IMFAR

INTERNATIONAL MEETING FOR AUTISM RESEARCH

Annual Meeting of the International Society for Autism Research (INSAR)

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International Meeting for Autism Research

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ABSTRACT BOOK

Keynote Address
100 - Who Owns Autism? Exceptionalism, Stigma, and Stakeholders
9:00 AM - 10:00 AM - Grand Ballroom

Speaker: R. R. Grinker, George Washington University, Washington, DC

This presentation focuses on critical themes and challenges in the cultural study of autism spectrum disorder (ASD). First, in clinical, research, and advocacy settings ASD has emerged as a singular and powerful construct that encompasses an increasing number of heterogeneous phenomena. What forces made this category possible? How did it become both a valid and unstable construct? Second, the growth of genetic and other biomedical perspectives on ASD risks reducing ASD to biology alone, and, as a consequence, masking the fact that scientific representations express cultural values about diversity and disability. Difference constructed on the molecular level is still difference, no less stigmatizing and socially consequential because of its biological source (and perhaps even more so). How can we integrate both the biological and sociocultural aspects of ASD into research? Third, ASD is now, in some respects, a commodity that circulates in an industry of "stakeholders," such as therapists, producers of high-cost diagnostic tools, and advocacy organizations. Indeed, as health professionals are discovering in low- and middle-income countries, few diagnostic categories cost as much as ASD. How does the economy of ASD influence the science of ASD?

9:00 **100.001** Who Owns Autism? Exceptionalism, Stigma, and Stakeholders
R. R. Grinker, *George Washington University, Washington, DC*

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Panel Session
101 - Autism and Society: Taking Stock of the History and Meaning of Autism Research
10:30 AM - 12:30 PM - Grand Ballroom B

Panel Chair: Roy Grinker*, George Washington University, Washington, DC*

Current debates about the present and future of autism research generally focus on scientific discovery and are fitted into the framework of the scientific method. This panel departs from convention to "take stock" of the field, and explore autism research as a system of knowledge and practices in social, historical, and economic context. The questions at the core of this panel concern the various and sometimes contradictory aspects of the field of autism research: How has the definition of autism changed over time for a range of individuals, communities, and audiences, and what factors led to those changes? What kinds of authority (e.g., institutional, bureaucratic, academic, legislative, familial) have structured, and been structured by, scientific representations of autism? Is autism a disease, a disability, or an aspect of a 'normal' range of human variation? Is autism singular, or do the boundaries and definitions of the category constrain the ability of researchers and clinicians to address the dimensions of autism as outcomes of a common set of developmental pathways shared by all humans? Speakers from the fields of anthropology, disability rights, linguistics, and epidemiology will employ historical, ethnographic, philosophical, and public health perspectives to explain the dramatic changes in the field of autism research over the past several decades and outline possibilities for the future.

10:30 **101.001** Trends in the Prevalence of Intellectual Disability and Autism Spectrum Disorder
M. S. Durkin, *Population Health Sciences, University of Wisconsin-Madison, Madison, WI*

Background: Changes in the diagnosis and treatment of just one psychiatric condition can have an important effect on the diagnosis and treatment of others. This presentation provides a historical review of the epidemiology of intellectual disability (ID) and autism (ASD) in relation to each other. Objectives: We discuss possible explanations for declines in ID prevalence include the expansion of

170 **174.170** Abnormal Expression of a SERT-Binding Protein, NSF, in Autism: Implications for Pathophysiology in Autism

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Background: Change in serotonin transporter (SERT) function has been implicated in autism. SERT function is influenced by the number of transporter molecules present at the cell surface, which is regulated by various cellular mechanisms including interactions with other proteins. Thus, we searched for novel SERT-binding proteins and investigated. As we presented at the IMFAR 2014, *N*-ethylmaleimide-sensitive factor (NSF) was identified as a novel SERT-binding protein. NSF co-localized with SERT at the plasma membrane, and NSF knockdown resulted in decreased SERT expression at the cell membranes and its uptake function in HEK293-hSERT cells. In addition, NSF endogenously co-localized with SERT and interacted with SERT in mouse brain.
Objectives: The objectives of this study were to address whether expressions of SERT and NSF were changed in autism and whether these expression correlate with clinical variables and symptom profiles.
Methods: We examined the mRNA expression of SERT (SLC6A4) and NSF in the post-mortem brains from 7 subjects with autism and 11 healthy age- and sex-matched control subjects, and in the lymphocytes from 30 male subjects with autism and 30 male healthy age-matched control subjects by quantitative real-time PCR. Additionally, we evaluated the relationships between these expression levels and clinical variables and symptom profiles.
Results: While *SLC6A4* expression was not significantly changed, *NSF* expression tended to be reduced in post-mortem brains, however this potential trend is not statistically significant, and was significantly reduced and correlated with the severity of the clinical symptom in lymphocytes of subjects with autism.
Conclusions: A possible role for NSF in the pathophysiology of autism, through modulation of SERT trafficking, is suggested.

171 **174.171** Austism Spectrum Disorder and the Brain-Gut-Microbiome Axis

N. E. Furland[1,2] *and* ***M. T. Sindelar***[2], *(1)INIBIBB-CONICET-UNS, Bahia Blanca, Argentina, (2)Emily Fenichel Foundation, Bahía Blanca, Argentina*

Background: Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder where a high frequency of gastrointestinal (GI) dysfunction (e.g., constipation, diarrhea, bloating, gas, and history of reflux) is reported. However, the mechanism underlying GI tract defects in autistic children as well as the association between abnormal GI structure and function with ASD is yet to be clearly understood. GABA and serotonin functions as key neurotransmitters at both, the central nervous system and the gastrointestinal tract, and there is accumulating evidence pointing to a critical role for the gut microbiome in regulating normal functioning of tryptophan metabolism and the GABAergic system. There is also substantial overlap between ASD behaviours that could be influenced by the gut microbiota.
Objectives: the aim of this work is to analyse and identify differences on fecal microbiota (as a proxy for gut microbiota), some neurotransmitters levels and SCFA (short chain fatty acids) between autistic children and healthy donors. If the unique microbial flora or metabolic profile is found to be a causative or consequent factor in GI disorders in ASD, it may have implications with regard to a specific diagnostic test, its epidemiology, and therapeutic targeting of the gut microbiota as a viable treatment strategy for ASD
Methods: we analyzed Serotonin and Dopamine, both neurotransmitter monoamines involved in modulating adult cortical plasticity, also GABA and SCFA (short chain fatty acids) profile in fecal samples in a cohort of 30 patients that met DSM V criteria for autism based on ADOS and their typical developed (TD) siblings. The control sample consisted of 35 healthy donors, sex-matched with the case sample.
Results: Autistic patients have a unique microbiome consisting of more clostridial species. Half of all autistic children with gastrointestinal dysfunction were found to have Sutterella, a bacteria which is absent in no autistic children with gastrointestinal dysfunction. Our results show that microbiota and metabolic profiles from ADS children significantly differ from their healthy siblings and controls and suggest a potential correlation with gastrointestinal dysfunction.
Conclusions: Differences in microbiota and some metabolites levels found in ADS children stools versus controls correlates with GI distress. Also CNS neurotransmission can be profoundly disturbed by the gut microbiome in ASD.

172 **174.172** Dynamic Gene Network Analysis of Neuronal Differentiation Identifies Novel Gene-Network Clusters Specifically Enriched for Autism Risk Genes

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