbom, A., Balogh, G., Török, Z., Maslyanko, A., Fiszer-Kierzkowska, A., Lisowska, K., Slotte, P.J., Sistonen, L., Horváth, I., **Vigh, L.** (2007) Hyperfluidization-coupled membrane microdomain reorganization is linked to activation of the heat shock response in a murine melanoma cell line. **Proc Natl Acad Sci USA 104:** 7945-7950.

Vigh, L., Horváth, I., Maresca, B., Harwood, J.L. (2007) Can the stress protein response be controlled by ‚membrane-lipid therapy'? **Trends Biochem Sci 32:** 357-363.

Török, Z., Tsvetkova, N.M., Balogh, G., Horváth, I., Nagy, E., Pénzes, Z., Hargitai, J., Bensaude, O., Csermely, P., Crowe, J.H., Maresca, B., **Vigh, L.** (2003) Heat shock protein coinducers with no effect on protein denaturation specifically modulate the membrane lipid phase. **Proc Natl Acad Sci USA 100:** 3131-3136.

Vigh, L., Literáti, P.N., Horváth, I., Török, Z., Balogh, G., Glatz, A., Kovács, E., Boros, I., Ferdinándy, P., Farkas, B., Jaszlits, L., Jednákovits, A., Korányi, L., Maresca, B. (1997) Bimoclomol: a nontoxic, hydroxylamine derivative with stress protein-inducing activity and cytoprotective effects. **Nat Med 3:** 1150-1154.