

Atypical infant cries among incipient ASDs, developmentally delayed individuals, and language-impaired individuals

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Abstract

Due to familial factors, younger siblings of children with Autism Spectrum Disorder (ASD) are at an increased risk for developing the disorder^{1,2}. Although behavioral symptoms of ASD typically emerge during a child's second year,³ recent work on younger siblings demonstrates that prodromal features of ASD are present already within the first months of life. These features include atypical attention toward stimuli relevant to social engagement, such as faces and speech sounds.^{4,5} Similar deficits were observed in clinic-referred toddlers suggesting continuity of social attention impairments in ASD from prodromal to early syndromal stages.^{6,7} This presentation will review: (1) the methodological underpinnings of prospective high-risk sibling studies and findings on patterns of autism onset in infancy, and (2) experimental studies on endo- and exogenous attention to multimodal social stimuli conducted during prodromal and early syndromal stages of the disorder.

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Speaker 2: Gianluca Esposito, Italy

Title: Atypical infant cries among incipient ASDs, developmentally delayed individuals, and language-impaired individuals

Abstract

To better understand social communication during early human development, a growing literature is assessing the vocal production of children with Autism Spectrum Disorders (ASD). Previous studies have provided preliminary evidence that disruptions in cry acoustics may be part of an atypical vocal signature of autism early in life.

In the current research we investigate the acoustic characteristics of cries elicited during real life events as well as cries elicited in experimentally standardized social interaction contexts (i.e. the Strange Situation Procedure -SSP).

Using these approaches, we found that 15-month-olds at high risk for ASD had atypical acoustical patterns of distress vocalization (e.g. shorter cry utterances, higher fundamental frequencies). Then, next step was to assess using multiple neuroimaging and electrophysiological techniques (EEG, fMRI, TMS,

EKG, EMG, GSR, etc) the effect on parental perception of ASD distress vocalizations. Perceived distress engendered by ASD cries related to increased activation in brain regions associated with emotional processing.

Speaker 3: Elodie Ey, France

Title: Subtle abnormalities in the vocal behavior of mouse pups mutated in *Shank2*

Elodie Ey, Thomas Bourgeron

Abstract

Mutations in genes coding for synaptic proteins were shown to increase susceptibility to autism spectrum disorders (ASD)^{1,2}. Recently, the synaptic scaffolding protein PROSAP1/SHANK2 has been associated with ASD^{3–6}. The mouse model lacking *Shank2* displayed abnormal glutamatergic receptor expression and neurotransmission. Abnormalities in body weight as well as in vocal behavior emerged in the first two weeks of life of *Shank2*^{-/-} pups. We highlighted a different profile in the emission rate of pup isolation calls between *Shank2*^{-/-} mice and their wild-type littermates. We did not highlight any significant genotype-related differences in the vocal repertoire used, in the organization of call types and in the acoustic structure. In this mouse model, subtle abnormalities in usage of ultrasonic vocalizations during development precede impairments in social communication in adulthood. Indeed, in adult *Shank2*^{-/-} mice, impairments in social interactions emerged together with abnormalities in usage and structure of ultrasonic vocalizations^{7,8}. Together with other mouse models of ASD, the *Shank2*^{-/-} mice provide a comprehensive framework to identify new knowledge-based treatments.

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Speaker 4: Noboru Hiroi, USA

Title: Structure and function of neonatal social communication in a genetic mouse model of autism

Abstract

A critical step toward understanding autism spectrum disorder (ASD) is to identify both genetic and environmental risk factors. A number of rare copy number variants (CNVs) have emerged