



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, 2/4/16, 4 PM, coffee at 3:45 PM
Weill Auditorium

“Mitochondria, Motors, and Microtubules in Neurons and Mitotic Cells”

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Abstract:



The motility of mitochondria allows cells to regulate the fusion, fission, and distribution of mitochondria. In neurons, the need for mitochondrial movement is particularly great because the vast majority of the cell volume and of cellular energy expenditure occur in the axons and dendrites that can extend a meter or more from the nucleus. This presentation will discuss the diverse mechanisms by which mitochondrial motility is regulated in both neuronal and non-neuronal cells. The motor complex that attaches the kinesin and dynein motors to the mitochondrial surface is the control point for regulation by cytosolic Ca^{2+} , glucose and mitochondrial damage. Regulation of this complex appears to be an early step in preparing damaged mitochondria for clearance by mitophagy and the relevance of this pathway for Parkinson's disease will be discussed. Finally, because mitochondria must stay clear of the spindle apparatus in order for cells to proceed correctly through mitosis, regulation of this complex also plays a crucial role in mitotic cells.

Recent relevant publications:

- 1) Wang, X., Winter, D., Ashrafi, G., Schlehe, J., Wong, Y.L., Selkoe, D., Rice, S.J., Steen, J., LaVoie, M.J., and Schwarz, T.L. (2011) PINK1 and Parkin target Miro for phosphorylation and degradation to arrest mitochondrial motility. *Cell* 147:893-907
- 2) Pekkurnaz, G., Trinidad, J.C., Wang, X., Kong, D. and Schwarz, T.L. (2014). Glucose Regulates Mitochondrial Motility via Milton Modification by *O*-GlcNAc Transferase. *Cell*, 158:54-68.
- 3) Ashrafi, G., Schlehe, J. S., LaVoie, M. J., & Schwarz, T. L. (2014). Mitophagy of damaged mitochondria occurs locally in distal neuronal axons and requires PINK1 and Parkin. *The Journal of Cell Biology*, 206(5), 655–670.



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