



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, 12/18/14, 4 PM, coffee at 3:45 PM
Weill Auditorium

“The Cerebellum, Sensitive Periods, and Autism”

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Abstract:



Cerebellar research has focused principally on adult motor function. However, the cerebellum also maintains abundant connections with nonmotor brain regions throughout postnatal life. Clinical and nonhuman experimental evidence suggest that the cerebellum may guide the maturation of remote nonmotor neural circuitry and influence cognitive development, with particular relevance for autism. Modern optogenetic and pharmacogenetic tools open the possibility of perturbing identified cell types at defined times during development. We are performing experiments to test two hypotheses: (a) specific cerebellar zones influence neocortical substrates for social interaction and cognitive function, and (b) sensitive-period disruption of such internal brain communication can account for autism's key features. The tools that we bring to bear upon these problems include multiphoton microscopy to image neural activity, pharmacogenetics to perturb activity, and quantitative behavioral monitoring.

Recent relevant publications:

S.S.-H. Wang, A.D. Kloth, and A. Badura (2014) The cerebellum, sensitive periods, and autism (Perspective). *Neuron*, 83:518-532. doi:10.1016/j.neuron.2014.07/016

X.R. Sun, A. Badura, D. A. Pacheco, L.A. Lynch, E.R. Schneider, M.P. Taylor, I.B. Hogue, L.W. Enquist, M. Murthy, S.S.-H. Wang (2013) Fast GCaMPs for improved tracking of neuronal activity. *Nature Communications*, 4:2170. doi:10.1038/ncomms3170.

C. Piochon, A.D. Kloth, G. Grasselli, H. Titley, H. Nakayama, K. Hashimoto, V. Wan, D.H. Simmons, T. Eissa, J. Nakatani, A. Cherskov, T. Miyazaki, M. Watanabe, T. Takumi, M. Kano, S.S.-H. Wang, and C. Hansel (2014). Cerebellar plasticity and motor learning in a copy number variation mouse model of autism. *Nature Communications*, in press.



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