

Protecting your Proteome: The Upside of Folding Under Stress

Kevin Strange, Ph.D.President
MDI Biological Laboratory **Salisbury Cove, Maine**

Friday, November **14**th **2014** 12:00-1:00 p.m.

Alfond 113 UNE, Biddeford Campus

Lunch will be provided

Hosted by: Edward Bilsky, Ph.D.

Sponsored by: The Office of Research and Scholarship



Dr. Kevin Strange became the MDI Biological Laboratory's first president in July 2009 after a distinguished career as an NIH-funded biomedical scientist and leader at Harvard Medical School and Vanderbilt University School of Medicine. Dr. Strange is leading a dramatic transformation of the Institution's biomedical research and educational programs. He began his tenure by refocusing the Institution's research program and recruiting a new generation of multidisciplinary scientists to

understand the genetic mechanisms of tissue repair, regeneration, and aging. These scientists are defining how diverse animal models rapidly replace damaged hearts and nervous systems and lost limbs, and how gene activity influences lifespan and the degenerative changes that occur during aging. Only three years after establishing this new research focus, the MDI Biological Laboratory was recognized by the National Institutes of Health as a center of excellence in regenerative and aging biology and medicine.

The maintenance of protein function or "proteostasis" is mediated by the tightly integrated and highly conserved activities of gene transcription, RNA metabolism and protein synthesis, folding, assembly, trafficking, disassembly and degradation. Protein structure is inherently unstable and is readily perturbed by numerous physiological and pathophysiological challenges including diverse environmental stressors. As aptly noted by Ghosh and Dill, "cells live on the edge of a proteostasis catastrophe" (Biophys J 99:3996-4002, 2010). In his seminar, he will discuss how they are using the nematode C. elegans to develop an integrated understanding of the genetic, molecular and cellular pathways by which animal cells manage protein damage induced by water stress, and how signals associated with proteostasis mechanisms activate cellular stress response pathways essential for survival.



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