Abstract: This literature review examines the history and pertinent research on Autism, a brain development disorder characterized by social impairment, communication difficulties and ritualistic behavior, and Theory of Mind, the ability for one to impute mental states to the self and to others. An introduction to these topics is followed by an investigation of whether Theory of Mind is missing in cases of autism, whether it is truly a core deficit of the disorder and what the ramifications are if this is the case. Also examined is whether this deficit is also present in cases of Asperger's syndrome, where language is not delayed and IQs are usually high, as there has been controversy in this area. Possible biological connections are examined, including the Mirror Neuron system. A look at the world of treatment and adaptive technology solutions for the deficit is also undertaken.
Introduction

Theory of mind (ToM), the ability to impute mental states to the self and others and make reasoned decisions based on this information, is a cognitive skill usually found in typical children by the age of four or five years old. It is now believed to be a core deficit in cases of autism, a neurological disorder characterized by social and communication impairment, repetitive behavior and delayed speech. A great deal of research has been done in the last thirty years that focuses on this area. Do autistic children truly have an inability to imagine what someone else is thinking? If so, what does this tell us about the autistic child's brain? Are higher functioning people with autism and people with Asperger's, a condition on the autism spectrum with no delays in speech or cognitive development, also without ToM? What are the implications when you lack these skills? It is generally accepted that autism is biological in origin, so is there an area in the brain where ToM is processed that's defective in cases of autism? What biological component leads to this psychological phenomena? Is there a way to remedy this situation and teach ToM to children who do not have it develop naturally?

In this review all of these questions are examined. The topics of autism and theory of mind are introduced and then brought together in an examination of the work done on this topic over the last thirty years. A look at the discovery of the phenomena and possible reasons for why it exists are undertaken. Asperger's and high functioning people with autism are also examined as far as ToM is concerned, as their stories differ and the debate as to whether they show the same deficits as classic autism is more heated. An inspection of some of the biological reasoning for this condition, a consideration of the neurology, looks at the possible existence of a neural substrate that could be responsible for ToM. The mirror neuron system that may play a deep role in social cognition, ToM and autism are examined. An analysis of treatment and education tools to address this deficit follows.
The story of ToM deficits in cases of autism is part neuroscience, part psychology, part medicine and part detective story. At the end of the day, it's the story of researchers and scientists trying to get to the root causes of autism so we might understand this condition better and, perhaps, find a way to stop it.

**Autism**

In 1943 Dr. Leo Kanner published a report on eleven of his patients at a Baltimore clinic. Entitled, “Autistic Disturbances of Affective Contact.” (Kanner, 1943), this seminal paper became the base for all study on what came to be known as autism. It is a disorder characterized by deficits in three major areas of behavior - social, communicative and the display of repetitive or restricted behaviors. The DSM-IV-TR lists the essential features as “the presence of markedly abnormal or impaired development in social interaction and communications and a markedly restricted repertoire of activities and interests” (APA, 1994). The social problems include less eye contact, less attention to social stimuli and difficulty in learning and using the social skills needed to function in society. The communication problem is due to a significant delay in language development seen in classic cases of autism. This varies on a case by case basis and if speech does develop it is often joined by echoalia, a simple repeating of what's just been said by someone else. Lastly, and perhaps most often identified with autism, is the repetitive or ritualistic behaviors that manifest. Behaviors such as wanting the same foods and cloths everyday, organizing things into lists and insisting on sameness, resisting any change in routine or schedule. All of these symptoms are the hallmarks of classic autism.

In the early part of the last century autistic children were often misdiagnosed as being schizophrenic (Grinker, 2007). Four times as many males have the condition than females. While
autism was once thought to be very rare (4 in 10,000), autism is now thought to be very common (possibly 1 in 166) (Sicile-Kira, 2004). This is likely due to the fact that autism is now seen as a spectrum of disorders, from classic autism (sometimes called Kanner's autism) to Asperger's Syndrome to PDD-NOS, Pervasive Developmental Disorder Not Otherwise Specified. Autism is diagnosed based on behavioral symptoms and not with any objective biological means, a situation that would also bolster prevalence numbers.

The cause is almost surely a mixture of biological and environmental factors with genetics playing a large role. In the 1960s, psychoanalyst Bruno Bettelheim portrayed autistic children as unreachable and blamed their condition on unaffectionate “refrigerator” mothers. Because children with autism often look perfectly normal physically it was thought that the condition was wholly psychogenic, or purely in the mind (Baron-Cohen, 2008). Brain scanning technology has allowed science to probe the autistic brain and discover that there are many physical differences when compared to typical brains. Children with autism will have more grey and white brain matter as youngsters. Eventually, the grey matter will stop increasing but white matter will continue to increase, giving an autism patient more connective white tissue than “neurotypical” people throught their lives. Often the amygdala and the hippocampus of the autistic patient are smaller than average. This may play a role in some of the social deficits seen later in an autistic child's life. Recently, there has been a scare concerning child vaccination and whether vaccines could play a role in autism. Spurred on by one faulty study, the idea that vaccines can cause autism has no evidence behind it and is now seen as biologically implausible (Paul, 2009).

Very few people with severe to moderate autism live independently, but some are very successful (Howlin, Goode, Hutton & Rutter, 2004). Temple Grandin, an autism advocate and author
who doubles as a doctor of animal science at Colorado State University. Peter Tork of the Monkees rock and roll band has Asperger's Syndrome. Jason McElwain, a young man with autism who was the team manager for his high school basketball team, got a chance to actually play at the very end of the team's last game when he was a senior. With only four minutes left in the game he scored twenty-three points and hit a school record six three pointers, lighting the basketball world on fire in the process (McElwain & Paisner, 2008). Unfortunately, stories like those above are few and far between.

There remains no cure for autism. New treatment techniques and early intervention strategies give hope for the future, but the prognosis for a patient with autism is mixed. Sometimes a child can “recover” or lose their diagnosis, but this is rare and in most cases due to a faulty initial diagnosis. If one acquires speech and has an IQ above 50 their chances for the future improve drastically, but below those figures resides a life of dependence. Autism is a devastating and debilitating condition that continues to perplex the fields of medicine, neurology and psychology.

**Theory of Mind**

In 1978 a research paper was published that asked the question whether a chimpanzee could imagine what someone else was thinking and use this information to alter its behavior (Premack & Woodruff, 1978). In this paper, the ability to impute mental states to the self and to others was given a name, “Theory of Mind.” It's most often described as, “The ability to put yourself in someone else's shoes” and usually develops in typical children by four or five years old. It is considered a high-order cognitive feat. Many claim to this day that ToM is present in chimpanzees (Call & Tomasello, 1998) as well as in humans.

Early in an infant's life there are many mechanisms that help us support social cognitive
understanding, things like joint attention, gaze following and monitoring goal-directed actions. As these mechanisms allow us to gain information the developing infant will soon start making inferences and begin to expect people to start looking for items where they last left them at fifteen months (Goswami, 2008). In order to do this one must be able to represent another person's thoughts in their minds, they must have a way of treating this abstract information as an object in their mind. This is called metarepresentational ability.

In his 1987 paper, “Pretense and Representation: The Origins of “Theory of Mind”, Dr. Alan Leslie examined the ability of infants to pretend and still manage to avoid the contradictions with reality as the infant dealt with pretense (Leslie, 1987). He presents the idea of primary representations, our ideas that represent the realities of our world. In order for a child to pretend, the example used in the paper is to have a child pretend that a banana is a telephone, the child needs to decouple from the primary representation and form a secondary representation that's not necessarily in line with reality, a representation of a representation, or a metarepresentation. This secondary representation does not reflect the real world as the primary does, it is not veridical as a banana is certainly not a phone. It shows that the holder is able to suspend the primary representation, quarantining it, and use this secondary representation for the pretend play.

What does this have to do with ToM? Leslie states that when one thinks of representation in terms of mental states, people's beliefs and desires, they need to deploy metarepresentations because reality may or may not be in use. The example used is, “John believes it's raining and jumps into a shop doorway because he doesn't want to get wet.” It doesn't matter if it's raining or not, John believed it was and this belief dictated his actions. Leslie posits that a secondary representation must be at play here for us to consider this situation properly and that to use this “opaque” logic to impute actions is to
employ ToM. He believes that pretend play is the beginnings of ToM as both participants in a banana phone game are sharing the pretense that the banana is a phone. A child understands that another is representing the banana as a phone for the game just like they are, and therefore have some insight as to what the other child is thinking.

It is generally agreed that ToM is not present at birth and develops slowly (Goswami, 2008). While the ability to have two different representations of the same thing in one's mind is the beginning of ToM, the situation quickly becomes more complex. Leslie states that an example of complex reasoning across metarepresentational structures is the ability of a child to predict the behavior of someone who holds a false belief (FB) (Leslie, 1987).

The philosopher Daniel Dennett of Tufts University, in his book “Brainstorms”, was discussing the original ToM chimpanzee paper of Premack & Woodruff and stated that the only convincing evidence that he could think of for an ape successfully attributing a mental state to another is if they could predict the actions of someone who held a belief that wasn't true (Dennett, 1978). He said that anyone can predict a belief based on reality, but if we can figure out what someone would do if they were in possession of bad information, that would show that you that they were representing another person's mental state in their minds.

Along these lines, Heinz Wimmer and Joseph Perner devised a test, a “false belief” test, that has come to be seen as the classic ToM test for children. That children aged three to four years old have no understanding of FB was a belief that was widely held in the early eighties (Wimmer & Perner, 1983). Understanding FB was considered a qualitative shift in cognitive development and Wimmer and Perner looked for a way to detect this shift. They devised a test where a child would hear the story of Maxi, a story character that leaves a piece of chocolate in a cupboard. Before going out to play Maxi leaves his
chocolate in cupboard X. While he is outside his mother comes along and moves his chocolate to
cupboard Y. Maxi then comes back inside the house after playing. The children being told the story
are then tested by being asked where Maxi is going to look for his chocolate. This is the key point,
where a child has to choose between what he/she knows is reality, that the chocolate resides in cupboard
Y or impute from Maxi’s false belief that Maxi is going to look in cupboard X. The results of the study
showed that children at or under the age of four performed at chance, no better, while children aged five
to six years old performed at a 92% success rate and said that Maxi would look for the chocolate in the
last place he knew it to be, cupboard X. It seemed that the ability to represent a false belief was
something that did not develop until one was five years old.

It was not long before people began to express worry about the Maxi FB test. Some stated that
it had a reliance on language that a three year old might not be able to negotiate and that other research
was showing vestiges of ToM in children much younger than four years old. Was the Wimmer &
Perner FB test the end all be all of ToM testing or was it fundamentally flawed? The truth likely lies in
the middle as passing a FB test does show a certain level of ToM but does not mean the phenomena is
completely absent if one fails it. The language objection is something we examine later in this review
as a delay in language is a defining symptom in cases of autism and the use of FB tests in autistic
spectrum disorders (ASDs) is not without controversy. Other mental representation tests, Flavell's
appearance-reality tests (Flavell, Flavell & Green, 1983) and the “False Photo” test of Zaitchik (1990),
are widely used tests to see if a child has issues with representations, but the FB test is still considered
the gold standard for testing belief representation and ToM.

Having a ToM and passing the false belief test is considered a milestone in social cognitive
development. In the early eighties, when a growing awareness of autism and ASDs began, people
began to make a connection. If a core symptom of autism is a lack of social skill and social
development and one of the key milestones for social cognitive development is ToM and false belief
understanding, then it might be informative to see how ToM develops in cases of autism when
compared to typical children.

Autism & Theory of Mind

In “Does the autistic child have “theory of mind”?, a research paper by Simon Baron-Cohen,
Alan Leslie and Uta Frith (1985), the authors set out to discover if ToM was deficient in cases of autism
and if it was, to show that it was a phenomena that was specific to autism and independent of mental
retardation. The theory held by the authors was that children with autism lacked the ability to create
and use secondary representations and that this deficit was not due to mental retardation, but to an as
yet undetermined deficit in a ToM mechanism unique to autism. A few things lead them to this
hypothesis. One, if Leslie’s connection of secondary reps and pretend play was on track (This paper
was published two years before Leslie's 1987 Metarepresentation paper, but major parts of it are
included here in 1985, he had already developed the major ideas) then a lack of secondary
representations would help explain the lack of pretend play in cases of autism. Pretend play is not
lacking in classic mental retardation cases. Even high IQ cases of autism lack pretend play but severely
retarded Down's syndrome children do not (Hill & McCune-Nicholich, 1981). Secondly, a lack of
secondary representations would lead to poorly developed ToM and the social ineptness that this can
lead to. Social ineptness is a hallmark of autism. People with autism often report not understanding
how others think. One man was quoted as saying, “other people seem to have a special sense by which
they can read other people's thoughts” (Frith, Morton & Leslie, 1991). A lack of ToM could go a long way towards explaining some of the social deficits that appear in autism but not in other forms of mental retardation, such as the extroverted exuberance seen in cases of Down's syndrome.

Many other cognitive deficits had been proposed to explain the behavioral symptoms of autism in the past (Frith, et al, 1991). They included object permanence and perception, but when these aspects were investigated nothing solid could ever be found. ToM seemed tantalizingly like a cognitive deficit that could reasonably explain a good deal of the behavioral symptoms that appeared in autism.

The method of the aforementioned Baron-Cohen, et al., (1985) paper was simple. Twenty autistic children were administered a false belief task, with fictional Sally and Anne dolls taking the place of Maxi and his mother and a marble taking the place of the chocolate. As a control, twenty-seven clinically normal children were tested as well as fourteen children with Down's syndrome. The results were striking.

Twenty-three of Twenty-seven (85%) typical children and twelve of the fourteen Down's children (86%) passed the FB test while sixteen of twenty children (80%) with autism failed it. All of the children were tested for reality (where is the marble now?) and for memory (Where was the marble?) and all passed. There seemed to be no misunderstanding of language used during the tests, it seemed that the children with autism could not differentiate between their own knowledge and the doll's knowledge. The results seemed to confirm the hypothesis that there is a specific ToM deficiency in cases of autism.

Further confirmation of this result appeared in 1989 with the “Smarties” test (Perner, Frith, Leslie & Leekam, 1989), where a child is asked what will appear in a box clearly labeled “smarties.” Once the box is opened it is revealed that the box is full of pencils. The child is then asked two belief
questions. What did they think was in the box before it was opened, to which the typical child answers “smarties”, and what will the next child to walk in the room think is in the box? The answer given by a normal child with typical ToM will also be “smarties.” When this test was administered to autistic children they replied “pencils” to both questions, reflecting the reality as they knew it at that time and not their previous false belief or the false belief of the next child that would walk into the room.

Further investigation into this phenomena has shown that verbal ToM FB tasks can be passed by typical five year olds and still failed by adolescent autistics (Roth, 1991).

Soon, questions about the FB test's reliance on language began to crop up. If delayed language development is a hallmark of autism, people argued, why are you administering an FB test that relies on the understanding, some would say nuance, of language? How could you use FB tests like Maxi's to test autistic children without language? An estimated one-third of autism cases are without language (Colle, Baron-Cohen & Hill, 2007). Can FB ToM tests be trusted if a full one third of the autism population cannot be tested in these studies? These were valid issues and in order to combat them there have been some non-verbal tests developed. Picture sequences have been used to test ToM ability (Baron-Cohen, 1995) as well as a recent method that was used to test autistic children and children with cases of selective language impairment (Colle, et al., 2007). ToM deficiency in cases of autism were confirmed in both cases.

It seems that ToM specifically is what is at issue here as children with autism pass many other perception and cognition tests they would be expected to fail if they were merely arrested in their development. They pass the perspective taking tests of the “three mountians” and the “line of sight” (Baron-Cohen, et al, 1985) and also pass Zaitchick's false photograph test (Leslie, 1992) that does not test belief, but the recognition that photos age.
ToM, considered a higher-order cognitive function, has lead some to attempt to dig even deeper, positing that a deficit in a child's basic attentional ability as young as eighteen months old may lead to the poorly developed ToM seen in the studies. In one study, a new method to screen autism at eighteen months old was used to identify and study twelve autistic infants to examine their attention shifting. It was found that even at twenty months old these children showed attentional deficits that might be responsible for the deficits in ToM seen later in their development (Swettenham, Baron-Cohen, Charman, Cox, Baird, Drew, Rees and Wheelwright, 1997). This could also explain the failure of autism infants to maintain joint attention (Baron-Cohen, 1995).

What does the discovery of a ToM deficit in cases of autism mean? In a disorder with multiple possible biological causes and a myriad of different behavioral outcomes, these studies may have found something that all ASDs share, a core deficit in a cognitive function. Dr. Uta Frith, John Morton and Alan Leslie explain it well by examining autism at four different levels (Frith, et al., 1991). The biological aspects of the disorder lead to cognitive deficits which lead to behavioral symptoms that lead, finally, to social impairment as the outcome. With multiple possible biological reasons (genetic, disease, etc), multiple behavioral outcomes (language delay, no pretend play, etc) and many social outcomes (inability to communicate, social ineptness) it seems that the only thing that is keeping autism as a single diagnosis is the facet that is shared by all cases, a lack of fully developed ToM at the cognitive level. It forms a bottleneck that leads one to believe that all of the possible biological reasons may be attacking a single system that's responsible for ToM and that this system's lack of proper development is responsible for most of the behavioral outcomes. If true, this could lead researchers in fruitful directions and help focus a search for future understanding or a cure.

The implications of a modular ToM system will be discussed later when the biology of autism
and ToM is discussed. First, an examination of an aspect that can do great damage to the framework given above needs to be discussed. It is generally believed that there is a core deficit in ToM in classic autism, but does the same apply to all disorders on the autism spectrum, specifically cases of Asperger's?

**Asperger's**

In 1944, a year after the publication of Leo Kanner's paper on autism, a doctor named Hans Asperger published a paper in his native German about a high functioning group of children with many of the symptoms of classic autism, but some differences as well. Imagine a case of autism without the delay of language and with an IQ at or above the average. Having an excellent memory and an attention for detail this group of children still had some of the symptoms of classic autism, a desire for sameness, ritualistic behavior and narrow interests. In 1981 when Dr. Lorna Wing suggested that autism was a spectrum and not a categorical diagnosis, she brought Dr. Asperger's ideas to the English speaking world (Woodbury-Smith & Volkmar, 2008). In 1994 Asperger's syndrome became a diagnosis in its own right (APA, 1994) and is considered the highest functioning condition in the autistic spectrum of disorders.

Noting that some of the key symptoms of Asperger's, ritualistic behavior, desire for sameness, are some of the aspects of autism that are not clearly defined as resulting from a lack of ToM, the scientific community began to wonder if the deficit in ToM found in classic autism really existed in cases of Asperger's. If it didn't, then the idea that lacking ToM was a core deficit across the entire Autism spectrum of disorders would be in jeopardy and the framework laid out by Frith, Morton and Leslie would crumble.
A Bowler study (1992) found that adults with Asperger's not only passed ToM tests, they passed second-order ToM tests, where a subject is asked to reason about the thoughts of another person. When asked to explain situations they did not use mental state terms, but they did score just as well as the controls in the study's experiments (Bowler, 1992). If this were true it would lead one to admit that a ToM deficiency is not a core deficit across the ASDs. The idea that there is a convergence across the spectrum at the cognitive level of the disorder would disappear and justification for calling all of the conditions along the spectrum by the single term “autism” would be questioned (Frith, et al., 1991).

Dr. Simon Baron-Cohen, Professor of Developmental Psychopathology in the Departments of Psychiatry and Experimental Psychology and Director of the Autism Research Centre at the University of Cambridge, came to the theory's rescue. He noted what he saw as a flaw in the Bowler study. Adults with Asperger's syndrome were passing second-order ToM tests that were actually designed to probe for ToM in a typical child at the age of six. Children over the age of six will often produce ceiling effects if these second-order ToM tests are used. He stated that neither first nor second-order tests were truly complex tests of ToM and that, “finding a 30-year-old individual with autism, of normal intelligence, who can pass a theory of mind test at the level of a normal six-year-old does not lead to the conclusion that they are necessarily normal in this domain” (Baron-Cohen, Jolliffe, Mortimore & Robertson, 1997).

What was needed was a test to detect a ToM deficiency in adults. The Happe Strange Story task was introduced in 1994, and tests a subject's ability to understand a non-literal statement (Happe, 1994). This test was aimed at eight or nine year olds and was seen as a being more advanced than previous ToM tests. Dr. Happe found that adults with autism or Asperger's syndrome did have trouble with mental state stories while the controls in the group did not.
Baron-Cohen went further, developing the “Eyes Task”, where a subject has to infer the mental state of a person from just looking at a picture of the person's eyes (Baron-Cohen, et al., 1997). When this test was used, adults with autism and Asperger's were significantly impaired in ToM functioning. Therese Jolliffe and Simon Baron-Cohen returned to the Happe test and replicated her results in 1999 (Jolliffe & Baron-Cohen, 1999). The failure of autism and Asperger's subjects to perform on the “Eyes task” has been replicated as well (Kaland, Callesen, Moller-Nelson, Mortensen & Smith, 2008). New higher level ToM tests continue to be developed, like the ATOMIC, The Animated Theory of Mind Inventory for Children, that uses a computerised format that takes advantage of the special interest in computing that exists in the Asperger's community. ToM measures are lacking when this test is administered to adults with Asperger's as well (Beaumont & Sofronoff, 2008). There were some questions as to whether people with Asperger's syndrome had the same ToM deficiency seen in classic autism in the years after the ASD-ToM theory was publicized, but further study has lead to the conclusion that while newer tools were needed to detect it, a ToM deficit does indeed exist in cases of Asperger's syndrome.

The Biology

At the very end of his seminal paper on autism, Leo Kanner finished his study (1943) with a rather subjective but telling note. He wrote, “One other fact stands out prominently. In the whole group, there are very few really warm hearted fathers and mothers... they are persons strongly preoccupied with abstractions of a scientific, literary or artistic nature, and limited in genuine interest in people.” Not as bad as Bruno Bettelheim, but one doubts whether such a seemingly subjective thing could be written in a published peer reviewed article today.
The reason the Kanner passage is brought up here is to discuss the point that autism and ASDs have long been thought, even in the days of Kanner, to have a large biological and/or genetic component involved in the cause. Twin studies and the fact that autism runs in families is convincing evidence of this phenomena (Baron-Cohen, 2008). Also seen within families that have members with autism is likely what Kanner is describing above, the Broader Autism Phenotype or BAP. This is when members of the immediate family of children with autism often show mild manifestations and some characteristics of autism, things like being socially withdrawn or mildly obsessive (Baron-Cohen, 2008).

What does this have to do with ToM? If ToM is a core deficit in autism then one might expect to see lowered ToM levels in family members as well. Just this phenomena was looked for and found in a study where relatives of individuals with autism exhibited lower than normal rates of ToM features such as reasoning about other's emotions and on mental state reasoning tasks (Gokcen, Bora, Erermis, Kesikci & Aydin, 2009). Knowing that deficient ToM is a trait that's also part of the BAP leads one to the question, “is there a physical basis for ToM deficits?”

Alan Leslie has proposed the idea of a domain specific processing module for ToM and understanding mental states in the brain (Leslie, 1992). If this theory of mind mechanism was part of the brain and it were damaged or developed abnormally, one might expect to see cases like those of autism. Children with autism can pass other tasks that require sophisticated reasoning, it's just belief that they have problems with, so the idea is reasonable. Leslie puts it this way, “I have argued that the normal and rapid development of theory-of-mind knowledge depends on a specialized mechanism that allows the brain to to attend to invisible mental states. Very early biological damage may prevent the normal expression of this theory-of-mind module in the developing brain, resulting in the core
symptoms of autism.” (Leslie, 1992)

This leads to the next question, is there really a neural substrate, something physical and tangible, in the brain for reasoning about beliefs? The idea that there is a specialized region in the brain just for representing beliefs has become very popular (Goswami, 2008). The massive leaps forward in brain imaging technology have allowed scientists and researchers to begin to probe the brain in new and unique ways. One can now take pictures of the brain and monitor its activity as it performs certain tasks. It was during an experiment of this type with monkeys that a system was identified that may have a great deal to do with ToM development, the Mirror Neuron system (MNS).

While studying the brain activity of primates the mirror neuron system was discovered when the subject monkeys were engaged in object directed actions (Iacoboni & Dapretto, 2006). These neurons fired not only when the monkey performed the action but also when the monkeys observed someone else performing the same actions. This was striking because it lead the scientists to believe that the MNS might have something to do with the way primates learn to imitate or code intentions. In the mid 1990s PET scans provided evidence for an MNS in humans. The suggestion was made that a fully developed MNS system is needed for early imitation and that a dysfunction in this system could lead to the deficits that constitute autism (Williams, Whiten, Suddendorf & Perrett, 2001).

Iacoboni’s (2009) contention is that if MNs fire when we watch an action and when we perform the action they likely have something to do with imitation. Not only that, but further studies have discovered that the MNS seems to be sensitive to biological beings, only firing for people performing actions and not machines, and that the same action with differing intentions fires off different neurons. Different mirror neurons fire when someone reaches out to drink from a cup of coffee than the neurons that fire when someone is reaching to pick up the same cup and put it in the sink. This was fascinating
because understanding intentions can be thought of as imputing a state of mind for the person performing the action, a definite vestige of ToM.

Could the MNS be the ToM mechanism proposed by Leslie? Currently this explanation is completely speculative (Goswami, 2008) and a few pieces of the puzzle, such as the fact that via echoalia and other behaviors autistic children are excellent imitators (Baron-Cohen, 2008), work against this theory. Still, many new studies continue to tie a dysfunction in the MNS to autism (Oberman & Ramachandran, 2007). As techniques for studying the brain continue to improve more information about the connection between the MNS and ToM is sure to be on the way.

Treatment and Adaptive Technology

Now that it has been established that people with ASDs, including people with Asperger's syndrome, have a diminished ToM and that the reason might be biological in origin there still remains one question, what can we do to improve the situation?

Early intervention in autism has been shown to improve cognitive skills and to affect progress in cognitive levels reached (Itzchak, Lahat, Burgin & Zachor, 2008) so the earlier in a child's life one looks for treatment and aids the better the future situation will be. To aid ToM performance there is one area where good results have been found, computer instruction. Unfortunately, using these methods usually require a child to be older, at least old enough to operate a mouse and a keyboard.

For young children there are DVDs and animation programs to improve ToM. A video series called “The transporters” features cars that move along in a fictional city, each with a face grafted onto the front of the vehicle (Baron-Cohen, 2008). As the cars go into different situations their facial expressions change and they talk to each other about how they feel in mental state terms.
Once children become a little older they can begin to use a computer. Many computer training programs and adaptive technology solutions targeted at improving ToM are in use today (Bernard-Opitz, Sriram & Nakhoda-Sapuan, 2001). One drawback noted is that working alone with a computer may not be the most social of experiences, but the skills learned in this one on one pressure free environment are preferred by people with ASDs (Siegal, 2003). Other advantages to using computer aided instruction for ToM training are that it's easier to screen out unnecessary sensory information when concentrating on the computer screen and that computers have explicit routines and clear step by step processing. Both these features are perfect for people with ASDs. Computer instruction is so widely favored among people with ASDs that it has been reported that they often prefer using the computer to playing with toys (Silver & Oakes, 2001).

Because people with ASDs have low ToM and difficulty recognizing or understanding emotions in others, computer training provides “Emotion training.” Software was developed and tested in England that displays a face and lets the user choose what emotion is being displayed. There is no pressure or score when going through a session. Wrong answers are just met with a small sign that says, “try again.” After twenty correct answers the student can go on to the next level. Here they are presented with a picture, a small story, and a question asking them how they would react to the situation. Again there is no pressure and no score. The session ends when twenty correct answers are tallied. A sample of twelve people with ASDs used the experimental ‘Emotion Trainer’ software and all displayed significant improvements in emotion recognition (Silver & Oakes, 2001). One piece of intervention software, Gaining Face (http://www.ccoder.com/GainingFace/), is a public program written to help children with autism or Asperger's to learn about facial expressions. While useful, this program has no published evidence or research as to its effectiveness.
Technology solutions for ToM and understanding others in social situations are not tied to the desktop, mobile devices are coming into wide use as well. One of the more interesting projects is the PARLE (Portable Affect Recognition Learning Environment) project (Bishop, 2003). People with autism tend to take things literally, too literally. If I were to say, “I would kill for a glass of lemonade”, a person with autism would likely be frightened, especially if they were holding a pitcher of lemonade. People with an ASD are sometimes made to feel socially inadequate when they aren't able to understand such idioms and begin to avoid situations where they might happen. This begins a downward spiral that hurts the social growth of the person with an ASD. PARLE provides a way to begin to combat this and begin improving a person's ToM.

PARLE takes techniques from Social Skills Training and put them in a hand held device. When involved in a social situation a person with ASD can take a step back, consult the PARLE system and get a response they understand. For example, when asking for help in a grocery store a man with an ASD might become quiet all of a sudden. The woman helping him says, “What's the matter, the cat got your tongue?” As you can imagine, a literal interpretation of this sentence would be a bit confusing. Enter PARLE. The user can enter the phrase into a mobile Internet based phone that connects to a database system that fields the man's query. The database then responds, “you appear quiet, why?” This is something our man in the store can understand. He can respond to the woman properly now and their social interaction can continue. Such social learning would be enormously beneficial to a person with an ASD, expanding their zone of comfort and allowing them to experience more in their lives. This kind of insight could improve ToM as well. They could be confident that they will be able to react and respond properly to a host of social situations, learn about the mental states of others and socialize more as a result.
Through the use of DVDs, computer aids and social training the lack of ToM seen in ASDs is being addressed specifically, a positive result that has come from the research in this area over the last thirty years.

Conclusion

After an examination of the scholarship of the last thirty years on the subject of autism and theory of mind, one thing seems clear. There does indeed exist evidence that a lack of a fully functioning theory of mind is a core deficit in cases of autism. While there existed some controversy as to whether cases of Asperger's syndrome showed the same deficit, further study revealed that this is likely the case as well, that the ToM deficiency stretches across the entire autism spectrum. After the discovery of this phenomena further testing with more advanced ToM tests have confirmed these findings many times. In a disorder with multiple possible biological causes and many different behavioral outcomes it seems that a lack of fully developed ToM at the cognitive level is what really defines the ASDs.

This has lead the scientific community to look for a biological component to this puzzle, a physical mechanism that maybe responsible for functional ToM in humans. Mirror neurons may very well be that mechanism. Computer assisted training has proven to be a successful method for combating some of the social outcomes that result from the ToM deficit. Computer applications that help people with autism understand emotions and the mental states of others show positive results and newer mobile applications let children and adults with autism take their solutions on the road with them. The discovery of this core deficit has allowed educators to focus on this aspect of cognitive functioning and tailor their computer solutions accordingly.
By recognizing and studying this aspect of cognitive development the research community have provided a guide for treatments, understanding for families and people with autism and a direction forward for future research. Hopefully, someday, research of this kind can lead to the detection of the root cause of ASDs.
References


