MSN*seminars*

presents

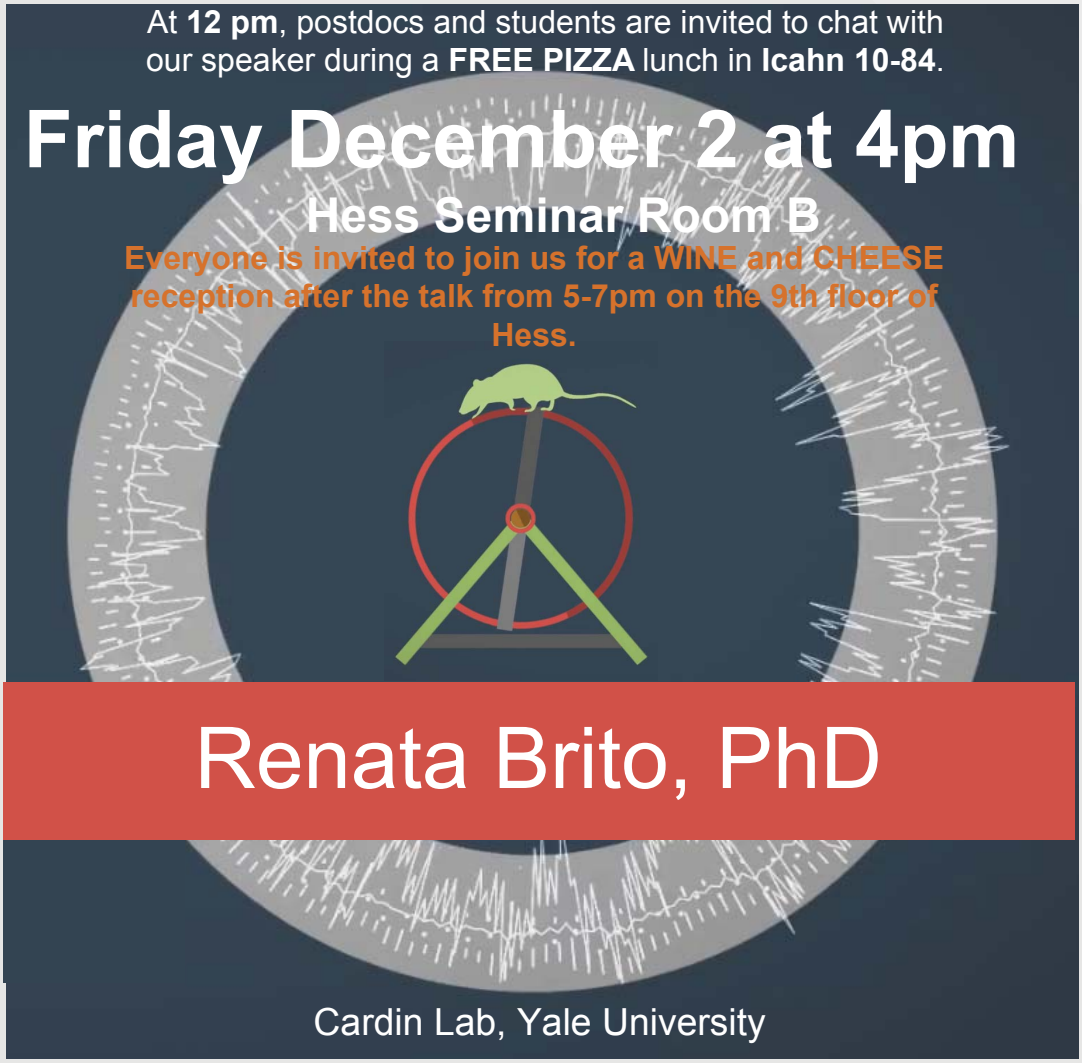
Developmental dysfunction of VIP interneurons impairs cortical circuits

At **12 pm**, postdocs and students are invited to chat with our speaker during a **FREE PIZZA** lunch in **Icahn 10-84**.

Friday December 2 at 4pm

Hess Seminar Room B

Everyone is invited to join us for a WINE and CHEESE reception after the talk from 5-7pm on the 9th floor of Hess.



Renata Brito, PhD

Cardin Lab, Yale University

Neuregulin 1 (NRG1) and its interneuron-specific tyrosine kinase receptor ERBB4 are risk genes for schizophrenia, and the Nrg1/ErbB4 pathway is important for normal cortical development. Using a conditional ErbB4 deletion model, we directly tested the role of VIP-expressing interneurons in schizophrenia-related deficits in vivo.

ErbB4 removal from VIP interneurons during development leads to changes in their activity, along with severe dysregulation of the temporal organization and state-dependence of cortical activity. Animals in which VIP interneurons lack ErbB4 exhibit behavioral abnormalities, reduced cortical responses to sensory stimuli, and impaired sensory learning. Our data support a key role for VIP interneurons in normal cortical circuit development and suggest that their disruption contributes to pathophysiology in the ErbB4 model of schizophrenia.

Icahn School of Medicine at Mount Sinai

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