Linguistic Portfolios

Volume 1 Article 3

2012

Neurobiological Underpinnings and Linguistic Manifestations in Autism Spectrum Disorders

Jill Bayliss
St. Cloud State University

NEUROBIOLOGICAL UNDERPINNINGS AND LINGUISTIC MANIFESTATIONS IN AUTISM SPECTRUM DISORDERS

JILL BAYLISS

1.0 Introduction:

Autism is a neurological developmental disorder that shows lifelong effects and is most evidently disruptive to sociocommunicative development (Solomon, 2008). Since it is a developmental disorder, it is often evident in early childhood. According to Lord and Spence (2006), a diagnosis is usually possible by the age of two. It is a pervasive disorder, affecting many areas of the brain, and is often characterized by impairments in both brain structure and in social behavior. Some of the most evident impairments in this disorder are problems with language and communication, deficits in social reciprocity, and behaviors that appear repetitive or formulaic. These repetitive behaviors often include hand flapping and rocking back and forth (Solomon, 2008). Lord and Spence note that laboratory studies have resulted in an “autistic phenotype” used to identify autism. This phenotype includes irregular eye gauze and use of gestures, language delays, the reversal of pronouns, the lack of creative or imaginative play, and echolalia. That latter is defined as the repetition of sounds and utterances.

1.1 Diagnosis and Cause of Autism

Autism is often hard to diagnose because it includes such diverse symptoms and has many symptoms that are similar to other disorders. In addition to this, there were many changes made to the criteria for diagnosing autism in the *Diagnostic and Statistical Manual of Mental Disorders* between the 1980 and 1994 versions. Due to the similarity of autism symptoms to other disorders, autism is often placed in a group with comparable disorders under the header of Autism Spectrum Disorders (ASD). In addition to autism, this group includes Asperger’s disorder, Rett syndrome, childhood disintegrative disorder, and any other all-encompassing developmental disorders that are not specifically classified (Eigsti & Schuh, 2008). Another reason why autism is hard to diagnose springs from the fact that it is a developmental disorder and, therefore, is often diagnosed first by schools and not by doctors. There are no medical tests that can be done to decisively diagnose autism or its spectrum disorders. Even though delays in development are apparent early on in a child’s life, autism is usually not diagnosed until years after the symptoms have started to appear. This can be caused by a variety of reasons including symptom severity, race, gender, and attitudes of the parents or teachers of the child (Mandell, Ittenbach, Levy, & Pinto-Martin, 2006).

Currently, the cause of autism is not known. There are a variety of factors that have been indicated as possible influences but none of these alone can be shown as an absolute cause of this disorder. Among these are both genetic and environmental factors. Many believe that individuals who develop autism are genetically susceptible to the disease while others think that childhood vaccinations or diets play a strong role. According to the Autism Society of America (2008), despite the different potential influences, it is widely believed that autism is caused by abnormalities in the structures and functions of the brain. However, due to the fact that no explicit cause has been identified, many new areas of research are being applied to autism.

1.2 Research in Autism

Research of autism spectrum disorders continues to expand as new concerns become apparent. For a time, one large concern was about autism being caused by vaccines. According to the Centers of Disease Control and Prevention (2009), many studies have addressed this issue and the majority of these have found no link between childhood vaccines and autism. Another large area of research taking place at the moment focuses on the possibility of genetic causes. The Autism Society of America (2008) states that researchers are "investigating a number of theories, including the link between heredity, genetics, and medical problems." This is supported by the fact that many families appear to show a pattern of autism spectrum disorders. However, no specific gene has been identified as a cause of autism so far. In terms of autism being linked to other medical conditions, it has been found that autism is more likely to occur in individuals with Fragile X syndrome, tuberous sclerosis, congenital rubella syndrome, and untreated phenylketonuria (CDC, 2007).

1.3 Prevalence

This increased amount of new research is also caused by an increased interest in this disorder, not just in the medical and scientific fields but in lay publications as well. This could be because the number of people diagnosed with autism has increased dramatically since the 1960's (Eigsti & Schuh, 2008). According to the Centers for Disease Control and Prevention, over 1.5 million people in the United States live with autism and the prevalence among children is thought to be 6-7 in every 1,000 (2007). It was also found that boys are approximately four times more likely to be diagnosed with autism than girls. This means that about 1 in 94 American boys live with this disorder.

With more people being affected by this disorder, and the absence of a known cause, autism is one of the most promising areas of research. As mentioned above, articles dealing with autism have been more common in the news recently. The New York Times published an article on March 19, 2009 about the seemingly heightened number of children diagnosed with autism coming from Somali homes in Minneapolis. According to the author, the Minnesota Department of Health is conducting a survey in an attempt to ascertain if this can be considered an outbreak or if it could be caused by a statistical fluke (McNeil, 2009). This happens often with diseases like autism that have no known cause and are not contagious. It becomes very difficult to tell why there are increased numbers of cases and what may be underlying each case. One explanation may simply be that autism is diagnosed differently today than it has been in the past. Doctors have more criteria to base their decision on. Moreover, this reported increase in autism cases could be influenced by the fact that the criteria for diagnosis have changed drastically. As a result, Eigsti and Schuh (2008) content that there is greater public awareness of autism, in addition to the possibility that there is actually a higher number of cases.

2.0 Autism and the Brain

Since this paper focuses on the potential biological causes of autism in the brain and its related linguistic manifestations, an area of research that is most relevant is neurobiology. Consequently, much new research is being conducted in the area of neuroscience to discover how autism effects brain structures and their functions.

2.1 Research Methods

The most useful methods used to study cognitive neuroscience are electrophysiological techniques and brain imaging techniques. Electroencephalography (EEG) is the most common of the electrophysiological approaches and is used to record electrical activity in the brain by means of placing electrodes on the scalp of the patient. Another of these methods is Magnetoencephalography with looks at neural activity by means of the magnetic fields that are created from activation of neurons. This differs from EEG because it is not distorted by the skull and scalp.

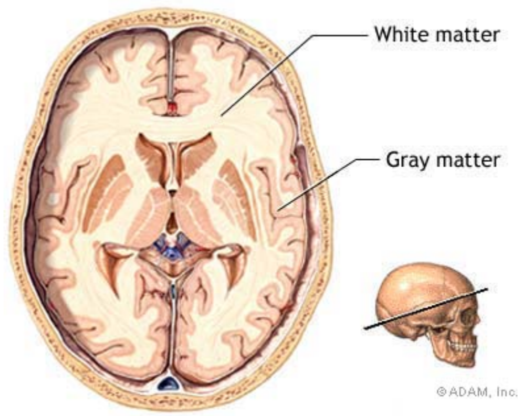
To look at the actual structures of the brain, imaging techniques are used. The most common of these is known as Magnetic Resonance Imagining (MRI) and is an extremely useful tool for studying developmental disorders such as autism. MRI provides precise anatomical images of the brain and is safe to use multiple times so that a picture of the neural developmental process can be formed. Another important technique is Diffusion tensor imaging (DTI). DTI derives its signal from the position of water molecules along axon bundles in the brain and is sensitive enough to detect the myelin that surrounds axons and augments the efficiency of brain signals. This becomes important when studying autism because language processing requires a high degree of coordination among brain structures and DTI can show where this connectivity is being affected. If this coordination of information is being affected structurally, it is expected that the processing of complex information will be greatly impaired (Minshew, Goldstein, & Siegal, 1997 as cited in Eigsti & Schuh, 2008).

Even though these techniques are profoundly important tools in neuroscience and can show detailed images of brain structures, they cannot display the mechanisms responsible for changes in the brain. Therefore, theories must be created as to why these changes occur.

2.2 Brain Structures

Studies have consistently found that there are differences in brain structure and function of patients with autism spectrum disorders when compared to healthy peers. One of the regular findings in neurobiological studies is an increase in cerebral volume of autistic patients. Some of these studies have found an increase in white matter and others have found an increase in gray matter (Eigsti & Schuh, 2008). White matter is composed of the myelinated nerve bundles or axons in the brain that are vital for communication between brain cells while gray matter includes mostly neural cell bodies and glial cells (see Figure 1). Although the findings of these studies can be inconsistent, increased volume of the brain in autism is a well-replicated discovery. This increased volume is measured by head circumference and is also known as macrocephaly. According to Courchesne, Carper, & Akshoomoff (2003), autistic children commonly have a smaller head circumference when they are born, but they grow faster and surpass their healthy peers by 14 months of age (as cited in Eigsti & Schuh, 2008).

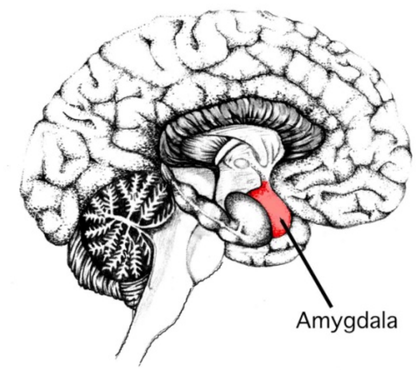
Figure 1: White matter vs. Gray matter



In addition to an overall increase in brain volume, the limbic system has been focused on in investigations of autism. Some of the main structures of the limbic system are the hypothalamus, the hippocampus, and the amygdala, but this system also includes structures such as the “subiculum, entorhinal cortex, septal nuclei, anterior cingulated gyrus, septum, and mamillary body” (Eigsti & Schuh, 2008, p.133). The limbic system is responsible for many of our emotions and for the formation of memories. In both children and adults with autism, autopsy studies have demonstrated that the limbic systems of autistic brains are distinguished by small and tightly packed cells compared to healthy brains. These reduced cells are consistent with arrested development (Bauman & Kemper, 1994 as cited in Eigsti & Shapiro, 2003).

Other studies have focused solely on the amygdala and documented abnormalities in this area of the brain (see Figure 2). The amygdala is an emotional center that is most commonly associated with feelings of fear and aggression but it also functions to recognize faces, evaluate situations, and identify social information such as the emotions of others. Although studies agree that there are structural abnormalities in the amygdalae of autistic patients, some have found increased volumes while others have documented decreased volumes. In any case, structural irregularities in the amygdalae of autistic patients could account for the lack of face recognition in toddlers and other problems with social behaviors common in autism spectrum disorders.

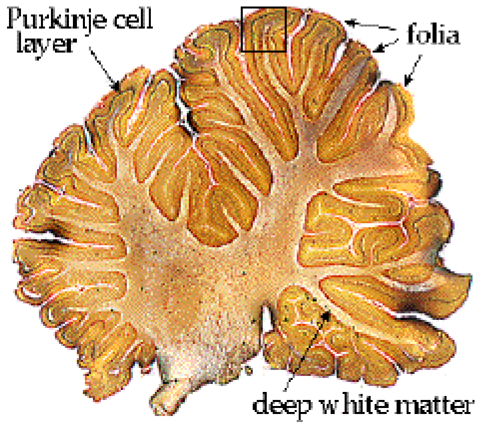
Figure 2: The Amygdala



Research projects taking place at the University of Washington have been using fMRI scans to study the amygdalae of adults with autism. In one study, a group of autistic adults and a group of healthy peers were shown a series of pictures of human faces. It was discovered that the amygdalae of the autistic adults stayed activated longer than the brains of the normally developed adults. According to the author, Kleinhans (2009), a decrease in activation of brain areas over time is associated with the learning process and is referred to as neural habituation. When overstimulation of the amygdala is observed, as in the autistic subjects, it suggests that the neurons in the amygdala are being fired more than would be expected in a healthy adult. This delayed process of neural habituation in autistic adults could explain many of the difficulties in social cognition present in autism. In addition to structural abnormalities in the amygdala, many of the smaller brain structures that have been found to be either increased or decreased in autistic patients are structures that connect to the amygdala (Eigsti & Schuh, 2008).

In addition to the amygdala, an abnormal cerebellum has often been found to be indicative of autism. However, MRI studies have found both increased and decreased volume of the cerebellum. This area of the brain is important for the coordination of movements within the body and is also partially responsible for motor learning. Perhaps more importantly, another study has found a decreased number of Purkinje cells in the cerebellum and the brainstem (Bauman & Kemper as cited in Eigsti & Schuh, 2008). Purkinje cells are large neurons with dense branches that play a central role in the circuitry of the cerebellum. In the study of Bailey et al. (as cited in Eigsti & Shapiro, 2003), it was stated that this scarcity of Purkinje cells is the most consistent neuroanatomical finding concerning the autistic brain.

Figure 3: Cerebellum showing layer of Purkinje cells



In a study done at the University of Washington, Sparks et al. (2002) combined all of these areas in one study of young autistic children from 3 to 4 years of age. They compared the brain structures of a group of autistic children, typically developing children, and children with a developmental delay. It was found that children with autism spectrum disorders had significantly increased brain volumes as compared to the other two groups. The volumes of the cerebellum, amygdala, and hippocampus were also measured and it was discovered that amygdalae and hippocampi of the autism group were increased bilaterally and were larger in volume consistent with the overall increase in cerebral volume. In addition, a subgroup of children with strictly classified autism was studied compared to the wider groups. These children were found to have enlarged amygdalae that were in excess of an overall increased brain volume (Sparks et al., 2002). These findings suggest that there is atypical brain development happening in children with autism.

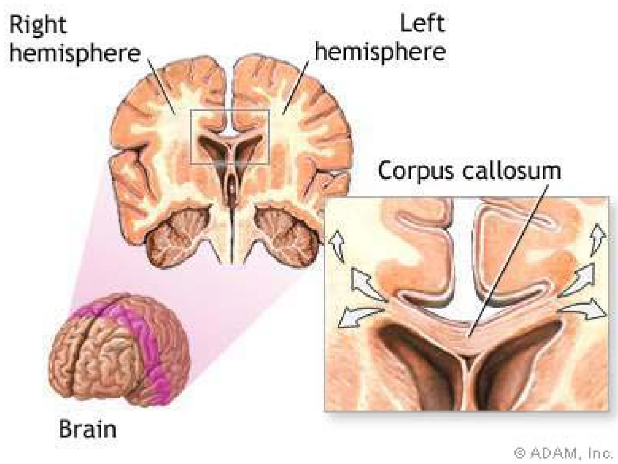
2.3 Functional Connectivity

Another region of the brain that is frequently found to be irregular in the autistic brain is the corpus callosum. This could be critical to language impairments in autism because the corpus callosum, "the largest and most prominent axonal pathway in the brain, is responsible for inter-hemispheric transfer of information," (Eigsti & Shapiro, 2003). Also, according to the review done by Eigsti and Schuh (2008), the differences in brain structure found in autism spectrum disorders may cause inefficient coordination between neural assemblies which can lead to problems in complex cognitive tasks. Therefore, much of the recent research has been concerned with studying the seemingly impaired nature of connectivity between brain structures as an underlying cause of language problems in autism. This integration of information across neural networks is commonly referred to as functional connectivity.

In the course of a normally developing brain, areas tend to become more specialized and less flexible. First, the brain goes through a building phase, during which cells migrate and axons are myelinated. Then, according to Eigsti and Schuh (2008) comes the pruning phase. This is where the connections between cells are refined by removing unnecessary or inappropriate synaptic projections. This pruning phase allows the brain to process more efficiently because it prevents unnecessary firing of axons. Eigsti and Schuh (2008) hypothesize that tf this phase of synaptic pruning develops differently in autism, it may result in excessive connections in the brain which could cause less efficient learning. One possibility is that synapses are pruned too early, preventing the connections in the brain to become refined.

This being the case, studies have examined the corpus callosum (see Figure 4) in the autistic brain to discover whether connectivity may be responsible for some of the characteristic impairments in autism. Based on the studies of Piven et al. (as cited in Eigsti & Shapiro, 2003), MRI scans suggest that body and posterior regions of the corpus callosum are considerably decreased in size compared to healthy individuals. This signifies that communication between brain hemispheres is less effective in autistic patients. There have also been studies conducted using Diffusion tensor imaging (DTI) to evaluate connectivity. These studies have found low

Figure 4: The corpus callosum



anisotropy in the corpus callosum, meaning that the arrangement of the water molecules along axons appears more disorganized. This could indicate that transmission of information of information along nerve bundles is less efficient (Alexander et al., as cited in Eigsti & Schuh, 2008). However, these findings were reflective of adult and adolescent brains. In the brains of children with autism, increased anisotropy was found, indicating that an early maturation of white matter was taking place especially in the frontal lobe and left hemisphere of the brain.

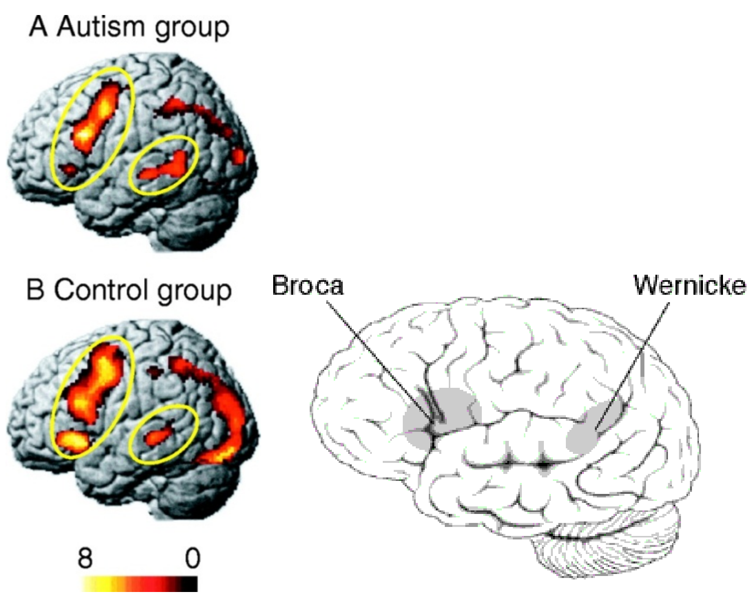
In a study by Boger-Megiddo et al. (2006), the corpus callosum of 3 and 4 year olds were examined. Similar to the study done by Sparks et al. mentioned above, a group of children with autism spectrum disorders was compared to a group of typically developing children and a group of children with developmental delays. This study found that although the corpus callosum in autistic children was not different in overall size compared to the typically developing children, it was disproportionally small when the volume of the whole brain was taken into account. Due to the fact that ASD children consistently have increased brain volume compared to healthy controls, the corpus callosum was seen to be smaller in the brains of young autistic children. Again, a subgroup of severely autistic children was also studied. This subgroup was found to have an even greater proportional reduction in corpus callosum size (Boger-Megiddo, 2006).

Issues with functional connectivity also tie into the findings about white matter in the autistic brain. Eigsti and Schuh (2008) cite the studies of Herbert et al. and Waiter et al. both from 2005, as documenting atypical distributions of white matter in the autistic brain. This is signaled by a decreased volume of white matter. The authors conclude that this supports the previous findings of underconnectivity between brain systems which can affect cognitive, perceptual, and motor circuits. Also cited is a study by Hendry et al. (2006) that examined the water content of brain tissues in school-age children with autism and found increased cerebral white matter throughout the brain and especially associated with the parietal and frontal lobes (Eigsti & Schuh, 2008). These abnormalities in white matter could also be a result of unnecessary connections in the brain that were kept due to a lack of developmental pruning of synapses.

2.4 The Brain and Language

Additional findings related to issues of functional connectivity are also related to areas of the brain that are important in language processing. For example, a recent study by Just et al. (2004) has demonstrated irregularities in connectivity using functional MRI. This study focused on high-functioning autistic adults and a group of healthy peers that were matched for verbal-IQ. Participants were presented with a sequence of active and passive sentence constructions and asked to identify the actor in each sentence. The high-functioning autism group was found to produce more activation in Wernicke's area compared to the control group and consistently less activation in Broca's area (sees Figure 5). Furthermore, the degree of synchronization between brain areas that were functioning to complete this task was seen to be consistently lower for the autistic participants, suggesting, again, the role of functional connectivity.

Figure 5: Wernicke's and Broca's activation in Just et al. study



Another recent study conducted by De Fosse et al. (2004) examined the brains of boys between the ages of 6 and 13. Included were groups of boys with normal development, boys with autism but unimpaired language, those with autism and impaired language, and those with a rare development disorder known as Specific Language Impairment (SLI). The brains of these boys were studied using MRI and differences were found in the structure of Broca's area. Broca's area is also known as the inferior frontal gyrus and is responsible for the production of language. Typically, in right-handed individuals, the left hemisphere of the brain dominates language production and processing which means that language specific areas like Broca's are visibly larger on the left side of the brain. However, the authors of this study found that in boys with autism and language impairments, there was still asymmetry between the hemispheres of the brain, but the inferior frontal gyrus of the right side was larger in size instead of that of the left side. This significant reversal in asymmetry of language areas was also found in the boys with Specific Language Impairment (SLI), suggesting a link between the two disorders. Furthermore, the degree of Broca's asymmetry was related to the severity of language problems. Curiously enough, the autistic boys who did not display language impairments had brain asymmetry that was similar to the normally developing controls. This could suggest a number of things including a potential phenotypic link between autism spectrum disorders and SLI, and the strong possibility that autism is not one disorder but has subgroups with overlapping symptoms (De Fosse et al., 2004). Given, it is appropriate to refer to it as autism spectrum disorders.

In addition to activation in Broca's area and Wernicke's area, a few studies have focused on white matter in the brain using diffusion tensor imaging. One study by Lee et al. (as cited in Eigsti & Schuh, 2008) discovered decreased anisotropy in the superior temporal gyrus and the temporal stem of autistic participants. Both of these areas are important in language and sound processing. This is so because Wernicke's area is contained in the superior temporal gyrus and is responsible for processing language sounds. Both of these areas are part of the temporal lobe which houses the auditory cortex.

In relation to the auditory cortex, Eigsti & Schuh (2008) hypothesize that one of the factors that could be causing language problems in autism spectrum disorders is the atypical processing of sounds—both speech and nonspeech. To support this, the authors state that autism is often characterized by either hypersensitivity or hyposensitivity to sound, and can include highly developed skills in the perception of music which are referred to as savant like abilities. If individuals with autism spectrum disorders are processing sound differently from normally developing individuals, it could clearly affect their language abilities.

In fact, many studies have been devoted to sound discrimination of autistic individuals. Sound discrimination is typically measured using mismatch negativity or MMN. This measures the difference between standard sounds and new sounds deviating from the standard sounds in the minds of the subjects (Eigsti & Schuh, 2008). Multiple studies working with autistic children aged 5-13 have measured MMN to elicit conclusions about sound discrimination. In these studies, the participants were presented with vowels, syllables, and simple or complex tones. Autistic participants usually showed greater MMN amplitude and a longer response time than controls. Eigsti and Schuh (2008) concluded that these findings show generally that the discrimination of speech, and particularly vowel sounds, is delayed in autism spectrum disorders. In very young children between the ages of 1 and 4, it was found by Kuhl et al. (2005) that the irregularity of sound processing correlated with symptom severity and that the autistic children had a preference for nonspeech sounds above speech sounds, which is typically in normally developing children.

3.0 Autism and Language

One of the major symptoms used to define autism is a deficit in language and communication skills. However, this may be misleading because not all individuals with autism spectrum disorders have language that is affected in the same way. Some may have language abilities that are hardly impaired while others may be completely nonverbal. The severity spans a large spectrum. According to the National Institute on Deafness and other Communication Disorders, some individuals with autism may be unable to speak, while others have rich vocabularies, but most have problems using communication effectively (2009). Solomon (2008) supports this noting that individuals with autism use language in a way that is unconventional and often hard to interpret for those who have developed normally. This can include utterances that are anywhere from slightly off topic to those that are completely cryptic to the listener. Eigsti and Schuh (2008) state that autism involves “marked abnormalities in acquisition and pragmatic language skills in as many as 25%-50% of individuals with the diagnosis.” The other 50% to 75% often fail to develop language skills of any kind. These nonverbal individuals are underrepresented in studies usually because the researchers have a desire to study the utterances of autistic individuals.

Even those children who develop language skills show significant delays in acquisition, usually producing their first words at an age of 38 months compared to normally developing children who first speak somewhere between 12 and 18 months (Eigsti, Bennetto, & Dadlani, 2006). However, in many cases, children who have started to show language progress early in life lose all ability by the age of 2 and revert to a nonverbal state.

3.1 Pragmatics

In the majority of those with autism, an area often affected is pragmatics. Pragmatics is defined as the way in which the meaning of speech is affected by context, by the intent of the speaker, or by structural ambiguity. Of those people that have some form of language ability, their utterances tend to lack content and provide no information (NIDCD, 2009). This is because autistic language frequently makes use of echolalia, which is the repetition of something heard previously. This could be the immediate repetition of something that was just said, a phrase taken from a movie or television commercial, or a stock phrase that the person uses whenever presented with a certain situation. For example, when asked by a parent: “Do you want something to drink?” and autistic child may repeat the question back instead of answering, or they may use the question every time they are asking for a drink. In this way, it is difficult for those with autism to contribute effectively to conversations. They may not respond to others attempts at communication but even when they do, autistic children often have trouble practicing turn-taking and sustaining a conversation.

Also in the area of pragmatics, a study done by Wang, Lee, Sigman, and Dapretto (as cited in Eigsti & Schuh, 2008), looked at the skills of high-functioning autistic children. This study showed that the autism group was less able to conclude whether a speaker was being sincere or ironic when the participants were given either prosodic clues (tone of voice) or context clues.

In another study done by Ochs et al. (as cited in Solomon, 2008), it was argued that the most challenging part of pragmatics for autistic children was indexicality. Indexicality exists when we use a behavior or utterance to indicate something else. For example, using "I" in conversation refers to whoever is speaking. The ability to infer what is being indicated relies on being able to recognize what is happening in social situations. This is often difficult for those with autism.

This may relate to the characteristic problem that children with autism display concerning pronouns. The reversal of pronouns is commonly observed in the speech of autistic children—referring to themselves as "you" while referring to their listener as "I," (Tager-Flusberg, 2000). While this pronoun reversal also happens in typically developing children, it is much more common in autism and is one of the features that is used to diagnose this disorder.

3.2 Prosody

Prosody in linguistics refers to the rhythm, stress, and intonation of speech. According to the National Institute on Deafness and other Communication Disorders (NIDCD), many individuals with autism have problems with intonation and rhythm, even though the majority has little or no problems with pronunciation. A few studies have focused on the prosody of the speech produced by autistic participants. Research done by Shriberg et al. (2001), looked at three groups of male speakers, 15 males with high-functioning autism, 15 with Asperger Syndrome, and 53 typically developing males. According to the results of this study, significantly more of the men in the high-functioning autism group and the Asperger group showed utterances that were considered inappropriate in the areas of phrasing, stress, and resonance. They also displayed more residual articulation distortion errors.

3.3 Semantics

Semantics has everything to do with the meanings of words and sentences. This is another of the areas that the NIDCD mentions as a problem in those with autism spectrum disorders. One study looking at this area of language processing was done by Harris et al. (as cited in Eigsti & Schuh, 2008) where adults with autism were presented, visually, with a series of words. These adults showed atypical language-related responses in Broca's area while processing these words semantically versus perceptually. The participants in this study also revealed decreased brain activation when responding to concrete versus abstract words.

In another study conducted by Gaffrey et al. (2007), adults with autism were tested on their ability to sort a list of words into colors, feelings, or tools. Again, the autism group showed less activation in Broca's area than the control group. However, there was more activation in areas of the brain responsible for visual processing which could mean that perception plays an important role during semantic decisions in those with autism.

3.4 Grammaticality

One area of language acquisition that is not often studied is the grammar abilities of children with autism spectrum disorders. Grammaticality is something like a linguistic intuition that speakers have about their language that tells them when utterances or sentences are well-formed compared to when they are not. It is most closely related to the area of syntax.

One recent study by Eigsti, Bennetto, and Dadlani (2007) took an in-depth look at the syntactic and high-level discourse abilities of autistic children. The children studied were five years of age. From the results of this study, the authors concluded that there were clear differences in the language abilities of the autistic children compared to those of the typically developing controls. They expressed that the deficits went beyond what would be expected based on the levels of development. Specifically, there were syntactic delays, a higher production of jargon (words with no meaning), and problems managing discourse. In a follow-up study published in 2009, Eigsti and Bennetto tested older children with autism. A group of autistic children and adolescents aged 9 to 17 were given a grammaticality judgment task and compared to a control group of typically developing children of the same ages. It was found that the autistic participants were noticeably less sensitive than controls, especially for sentence constructions with third person singular and present progressive marking. Also, performance level was tied to the length of sentences given (Eigsti & Bennetto, 2009).

4.0 Conclusions

Clearly, the language of those individuals with autism spectrum disorders differs in many areas from those with typical patterns of development. This difference becomes apparent not only in the way language is used, but also in the way language is processed and acquired. The world of autism research would benefit greatly from enhanced studies of children and adolescents to discover the ways that language is being acquired and utilized in these various linguistic areas.

Also, because many areas of the brain have been implicated in autism, it would be beneficial to further strengthen the ties between the areas of the brain that are affected and the impact that this has on language abilities. This may be especially useful considering that language and communication is one of the signature deficits in autism spectrum disorders and has recently been offering up new perspectives on autism and its potential causes. This would also be useful because, generally, very little is known about the intersection between linguistics and biology, both in the field of genetics and neuroanatomy. Consequently, the field of cognitive science is proving to be useful in bringing together linguistics and biology, as well as computer science and psychology. Therefore, research in this area holds promise.

With my own background in linguistics and biology, I hope to contribute further to research that ties these two fields together. A multidisciplinary look at these disorders will be able to yield much new information and develop a connected understanding of the areas affected. Hopefully having a broader understanding of these connections will help in the lives of individuals who live with these disorders.

ABOUT THE AUTHOR

Jill Bayliss graduated from Saint Cloud State University with a BA in Linguistics and a minor in Biology. She hopes to pursue a Ph.D. in Cognitive Linguistics or Neuroscience. She wrote this paper for her senior thesis under the supervision of Dr. Ettien Koffi.

References

Autism and communication. (2009). *National Institute on Deafness and Other Communication Disorders*. Retrieved 23 March 2009, from http://www.nidcd.nih.gov/health/voice/autism.asp

Autism information center: Vaccines and autism. (2009). *Centers for Disease Control and Prevention*. Retrieved 25 April 2009, from http://www.cdc.gov/nbddd/autism/vaccines.htm

Boger-Megiddo, I., Shaw, D.W., Friedman, S. D., Sparks, B.F., Artru A.A., Giedd, J.N., et al. (2006). Corpus callosum morphometrics in young children with autism spectrum disorder. *Journal of Autism and Developmental Disorders,* 36 (6), 733-739. Abstract retrieved from PubMed Database online.

De Fosse, L., Hodge, S. M., Makris, N., Kennedy, D. N., Caviness Jr, V. S., McGrath, L., et al. (2004). Language-association cortex asymmetry in autism and specific language impairment. *Annals of Neurology*, 56 (6), 757-766. Abstract retrieved from Wiley InterScience database online.

Eigsti, I., Bennetto, L., Dadlani, M. B. (2007). Beyond pragmatics: Morphosyntactic development in autism. *Journal of Autism and Developmental Disorders*, 37, 1007-1023. Abstract retrieved from SpringerLink database online.

Eigsti, I. & Bennetto, L. (2009). Grammaticality judgments in autism: Deviance or delay. *Journal of Child Language*. doi: 10.1017/S0305000909009362

Eigsti, I. & Schuh, J.M. (2008). Neurobiological underpinnings of language in autism spectrum disorders. *Annual Review of Applied Linguistics: Neurolinguistics and Cognitive Aspects of Language Processing*, 28, 128-149.

Eigsti, I. & Shapiro, T. (2003). A system’s neuroscience approach to autism: Biological, cognitive, and clinical perspectives. *Mental Retardation and Developmental Disabilities Research Reviews*, 9, 205-215. Online version, retrieved from http://eigsti.psy.uconn.edu/Eigsti&Shapiro2003.pdf

Just, M. A., Cherkassky, V. L., Keller, T. A., Minshew, N. J. (2004). Cortical activation and synchronization during sentence comprehension in high-functioning autism: evidence of underconnectivity. *Brain*, 127 (8), 1811-1821. Retrieved April 26, 2009, from http://brain.oxfordjournals.org

Kuhl, P. K., Coffey-Corina, S., Padden, D., & Dawson, G. (2005). Links between social and linguistic processing of speech in preschool children with autism: Behavioral and electrophysiological measures. *Developmental Science*, 8 (1), F1-F12.

Lord, C., & Spence, S. (2006). Autism spectrum disorders: Phenotype and diagnosis. In S. Moldin and J. Rubenstein (Eds.), *Understanding autism: From basic neuroscience to treatment* (pp. 1-23). New York: CRC Taylor and Francis.

Mandell, D.S., Ittenbach, R.F., Levy, S.E., & Pinto-Martin, J.A. (2006). Disparities in diagnoses received prior to a diagnosis of autism spectrum disorder. *Journal of Autism and Developmental Disorders*. Published online December 8, 2006.

McNeil Jr., D. G. (2009, March 16). An outbreak of autism, or a statistical fluke? *The New York Times*. Retrieved from http://www.nytimes.com

Schwartz, J. (2009, March 19). Heightened level of amygdala activity may cause social deficits in autism. *EurekAlert!* Retrieved from http://www.eurekalert.org/pub_releases

Shriberg, L. D., Paul, R., McSweeny, J. L., Klin, A., Cohen, D. J., Volkmar, F. R. (2001). Speech and prosody characteristics of adolescents and adults with high-functioning autism and Asperger syndrome. *Journal of Speech, Language, and Hearing Research*, 44, 1097-1115. Abstract retrieved from JSLHR online at http://jslhr.asha.org

Solomon, O. (2008). Language, autism, and childhood: An ethnographic perspective. *Annual Review of Applied Linguistics: Neurolinguistics and Cognitive Aspects of Language Processing*, 28, 150-169.

Sparks, B.F., Friedman, S.D., Shaw D.W., Aylward, E.H., Echelard, D., Artru, A.A., et al. (2002). Brain structural abnormalities in young children with autism spectrum disorder. *Neurology*, 59 (2), 184-192. Abstract retrieved from PubMeb Database online.

Tager-Flusberg, H. (2000). Understanding the language and communicative impairments in autism. *International Review of Research on Mental Retardation: Special Issue on Autism*.

What causes autism. (2008). *Autism Society of America*. Retrieved 22 April 2009, from http://www.autism-society.org/site/PageServer?pagename=about_whatcauses