BIOLS SEMINAR SERIES

北京生命科学研究院精品讲座

报告时间：2013年6月26日（星期三）下午14:00

报告地点：中国科学院生物物理所9501会议室

报告题目：Potassium Channel Gating by Calcium and Calmodulin.

报 告 人：Richard W. Aldrich. Member of National Academy of Sciences, USA.
Section of Neurobiology, The University of Texas at Austin.

欢迎广大科研人员和研究生光临！

Prof. Aldrich received his Ph.D. in Neuroscience from Stanford University in 1980, after which he did postdoctoral work at Yale University. He joined the faculty at Yale before returning to Stanford in 1985 where he served as Chair of Department of Molecular and Cellular Physiology from 2001 to 2004. He was a member of the Howard Hughes Medical Institute from 1990 until moving to The University of Texas in 2006, where he is Professor and Chair of the Section of Neurobiology in the School of Biological Sciences and the Karl Folkers Chair II in Interdisciplinary Biomedical Research. Prof. Aldrich has served on the council and as president of the Society of General Physiologists, and was a Fellow and president of the Biophysical Society. He was elected member of the National Academy of Sciences in 2008 and member of American Academy of Arts and Sciences in 2011. He is also on Editorial Board of *PNAS*, *eLife*, *PLoS Biology* and *PLoS One*, etc.

Prof. Aldrich, an ion channel biophysicist, studies the structure and function of proteins involved in interand intracellular communication. He pioneered the study of physical mechanisms that determine whether ion channels are open or closed. He demonstrated a “ball and chain” mechanism for shutting off potassium channels, and delineated how depolarization and calcium operate jointly to open calcium-activated potassium channels, thus illuminating both channel inactivation and activation. His laboratory developed a quantitative model of this process that is applicable to the study of other proteins.

Key Publications:

2010. An auxiliary LRRC26 protein allows BK channel activation at resting voltage without calcium. ***Nature***. 466:513-517.
2009. Convergent evolution of alternative splices at domain boundaries of the BK channel. ***Annual Review of Physiology***. 71:19-36.
2006. Local potassium signaling couples neuronal activity to vasodilation in the brain. ***Nature Neuroscience***. 9:1397-1403.
2006. BK calcium-activated potassium channels regulate circadian behavioral rhythms and pacemaker output. ***Nature Neuroscience***. 9:1042-1049.
2005. BK channel β4 subunit reduces dentate gyrus excitability and protects against temporal lobe seizures. ***Nature Neuroscience***. 8:1752-1759.
2002. Calcium activation of BK_{Ca} potassium channels lacking calcium bowl and RCK domains. ***Nature***. 420:499-502.
2000. Vasoregulation by the beta1 subunit of the calcium-activated potassium (BK) channel. ***Nature***. 407:870-876.
1994. Molecular determinants of voltage-dependent inactivation in calcium channels. ***Nature***. 372:97-100.
1993. Functional stoichiometry of Shaker potassium channel inactivation. ***Science***. 262:757-759.
1990. Restoration of inactivation in mutants of Shaker potassium channels by a peptide derived from ShB. ***Science***. 250:568-571.
1990. Biophysical and molecular mechanisms of Shaker potassium channel inactivation. ***Science***. 250:533-538.
1988. Effect of Forskolin on voltage-gated K+ channels is independent of adenylate cyclase activation. ***Science***. 240:1652-1655.
1987. Single-channel and genetic analyses reveal two distinct A-type potassium channels in Drosophila. ***Science***. 236:1094-1098.
1983. A reinterpretation of mammalian sodium channel gating based on single channel recording. ***Nature***. 306:436-441.

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