PHYSIOLOGY & MEMBRANE BIOLOGY

SCHOOL OF MEDICINE

UNIVERSITY OF CALIFORNIA AT DAVIS

DISTINGUISHED SPEAKER SERIES

Donald W. Hilgemann, Ph.D.
Professor of Physiology
Roy & Christine Sturgis Chair in Biomedical Research
UT Southwestern Medical Center at Dallas

"New Perspectives on PIP_2 in cardiac myocytes"

Friday, November 7, 2008

10:00 am

Genome and Biomedical Sciences Facility **Auditorium**

Room 1005

Refreshments Will Be Served

PIP_2 is similar to GTP in being a metabolized cofactor in the regulation of numerous cellular processes. Direct effects of PIP_2 on ion channels and transporters have recently received much attention. But in heart, PIP_2 levels do not change much im response to hormones and activity changes. A provocative but unsubstantiated hypothesis is that local PIP_2 signals occur in myocytes because PIP_2 diffusion is highly restricted. Our perspective is that PIP_2, in general, serves to activate many cardiac channels and transporters when they are inserted in the sarcolemma, while lack of PIP_2 on internal membranes keeps them inactive during trafficking and processing. In addition, however, PIP_2 metabolism is part-and-parcel of membrane trafficking and the turnover of membrane cytoskeleton. In this context, we have established new models of PIP_2-dependent endocytosis in fibroblasts and cardiac myocytes. These models are unique in allowing rapid control of the cytoplasm while monitoring membrane fusion and retrieval processes at high resolution. Powerful Ca^{++}- and PIP_2 -activated endocytic processes can remove 50% of the cell surface in <10 s with no involvement of clathrin. Cardiac Na/Ca exchangers are prone to removal by these processes, and endocytosis during ischemia-reperfusion episodes may importantly control the Ca^{++} and Na^{+} loads in cardiac myocytes that ultimately determine myocyte fate.

Contact Information:

Alla Fomina, Ph.D.
affomina@ucdavis.edu
Yu-Fung Lin, Ph.D.
yflin@ucdavis.edu