A Review: Neurodevelopment and Neuroanatomy of Individuals with Autism Spectrum Disorder; Implications for Therapist

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Abstract

This is an in-depth review of current research literature on the neurodevelopment and neuroanatomy of individuals with Autism Spectrum Disorders (ASD). To gain a better understanding of ASD, researchers have turned to the brain to seek answers. Currently, researchers are exploring different sections of the brain to determine if there is overgrowth, undergrowth, or abnormalities that would lead to the symptoms of autism. Researchers have found significant differences in certain parts of the brain, while no differences in other parts. Through the results of these studies, researchers, therapists, and other individuals who work with ASD clients are gaining a better, more in-depth understanding of the etiology of ASD, the usefulness of early intervention, as well as techniques and strategies to be used in treatments.
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A Review: Neurodevelopment and Neuroanatomy of Individuals with Autism Spectrum Disorder; Implications for Therapist

Autism has been around for centuries. Some individuals in the field speculate that famous people in history were on the autism spectrum. A few of the most well known are Albert Einstein, Sir Isaac Newton, Ludwig van Beethoven, Wolfgang Amadeus Mozart, Lewis Carroll, and Vincent van Gough. Although it may never be known whether these individuals were on the autism spectrum, we do know how prevalent autism has become in our society.

There has been an increase in autism spectrum disorders (ASD) diagnosis over the past 25 years. Because of this increase, there is a need for refined diagnostic tools, more services and treatment options, as well as more research. Researchers are trying to gain a better understanding of the etiology,

Autism begins before 3 years of age, but exactly when it begins is unknown. Autism is hypothesized to be a complex disorder involving brain growth and perhaps brain maturation (Lainhart, 2003). Researchers generally feel that autism is a malfunction of the central nervous system (New Dictionary of Cultural Literacy, n. d.). Additionally, research efforts have aimed at pinpointing biological markers to facilitate early diagnosis and intervention, identification of such markers also help to better understand the pathomechanisms leading to these developmental anomalies (Dissanayake, Bui, Huggins, & Loesch, 2006).

This literature review will cover a portion of the current research available on the neurodevelopment and neuroanatomy of individuals with autism. Specifically, it reviews research on brain overgrowth, connectivity, volume, and head circumference, as well as
specific brain structures and directions for further research. In addition, it addresses implications for therapy and the need for therapist to adapt their therapeutic approaches when working with ASD clients.

**Definition of Autism**

The word autism comes from the Greek word *autos* meaning “self” and combined with the suffix *ismos* meaning “of action or of state” (*Online Etymology Dictionary*). Leo Kanner and Hans Asperger first used the word autism in 1940s. Both men used the term in their individual publications, Kanner in 1943 and Asperger in 1944, but neither knew the etiology (Sicile-Kira, 2004). Today, the term autism refers to a spectrum of disorders with specific characteristics, and researchers are still trying to determine the etiology.

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that is variable in expression (*Merriam-Webster's Medical Dictionary*) and with an estimated genetic origin of 90% (Palmen et al., 2005). ASD is comprised of several different phenotypes each of which has the same triad of deficits. These deficits are of impaired social interaction, impaired communication, and restricted interests and repetitive behaviors (Belmonte et al., 2004). Other ASD characteristics are a deficit of executive function (Ozonoff et al., 1991), complex information processing (Minshew et al., 1997), theory of mind (Baron-Cohen et al., 1985), and empathy (Baron-Cohen, 2002).

**DSM-IV-TR Criteria**

The *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)* (American Psychiatric Association, 2000) is what mental health professionals us to diagnose their clients. The most current edition was published 1994, but the newest
revision was published in 2000. A new edition is currently in the works and the projected release date is in 2012.

In the *DSM-IV-TR*, the two major diagnoses under the category of Autism Spectrum Disorders (ASD) are autistic disorder and Asperger disorder. The criteria for both disorders are as follows:

299.00 Autistic Disorder:

- Qualitative impairment in social interaction
- Qualitative impairment in communication
- Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities

299.80 Asperger’s Disorder:

- Qualitative impairment in social interaction
- Restricted, repetitive, and stereotyped patterns of behavior, interests, and activities
- Clinically significant impairments in social, occupational, or other important areas of functioning
- No clinically significant delay in language
- No clinically significant delay in cognitive development of age-appropriate self-help skills, adaptive behaviors, and curiosity about the environment in childhood

In order for individuals to be diagnosed with ASD, they must meet a specific number of the criterions from the *DSM-IV-TR*. Often time the clinician will use additional assessment tools, inventories, and questionnaires. Ultimately the clinician makes the decision as to rather or not individuals have ASD.
The Brain

Neurodevelopment

Overgrowth. Brain overgrowth in autism is an area researchers are trying to understand and determine its effects on autism. Current studies have shown brain overgrowth occurring during the first two years of life and at the time when the formation of cerebral circuitry is at its most abundant and vulnerable stage (Courchesne, 2004). The most significant overgrowth occurs in cerebral, cerebellar, and limbic structures of the brain. These structures manage higher-order cognitive, social, emotional, and language functions (Courchesne, 2004).

A study by Ghaziuddin, Zaccagnini, Tsai, & Elardo (1999), compared megalencephaly in autism to megalencephaly in attention deficit hyperactivity disorder (ADHD. Megaloencephaly is an abnormal enlargement of the brain (Stedman's Medical Dictionary). The researchers compared 20 male subjects with autism with 20 male controls with ADHD (Ghaziuddin, Zaccagnini, Tsai, & Elardo, 1999). Four of the subjects and five of the controls had evidence of megalencephaly. Additionally, the four subjects with megalencephaly were also hyperactive and impulsive like their counter parts in the control group.

These findings suggest that megalencephaly may not be specific to autism (Ghaziuddin, Zaccagnini, Tsai, & Elardo, 1999) and may have to do more with other neurological disorders. In addition, when megalencephaly is present, it may indicate the presence of additional symptoms such as hyperactivity and impulsivity (Ghaziuddin, Zaccagnini, Tsai, & Elardo, 1999). More research is needed to determine the correlation between ASD, ADHD, and megalencephaly.
Other studies have also found that the frontal cortex and white matter show the greatest early overgrowth followed by slowing of growth (Carper et al., 2002). These regions also show sharply reduced growth thereafter (Courchesne & Pierce, 2005). These studies will be discussed in more depth in section Frontal Cortex and Gray and White Matter.

Connectivity. The brain is a complex system of neural connections. These connections are responsible for sending signals and messages between the different parts of the brain and the rest of the body. Problems arise whenever there is a disruption in the connection. Researchers are trying to determine how the connectivity in the brain influences autism.

Scientist can differentiate local connectivity within neural assemblies. They are able to discriminate long-range connectivity between functional brain regions (Belmonte et al., 2004). Scientist can also separate the physical connectivity associated with synapses. In addition, they can separate the pathways from the computational connectivity associated with information transfer (Belmonte et al., 2004).

Just and colleagues (2004), did a study to determine if the connectivity in the language part of the brains was different between a group of high-functioning autistic participants and a control group. The two groups were measured using functional MRI during a sentence comprehension exercise.

They determined that in two of the key language areas, Wernicke's and Broca's area, the two groups differed in the distribution of activation. The autism group produced reliably more activation than the control group in Wernicke's area. However, in the Broca's area the autistic group had reliably less activation than the control group.
Furthermore, the functional connectivity between the various participating cortical areas was consistently lower for the autistic group than the control group. Their findings suggest that in the brain of individuals with autism, high local connectivity may develop in tandem with low long-range connectivity. Additionally, the neural basis of disordered language in autism involves a lower degree of information integration, as well as a lower synchronization across the cortical network for language processing (Just et al., 2004).

Recent attempts at a theoretical synthesis have focused on abnormal neural connectivity, and there seems some disagreement as to whether this abnormality involves a surfeit (Rubenstein and Merzenich, 2003; Belmonte et al., 2004) or a deficit (Brock et al., 2002; Just et al., 2004) of connectivity. Courchesne (1997), found that deficits of long-range connectivity and coordination of cognitive functions in the cerebellum. Likewise, Brock et al. (2002) proposed that underconnectivity between separate functional brain regions in autism may reflect in a lack of EEG synchrony in the gamma band. Moreover, high physical connectivity and low computational connectivity may reinforce each other by failing to differentiate signal from noise (Rubenstein and Merzenich, 2003; Belmonte et al., 2004).

Lewis and Elman (2008) hypothesized that a deviant brain growth direction will lead to deviant patterns of change in cortical connectivity. They proposed that the differences in brain size during development would alter the relative cost and effectiveness of short- and long-distance connections. Additionally would influence the growth and retention of connections.

They proposed that a reduced brain size would favor long-distance connectivity, while brain overgrowth would favor short-distance connectivity. In addition, they
proposed that inconsistent growth, as seen in autism, would result in potentially disruptive changes to established patterns of functional and physical connectivity during development.

To explore this hypothesis, Lewis & Elman (2008) used neural networks, which modeled interhemispheric interaction. These neural networks were grown at the rate of either typically developing children or children with autism. They analyzed the influence of the length of the interhemispheric connections at multiple developmental stages.

Lewis and Elman (2008) found that the networks that modeled autistic growth were less affected by removal of the interhemispheric connections than those that modeled normal growth. These results indicated a reduced reliance on long-distance connections for short response times. This difference increased considerably at approximately 24 simulated months of age. These results supported their hypothesis that the deviant growth trajectory in autism spectrum disorders may lead to a disruption of established patterns of functional connectivity during development, with potentially negative behavioral consequences, and a subsequent reduction in physical connectivity.

Brain volume. To further understand autism, researchers are turning to the brain volume of individuals with autism. Researchers compare the brain volume of individuals with autism to neurologically typical individuals. Recent studies suggest that early in development there is an increased brain volume, while other studies suggest a decrease in brain volume later in development.

Whole-brain and specific regions. MRI studies in young children with autism revealed excessive volume of cerebrum, cerebral white matter (Courchesne et al.,
2001; Sparks et al., 2002; Herbert et al., 2003), or an increased total brain volume (Piven et al., 1995; Aylward et al., 2002). Another MRI study of toddlers with autism found that brain volume was 10% greater than average, with 90% of these toddlers having volumes exceeding normal average, and occurring by 2-years-old (Courchesne et al., 2001, Courchesne et al., 2003, Sparks et al., 2002). Furthermore, a MRI study analyzed the neuroanatomy of 2-5-year-old girls and boys with autism, and found that both the boys and girls with autism had significantly abnormally enlarged whole brain volume (Bloss & Courchesne, 2007).

Rojas and colleagues (2006) did a study on the whole brain and correlated regional volume changes with several autism symptoms. They performed MRI scans on 24 males with ASD and compared those to scans from 23 neurologically typical male subjects. They used voxel-based morphometry (VBM) to analyze the regional gray matter volume.

Rojas et al. (2006) found in the autism group that the volumes of the medial frontal gyri, left pre-central gyrus, right post-central gyrus, right fusiform gyrus, caudate nuclei, and the left hippocampus were larger than in the control group. They additionally found some regions of the cerebellum exhibited smaller volumes in the autism group. Furthermore, significant partial correlations were found between the volumes of the caudate nuclei, multiple frontal and temporal regions, the cerebellum, and a measure of repetitive behaviors. Their results also revealed that the caudate, cerebellar, and precuneus volumes, as well as with frontal and temporal lobe regional volumes were associated with social and communication deficits in the group with autism. Rojas et al.
(2006), concluded that VBM was sensitive to associations between social and repetitive behaviors and regional brain volumes in autism.

Sparks and colleagues (2002) explored the specific gross neuroanatomic substrates of the brain morphometric features in 45 3-4-year-old children with ASD. They compared the subject group with age-matched control groups of 26 typically developing (TD) children and 14 developmentally delayed (DD) children. Sparks et al. (2002) use three-dimensional coronal MRI to measure the volumes of the cerebrum, cerebellum, amygdala, and hippocampus. The volumes were then analyzed with respect to age, sex, volume of the cerebrum, and clinical status.

The children with ASD were found to have significantly increased cerebral volumes as compared to the children with TD and DD (Sparks et al., 2002). Additionally, Sparks et al. (2002) found the cerebellar volume for the ASD group was also increased in comparison with the TD group. However, the increase was proportional to overall increases in cerebral volume. Moreover, the measurements of amygdalae and hippocampi in the ASD group revealed enlargement bilaterally that was proportional to overall increases in total cerebral volume. In addition, the DD group had smaller cerebellar volumes than both of the other groups (Sparks et al., 2002). They concluded that the results suggested abnormal brain growth early in the developmental processes of autism.

Herbert et al. (2003) conducted a study on 17 high-functioning boys with autism and 15 neurologically typical boys. These two groups were compared using a MRI whole-brain morphometric profile. This profile includes both total brain and major brain region volumes.
In their study (Herbert et al., 2003) neuroradiologists reviewed the MRI scans of both groups and determined the brains of all subjects to be clinically normal. The entire brain was segmented into several different sections and those sections were divided into subsections. After which volumes were formulated for each region and then compared between both groups. Lastly, Herbert et al. (2003) used factor analysis to group brain regions based on their intercorrelations.

Herbert and colleagues (2003) discovered the volumes were significantly different between groups overall. In the group with autism, the diencephalon, cerebral white matter, cerebellum, and globus pallidus-putamen were all significantly larger than the neurologically typical group. They concluded the morphometric profile of the brain of boy with autism suggests there is an overall increase in brain volumes as compared with controls. Additionally, results suggest that there may be differential effects driving these differences, and the cause is unknown, and further investigation is needed.

In a cross-sectional design, Aylward et al. (2002) reported brain volume in autism to be larger than normal in 8-12-year-olds, but not in adolescents and adults. Also using a cross-sectional design, Courchesne et al. (2001) found abnormally enlarged brain volume in 2-4-year-olds, but not 5-16-year-old children with autism. Several other recent MRI studies reported statistically non-significant differences in autistic brain volume at 4-11-years-old (Carper et al., 2002), 5-13 years (Kates et al., 2004), 7-11-years-old (Herbert et al., 2002, 2003, 2004), 8-45-years-old (Hardan et al., 2003), 17-47-years-old (Rojas et al., 2002) and 18-43-years-old (Carper & Courchesne, 2005). The absence of significant brain volume difference in older children and adults with autism (Courchesne et al., 2001; Aylward et al., 2002) may suggests that early hyperplasia in autism is
followed by a plateau during which brain growth in neurologically typical individuals catches up.

Hardan, Minshew, Mallikarjuhn, and Keshavan (2001) obtain measurements of the brain volume in a sample of 16 males with autism and 19 neurologically typical males. The third, fourth, and lateral ventricles, along with intracranial and cerebral volumes were obtained through MRI scans for both groups. Their results found that the mean cerebral and third ventricle volumes were significantly greater in the group with autism than in the controls. These findings were consistent with previous studies.

**Biological bases.** Previous studies on individuals with autism have reported brain volume increase of approximately 5%, especially in children, as compared to neurologically typical control groups. Palmen et al. (2005) hypothesized that if this brain volume increase is genetically determined, then biological parents of individuals with autism might be expected to also show brain or intracranial enlargement. They suggested that identifying structural brain abnormalities under genetic control was importance as it could represent endophenotypes of autism.

In their studies, Palmen and colleagues (2005) used quantitative anatomic brain MRI of different intracranial areas in biological, non-affected parents of individuals with autism and in healthy, closely matched control subjects. Palmen et al. (2005) measured the total brain volume, as well as the frontal, parietal, temporal, and occipital lobe volumes. The cerebral and cortical gray and white matter where also measured along with the cerebellum, laterals ventricle, and third ventricle.

Palmen et al. (2005) found no statistically significant differences between the two groups in any of the brain volumes. They concluded that since the intracranium was not
enlarged in the subject group, it is unlikely that their brain volumes had originally been
enlarged and have been normalized. Additionally, increased brain volume in autism
might be caused by the interaction of parents’ genes, possibly with an additional effect
of environmental factors. On the other hand, increased brain volumes might reflect
phenotypes of autism.

**Neurotrophins.** At the biochemical level, there is a link between enlarged brain
volume and increased levels of neurotrophins (Akshoomoff, Pierce, & Courchesne,
2002, Courchesne et al., 2001). The neurotrophins were found in the neonatal blood
spots of infants who later received a diagnosis of autism (Nelson et al., 2001). These
factors have been known to increase neuronal survival (Lewin & Barde, 1996).
Additionally, they play an important role in central synapse formation, as well as
maturation (Vicario- Abejon, Owens, McKay, & Segal, 2002).

In addition, there is evidence that the neurotrophic factors also play an important
role in immunomodulation (Nassenstein et al., 2003), angiogenesis (Kraemer &
Hempstead, 2003), and body metabolism (Chaldakov, Fiore, Hristove, & Aloe, 2003).
Furthermore, the evidence for the role of neurotrophins and neuropeptides in bone
metabolism and growth (Garcia- Castellano, Diaz-Herrera, & Morcuende, 2000)
suggests that the increased levels of these factors may affect both brain development,
as well as linear growth in autism.

**Head circumference.** Researchers are trying to determine if head growth is an
indicator of autism. Lainhart (2003) suggested that if increased head growth in infancy is
a risk factor for autism, then it must precede the onset of the disorder. Increased rate of
head growth during infancy might precede parental and clinical recognition of autism
even if the underlying neurological differences are already present. Recognition of a disorder is not always coincident with its onset. Several research studies explored the possibility of head circumference as being either an identifying or a determining factor in autism.

**Infancy.** It appears that at birth HC is within normal range or is slightly smaller than normal for the majority of autism cases. Macrencephalic HC at birth in autism is uncommon, occurring in approximately 6% of all autism cases (Courchesne & Pierce, 2005). Different research groups have found similar average newborn HC size in autism (Courchesne & Pierce, 2005). The average HC at birth in autism was 34.7 cm in Gittberg and de Souza (2002) study, 34.41 cm in Lainhart et al. (1997) study, and 34.65 cm in Courchesne et al. (2003) study, as well as Huttman et al. (2002) reporting the same average range for their sample.

Some studies reports a very small percentage of autistic newborns have an extremely large HC. For instance, excessive HC at birth in autism was reported for 4 of 42 (Gillberg & de Souza, 2002), 5 of 206 (Mason-Brothers et al., 1990), 3 of 51 (Lainhart et al., 1997), and 1 of 15 (Courchesne et al., 2001) cases. Among a sample of autistic children who were pre-selected because of having clinical macrencephaly, only 1 of the 18 had macrencephaly at birth, 1 of the 18 had microcephaly at birth, and the remaining 16 were normencephalic (Stevenson et al., 1997).

Courchesne, Carper, and Akshoomoff (2003) conducted a retrospective analysis of head circumference (HC) measurements. The findings suggested that much of the overgrowth occurred postnatally within the first 6-14-months (Courchesne et al., 2003). This overgrowth seemed to coincide with what is normally a period of exuberant
synaptogenesis, dendritic arborization, and ongoing myelination (Courchesne et al., 2003). Courchesne, Carper, and Akshoomoff (2003) also reported that 59% of the infants diagnosed with ASD showed a HC increase of 1.5-2 S.D. or more across the first year of life as compared to 6% of normal infants. This overgrowth was concluded by about 2 years of age.

Their study also measured for correlations between HC in the first year of life and later neuroanatomical outcome (Courchesne, Carper, & Akshoomoff, 2003). They found that in the infants with ASD, the HC size at birth and HC overgrowth by the end of the first year of life were strongly correlated with abnormal cerebellar and cerebral volumes at 2-5-years-old. Courchesne, Carper, and Akshoomoff (2003) concluded that the clinical onset of autism appears to be preceded by two phases of brain growth abnormality. The first abnormality is a reduced head size at birth, and the second is an overgrowth in head size between 1 to 2 months and 6 to 14 months. Abnormal rates of head growth may serve as an early detection for autism.

Using linear mixed-effects models, Dissanayake, Bui, Huggins, and Loesch (2006) compared the HC, height, and weight measurements of three infant groups. The measurements were extracted from health records over the first 3 years of life for 16 children with high-functioning autism (HFA), 12 children with Asperger disorder (AsD), and 19 typically developing children. They found that the HFA and AsD groups were similar in their HC, height, and weight, and had a higher growth rate than the control group. Dissanayake, Bui, Huggins, and Loesch (2006) concluded that the results indicate that in autism, growth dysregulation is not specific to the brain but also involves growth in stature.
**Childhood.** Gillberg and de Souza (2002) did a study of head circumference (HC) in children with Asperger disorder (AsD), lower functioning autism, and typically developing children. Their study determined that the children with AsD were more likely to have an enlarged HC at birth as compared to the other two groups. Moreover, the AsD group was likely to have a larger HC later in development.

A study by Barthotomeusz and colleagues (2002) tested the correlation between HC and brain volume in children with autism and a control group. They used MRI to gather the data. Their study reported that in both children with autism and the control group, HC was an accurate index of brain volume in young children (Barthotomeusz et al., 2002).

The objective in a study by Hazlett et al. (2005) was to examine brain volume and HC in children with autism as compared with a control group. At the first time point in an ongoing longitudinal MRI study of brain development in autism, they preformed a cross-sectional study of brain volume on 51 children with autism and 25 control children between 18 and 35 months of age. They also gathered retrospective longitudinal HC measurements from birth to 3-years-old of 113 children with autism and 189-control group of children.

Hazlett et al. (2005) found significant enlargement in cerebral cortical volumes in the ASD group. In addition, the ASD group had enlargement in both white and gray matter. This enlargement was generalized throughout the cerebral cortex. It was also determined that the HC appeared normal at birth, but followed by a significant increased rate of HC growth beginning around 12-months-old. Finally, they concluded that indirect
evidence suggests that this increased rate of brain growth in autism may have its onset postnatally in the latter part of the first year of life.

Head growth rates are often accelerated in autism, so to explore this further Sacco et al., (2007) devised a study aimed at defining the clinical, morphological, and biochemical correlates of HC in autistic patients. HC were measured in 241 individuals with ASD, 3-16-years-old. They assessed clinical parameters using the Autism Diagnostic Observation Schedule, Autism Diagnostic Interview- Revised, Vineland Adaptive Behavioral Scales, intelligence quotient (IQ) measures, and an ad hoc clinical history questionnaire. Sacco et al., (2007) also recorded the height and weight of the participants, as well as their serotonin blood levels and peptiduria.

Sacco et al., (2007) identified that the distribution of HC was significantly skewed toward larger head sizes. Additionally, macrocephaly was part of a broader macrosomic endophenotype, which was characterized by highly significant correlations between HC, weight, and height. Likewise, a HC >75th percentile was associated with more impaired adaptive behaviors and with less impairment in IQ measures, as well as less impaired motor and verbal language development. Moreover, large HC were significantly associated with a positive history of allergic/immune disorders both in the patient and in first-degree relatives. Their study demonstrates the existence of a macrosomic endophenotype in autism. Moreover, it points toward pathogenetic links with immune dysfunctions that they speculate either lead to or are associated with increased cell cycle progression and/or decreased apoptosis.

Postmortem. Redcay & Courchesne, (2005) conducted a postmortem study on individuals who had been diagnosed with ASD. They reviewed reports of autism head
circumference (HC), MRI, and postmortem brain weight (BW) were identified and analyzed. Next, Redcay & Courchesne, (2005) calculated percent difference from normal values and standardized mean differences to compare brain size across studies and measurement methods.

Their results revealed a largely consistent pattern of brain size changes. In autism, they found there was a slight reduced of brain size at birth, followed by a dramatic increased within the first year of life, and followed by a plateauing of brain size by adulthood. Redcay & Courchesne, (2005) findings reveal a period of pathological brain growth, occurring in the first years of life, before the typical age of clinical diagnosis.

**Genes.** The HOXA1 gene has a critical role in the development of hindbrain neural structures. Conciatori and colleagues (2004) found an association between the HOXA1 gene and autism. In a combined case-control and family-based association design, Conciatori et al. (2004) showed that the HOXA1 A218G polymorphism contributed significantly to findings of large HC in individuals with autism. It is unclear if this genotype-phenotype correlation was specific to autism, or related to large HC. Their study supports the usefulness of HC as a potential endophenotype in autism.

**Neuroanatomy**

**White and gray matter.** The white and gray matters of the brain are responsible for passing information between the cell bodies. Researchers have conducted studies to determine if there are any abnormalities of the brain matter in autism. Furthermore, if there are abnormalities, are they isolated to specific brain regions or the whole brain?
Past studies determined that the brain sizes of individuals with ASD were inversely correlated with the ratio of inter-hemispheric white matter to gray matter (Jancke et al., 1997; Jancke et al., 1999; Rilling & Insel, 1999). An increase in both white and gray matter volumes have also been reported, with especially pronounced increases of 18% in cerebral and 39% in cerebellar white matter (Courchesne et al., 2001). They also found that the maximum cerebral gray matter volume was reached in individuals with autism by 2-4 years of age, which was about 4-6 years earlier than typically developing children (Courchesne et al., 2001).

In a study of cerebral white matter, researchers discovered that the subregion with the greatest deviation from normal was the white matter underlying the prefrontal cortex (Herbert et al., 2004). Another white matter study of individuals with autism reported abnormalities in white matter diffusion patterns (Barnea-Goraly et al., 2004). These abnormalities appeared in the medial and dorsolateral frontal regions, temporal lobes, temporoparietal junction, and anterior regions of the corpus callosum. Carper et al. (2002), Walter et al. (2005), Buxhoeveden et al. (2006) conducted similar studies and found abnormalities in the corpus callosum, and suggested it may play a role in the pathophysiology of ASD.

In a fully automated voxel-based whole brain volumetric analysis, McAlonan et al. (2005) compared children with ASD and age-matched controls. They hypothesized there would be similar brain structural changes to prior studies and there would be structural dysconnectivity in the children with ASD.

Findings indicated that the children with ASD had a significant reduction in total gray matter volume, especially in the ventral and superior temporal regions, and
significant increase in cerebral spinal fluid volume (McAlonan et al., 2005). Like wise, there was a reduction of the white matter in the cerebellum, left internal capsule, and fornices. Palmen et al. (2005) and Hazlett, Poe, Gerig, Smith, and Piven (2006) conducted similar studies, which had the same findings. The findings suggest abnormalities in the anatomy and connectivity of limbicstriatal 'social' brain systems may contribute to the brain metabolic differences and behavioral phenotype in autism (McAlonan et al., 2005; Palmen et al., 2005; Hazlett et al., 2006).

Wassink and colleagues (2007) conducted a longitudinal brain MRI study to evaluate whether 5-HTTLPR, a functional promoter polymorphism of the serotonin transporter gene SLC6A4, influenced cerebral cortical structure volumes in 44 male children, 2-4-years-old with autism. They found that 5-HTTLPR genotype influenced gray matter volumes of the cerebral cortex. Additionally, there was an influence of gray matter in the frontal lobe. They concluded that the SLC6A4 promoter polymorphism 5-HTTLPR did influences cerebral cortical gray matter volumes in young male children with autism.

**Lobes and cortices.** The brain has several subdivision called lobes and cortices. Each section has a different role and responsibility in the central nervous system. Certain cognitive and behavioral deficits suggest that there may be several lobe and cortex abnormalities in patients with autism. However, little anatomical research is available to verify or refute this (Carper & Courchesne, 2000), so researchers are focusing on this area.

**Cerebral cortex.** The cerebral cortex is the extensive outer layer of gray matter of the cerebral hemispheres. It is responsible for higher brain functions, coordination of
sensory information, voluntary muscle movement, learning, thought, reasoning, memory, and the expression of individuality (The American Heritage New Dictionary, n.d.). Investigations of the abnormal brain overgrowth in individuals with autism, which occurs within the first years of life, is due to enlargement of cerebral, cerebellar, and limbic structures (Carper and Courchesne, 2000; Carper et al., 2002; Courchesne et al., 2001; Sparks et al., 2002). Later sections address the cerebellar and limbic structures.

In a previous cross-sectional study, Carper et al. (2002) found abnormal enlargement of cerebral cortex and cerebral white matter volumes in 2-3-year-old children with autism. They also found abnormally slow rates of volume change across later ages. Additionally, in neuropathological studies of cerebral cortex in autism, researchers found abnormalities of synaptic and columnar structures (Williams et al., 1980; Casanova et al., 2002).

Researchers have found that the cortical areas most affected are precisely those broadly projecting, phylogenetically, and ontogenetically late-developing regions (Belmonte et al., 2004). These regions are essential to complex cognitive functions such as attention, social behavior, and language (Belmonte et al., 2004). Like wise, in neurobehavioral studies of individuals with autism, researchers have discovered associations between cerebellar anatomic abnormalities and certain motor, cognitive, and social deficits (Haas et al., 1996; Harris et al., 1999; Townsend et al., 1999; Pierce and Courchesne, 2001). Moreover, recent genetic (Gharani et al., 2004; Vaccarino et al., 2009) and MRI-behavior correlation (Akshoomoff et al., 2004; Kates et al., 2004) studies suggest that cerebellar abnormality may play a more central role in autism than previously thought.
**Frontal lobe and cortex.** The frontal lobe is the largest and forward most lobe of each cerebral hemisphere in the brain (*The American Heritage Science Dictionary*, n.d.). It is responsible for the control of skilled motor activities, mood, and the ability to think and reason. Within the frontal lobe, there are two subdivisions, the dorsolateral and medial frontal cortices, and their role is higher-order cognitive, language, speech, and social functions (Courchesne & Pierce, 2005). Since individuals with autism deficits in these areas, researches have conducted studies to determine the cause of the deficits. Evidence from previous behavioral, imaging, and postmortem studies indicates that the frontal lobe develops abnormally in children with autism. It is not yet clear to what extent the frontal lobe is affected (Carper & Courchesne, 2005).

A study by Carper and Courchesne (2000) showed a frontal lobe cortex volume increase in a subset of patients with autism. In addition, they found that the increase correlates with the degree of cerebellar abnormality. Carper and Courchesne (2000) concluded that the evidence of concurrent structural abnormalities in both the frontal lobe and the cerebellum has important implications for understanding the development and persistence of ASD (Carper & Courchesne, 2000), and there needs to be more research.

Another study by Carper and Courchesne (2005) measured cortical volume in four subregions of the frontal cortex in 2-9-year-old boys with autism and neurologically typical boys. The dorsolateral region showed a reduced age effect in the boys with autism when compared with control subjects. There was a predicted 10% increase in volume from 2-9-years-old as compared with a predicted 48% increase for control subjects. Additionally, there was a significant enlargement of the dorsolateral and
medial frontal regions in the 2-5-year-old boys with autism. However, they found no significant differences in the precentral gyrus and orbital cortex. Carper and Courchesne (2005) concluded that their data indicated regional variation in the degree of frontocortical overgrowth with a possible bias toward later developing or association areas.

Buxhoeveden et al. (2006) found cell minicolumns to be narrower in frontal regions in brains of individuals with autism as compared with controls. Within the frontal cortex, dorsal and orbital regions displayed the greatest differences while the mesial region showed the least change. They also found that in contrast to the controls, the minicolumns in the brain of 3-year-old children with autism were indistinguishable from those of adults with autism in two of three frontal regions. They suggested that it might have been due to the small size of the columns in the adult autistic brain rather than to an accelerated development. Buxhoeveden et al. (2006) concluded that the presence of narrower minicolumns supports the theory that there is an abnormal increase in the number of ontogenetic column units produced in some regions of the brain of individuals with autism during corticoneurogenesis.

Levitt et al. (2003) examined different brain regions in a group of 11-year-olds boys with autism. They found abnormality of the dorsolateral frontal, medial frontal and temporal regions. They also found anterior and posterior shifting of several frontal and temporal sulci, with the greatest deviation from normal being superior frontal, inferior frontal and superior temporal sulci and the Sylvian fissure.

Carper et al. (2002) assessed whether the volume abnormalities were limited to particular cerebral regions or are pervasive throughout the cerebrum. They used MRI
scans to quantify volumes of frontal, temporal, parietal, and occipital lobe regions of 38 boys with autism and 39 normal control boys between 2-11-years-old. Several regions showed signs of gray matter and white matter hyperplasia enlargement, as much as 20%, in 2-3-year-old patients. Additionally, they found that the frontal lobe showed the greatest enlargement, where as the occipital lobe was not significantly different from the controls.

**Orbitofrontal cortex.** The orbitofrontal cortex is part of the frontal lobe. It is involved in multiple psychologic functions, such as emotional and cognitive processing, learning, and social behavior (Hardan et al., 2006). These functions are variably impaired in individuals with autism.

Recent evidence has implicated the orbitofrontal cortex (OFC) in the pathophysiology of social deficits in autism. Girgis et al. (2007) conducted a MRI-based morphometric study of the OFC involving 11 children with autism and 18 controls. They found a decreased gray matter volume in the right lateral OFC in the autism group. Additionally, they observed correlations between social deficits and white, but not gray, matter structures of the OFC. Their findings support the role of OFC in autism and warrant further investigations of this structure using structural and functional methodologies.

Hardan et al., 2006 examined the size of the OFC, and its medial and lateral subdivisions of 40 individuals with autism and 41 controls. They preformed MRI scans on the two groups and analyzed. From the data, Hardan and colleagues (2006) found no differences between the individuals with autism on any of the OCF measurements.
However, Hardan and colleagues (2006) compared the group with autism and the controls, and found a smaller right lateral orbitofrontal cortex in children and adolescents with autism. Furthermore, they observed a larger right lateral orbitofrontal cortex in adult patients. In addition, they discovered a positive relationship between circumscribed interests and all orbitofrontal cortex structures in the autism group. These findings suggest the absence of global volumetric abnormalities in the orbitofrontal cortex in autism, as well as indicate that there may not be a relationship between the functional disturbances in this structure and anatomic alterations.

**Medial temporal lobe.** MRI studies show that there is enlargement of the temporal cortex at 2-4 years of age in children with autism and failure to grow thereafter (Carper et al., 2002). Part of the temporal cortex is the medial temporal lobe. Researchers have discovered abnormalities in this section of the temporal cortex in individuals with autism.

Salmond and colleagues (2005) examined the memory profile of children with ASD and a control group, as well as the relationship to structural abnormalities. The two groups completed a comprehensive neuropsychological memory battery and underwent MRI for the purpose of voxel-based morphometric analyses. Additionally, to further examine the role of the medial temporal lobe in ASD, they explored correlations between cognitive and behavioral test scores and quantified results of brain scans.

Salmond and colleagues (2005) found a selective deficit in episodic memory with relative preservation of semantic memory. Voxel-based morphometry revealed bilateral abnormalities in several areas implicated in ASD including the hippocampal formation. Furthermore, Salmond et al. (2005) found a significant correlation between parental
ratings reflecting autistic symptomatology and the measure of gray matter density in the junction area involving the amygdala, hippocampus and entorhinal cortex. Their data revealed a pattern of impaired and relatively preserved mnemonic function that is consistent with a hippocampal abnormality of developmental origin. Moreover, the structural imaging data highlight abnormalities in several brain regions previously implicated in ASD, including the medial temporal lobes.

*Anterior cingulate cortex.* The anterior cingulate cortex (ACC) is a brain region that assists the corpus Collosum in communication between the left and right hemispheres. Functional imaging studies show that the ACC is associated with integrating information with emotional overtones (Bush et al., 2000). The ACC helps in anticipating and monitoring complex information and processing of conflicting information. Furthermore, it assists in viewing personally important faces and experiencing a feeling of social exclusion (Bush et al., 2000).

Researchers have discovered structural and functional abnormalities of the ACC in individuals with autism (Courchesne & Pierce, 2005). Reported abnormalities have also included abnormal enlargement of the ACC by 2-4-years-old (Carper et al., 2002), reduced ACC volume in adult with autism (Haznedar et al., 1997), as well as underdeveloped minicolumns in the ACC (Buxhoeveden et al., 2006). Research studies have also found that lesions of the ACC lead to autism, decreased motivation, decreased interest in novel information, and a reported reduced "will to act" (Allman et al., 2002).

Pierce et al. (2004) used fMRI scans of individuals with autism and compared them to scans of a control group. They examined the brain regions that are functionally
responsive during the presentation of socially familiar and significant faces. Those with autism showed an absence of medial frontal lobe activity extending from ACC into frontopolar area 10.

Courchesne and Pierce (2005) reviewed MRI, postmortem, diffusion tensor imaging, PET, fMRI and MR spectroscopy studies of individuals with autism and found consistent reports of abnormalities in the ACC. In another postmortem study of individuals with autism, researchers found a small neuron size and increased neuron packing density in the ACC, with the abnormalities being interpreted as evidence of arrest in development (Kemper & Bauman, 1998). All of these findings suggest that abnormalities of the ACC have a significant effect on autism.

Cerebellum. The cerebellum is the trilobed structure of the brain that sits beneath the occipital lobe. It is responsible for the regulation and coordination of complex voluntary muscular movement, the maintenance of posture, and balance (The American Heritage Dictionary, n.d.). Several past neuropathological and neuroimaging studies have found anatomical abnormalities in the cerebellum in individuals with autism (Carper & Courchesne, 2000). Some researchers suggest that the cerebellum is the most common site of anatomic abnormality in autism (Courchesne & Pierce, 2002).

MRI morphometry reveals hypoplasia of the cerebellar vermis and hemisphere studies report reductions in numbers of cerebellar Purkinje cells (Belmonte et al., 2004). Other MRI and postmortem neuropathological studies have implicated the cerebellum in the pathophysiology of autism. MRI studies of the cross-sectional area of the vermis have found both decreases and no difference in autism groups. Volumetric analysis of
the vermis may provide a more reliable assessment of size differences (Scott, Schumann, Goodlin-Jones, & Amaral, 2009).

Scott, Schumann, Goodlin-Jones, and Amaral (2009) conducted a volumetric analysis of the structure of the whole cerebellum and its components in children and adolescents with ASD. They acquired structural MRI scans male participants with ASD and typically developing children. Findings showed a decrease of total vermis volume in the ASD group when compared to the control. Their findings were congruent with past studies.

Previous clinical observations suggest abnormalities within the cerebellum may explain the severe behavioral deficits and increases in seizures in autism. To explore this further, Walker, Diefenbach, and Parikh (2007) created a study, which used a rodent model for the autism-like behaviors. In addition, they explored the possibility for limiting autism-like behaviors via antiseizure brainstem and cerebellar circuitry. Their findings suggest that specific neuronal populations within the cerebellum are responsible for mediating exploration behavior. In addition, these neuronal populations are similar to the circuitry involved in limbic motor seizures in that they are sensitive to brainstem inhibition. Furthermore, their results suggest utilizing connection as a form of treatment to control behavioral deficits seen in autism.

Cleavinger and colleagues (2008) studied the detailed morphometric analysis of the cerebellum in individuals with autism. They acquired quantitative MRI scans from the group with autism and compared them to neurologically typical controls. Additionally, they measured the total cerebellum volumes and surface areas of four
lobular midsagittal groups. Cleavinger et al. (2008) found that in autism the cerebellar structures proportional to head size and did not differ from typically developing subjects.

Although motor deficits are common in autism, the neural correlates underlying the disruption are unknown. In order to gain an understanding of these deficits, Mostofsky et al. (2009) used fMRI to examine neural activation and connectivity during sequential, appositional finger tapping in 13 children with high-functioning autism (HFA) and 13 typically developing children. They discovered that the HFA group demonstrated diffusely decreased connectivity across the motor execution network relative to control children.

Mostofsky and colleagues (2009) concluded the decreased cerebellar activation in the HFA group might reflect difficulty shifting motor execution from cortical regions associated with effortful control to regions associated with habitual execution. Additionally, they concluded that decreased connectivity in the HFA group might reflect poor coordination. These findings might help to explain impairments in motor development in autism, as well as abnormal and delayed acquisition of gestures important for socialization and communication. Motor deficits may be an early sign of abnormal development (Mostofsky et al., 2009).

**Thalamus.** The thalamus is a large ovoid mass of gray matter that relays sensory messages to the cerebral cortex (The American Heritage Dictionary, n.d.). It assists in the coordination of nerve impulses relating to the senses of sight, hearing, touch, and taste. Researchers are interested in determining if there are abnormalities in the thalamus that could help explain autism.
Hardan et al. (2006) conducted a study to examine the volume of the thalamus in autism. Additionally, Hardan and colleagues (2006) wanted to investigate the effect of brain size had on the thalamus. This was in an attempt to replicate a previous study that reported a relationship between brain volume and thalamus. In addition, they examined the relationships between thalamic volumes and clinical features.

Volumetric measurements of thalamic nuclei were performed on MRI scans obtained from 40 individuals with HFA and 41 healthy controls (Hardan et al., 2006). The researchers observed no differences between the two groups for unadjusted thalamic volumes. Additionally, they observed no linear relationship between the total brain volume and thalamic volume in the HFA group. Furthermore, no correlations were observed between thalamic volumes and clinical features. Hardan et al. (2006) stated their findings were consistent with previous reports of an abnormal brain size effect on the thalamus in autism, as well as supported the possibility of abnormal connections between cortical and subcortical structures in this disorder.

The objective of Hardan et al. (2008) study was to also examine the relationship between thalamic volume and brain size in individuals with Asperger's disorder (ASP). Volumetric measurements of the thalamus were performed on MRI scans obtained from 12 individuals with ASP and 12 controls. Hardan et al. (2008), found a positive correlation between total brain volume and thalamic size in controls, but not in ASP subjects. These findings were consistent with previous studies. Hardan and colleagues (2008) concluded that there was an abnormal relationship between the thalamus and its projection areas in ASP.

**Corpus callosum.** The corpus callosum (CC) is the arched bridge of nervous
tissue that connects the left and right cerebral hemispheres. It communication between the two hemispheres (The American Heritage Dictionary, n.d.), and is an index of interhemispheric connectivity (Keary et al., 2009).

Hardan, Minshew, and Keshavan, (2000) measured the size of the seven subregions of the CC in individuals with autism to determine if there were any abnormalities, and they found that the areas of the anterior subregions were smaller. Vidal et al. (2006) also conducted a volumetric study of the CC and found similar abnormalities, concluding that there was abnormal cortical connectivity in autism. In another volumetric study, Keary et al. (2009) examined the size of the CC in individuals with autism to determine the relationship between the structure and cognitive measures linked to interhemispheric functioning. Like the previous studies, Keary et al. (2009) found reductions in total CC volume and in several of its subdivisions, as well as a relationship between volumetric alterations and performance on several cognitive tests.

In a MRI study of 14 individuals with autism and 28 controls, Casanova et al. (2009) found macroscopic morphological correlates to recent neuropathological findings. These findings suggest a minicolumnopathy in autism. They observed that the individuals with autism manifested a significant reduction in the aperture for afferent/efferent cortical connections. Furthermore, they observed a reduced size of the gyral window that directly correlated to the size of the CC. Casanova et al. (2009) suggested that their findings might help explain abnormalities in motor skill development and differences in postnatal brain growth.

**Amygdala and hippocampus.** The brain structures of the amygdala and hippocampus are part of the limbic system. The limbic system is a group of
interconnected structures that are located beneath the cortex and are associated with emotions, memory, motivation, and various autonomic functions (The American Heritage Science Dictionary, n.d.). Previous neuropathologic studies of the limbic system in autism have found decreased neuronal size, increased neuronal packing density, and decreased complexity of dendritic arbors in hippocampus, amygdala, and other limbic structures (Aylward et al., 1999).

Juranek and colleagues (2006) evaluated the volumes of the amygdala, hippocampus, and whole brain MRI scans of children with autism, and identified a significant neurobiologic relationship between symptoms of anxiety and depression with amygdala structure and function. Their results highlight the importance of characterizing comorbid psychiatric symptomatology in autism.

Past studies report an enlargement of the amygdala in children with autism (Sparks et al., 2002; Schumann et al., 2004), but not in adolescents or adults with autism (Aylward et al., 1999; Pierce et al., 2001; Schumann et al., 2004). Researchers are trying to determine the correlations between the amygdala and autism, as well as what causes the changes structural sizes.

Other abnormal structures. Several other research studies have discovered abnormalities of different brain structures in individuals with autism. Langen, Durston, Staal, Palmen, and van Engeland (2007) found an enlargement of the caudate nucleus, and Hardan, Muddasani, Vemulpalli, Keshavan, and Minshew, (2006) discovered increases in total cerebral sulcal, gyral thickness, and in temporal and parietal lobes. Additionally, Hardan, Kilpatrick, Keshavan, and Minshew (2003) observed structural abnormalities of the basal ganglia, which may explain motor deficits related to autism.
Van Kooten et al. (2008) found the fusiform gyrus and other cortical regions supporting face processing were hypoactive in patients with autism. In addition, they discovered neuropathological alterations, especially in neuron density, abnormalities in total neuron number and mean perikaryal volume. Furthermore, this study may provide important insight about the cellular basis of abnormalities in face perception in autism.

**Discussion**

**Directions for Further Research**

Researchers have made strivess towards understanding autism, however, additional research is necessary. In the majority of the previous studies were comprised of Caucasian male children. There is a need to have more studies encompassing a wider age range, greater cultural diversity, and more female participants. Additionally, future studies should strive for larger sample sizes in order to increase the validity.

In order to discover the causes of autism, early-warning signs, and effective treatments of autism (Courchesne, 2004), future research should focus on elucidating the neurobiological defects that underlie brain growth and structural abnormalities in autism. Focusing on the abnormalities that appear during the critical first years of life is essential. Future research focusing on the early process of brain pathology will also be critical to elucidating the etiology of autism (Redcay & Courchesne, 2005).

Moreover, there is also a need for more data on head circumferences at birth, postmortem studies, as well as additional longitudinal neuroimaging and neuropathologic studies (Harden et al., 2001). These studies will help to provide a better understanding of the complexities underlying increased brain size in autism. Furthermore, similar studies of other pervasive developmental disorders are also
necessary before a case can be made that these developmental neuroanatomic abnormalities are specific to autism (Courchesne et al., 2001). Through gathering quantifiable data of the cross-sectional population of those with autism, researchers will come closer to knowing the exact etiology of autism.

Implications for therapist

The current research concludes that there are significant developmental and structural differences in brains of individuals with autism. Although the full extent of these abnormalities is unknown, it is known that these abnormalities cause the brain to function differently. Individuals with ASD process information differently, have difficulties with executive functioning, struggle with perspective taking, and have sensory issues. Knowing these issues provides therapists with an understanding of their clients with ASD, which they can share, with clients. Educating clients with ASD about their brain differences can help clients understand themselves and accept their differences.

Since individuals with ASD brains function differently, therapists must work with them differently. Therapists are unable to use all of the same tools, techniques and strategies as they use with their neurologically typical clients. They have to focus on different route to the same goal and tailor their approach.

Therapists have to be more patient and understanding since progress is often times slower with clients on the autism spectrum, but therapists cannot fragilize their clients and must still hold them accountable for their actions. It is crucial that therapists set extremely clear and concise rules and boundaries since clients with ASD struggle with what is socially appropriate for a therapist/client relationship, but are good rule followers. These rules and boundaries may have to be reiterated or even written down
because often clients with ASD struggle with memory, which is linked to differences in their brains.

The way in which therapists communicate with their ASD clients is somewhat different than they would with other clients. Communication has to be clear, concise, direct, and to the point. Therapists are unable to allude, perspective taking, or ask “what if?” questions because clients with ASD struggle with Theory of Mind and executive functioning. Since individuals with ASD are analytic thinkers, therapist need to use logic rather than emotions and empathy in therapy.

Research suggests that the neurologic differences in individuals with autism cause them to be visual processors and learners. Writing things down is a great way for therapists to explain information to their clients with ASD. In addition, having paper or a dry erase board clients can use to write and draw on can help them explain there their thoughts and process information.

Using visual cues (e.g. stop signs, pictures, objects, etc…) are a useful tool to teach clients communication skills, social interactions, and how to regulate themselves. One useful visual tool is the 0-10 scale with zero being the lowest end of the scale and 10 being the highest. Having the scale gives clients with ASD a numerical value to emotions and behaviors, which they often struggle with understanding and processing. Finding out what visual aides clients with ASD need will enhance the therapeutic process.

Since individuals with ASD have sensory issues caused by their brain abnormalities, as a therapist it is important to be accommodating to their sensory needs. For instance, some clients may be sensitive to lighting, so it is necessary to meet their
needs by either making it brighter or dimmer in the room. In addition, some clients may
be sensitive to sounds and noises, so having appropriate noise control (e.g. white
noise) is important. Having different sensory objects clients can fidget with (e.g. slinky,
Koosh ball, Play-Doh, etc…) can allow them to focus and process information. Even
having a weighted blanket can help some clients calm themselves and be able to
express their thoughts better. Addressing clients’ sensory needs in another way in
which therapists can optimize therapy sessions.

It is essential for therapists to know the neurological differences in their clients
with ASD. It is also important for therapist to understand the impact these differences
have on their clients. Tailoring the therapeutic approaches, tools, and techniques with
maximize the therapeutic outcome for the clients and will optimize their success.

**Application for Adlerian Therapists**

Adlerian therapists are known for adhering to specific theoretical approaches and
therapeutic techniques. These approaches and techniques can be used with a variety of
different kinds of clients. Adlerian therapists take a holistic approach, do not focus on
the diagnostic labels, and encourage their clients. When it comes to clients with ASD,
Adlerian therapists have to alter, change, and tailor their techniques and approaches to
the individual client.

Frequently clients with ASD have been pamper and/or fragilized; they is they
have been treated as if they are fragile, incompetent, been catered to, and given few
responsibilities or accountability., Other clients, especially those who have been
diagnosed as adolescents and adults may have had a long history of being held to
expectations that they do not understand and have been unable to meet. Client with
both these experiences are often discouraged, striving for significance, have a hesitating attitude, feel inferior, and struggle with depression and anxiety. Additionally, clients with ASD are often perfectionist, analytic, and reason with logic.

There are several Adlerian strategies, tools, and techniques to assist clients with ASD to work through their issues. Please note the following suggestions cannot be used on all clients with ASD. Ability to use these techniques depends on clients' individual developmental levels, may work best with higher functioning clients, and all techniques have to be tailored to the individual.

**Pampering.** As Adlerian therapists, we cannot pamper or fragilize our clients with ASD. Although their brains function differently and they may have disabilities, they are to have developmentally appropriate responsibilities, held accountable for their actions, and unable to use their disorder as a crutch. Providing appropriate rules, boundaries, and expectations, and maintaining consistency will help to ensure pampering and Fragilizing do not occur.

**Encouragement.** Some clients with ASD struggle with low self-esteem, feelings of rejection, and do not have a place or purpose in the world. They may feel as though they do not belong and are outsiders. Encouragement is essential to working with these discouraged clients. In addition, assisting clients in determining and writing down their strengths, abilities, and successes can be validating and encouraging. Along with encouraging clients, affirmations, positive attitude, and believing in clients is also important.

**Courage to be imperfect.** Another technique to assist clients in the therapeutic process is to teach them how to have the “courage to be imperfect.” Since clients with
ASD are often perfectionist, and sometimes to an extreme, teaching them ways to be imperfect may help reduce their anxiety, become more self-accepting, and lower stress levels.

One way to teach some clients with ASD how to be imperfect is through games. Creating a simple game with clear rules and having the main object to be imperfect. This may create an environment in which clients are more open, accepting, and able to processes the idea of imperfection.

Another way to teach how to be imperfect is through the use of social stories. Social stories are short and concise stories that are meant to teach, prepare, and assist in processing a variety of behaviors, expectations, and experiences. Creating a social story can be a useful tool that clients can reuse whenever needed. The social story could be used to inform clients that imperfection is acceptable, as well as specific statements to repeat to themselves and behaviors they can do whenever they feel anxious or upset because something is imperfect.

Assigning homework can also be useful when teaching clients how to be imperfect. Homework helps clients to apply what they learned in therapy sessions to their everyday lives. An example of homework might include having the client tally the number of times they experience imperfection in themselves and others, in everyday life. At the beginning of the subsequent session review the tallied imperfections, address accepting these imperfections, and discuss the differences between perfection and good enough. Encouraging and affirming clients on their continual progress, no matter how fast or slow, is necessary during this therapeutic process.

**Forward movement.** Determining what drives and motivates clients (e.g. intrinsic
and extrinsic motivators) with ASD is essential in creating forward movement. Once these drives and motivations are known and understood, they can be used to facilitate movement.

Forward movement is often slow with some clients with ASD and is necessary to be additionally patient. Successful forward movement comes in little victories that need to be noticed, pointed out to clients, and celebrated. Creating a simple progress chart that is visual and tangible is one way to track clients’ movement. This could be helpful to show clients how far they have come since the time they began. Looking back, acknowledging, and celebrating their success will help facilitate additional forward movement, motivational for the clients, and encouraging to them.

**Black and white thinking.** Since individuals with ASD have neurological differences that contribute to their logical and analytical mindset, it creates an all or nothing, black and white way of thinking. This way of thinking can cause distress in some clients because they are unable to see other alternatives to problems or situations. Difficulty seeing the gray area can be detrimental to clients with ASD in their relationships with others.

An example of how to teach about the gray areas is through the following activity. On a dry erase board, or paper, draw three columns titled “Black,” “Gray,” and “White.” In the “Black” column write down a list of words, which have clear opposites and, write the opposite words in the “White” column. It is the clients’ job to try and think of what word goes in the middle “Gray” column (see Figure 1 in the Appendix for an example). Clients may need help thinking of words for the “Gray” column, but be sure to give them time to think.
After completing a few rows, have the clients decide on words to go in the “Black” and “White” column, and then fill in the “Gray” words. Before, during, and at the end of the activity discuss with clients how there are gray areas in life, situations, and problem solving. Also, address logically why gray areas are important and necessary. This activity can help clients to visually see other alternatives.

**Life Style Analysis.** An assessment tool most Adlerian therapists use is the Life Style Analysis (LSA). This tool is useful in determining and understanding clients, their core values, mistaken beliefs, and a variety of other things in their lives. Although the LSA works on most clients, it cannot be administered the same way to clients with ASD. It has to be tailored to the individual client and some sections of the LSA may be uncompleted.

**Early Recollections.** Taking Early Recollections (ERs) from clients with ASD is different than taking ERs from neurologically typical clients. When asking for an early memory, the therapist has to be more specific than just asking for any memory because it can be obscure, unclear, and overwhelming to some clients with ASD. For instance, ask, “Tell me a memory of you playing when you were seven years old.” Also, therapists are unable to transform ERs of clients with ASD. It is also too obscure and requires different processes of thinking that clients with ASD struggle with and are often unable to perform. Even though ERs are slightly different in individuals with ASD, they can still be used to uncover current belief systems, mistaken beliefs, and most all other uses which ERs are typically used for.

**Life Task.** The most successful way to assess Life Task in individuals with ASD is
to use scaling (i.e. 1-5 or 1-10). Writing down the numbers or drawing number points on a line is helpful for clients to see. Giving the different areas of Work, Family/Friends, Significant Other/Love, Self-Care, and Spirituality a numeric value assist in determining what areas clients need to work on while being visual, concrete, and easier to process.

“The Question”. “The Question” is a diagnostic tool Adlerians use to determine what the current issues are in clients’ lives. It can be very useful and informative. The therapist will ask clients some variation of “You fell asleep and while you were sleeping a miracle happened. When you woke up what would be different?”

Asking a client with ASD “The Question” unfortunately does not work as well as it does with neurologically typical clients. Some very high function clients might be able to answer it, but will need additional help understanding it, processing it, and coming up with answers. Often time clients with ASD will answer the question with, “I do not know.” They honestly do not know how to answer the question because to them it has not happened yet so they do not know what the future holds. They are truly unable to process or think that abstractly. Since their brains function differently, this type of technique is too obscure and requires a form of brain processing that is difficult and challenging for them. Instead, therapist need to focus on using more logical, concrete, visual, and tangible means of diagnostic tools when working with clients with ASD.

Conclusion

For almost three decades, there has been an increase in the number of individuals diagnosed with ASD. There has been a need for refined diagnostic tools, more services and treatment options, as well as more research because of this increase
in ASD diagnosis. Researchers have been trying to gain a better understanding of ASD etiology,

Although the exact etiology of autism is still not known, researchers have made strides in understanding the neurologic basis for autism. Because of the past research, it is now known that there is overgrowth in the brain causing great brain volumes and head circumferences of individuals with autism, which occurs in the first few years of life and is followed by a normalizing period of development. Additionally, research has discovered abnormalities in the neurologic connection, patterns of white and gray matter, as well as developmental and structural abnormalities in specific brain regions. There is a need for more research in order to fully understand the complex developmental and structural abnormalities in autism, the role these abnormalities play, and to determine the exact etiology of autism.

It is essential for all therapists working with clients on the autism spectrum to know and understand the current research, how it affects clients, and be able to implement what they have learned. Therapists are responsible for tailoring their therapeutic approaches to the individual client. They also have to be creative and adaptive with their assessment and techniques. Treating clients with ASD respectfully, giving encouragement, being consistent, and having obtainable expectations will assist in facilitating change, growth, and forward movement, as well as create a positive environment for success.
References


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Cleavinger, H. B., Bigler, E. D., Johnson, J. L., Lu, J., McMahon, W., Lainhart, J. E.


Hardan, A. Y., Girgis, R. R., Lacerda, A. L. T., Yorbik, O., Kilpatrick, M., Keshavan, M.


Lainhart, J. E., Piven, J., Wzorek, M., Landa, R., Santangelo, S. L., Coon, H., …


McAlonan, G. M., Cheung, V., Cheung, C., Suckling, J., Lam, G. Y., Tai, K. S.,


Palmen, S. J. M. C., Hulshoff Pol, H. E., Kemner, C., Schnack, H. G., Durston, S.,


Appendix

*Figure 1.* This is an example of how to create the “Black, Gray, and White” activity. This activity can be used in helping to teach clients with Autism Spectrum Disorders other alternatives and gray areas in life.

<table>
<thead>
<tr>
<th>Black</th>
<th>Gray</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cold</td>
<td>Cool/Warm</td>
<td>Hot</td>
</tr>
<tr>
<td>Happy</td>
<td>Content</td>
<td>Sad</td>
</tr>
<tr>
<td>Starving</td>
<td>Full/Satisfied</td>
<td>Stuffed</td>
</tr>
<tr>
<td>Tall</td>
<td>Average</td>
<td>Short</td>
</tr>
<tr>
<td>Hate</td>
<td>Like</td>
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<tr>
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