2011 Winner

Tiago Branco, M.D., Ph.D.

Postdoctoral Research Fellow
University College London

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Tiago Branco received his M.D. from Lisbon University in 2002. He then joined the Wellcome Trust Four Year Ph.D. Programme in Neuroscience at University College London (UCL), where in the group of Dr. Yukiko Goda he focused on neurotransmitter release properties of individual synapses. After receiving his Ph.D. he moved to Dr. Michael Hausser's laboratory, where he has been a postdoctoral research fellow since 2007. He has applied electrophysiological, optical and modelling techniques to investigate how dendritic integration contributes to single neuron computations. He plans to combine this approach with molecular methods to investigate the role of dendrites in controlling animal behavior.

The Language of Dendrites

Animal survival depends on the ability to analyze the environment and act upon it. This requires processing information from the outside world and using it to produce an appropriate behavior. How does the brain achieve this? Information arrives at neurons in the form of synaptic input delivered to dendrites—protrusions from the cell body separating the input from the action potential initiation zone. In his studies Dr. Branco has investigated how the properties of dendrites might be used by single neurons to integrate information and perform specific computations. In particular, he has focused on the ability of dendrites to discriminate betweeen different temporal sequences of input, a fundamental computation for successfully interacting with a dynamic environment. Using two-photon glutamate uncaging to activate synapses with precise spatial and temporal control, Dr. Branco has shown that the presence of NMDA receptors allows dendrites to efficiently discriminate multiple input sequences. In addition, this property also gives dendrites the ability to use different computational strategies depending on input location. These findings give insight on how the brain performs the computations that underlie behavior, and suggest that even single neurons can solve complex computational tasks.

2011 Finalist

Aaron D. Gitler, Ph.D.

Assistant Professor
Department of Cell and Developmental Biology
Perelman School of Medicine at the University of Pennsylvania

Aaron Gitler is an assistant professor in the Department of Cell and Developmental Biology at the University of Pennsylvania School of Medicine. Dr. Gitler received his Ph.D. in cell and molecular biology from the University of Pennsylvania working with Jonathan Epstein on endothelial cell signaling pathways in cardiovascular development. In postdoctoral research with Susan Lindquist, at the Whitehead Institute for Biomedical Research, he performed high-throughput genetic screens in yeast for modifiers of toxicity associated with the Parkinson's disease protein alpha-synuclein. His group at the University of Pennsylvania is combining yeast and human genetics to elucidate novel pathways involved in neurodegenerative disease, focusing on the role of RNA-binding proteins in the motor neuron disease ALS.

New Insights into Human Neurodegenerative Diseases

The goal of my laboratory is to elucidate the mechanisms of human neurodegenerative diseases by defining critical genes and cellular pathways affected by aggregation-prone human disease proteins. We are harnessing the budding yeast as a model system to study mechanisms underpinning protein-misfolding diseases. Our approach has been to construct yeast models to study human neurodegenerative disease proteins and to perform high-throughput genome-wide screens for modifiers of toxicity. We have focused on the amyotrophic lateral sclerosis (ALS) disease protein TDP-43 and discovered several potent modifiers of TDP-43 aggregation and toxicity. We have begun to extend these findings to human disease, discovering that mutations in ataxin 2, the human homolog of one of the yeast TDP-43 toxicity modifier genes, are associated with increased risk for ALS in humans. These findings show the power of simple experimental model systems for providing completely new insight into mechanisms of complicated human diseases.

2011 Finalist

Roger L. Clem, Ph.D.

Postdoctoral Fellow
Johns Hopkins University

Roger Clem is a postdoctoral fellow in the Department of Neuroscience at The Johns Hopkins University School of Medicine. He received his Ph.D. at Carnegie Mellon University under the mentorship of Alison Barth. In his doctoral work, he investigated synaptic mechanisms of sensory-induced plasticity in the neocortex. Since joining the laboratory of Rick Huganir, Dr. Clem has examined the role of AMPA-type glutamate receptor trafficking in emotional memory. His work explains how fear memories can be permanently weakened through behavioral training, a process akin to software uninstall routines, and provides new molecular targets for alleviating emotional trauma. Dr. Clem has accepted an appointment to Assistant Professor of Neuroscience at the Mount Sinai School of Medicine, where he will investigate synaptic processes in memory formation and updating.

An Uninstall Function for Fear Memories

Memory is the essence of human identity. For some, however, a painful past is like a debilitating emotional storm lurking behind the slightest reminder. Coping in the aftermath of trauma can be aided by pharmacology and psychological therapy, but these approaches leave underlying emotion subject to resurface when least expected. In general, this is thought to reflect the relative imperviousness of memories once encoded in the brain. By understanding the dynamic molecular events at sites of fear memory storage, however, Dr. Clem has discovered that malleability of fear memories is subject to key molecular determinants. A provisional window of opportunity allows fear memories to be permanently weakened through the rearrangement of neuronal AMPA-type glutamate receptors by simple re-exposure to a threatening cue. Thus, the brain has self-editing repertoires that allow for adaptive updating of memories, and that could heavily inform our treatment of emotional disorders.