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, 4/2/15, 4 PM, coffee at 3:45 PM
Weill Auditorium

"Is the Function of APP Relevant to the Pathogenesis of Dementia?"

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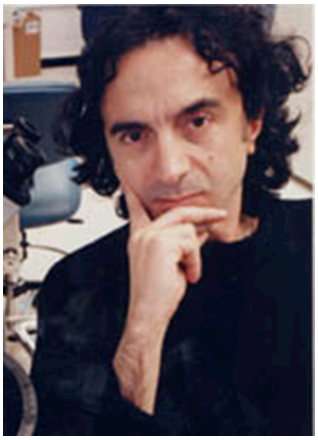

Abstract:

During the last 15 years, my lab has used genetic approaches to dissect the mechanisms of age-related neurodegeneration occurring in Alzheimers's disease (AD), Familial British dementia (FBD) and Danish dementia (FDD) as well as the biological function of APP and BRI2 in the CNS. Through multidisciplinary analysis of *Bri2*-KO, FDD_{KI} and FBD_{KI}, and App_{KI} mice our studies demonstrated that loss of BRI2 causes progressive memory and synaptic transmission impairment that are dependent on a C-Terminal fragment of APP. In addition, we have made considerable progress in understanding the function of APP and BRI2 in synaptic transmission. In this talk, I'll present some of our recent unpublished data focusing on the mechanisms of synaptic plasticity regulation by APP.

Recent relevant publications:

1. APP is cleaved by Bace1 in pre-synaptic vesicles and establishes a pre-synaptic interactome, via its intracellular domain, with molecular complexes that regulate presynaptic vesicles functions Del Prete D, Lombino F, Liu X, **D'Adamio L**. PLoS One. 2014 Sep 23;9(9):e108576.
2. An Intracellular Threonine of Amyloid-β Precursor Protein Mediates Synaptic Plasticity Deficits and Memory Loss. Lombino F, Biundo F, Tamayev R, Arancio O, **D'Adamio L**. PLoS One. 2013;8(2):e57120.
3. Tyr682 in the Aβ-precursor protein intracellular domain regulates synaptic connectivity, cholinergic function, and cognitive performance. Matrone C, Luvisetto S, La Rosa LR, Tamayev R, Pignataro A, Canu N, Yang L, Barbagallo AP, Biundo F, Lombino F, Zheng H, Ammassari-Teule M, **D'Adamio L**. Aging Cell. 2012 Dec;11(6):1084-93.

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