Cognitive neuroscience is now able to provide a lens through which to view severe psychological disorders; in particular, theory of mind presents a way to envision the silent inner realm of the autistic child. Autism is a pervasive developmental disorder characterized by impairments in behavior and cognition including reduced social interaction, impairment in both verbal and nonverbal communication, marked absence in imaginary or “pretend” play, and a severely restricted range of interests. What causes these unique deficits? David Premack originally coined the term “theory of mind” to denote the human ability to represent and make inferences about the mental states of the self and others, encompassing desires, intentions, and beliefs (Gazzaniga et al., 1998). Researchers point to a deficit in theory of mind to account for behavioral impairments in autism. “The autistic child . . . does not form a mental image of what can go on in other people’s heads and this stems theoretically from a failure in thinking about his or her own mental states” (Trevarthen et al., 1996, p. 52). This paper attempts to integrate the research showing that pervasive impairments in theory of mind are correlated with damage to the parts of the brain related to higher cognitive functions, thereby profoundly affecting the autistic child’s functioning in a social world.

Deficits in Theory of Mind

As defined by Baron-Cohen in 1985, theory of mind refers to the process of attributing mental states to others in an effort to explain and predict social behavior (Happé, 1995). Mental states are “propositional attitudes . . . such as ‘think’, ‘hope’, ‘intend’, ‘wish’, and ‘believe’” (Happé, 1995, p. 39). Specifically, theory of mind relates to two main abilities: to “attribute a mental state or belief to another person [first-order attribution], and, at a higher level, predict what a person thinks about another person’s beliefs or thoughts [second-order attribution]” (Temple, 1997, p. 305). In normally developing children, theory of mind manifests itself between the ages of three and five (Gazzaniga et al., 1998). Children then recognize that others have a unique mental state which includes beliefs and desires about the world (Happé, 1995). In order to recognize mental states, the child must also see that the same awareness applies to the self; however, this recognition develops much earlier, between 18 and 24 months (Gazzaniga et al., 1998). Sigman and Capps (1997) define theory of mind as a relative progression in social understanding that stems from an intrinsic ability to conceive of one’s own thoughts and those of others in terms of mental states. To achieve the understanding, a child must recognize that other people have knowledge, beliefs, and desires different from one’s own. Only then can the child become interested in differing points of view.

Leslie was the first researcher to relate behavioral deficits to cognitive skill deficits in autistic children (Happé, 1995). Looking at the incidence of pretend play, Leslie notes that in order for a normal child not to confound his/her knowledge of the world, that child must have the ability to form representations about reality. In particular, the healthy child has “not only primary representations of things as they really are in the world (with a premium on accuracy and truth), but also metarepresentations which are used to capture pretending” (Happé, 1995, p. 39). A metarepresentation includes the formation of an “expression” not based in reality in an attempt to understand an agent and the object to which that agent refers (Happé, 1995). Because the ability to pretend is distinctly difficult, Leslie theorizes that autistic children are impaired in their ability to form metarepresentations. Metarepresentations are vital components in the representation of mental states, both of the self and of others. If autistic children are deficient in metarepresentations, it follows that they cannot realize the existence of a mental state beyond their own. They are unable to “mind read” like their healthy counterparts. The severe limitation in theory of mind is an obstacle to understanding the behavior of others (Happé, 1995).
Theory of mind is not easily measured by observation or overt behavior (Temple, 1997). Quantifying it is a challenging task because there are no clear ways to gauge if a child is thinking about another’s mental state. In 1978, philosopher Daniel Dennett pointed to the recognition of one’s “false beliefs” as a method for understanding and predicting that person’s mental state and his/her resultant behavior (Happé, 1995). Tests that measure these beliefs do exist and usually involve a story centered on a character. Knowledge of false beliefs is tested in such a way that the subject cannot interpret the behavior of the character through the use of personal convictions or the reality of the situation; instead, attribution to mental states of the model is required.

Regarded as the most influential test for deficits in theory of mind, the Sally–Ann Task provides a tool with which to measure a child’s ability to recognize false belief through the first-order attribution. Wimmer and Perner designed this cognitive analysis in 1983:

“In this task the child is shown two dolls, one called Sally, and one called Ann; Sally has a basket and Ann has a box. The child watches as Sally places her marble in the basket and goes out. While she’s out, naughty Ann moves Sally’s marble from the basket to her own box, then she goes out. Now Sally comes back in. The child is asked the question, ‘Where will Sally look for her marble?’” (Happé, 1995, p. 40).

The child is asked to make an inference about the mental state of a character; in this case, it is an inference about the character’s belief of the world. The incorrect response is that Sally will look for the marble in the box, thus relying on his own mental state to make the determination. Healthy and Down’s syndrome children over the age of four tend to be successful on these tasks by making correct attributions, while those under four base their judgments in reality and on personal knowledge (Sigman & Capps, 1997). Down’s syndrome children are tested to exclude the possibility that the characteristic low IQ of both Down’s syndrome and autistic children does not cause the error. Baron-Cohen finds that 80 % of children with autism fail this test (Happé, 1995), while Ozonoff finds a 50 % failure rate (Temple, 1997).

Another first-order attribution task is the “Smarter’s” test (see appendix i), in which a child must infer what another child will believe about the world, knowing this belief will be false. Autistic children tend to fail this task, while healthy and Down’s syndrome children again pass after the age of four (Happé, 1995). The numbers show that children with autism seem unable to differentiate in first-order attribution. The ability to recognize another’s differing view about the world is impaired (Sigman & Capps, 1997).

Examining second-order attribution, or the ability to decipher what one person thinks about another person’s beliefs or thoughts, requires a different test. A story is told about two children in the park (see appendix ii). The level of second-order attribution skill is measured through questions about the story (Temple, 1997). This task involves the ability to recognize one person’s (false) belief about another person’s belief of the world. Healthy children understand this type of representation between the ages of five and seven. Baron-Cohen posits that autistic children have a 100 % failure rate for this task (Happé, 1995) while Ozonoff and colleagues find an 87% failure rate (Temple, 1997); this ability is severely impaired. Many researchers point to an inability to understand one’s own mental state as an explanation for the inability to understand the mental states of others (Trevarthen et al., 1996).

These deficits do not appear to be a mere developmental delay in young children with autism; often attribution abilities never develop (Temple, 1997). However, several high functioning children with autism have passed tests of representation. Autistic children able to pass some of these tests may do so because they use skills other than mental representation to work their way through the situations. Some feel that these children find a “task-specific” strategy to solve these tests. In the real world, however, deficits impair social functioning (Happé, 1995). Task-specific strategies also break down in the face of more difficult attribution activities. Autistic children are adept at utilizing pictorial representations of the world because these do not require an understanding of how another person thinks (Happé, 1995). Suggesting that autistic children have the capability to use non-mentalistic forms of representation in understanding the world implies that the central deficit is mentalization ability (Gazzaniga et al., 1998). In 1993, Perner proposed that autistic children have theory of mind genetically, but lack the genetic predisposition to create an information system with which to build theory of mind (Temple, 1997).
Neurological Correlates and Theory of Mind

"Autism as a form of neuropathology seems to be highly specific: an impairment in the brain system’s underlying theory of mind” (Baron-Cohen, cited in Gazzaniga et al., 1998, p. 544). Biologically, theory of mind and other higher-order cognitive functions develop with maturation of the frontal lobes of the brain (Zilbovicius et al., 1995). The construction of metarepresentations depends primarily on metabolic activity in the frontal lobes (Temple, 1997). Specifically, the prefrontal cortex is linked to theory of mind. Lesions in this area of the brain result in deficiencies in planning ability, problem solving skills, and most importantly in the recognition of mental states in others (Bear et al., 1996). Positron emission tomography (PET) and single photon emission computed tomography (SPECT) scans show reduced blood flow to frontal areas in autistic children and young adults (Temple, 1997). Recent research provides evidence of delayed maturation of the frontal lobes in postnatal development. This regional delay in development is also related to delayed cerebral blood flow to the frontal lobes (Zilbovicius et al., 1995).

Zilbovicius and colleagues (1995) provide evidence for a transient hypoperfusion, or pervasive delay in development, in the frontal lobes of children with autism. At the global level in a healthy brain, rapid synaptic proliferation in infancy and early childhood is balanced by synapse elimination and stabilization throughout later childhood. Regional development of the brain follows a different course: there is rapid synaptogenesis in cortical areas followed by an evolution to an adult pattern between 16 and 24 months. The frontal cortex is the last region of the brain to evolve into adult patterns. In autistic children, there is strong evidence that this maturation is severely delayed. The hypoperfusion manifests itself between the ages of three and four, reaching normative values by age six or seven. Because regional maturation of the frontal cortex is positively correlated to development of specific cognitive skills, it follows that there should be a delay in the development of these cognitive tasks in autism. Specifically, cortical synapses in the prefrontal cortex mediate these cognitive tasks, corroborating the idea that autistic theory of mind deficits exist because of abnormalities in this region. Other abnormalities of the cortex include atypical metabolism levels and deviant patterns of cortical activation in response to sensory stimuli; this suggests abnormal cortical connectivity and activation within the prefrontal cortex (Zilbovicius et al., 1995). As a result, the shaping of neuronal connections is pervasively damaged by abnormal development of the frontal cortex in early childhood, perhaps between the ages of two and four. If the frontal cortex is delayed in its development, the neural networks connected to complex information processing are also unable to develop:

"Delayed metabolic maturation of the frontal lobes constitutes an important link between cognitive deficits and brain dysfunction in childhood autism . . . . The frontal lobes of autistic children do not follow a normal maturational pattern, and this may be one reason they fail to learn how to interact socially and to communicate normally” (Zilbovicius et al., 1995, p. 5).

Damage to the prefrontal areas early in development is also strongly linked to deficits in interpersonal role-taking and empathy (Temple, 1997).

In addition to the prefrontal cortex, other areas of the brain are implicated in the etiology of autism. Piven and colleagues (1995) note an increase in the size of lateral ventricles and in the density of brain tissue, resulting in increased brain size and volume. He hypothesizes that differences in neuronal density exist because of a decreased rate of neuronal death, increased neurogenesis, or increased production of blood vessels and glial cells in children with autism. The increased density of neurons in the limbic forebrain represents abnormal development of the brain (Piven et al., 1995). Interestingly, research on Einstein’s brain has revealed an increased density of glial cells per neuron in areas of the frontal cortex; Einstein presumably had a more intricate neural circuitry in these cortical areas (Bear et al., 1996). This may have interesting implications in relation to savant abilities and high IQ in some autistic children. It is also possible that deficits in the form of lesions are present in the mesolimbic cortex of the mesial frontal lobes. These deficits may be linked to deficits in motor skills, language, and goal-oriented behavior in autism (Temple, 1997). Courchesne et al. (1994) implicates the loss of Purkinje cells in the cerebellum, which leads to cerebellar hypoplasia (characteristically diminished development). The hypoplasia occurs early in development, specifically in the neocerebellar vermal lobules VI and VII (Sigman & Capps, 1997). These losses contribute to deficiencies in attention, intentional motor behavior, emotion, oculomotor function, dopaminergic activity and serotonergic activity, among other things (Courchesne, 1989).
Behavioral Correlates and Theory of Mind

Impaired theory of mind and the associated brain dysfunction is central to explaining the development of idiosyncratic abnormalities in autism. An intriguing manifestation of this deficit lies in the expression of extraordinary talent in "savant" abilities, presumably beyond the cognitive capacities of these individuals. In particular, autistic children may express such exceptional behaviors as "islets of abilities, good rote memory . . . and above-average IQ" (Happé, 1995, p. 43-44). These exceptional abilities do not require theory of mind. Instead, the essential component is primary representation, which merely requires a knowledge of the world, not of mental states. Although these non-mentalistic capabilities have not been explored, several methods of association are believed to be present. Data shows that autistic children have no difficulty with pictorial representation; perhaps savant abilities are based on this type of association. These children are adept at associating people with objects rather than mental states, another possible method of success for the "talented minority" (Happé, 1995). The presence of increased neural connectivity in both the autistic brain and Einstein's brain may account for high intelligence and savant abilities in autism. Some believe that Einstein had profound abilities in visual-spatial representation; if this is also the case with the autistic child, it may explain the child's capacity to employ pictorial representation in savant talents. Despite the presence of astounding abilities, devastating impairments that limit social interaction and normal development are more likely to occur.

The three most prominent behavioral deficits present in the autistic child are the absence of pretend play, limitations in social functioning, and lack of verbal and non-verbal communication skills. As Leslie noted, theory of mind is a cognitive ability that precedes the normal appearance of symbolic play in early childhood (Trevarthen et al., 1996). Although some autistic children can pass tests of first-order attribution, deficiencies in second-order attribution seem more pronounced and universal. Symbolic pretence does not appear in autistic children because of the inability to make these higher level metarepresentations (Trevarthen et al., 1996). Conversely, lack of pretend play reflects a lack of awareness of mental states, because imaginative skills require that the child recognize the internal mental states and individual thoughts of others. The child must also recognize his or her own mental state to engage in pretend play. This function is also impaired in autism, resulting in a complete absence of pretend play (Sigman & Capps, 1997).

The conception of mental states and the recognition of personal and external thoughts are essential to understanding the motives behind a social encounter and attributing them to the proper character. To develop socially, the child must recognize that others have beliefs about the world in the same way as they themselves do, but that these beliefs are inherently different from their own. Normatively, middle childhood signifies the period in which children become interested in external points of view (Sigman & Capps, 1997). This does not happen with autistic children. Theory of mind deficiency is directly related to social functioning because the differentiation of self-knowledge from the knowledge of others is tied to learning the conventions of society (Sigman & Capps, 1997). Leslie argues that the autistic child does not necessarily have a problem in every social situation, but rather, "has difficulties in social situations where it is necessary to take into account what someone else knows or expects" (Temple, 1997, p. 305). Second-order attribution skills are particularly related to the development of socioemotional relationships. "Autistic children's inability to perceive relationships between [persons] might contribute to their failure to develop social skills . . ." (Trevarthen et al., 1996, p. 51). Second-order attribution is imperative in both the development of empathy and the differentiation of the self from others in the cognitive, emotional, and social spheres (Sigman & Capps, 1997). Because these mentalizing capacities are hampered in autism, the autistic child cannot engage in such behaviors.

"The ability to mentalize is used not only for predicting how a person will behave, or what a person wants or thinks, but also for understanding what a person means" (Happé, 1995, p. 76). Inability to form mental representations is invariably manifested as the absence of communication skills in autism. Normative communication is more than verbal language; non-verbal forms play a major role in how we relate to other people. Those actions presented along with verbal speech are evidence for our intended meaning are termed "ostensive behavior" (Happé, 1995). Recognition of meanings behind ostensive behavior involves recognition of the mental state creating it. Because communication is a display of mental states, it follows that children with autism, impaired in their ability to represent mental states of the self and the other, are also impaired in their ability to engage
in communication. Happé has related the motivation behind communication to theory of mind. Through a series of stories, the autistic child is asked to interpret why a character says something that is not literally true. She finds that, although they respond with answers describing mental states, autistic children use terms inappropriate in context. Autism invokes a certain "idiosyncratic view of events, and the relative difficulty . . . of attributing mental states . . . makes constructing an elaborate and unusual physical explanation the preferred, easier, or perhaps only, option" (Happé, 1995, p. 81). The inability to understand minds in action is, on broader terms, an inability to understand minds in communication. Happé finds that autistic children have a very difficult time integrating elements of a social situation in order to elucidate the motivation of the speaker, resulting in an undermined ability to communicate ostensively.

Theory of mind presents an intriguing look into the silent world of the autistic child. It appears that these children do not have the capacity to see that others think about the environment in ways different from themselves. They cannot distinguish their own mental state, a task believed innate to a developing child. New and compelling research is breaking ground in an area that does a more thorough job of describing impairments than it does finding their cause. The evidence increasingly implicates neurobiological roots for the theory of mind deficit, and its resultant social, language, and imaginative impairments. Antonio Damasio is studying the idea of dual consciousness and its representation in the brain. He posits that there is a primary consciousness, termed the "protoself," which provides an understanding of the self in relation to the environment. There is also a secondary, extended consciousness. Patients with damage to the prefrontal lobe are still able to recognize the self; the protoself is still intact. With this damage, however, the second consciousness is destroyed, and problems in social behavior come into play. He believes the location of the second consciousness to be in the cingulate cortex (Nash, 1999). Perhaps, in autism the theory of mind deficit, manifested as social inaptitude, is actually the result of an impaired dual consciousness. Whether this is a viable theory is uncertain, but what is certain is that the future is wide open to research into the deficits characterizing autism using this novel perspective. Future research must delve further into the brain to glimpse at the dysfunction underlying the deficiency. Looking through the lens of cognitive neuroscience, perhaps there will one day be a way to help those children who may otherwise remain isolated for the course of their lives.

ACKNOWLEDGEMENTS

I would like to thank Professor Janine Scheiner for her assistance and support in preparing this article for publication.

REFERENCES


APPENDICES

Appendix i: Smaties test: First-order attribution
The child is asked to guess what a closed Smartie tube contains. Having answered ‘sweets’ or ‘Smaties’ the tube is opened to show the real contents, a pencil. The lid is then replaced and the child asked, ‘When Billy comes in, I’m going to show him the tube, closed up like I showed it to you. I’m going to ask him what he thinks is inside. What will he say?’

Cited in Happé, 1995, p. 42
Appendix ii: Second-order attribution test

This is Mary and this is John. Today they are in the park. Along comes the ice cream van. John wants to buy an ice cream, but he has left his money at home. He'll have to go home first and get his money before he can buy an ice cream. The ice cream man tells John, “It’s alright John, I’ll be here in the park all day. So you can go and get your money and come back and buy your ice cream. I’ll still be here.” So John runs off home to get his money.

But when John has gone home, the ice cream man changes his mind. He decides he won’t stay in the park all afternoon, instead he’ll go and sell ice cream outside the church. He tells Mary, “I won’t stay in the park, like I said. I’m going to the church instead”.

Comprehension check 1: Did John hear the ice cream man tell Mary that?

So in the afternoon, Mary goes home and the ice cream man sets off for the church. But on his way he meets John. So he tells John, “I changed my mind, I won’t be in the park, I’m going to sell ice cream outside the church this afternoon”. The ice cream man then drives to the church.

Comprehension check 2: Did Mary hear the ice cream man tell John that?

In the afternoon, Mary goes over to John’s and knocks on the door. John’s mother answers the door and says, “Oh, I’m sorry Mary, John’s gone out. He’s gone to buy an ice cream”.

Belief question: Where does Mary think John has gone to buy an ice cream?
Justification question: Why does Mary think that?
Reality question: Where does John really go to buy his ice cream?
Memory question: Where was the ice cream van in the beginning?
Cited in Happé, 1995: 69

ABOUT THE AUTHOR

Neha M. Shroff is an '02 majoring in Psychological and Brain Sciences and minorig in Genetics. She is currently involved with ArtCare and the EEG Lab in the Psychology Department. After graduation, Neha plans on attending medical school, where she will focus on pediatric oncology, eventually entering research and practicing as a doctor in the field.