

Towards Defining the Pathophysiology of Autistic Behavior

(Joint with "Synapses: Formation, Function and Misfunction")

Sponsored by Simons Foundation

April 11–15, 2010 • Snowbird Resort • Snowbird, Utah • USA

Scientific Organizers: Pat Levitt and Joseph Piven

PROGRAM FACULTY & TALKS

- Claudia Bagni**, Catholic University of Leuven, Belgium
Molecular Aspects of Mental Retardation: Insights from the Fragile X Syndrome
- Mark F. Bear**, Massachusetts Institute of Technology, USA
Activity Dependent Regulation of Glutamate Receptors and the Neurobiology of FXS
- Elizabeth M. Berry-Kravis**, RUSH University Medical Center, USA
Autism Phenotype in Fragile X Syndrome: a Door to Molecular Pathways and New Targeted Treatment Strategies
- Patrick Bolton**, King's College, London, UK
Overview of Medical Conditions Associated with the Autistic Phenotype
- Nancy M. Bonini**^o, University of Pennsylvania, USA
Human Neurodegenerative Disease: Insights from Drosophila
- Nils Brose**^o, Max Planck Institute of Experimental Medicine, Germany
Genetic Dissection of Neuroligin Function: From Synaptogenesis to Autism
- Elisabeth Dykens**, Vanderbilt University, USA
PWS and Others
- Daniel H. Geschwind**, University of California, Los Angeles, USA
Molecular Comparisons across Syndromes
- Michael E. Greenberg**^{*o}, Harvard Medical School, USA
Signaling Networks that Control Synapse Development and Cognitive Function
- Randi J. Hagerman**, University of California, Davis, USA
Molecular Mechanisms of ASD in the Premutation and the Full Mutation
- Kimberly M. Huber**^o, University of Texas Southwestern Medical Center, USA
Regulation of Synapse Number by Fragile X Mental Retardation Protein
- Pat Levitt**, Keck School of Medicine, USA
MET Receptor Tyrosine Kinase in ASD and Social-Emotional Circuit Wiring Relevant to Autism Spectrum Disorder
- Courtney Miller**, Scripps Research Institute, USA
Epigenetics and Memory
- Lisa M. Monteggia**, University of Texas Southwestern Medical Center, USA
Role of MeCP2 & HDACs in Regulating Synapse Function and Behavior
- Luis F. Parada**, University of Texas Southwestern Medical Center, USA
Mouse Models as Translational Tools to Discover Treatments for Autism Spectrum Disorders: Focus on Rapamycin
- Ben Philpot**^o, University of North Carolina at Chapel Hill, USA
Angelman Syndrome and Synaptic Plasticity
- Joseph Piven**, University of North Carolina at Chapel Hill, USA
Towards Defining the Phenomenology of Autism
- Carolyn Schanen**, A.I. duPont Hospital for Children, USA
Increasing Molecular and Phenotypic Complexities of the Chromosome 15q11.2-q13.3 Duplication Syndromes in Autism Spectrum Disorders
- Alcino J. Silva**, University of California, Los Angeles, USA
Tsc2 haploinsufficiency and gestational immune activation: interactive effects in mice
- Matthew W. State**, Yale Child Study Center, USA
Rare Structural and Sequence Variation in Autism Spectrum Disorders
- Moshe Szyf**, McGill University, Canada
How Early Life Experience Modifies the Epigenome and Affects Mental Health
- Toru Takumi**, Hiroshima University, Japan
Neurobiology of Chromosome 15 Copy Variants in Mice

*Keynote speaker. ^oJoint speaker. Program subject to change. Current as of February 9, 2010.

This is a joint meeting. Registration for one meeting allows participation in sessions of the other, pending space availability.



As one of the most common neurodevelopmental disorders, autism is recognized as heterogeneous in etiology, phenotype, behavioral trajectory and response to treatment. The overarching aim of this Keystone Symposia meeting will be to take advantage of our knowledge of etiologic heterogeneity by examining the phenomenology and pathophysiology of etiologically-defined autistic syndromes and contrasting this with what is known about idiopathic autism, to ultimately shape the development of treatment approaches informed by knowledge of the underlying pathophysiology. Goals: 1) Define more precisely the common and unique clinical features of syndromic and idiopathic autism. The field needs a realistic view of autism heterogeneity with regard to phenotype expression, longitudinal course and diversity in response to treatment; 2) Provide novel insight into the role of complex genetic mechanisms in autism; 3) Provide a basic understanding of and define the roles for epigenetics in understanding the causes of the autism; 4) Examine the common cellular mechanisms that underlie the autisms; 5) Re-examine the disconnection-synapse hypothesis of the autisms through a new perspective of factors that influence synapse formation and maturation.

PROGRAM PLENARY SESSIONS:

- Clinical and Phenotypes of Autism
- FXS
- 15q, CNV and Rare Syndromes
- PI3 Kinase Dysfunction
- Synaptic and Circuit Function in Neurodevelopmental Disorders (Joint)
- Epigenetic Modifiers of Neurodevelopmental Disorders

DEADLINES:

Early Registration: February 11, 2010

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