Dr. Dapretto is Professor of Psychiatry and Biobehavioral Sciences at UCLA. She received graduate training in both developmental psychology and behavioral neuroscience, and later acquired extensive expertise in neuroimaging methods before joining the faculty of the UCLA Semel Institute for Neuroscience and Human Behavior in 1999. Dr. Dapretto’s research focuses on characterizing both typical and atypical brain development. She has conducted pioneering work elucidating the neural correlates of core deficits in autism spectrum disorders (ASD) including the first multimodal investigations of altered developmental trajectories in ASD relating brain function and connectivity to both behavioral phenotypes and genetic risk. Her autism research has been supported by numerous awards by private foundations as well as NIH, and is consistently published in high profile journals (e.g., *Nature Neuroscience, Neuron, Brain, Science Translational Medicine, JAMA Psychiatry, Molecular Psychiatry*). Since 2007, as part of the NIH-funded Autism Center of Excellence (ACE) at UCLA, Dr. Dapretto has led the imaging projects in youth with ASD and in infants at high-risk for ASD. Her current research on autism is also supported by two other large-scale multi-site studies, also funded by NIH, which focus on girls with ASD and parsing the considerable heterogeneity observed in individuals with ASD. In addition to her autism research, Dr. Dapretto has long been interested in neurotypical development. Some of her early work focused on characterizing the neural basis of language, including some pioneering studies which examined developmental changes in the neural networks subserving language learning from childhood through adulthood. Dr. Dapretto has also conducted numerous studies on the neural correlates of key issues in adolescence (e.g., social exclusion, self concept development, social media influence), including some of the first neuroimaging studies to use a longitudinal approach and to examine the link between neural responsivity to emotional stimuli and behavioral outcome (e.g., onset of depressive symptomatology, resistance to peer pressure, risk taking, and prosocial behavior). Currently, Dr. Dapretto is involved in the NIH-funded Adolescent Brain Cognitive Development (ABCD) study, a large-scale multisite consortium on neurodevelopmental and behavioral predictors and consequences of substance abuse from late childhood through adolescence. Dr. Dapretto is also a site PI on another NIH-funded multisite project – the Lifespan Human Connectome: Development (HCP-D) – which aims to characterize the development of functional brain networks and brain-behavior relationships from childhood to young adulthood, with particular emphasis on adolescence as a key developmental transition.

**Presentation Title: Biomarkers and Heterogeneity of Autism Spectrum Disorder**

Under the single diagnostic umbrella term of Autism Spectrum Disorder (ASD), considerable variability is observed across affected individuals, extending well beyond differences in level of intellectual functioning. In addition to substantial clinical comorbidities and gender differences, this considerable heterogeneity likely reflects distinct etiological mechanisms. In this talk, I will argue that neuroimaging data may play a critical role in parsing the significant heterogeneity observed in ASD, a critical first step toward the development of more personalized and efficacious interventions. I will present findings from our neuroimaging studies indicating that individual differences in overall symptom severity, genetic risk, social functioning, and sensory processing atypicalities are meaningfully related to distinct neural signatures in youth with ASD. I will then present some of our recent work in infants at high familial risk for ASD. Contributing to a growing body of literature suggesting that early brain-based markers may be able to predict developmental outcome, our findings support a conceptual framework whereby the social impairments that characterize the ASD phenotype may result from earlier sensorimotor atypicalities that disrupt early attentional biases toward social stimuli, resulting in altered experience-dependent development and the emergence of overt ASD symptomatology. An imaging genetics approach and neuroimaging studies in young infants at increased risk for ASD may inform our understanding of the mechanisms associated with increased vulnerability vs. resilience and ultimately pave the way for more effective treatment and early interventions.