THE HARTWELL FOUNDATION

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Neurobiological Basis of Speech Patterns as a Biomarker for Autism Spectrum Disorder in Early Childhood

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that affects approximately 2% of children in the United States. It is characterized by hallmark deficits in social interaction that may contribute to significant behavioral challenges. One striking attribute of ASD is difficulty in verbal communication, typified by unusual intonation, pace, and emphasis when speaking, often described as monotonous, exaggerated, or even "singsong". Such non-phonetic patterns of speech (prosody) are used normally to convey emotion and meaning in language and contribute to supporting social interaction. Individual differences are quite variable, and children with ASD may have comorbid language or motor speech deficits that could be related to disrupted prosody. Sadly, atypical prosody can be a major barrier to clear and effective communication, adversely impacting interpersonal perceptions and reactions that hinder social, academic, and vocational opportunities. Fortunately, while prosody is a difficultly modifiable aspect of language and communication, it can be targeted to improve communication capabilities. Understanding prosody in ASD however, is critical for establishing meaningful interventions to correct such communication disorders. Although atypical speech prosody is distinct and immediately recognizable, current approaches to measurement rely typically on subjective ratings, which are inherently non-uniform. Neural mechanisms responsible for speech patterns are known to play a role in prosody, but despite the common appearance of prosodic deficits in ASD the neural basis of atypical prosody has yet to be determined. I hypothesize that characterizing the underlying neural basis of disrupted prosody in ASD will make it possible to identify biomarkers that will enable both early diagnosis and essential evidence-based interventions to improve communication outcomes. To address the fundamental gaps in our understanding, I will systematically characterize speech prosody among children with ASD and examine potential neural mechanisms underlying individual differences. Children with and without diagnosed ASD will first complete behavioral testing to characterize cognitive, speech, and language abilities, as well as custom experimental tasks probing speech prosody perception and production. Subsequently, magnetic resonance imaging (MRI) will be used to establish the neurobiological basis of speech prosody by identifying the extent of neural alteration in brain regions associated with linguistic, affective, motor speech, or some combination of these functions. If I am successful, the availability of reliable biomarkers for evaluating prosody will establish the groundwork for development of novel treatment paradigms to improve language and communication outcomes in children affected by ASD. With clinical translation, an evidence-based intervention will have the potential to minimize negative consequences of social communication deficits by improving social relationships and quality of life.