PROGRESS IN NEUROSCIENCE PINS

Seminar Series of the
Brain & Mind Research Institute
Weill Cornell Medical College (WCMC)
&
The Graduate Program in Neuroscience of
WCMC and Sloan Kettering Institute

Thursday, 4/28/16, 4 PM, coffee at 3:45 PM
Weill Auditorium

"Quantal Release and Its Requirements"

Robert Edwards, MD
Professor of Neurology & Physiology
UCSF School of Medicine

Abstract:

Quantal release by regulated exocytosis depends on the storage of neurotransmitter inside secretory vesicles. For classical transmitters, this requires transport from the cytoplasm, a process that generally involves exchange for lumenal H+. Coupling to H+ should restrict the activity of these transporters to acidic internal membranes such as synaptic vesicles, and prevent non-vesicular release when they reside at the plasma membrane as part of the synaptic vesicle cycle. However, glutamate transport depends on membrane potential, indicating the potential for non-quantal efflux across the plasma membrane. By developing the use of electrophysiology to study the vesicular glutamate transporters, we have identified a series of regulatory mechanisms with implications for both quantal and non-quantal release.

Recent relevant publications:

1. Bellocchio, E.E., Reimer, R.J., Fremeau, R.T.J., and Edwards, R.H. (2000). Uptake of glutamate into synaptic vesicles by an inorganic phosphate transporter. Science *289*, 957-960.
2. Hnasko, T.S., Chuhma, N., Zhang, H., Goh, G.Y., Sulzer, D., Palmiter, R.D., Rayport, S., and Edwards, R.H. (2010). Vesicular glutamate transport promotes dopamine storage and glutamate corelease in vivo. Neuron *65*, 643-656.
3. Goh, G.Y., Huang, H., Ullman, J., Borre, L., Hnasko, T.S., Trussell, L.O., and Edwards, R.H. (2011). Presynaptic regulation of quantal size: K(+)/H(+) exchange stimulates vesicular glutamate transport. Nat. Neurosci. *14*, 1285-1292.

Weill Cornell Medical College