**TITLE:** The Neurology of Social Cognition Deficits in Autism Spectrum Disorders

**ABSTRACT:** Deficits in social cognition are characteristic of autism spectrum disorders. This presentation will detail information from landmark and current neuroimaging studies that identify brain structures and areas contributing to normal social cognitive abilities and how those areas are compromised in function in autism spectrum disorders.

**Summary:** Recent imaging studies investigating the neurologic basis of social cognition have identified common areas and structures in the brain that support these abilities. Several of these studies relate their findings to the Theory of Mind, which postulates that social cognition is based upon the ability to infer emotions, beliefs, or intentions of other persons (Adolphs, 2009). This ability is notably in deficit in individuals with autism. Areas identified that contribute to normal social cognition include the subcortical amygdala, the insula, ventromedial and ventrolateral prefrontal cortices, the fusiform face areas, the superior temporal sulcus, and the mirror neuron system and the contributions of this system to the development of sympathy, empathy, and executive functions.

In this presentation, each of these areas or structures will be presented in terms of how they sub-serve normal social function and what the results of imaging of individuals with autism disorders suggest regarding these same areas and structures.

**The Amygdala:** Located in each temporal lobe, this structure is considered critical to arriving at correct social judgments. This is especially true of judgments based upon people’s faces (Adolphs, 2009; Damasio, 1999). The amygdala responds most effectively to negative facial perceptions. It is also involved in establishing the social significance of stimuli with both social and emotional significance (Berntson, et al 2007). Activation of the amygdala is correlated with activations of the superior temporal sulcus, the fusiform face area and the ventrolateral prefrontal cortex (Adolphs, 2009). As a component of the orbitofrontal limbic circuit, the amygdala is crucial for eye gaze direction, posture, and gestures that indicate appropriate social responses (Loveland, et al 2008). All of these may be in deficit in autism and autism spectrum disorders.

In subjects with autism spectrum disorders, the amygdala was underactivated but the superior temporal sulcus was normally activated. (Pinkham, et al 2007) This suggests that these individuals use feature and rule based strategies for facial processing without assigning normal emotional significance to the faces observed. In other words, faces may be perceived essentially as objects not as faces that represent beings (Pinkham, et al). Volumetric studies of amygdalae in autistic subjects reveal inconsistent findings, some studies report larger volume, others smaller (Sweeten, et al 2008; Verhoeven, et al, 2010).

**The Insula:** This island of cortex within the subcortex is thought to be crucial to provide the neurologic foundation for both the conscious experience of one’s own emotions and also allow an individual to experience empathy for others by helping to simulate the emotions of others. Empathy then often results in a response that allows us to generate speculations and
knowledge of other people’s minds, the foundation of the Theory of Mind. Empathy may also produce the action oriented feeling of sympathy in which we may help another who we perceive as suffering. (Adolphs, 2009). The relationship between the insula and the mirror neuron system, which has been hypothesized as in deficit in autism, may provide the explanation for the shallow feelings of empathy and sympathy prevalent in autism. Recent data suggest that there is hypoactivation of the insula in autistic subjects compared to controls when listening to emotional language (Anderson, et al 2009; Uddin, L. & Menon, V. 2009).

The Ventromedial (VMPC) and Ventrolateral Prefrontal Cortex (VLPC): The importance of the VMPC to normal cognitive and social functioning has been known since the unfortunate circumstances of Phineas Gage. The importance of the VMPC and VLPC to social cognition has been explored extensively both in brain damaged and later in autistic populations. Recent research demonstrates right hemisphere dominance for the role of the VMPC in the empathetic response in normal subjects (Adolphs, 2009; Shamay-Tsoory, et al 2003). Early damage to the VMPC results in more serious effects that later damage and according to Adolphs (2009), the early onset damage results in individuals who never acquire social knowledge, don’t have access to emotional processing, and therefore suffer from even larger social cognitive impairments as they mature.

The VMPC is part of a system that involves the cingulate cortex that allows the appreciation of ‘self’ by the VMPC as opposed to ‘other’ by the cingulate, thus supporting social skills and social cognition. For individuals with autism the VMPC is activated both for ‘self’ and for ‘other’. Those subjects whose VMPC made the least distinction between ‘self’ and ‘other’ were the most socially impaired. Those whose VMPC made the greatest distinction between ‘self’ and ‘other’ were the least impaired (Lombardo, et al, 2010).

The VLPC is part of a facial processing and social cognitive circuit which also includes the amygdala, the fusiform face area, and the superior temporal sulcus. Individuals with autism showed under activations of this entire circuit. The reduction of activation of the VLPC as well as the amygdala led the authors to speculate that individuals with autism are not able to assign emotional significance to faces and that coupled with the under activation of the other components of the circuit could result in the inability to make complex social judgments and thus contribute to significant deficits in social cognition abilities (Pinkman, et al, 2008).

Fusiform Face Area (FFA) and Superior Temporal Sulcus: Both these areas are more responsive to the human face than to any other visual stimuli. Normal functioning of both these areas is critical to normal emotional reaction and social interaction. Studies show that both of these areas are significantly under activated in autism (Humphreys, et al, 2009; Schultz, et al, 2003). These authors suggest that under activation of the FFA in autism reflects the core social cognitive deficit underlying the disorder.

The Mirror Neuron System (MNS): Researchers consider the MNS to be important for the development of theory of mind, empathy, and normal social functioning and communication.
Areas considered as integral to this system will be reviewed as they have been shown to be under activated in autism (Verhoeven, et al, 2010)

References for the Summary
(List does not include the references for the full presentation)


Learning Outcomes

By the end of the presentation participants will be able to:

1. Discuss how the brain structures and areas presented contribute to normal social cognition.
2. Describe how social cognition is impaired in individuals with autism spectrum disorders.
3. Discuss how deficits in those brain areas presented impair social cognition in individuals with autism spectrum disorders.

Bio Sketch:
Robert Logan, Ph.D. CCC-SLP, CCC-Aud, is Professor of Speech-Language Pathology at the University of Central Arkansas. He has presented at the national and international levels in the area of neurology and traumatic brain injury and the neurology of stuttering. He is the author of the text, The Three Dimensions of Stuttering: Neurology, Behavior and Emotion (2nd edition). His interests have recently tuned to autism and he is currently instructing the Doctoral Seminar, The Neurology of Autism Spectrum Disorders, for the University of Central Arkansas/University of Arkansas at Little Rock/University of Arkansas for Medical Sciences Consortium Doctoral Program in Speech-Language Pathology.

Sharon J. Jones, M.S./CCC-SLP is a clinician with nearly 20 years experience working with children with developmental disabilities. She is currently working towards her doctoral degree in Communication Sciences and Disorders, with a primary focus on autism spectrum disorders and a secondary focus on neurophysiology of autism spectrum disorders.