Abstract Submitted
for the 4CF10 Meeting of
The American Physical Society

Anomalous Diffusion and Weak Ergodicity Breaking of Kv2.1 Channels Observed by Single Molecule Tracking in Live Cells AUBREY WEIGEL, BLAIR SIMON, MICHAEL TAMKUN, DIEGO KRAPF, Colorado State University — The neuronal Kv2.1 potassium channel localizes into micron-sized clusters on the cell membrane. These clusters are essential for many cellular functions. However, the physical mechanism behind Kv2.1 cluster formation and maintenance is not yet understood. We are investigating the dynamics of Kv2.1 channels using single particle tracking in living cells. We dually label Kv2.1 channels with GFP and red quantum dots (QD). While the QDs enable accurate tracking of individual channels, the GFP provides characteristics of the cluster as an ensemble. Kv2.1 channel dynamics are analyzed in terms of their distribution of square displacements. Our results show that all Kv2.1 channels experience anomalous subdiffusion. Through direct comparison of the temporal averaged and ensemble averaged (average of squared displacements of all trajectories occurring at a specific time) mean square displacement distributions we observe that the Kv2.1 trajectories are non-ergodic. We further investigate possible mechanisms for the observed anomalous diffusion and weak ergodicity breaking by focusing on the continuous time random walk (CTRW) model, diffusion on a fractal and a combination of both. From our results we see that the Kv2.1 trajectories are best modeled by a CTRW in a fractal geometry.

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Date submitted: 10 Sep 2010 Electronic form version 1.4