B·DEBATE International Center for Scientific Debate BARCELONA

Sant Joan de Déu HOSPITAL MATERNOINFANTIL - UNIVERSITAT DE BARCELONA

Sant Joan de Déu Research Foundation

NILS BROSE

CV

PARTICIPANT AT:

CONNECTING THE GROWING BRAIN

UNDERSTANDING NEUROPAEDIATRIC DISEASES THROUGH SYNAPTIC COMMUNICATION

November, 26th-27th, 2015, Barcelona

Nils Brose, Director, Department of Molecular Neurobiology, Max Planck Institute of Experimental Medicine, Goettingen, Germany

Dr. Brose studied Biochemistry, Biology, and Physiology at the Universities of Tuebingen (Germany) and Oxford (UK). He received an MSc degree from the University of Oxford (UK), where he worked with Marianne Fillenz, and a PhD degree from the University of Munich (Germany), where he worked in the laboratory of Reinhard Jahn at the Max Planck Institute of Psychiatry. After postdoctoral training with Steve Heinemann (Salk Institute, La Jolla, CA, USA) and Tom Sudhof (UT Southwestern Medical Center, Dallas, TX, USA), Dr. Brose started his independent research program at the Max Planck Institute of Experimental Medicine (Goettingen. Germany), where he is currently the director of the Department of Molecular Neurobiology. Dr. Brose's research focuses on the molecular mechanisms of synaptogenesis and synapse function, and on the role of these processes in neuropsychiatric disorders.

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ABSTRACT

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Neuroligins at Inhibitory Synapses - from Synaptogenesis to Autism Spectrum Disorders

Members of the Neuroligin family of cell adhesion proteins are thought to regulate the generation, maturation, maintenance, and plasticity of synapses between nerve cells. Among the Neuroligin family members, the Neuroligin-2 and Neuroligin-4 isoforms are preferentially localised to inhibitory synapses, and loss-of-function mutations in Neuroligin-4 cause monogenic heritable forms of autism spectrum disorders (ASDs) in humans and ASD-like behavioral defects in mice. I will present biochemical, structural biology, cell biological, and electrophysiological data demonstrating that Neuroligin-2 and Neuroligin-4 regulate the recruitment of GABAA-receptors to nascent inhibitory synapses by activating the signaling/scaffold protein Collybistin. The main functional defects resulting from Neuroligin-4 loss in mice are subtle reductions in GABAA-receptor signaling in several brain regions, such as the CA3 region of the hippocampus, which lead to promient changes in oscillatory network activity. These data indicate that defects in GABAergic signaling and the consequent changes in network activity are causally involved in the ASD-like behavioral defects seen upon Neuroligin-4 mutation. Indeed, GABAergic dysfunction may be a common denominator of ASDs, because perturbed GABAergic signaling has been observed in several other ASD models.

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