

# FLEX CEUs



## Autism Spectrum Disorder



# OCCUPATIONAL THERAPY FOR AUTISM SPECTRUM DISORDER

Flex Therapist CEUs

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## INTRODUCTION

Autism Spectrum Disorder (ASD) is a developmental disability characterized by persistent social/communicative difficulty in multiple context areas. Autism was first identified in the 1960s as a much more severe condition which typically also included intellectual disability. At the time, autism was thought to be a rare condition, with only 4 to 5 cases diagnosed per 10,000 children. It wasn't until 1980 that autism was included in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III). Though initial clinical criteria for the diagnosis were narrow, the medical definition of autism has broadened to include a wider variety of impairments, behaviors, and deficits, thus beckoning the addition of "spectrum" to the condition's full name. The most recent edition of the DSM (DSM-V) has made changes to include all subtypes of autism under one diagnosis code (whereas previously Asperger's and other related types of autism were separated). However, three new levels of severity allow for a more specific diagnosis. Another update includes the mention of sensory processing deficits as a defining characteristic of ASD. The DSM-V also includes the related but separate diagnosis of Social Communication Disorder. Today, about 1 in 68 children is diagnosed with ASD. Autism appears to affect boys at a higher rate than girls, with a boy being 4.5 times as likely to be diagnosed with the condition. (CDC, 2016). Of importance to note is that this profound increase can be "largely attributable to broadening diagnostic criteria, younger age at diagnosis, and improved case ascertainment" (Main, 2012).

Besides just diagnostic criteria, our understanding of autism as a society has changed in recent years. Many individuals with autism prefer to think of the diagnosis as a "condition" instead of a disorder. These individuals consider themselves "neurodivergent" (in contrast to neurotypical) and consider autism as part of their identity, much like many modern Deaf people. They reject the idea of a "cure," or even many traditional therapies. Future policies and treatment should be decided with the input of this group. (Silva, 2013).

## DIAGNOSIS

ASD is typically evaluated by a multidisciplinary team, usually headed by a clinical psychologist. An evaluator will use DSM-V criteria to make a specific diagnosis of mild, moderate, or severe autism, and whether it includes accompanying intellectual impairment. Additionally, genetic and metabolic

conditions such as Rett Syndrome, Tuberous sclerosis, PKU, Fragile-X, Angelman, and Prader willi are also often related to ASD and will be considered in diagnosis. Though little information has been found about the cause of ASD, “multiple genes are thought to be involved in the pathogenesis.” (Dave, 2014).

Early detection of ASD is key. Symptoms often manifest within the second year of life and as such, screening for developmental disabilities at this age is recommended. Though not every child will be able to be diagnosed so early, some children already display significant communication and motor deficits that allow for early diagnosis. This early diagnosis is very valuable as it improves the prognosis of ASD overall. (Gambino, 2014).

The following is the full text diagnostic criteria from the DSM-5, reproduced here from the Autism Speaks website, 2016:

### **DSM-5 Diagnostic Criteria**

*A. Persistent deficits in social communication and social interaction across multiple contexts, as manifested by the following, currently or by history:*

- 1. Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.*
- 2. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures; to a total lack of facial expressions and nonverbal communication.*
- 3. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.*

***Specify current severity:*** Severity is based on social communication

*impairments and restricted repetitive patterns of behavior.*

*B. Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history:*

- 1. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).*
- 2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns or verbal nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat food every day).*
- 3. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interest).*
- 4. Hyper- or hypo-reactivity to sensory input or unusual interests in sensory aspects of the environment (e.g., apparent indifference to pain / temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).*

***Specify current severity:*** *Severity is based on social communication impairments and restricted, repetitive patterns of behavior.*

- C. Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).*
- D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.*
- E. These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. Intellectual disability and autism spectrum*

*disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual disability, social communication should be below that expected for general developmental level.*

*Note: Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger's disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for social (pragmatic) communication disorder.*

**Severity levels for autism spectrum disorder (Autism Speaks, 2016)**

<b>Severity Level</b>	<b>Social Communication</b>	<b>Restricted, Repetitive Behaviors</b>
<i>Level 3 "Requiring very substantial support"</i>	<i>Severe deficits in verbal and nonverbal social communication skills cause severe impairments in functioning, very limited initiation of social interactions, and minimal response to social overtures from others. For example, a person with few words of intelligible speech who rarely initiates interaction and, when he or she does, makes unusual approaches to meet needs only and responds to only very direct social approaches</i>	<i>Inflexibility of behavior, extreme difficulty coping with change, or other restricted / repetitive behaviors markedly interfere with functioning in all spheres. Great distress / difficulty changing focus or action.</i>
<i>Level 2 "Requiring substantial support"</i>	<i>Marked deficits in verbal and nonverbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions; and reduced or abnormal responses to social overtures from others. For example, a person who speaks simple sentences, whose interaction is limited to narrow special interests, and [who] has markedly odd nonverbal communication.</i>	<i>Inflexibility of behavior, difficulty coping with change, or other restricted / repetitive behaviors appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress and/or difficulty changing focus or action.</i>
<i>Level 1 "Requiring</i>	<i>Without supports in place, deficits in social communication cause</i>	<i>Inflexibility of behavior causes significant interference with</i>

<i>support”</i>	<i>noticeable impairments. Difficulty initiating social interactions, and clear examples of atypical or unsuccessful response to social overtures of others. May appear to have decreased interest in social interactions. For example, a person who is able to speak in full sentences and engages in communication but whose to-and-fro conversation with others fails, and whose attempts to make friends are odd and typically unsuccessful.</i>	<i>functioning in one or more contexts. Difficulty switching between activities. Problems of organization and planning hamper independence.</i>
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How a child visually interacts with their parent or caretaker may provide insight as to the possibility of autism. Even in early infancy, children are naturally inclined to look towards a caregiver’s face or eyes, a behavior that aids in social development. In children with autism, decreased eye contact is often one of the first red flags that development may be abnormal. “Unlike what happens with typically developing individuals, who consistently show activation of the fusiform gyrus for this kind of stimulus, people with autism show weak or no significant activation in this area, but show activation of areas not commonly associated with facial recognition. Thus one can say that individuals with autism 'see' faces using neural systems different from those used by people of typical development.” (Silva, 2013).

**THEORIES**

Three major theories exist that seek to explain possible differences in the brains of individuals with autism: the Theory of Mind, Executive Dysfunction, and Weak Central Coherence. (Silva, 2013.)

The Theory of Mind (ToM) is an ability that humans have that allows them to both realize they have individual thoughts, beliefs, and desires while simultaneously understanding others have these individual perspectives as well that are different than their own. (Silva, 2013.) This process can be seen on imaging studies, showing activation in various brain structures. (Silva, 2013.) In individuals with ASD, and even some other conditions, this ability is not as present. (Silva, 2013.) This is evidenced by decreased activity in those same brain structures. (Silva, 2013.) “The ability to understand their own emotional states, as well as those of

others, yet a consequence of deficits in ToM, is also impaired, which implies consequences on the ability to 'fit in,' hampering their participation in social contexts and contributing to the increased rates of depression among these individuals. Even with improvements in self-awareness resulting from experience and motivation, persistent difficulty in learning compensatory strategies in social communication is a barrier difficult to transpose.” (Silva, 2013.) It is thought that ToM develops alongside executive functions, such as cognitive flexibility, working memory, impulse control, and planning. (Silva, 2013.) As Executive Dysfunction is common in ASD, this is further evidence that underdeveloped ToM is a factor as well. (Silva, 2013).

Weak central coherence is another popular theory. It refers to an inability to see the big picture in any given situation, instead focusing on small details. (Silva, 2013.) “Seeing a wooden object in a rectangular shape, with a pair of doors with locks and handles, one immediately recognizes it as a closet, as well as eyes, nose, and mouth in a particular arrangement is a face. When there is a failure in this integrative mechanism it is said that there is a weak central coherence. A weak central coherence makes it hard for the individual to recognize the stimuli that surround him globally, making the world a series of parts, instead of an integrated whole, as in typical development subjects.” (Silva, 2013.) This cognitive style may underlie the communication difficulties that people with autism often have; often very direct and literal. Connections and information processing are typically done on a local rather than global level, leading to heavy focus on small details. (Silva, 2013.) Other disorders such as ADHD and intellectual disability share some of these features. (Silva, 2013.) With the advent of more advanced imaging technologies, and increased knowledge on how to read them, we will continue to learn more about brain differences of people with ASD.

## POSSIBLE CAUSES

### Pesticides

Autism currently has no known cause, though heavy research in this area continues to occur. One possible correlation that has been found is pesticides - recent studies have shown a potential link between early exposure to these chemicals and neurological development. Studies done on a particular insecticide, Imidacloprid, have shown an maladaptive effect on behavior in animal test subjects. Another study found that reported Imidacloprid exposure during pregnancy was two times as high in mothers of children with ASD versus typically developing children. The mechanism in which Imidacloprid interacts in the human body is also similar to organophosphates, which have previously been found to be linked with ASD. First available to the public in 1999, Imidacloprid is used in flea

and tick treatment as well as in agriculture. Though its negative effects are not fully known, its use has increased steadily since its arrival on the market. Current research shows that the pesticide can be absorbed dermally when petting animals, though it is unknown if this is a significant enough dose to reach the fetus. More studies need to be done on the direct neurotoxic effect on both fetuses and developing children. (Keil, 2014).

### Immune Dysregulation

Studies have also shown a link between immune system dysregulation and ASD. (Zerbo, 2014). “Several research groups have found that individuals with ASD have increased neuroinflammation in brain tissues, imbalances in immunoglobulins, including increased levels of plasma IgG4, reduced levels of total IgG or reduced levels of IgG and IgM, and imbalances in cytokine/chemokine levels.” (Zerbo, 2014). Like many possible causes of ASD, the interaction between the immune system and ASD are not well understood. Cytokines and chemokines are proteins that have various functions in the body, many of which relate to neurodevelopment. (Zerbo, 2014). A disruption in normal amounts of these proteins can cause problems during fetal development. (Zerbo, 2014). “For instance, in animal models, injection of the cytokine IL-6 or IL-2 into pregnant mice leads to neurodevelopmental abnormalities in the offspring including decreased prepulse inhibition and latent inhibition, attention, exploratory and social behaviors. These findings suggest that reported associations between maternal infections or inflammation during pregnancy and ASD could be mediated through a disruption in the balance of cytokine or chemokine levels. [One study] found elevated levels of MCP-1 and decreased levels of [Regulated upon Activation Normal T-cell Expressed and Secreted] (RANTES) in the newborn blood of children subsequently diagnosed with ASD. Levels of RANTES and MIP-1 $\alpha$  were also decreased in children later diagnosed with [developmental delay] compared to [general population] controls.” (Zerbo, 2014). The findings of this study indicate that it may be possible to identify ASD and other neurodevelopmental disorders from early immune system function alone.

### Oxidative Stress

Glutathione, or GSH, (L-g-glutamyl-L-cysteinyl-glycine) is another possible factor. It is an intracellular peptide that has multiple functions in the body, including detoxification, maintenance of intracellular redox balance, signal transduction, and apoptosis, and cysteine storage. It is also an antioxidant. Overall cell function and viability can be assessed by glutathione levels. “The ratio of glutathione:Glutathione disulfide (GSSG), or the glutathione redox ratio, is a sensitive index of oxidative stress, which can lead to a toxic imbalance between

the production and removal of reactive oxygen species (ROS). A shift in the glutathione redox ratio towards the oxidized state may lead to decreased cell proliferation, DNA damage, and increased apoptosis that could potentially affect neurological development in the early stages of life.” (Main, 2012). A decreased glutathione redox ratio has been found in many people with ASD. It is possible that this process may actually have a hand in causing autism. (Main, 2012).

These findings support the assertion that children with ASD are more likely to have lower glutathione levels overall. One study showed that while levels of serum homocysteine and cystathionine levels remain relatively similar in both neurotypical children and children with ASD, serum cysteine levels are generally lower in children with ASD, reinforcing the connection with glutathione. (Main, 2012) “As the bioavailability of cysteine is the rate limiting factor for synthesis of GSH, the lower cysteine levels detected in serum of children with autistic disorder could be an important factor leading to the lower levels of GSH observed in many children with this condition. The higher level of GSSG observed in the serum of many children with autism spectrum disorders is likely to truly reflect increased oxidative stress as there is no significant difference in GPx-1 activity in serum or platelets and GPx-1 is significantly lower in the erythrocytes of children with autistic disorder compared to controls. As cysteine itself may have strong antioxidant properties, its lower concentration in children with autistic disorder may contribute to increased oxidation of GSH” (Main, 2012). This information shows that many of these cells may require more GSH for optimal function. It is possible that a supplement including glutathione may benefit children with ASD. (Main, 2012).

#### **Brain Derived Neurotrophic Factor (BDNF)**

Brain Derived Neurotrophic Factor (BDNF) is a neurotrophin that may be involved in autism. It exists in the CNS and assists with neuronal survival, neurogenesis, and synaptic plasticity. Differences in BDNF levels in children with ASD and typically developing children have been noted. More research is needed to explore the role of BDNF in the pathology of autism. (Dave, 2014).

#### **Vitamin B<sub>6</sub>**

Plasma vitamin B<sub>6</sub> levels in children with autism are known to be higher than that of typically developing children. This may indicate a dysfunction within the cell itself to store or retain needed B<sub>6</sub>. B<sub>6</sub> is integral in the synthesis of many neurotransmitters such as serotonin, dopamine, and taurine, and as such, a lack of it could have a larger effect on neurological development. (Main, 2012).

## Genetics

Genetic research has been one of the most prolific areas in which information has been found to help us better understand the cause of ASD. During studies with identical twins, there was an 82-92% chance that if one twin was identified as having autism, the second would be as well. This is compared to a rate of only 1-10% in fraternal twins and 6-8% in non-twin siblings. Additionally, heritability rates have been estimated to be greater than 90%. No individual gene has been shown to be associated with autism, rather, it is likely that multiple genes are affected. It is hypothesized that these may even be different genes across different individuals - indicating that ASD may actually be multiple conditions that overlap to create similar pathology. (Main, 2012).

## Environmental Factors

Various factors such as heavy metals exposure, early viral infections such as meningitis, and gastrointestinal flora and function have also been linked to ASD. (Main, 2012).

## TREATMENT

### The Domain of Occupational Therapy

Occupational therapists primarily focus on assisting clients in gaining independence with functional tasks, or Activities of Daily Living (ADLs). Occupational therapists can help people with autism in many ways, though specific deficit areas will vary according to the individual. Collaboration with both the individual and their family, caregivers, teachers, or other support is integral to successful treatment. Services can focus on improving function, caregiver education, or overall quality of life. Evaluation of people with autism should focus on both strengths and challenges, and intervention plans should be tailored to the individual. Depending on setting, an occupational therapist may select strategies to support an individual with tasks at home, in school, or in the workplace. Treatment strategies may include both adaptive strategies such as changing the environment to better suit the individual, or habilitative methods such as identifying and strengthening basic skills needed to complete tasks. (American Occupational Therapy Association, 2011).

### **Ways an Occupational Therapist may address Autism Spectrum Disorder** (American Occupational Therapy Association, 2011).

- Evaluation of the condition's effect on daily living skills and

other age appropriate activities

- Sensory interventions including sensory integration or sensory processing, and developmentally appropriate sensory play activities
- Play interventions that focus on developing social skills
- Transition strategies, both on a micro (from a preferred to a non-preferred activity) and a macro (leaving middle school and starting high school) level
- Developing a plan for safe community mobility, secondary to physical and cognitive concerns
- Identify meaningful or necessary activities/occupations and adapt them as needed

Occupational therapists use a holistic model of treatment. When looking at the whole person, an occupational therapist may also identify potential factors that complicate an ASD diagnosis including epilepsy, gastrointestinal distress, or sleep issues. Referrals to other medical professionals skilled in addressing these conditions may aid in overall function. (Autism Speaks, 2016).

### Behavioral Therapies

In addition to OT, people with autism may also benefit from behavior therapy. (Autism Speaks, 2016) Two models for behavior therapy have been shown to be the most beneficial - Applied Behavior Analysis (ABA) and the Early Start Denver Model (EDSM). Other therapies such as Floortime, Pivotal Response Therapy, and Verbal Behavior Therapy may help too. (Autism Speaks, 2016) Due to the diverse nature of autism spectrum disorders, multiple approaches may need to be tried before the best fit is found.

Regardless of the type of behavior therapy chosen, several things have been found to be in common with successful programs. One facet is early intervention - generally speaking, the earlier behavior therapy services are started, the better. Early intervention usually refers to therapy services for children under the age of 3, but can include children up to 5 in some settings. (Autism Speaks, 2016) With this early intervention, more is better: most professionals recommend at least 25 hours per week of direct therapy. While this therapy is typically delivered by highly trained

therapists/teachers, it is also possible to train skilled paraprofessionals to aid in carrying out interventions under supervision. Another feature of successful programs is having well-written, specific, and measurable goals for the child, with progress checked regularly. These goals should mainly focus on the core areas affected by autism: communication, social skills, play skills, ADLs, and motor skills. Good programs will also give children opportunities to generalize these skills with typically developing peers. And lastly, the most successful programs focus on collaboration, both with the family and with the team of professionals seeing the child, including but not limited to the OT, SLP, and PCP. (Autism Speaks, 2016)

### Sensory Integration

Sensory processing deficits have recently been identified as a common component of ASD. One treatment approach many occupational therapists use is sensory integration, though more research is still needed on the efficacy of this treatment. Sensory Integration, originally developed by A. Jean Ayres, is based on the theory that sensory dysfunction in the neurological system has an effect on behavior, function, and development. “Ayres’s sensory integration enhances nervous system processing of sensation to provide a stable foundation for the formulation and execution of appropriate behavior. Classic Ayres’s sensory integration uses enhanced sensory experiences in the context of meaningful, self-directed activity to support a person’s ability to function adaptively and meet the contextual demands of daily occupations. Because vestibular, proprioceptive, and tactile sensations have powerful effects on the regulatory mechanisms of the nervous system, Ayres’s sensory integration uses these sensations to facilitate production of adaptive behavior. When using Ayres’s sensory integration, occupational therapists use clinical reasoning to guide the intervention process, individualizing the approach to match each child’s unique sensory processing abilities and challenges.” (Watling, 2007). Since many children with ASD have concurrent sensory deficits, this treatment style may be helpful for those who are hypo- or hyper-responsive to stimuli.

A pilot study was performed with the purpose of comparing effectiveness of sensory integration (SI) treatments compared to fine motor treatments. “The results of the study were mixed yet demonstrated significant changes in the autistic mannerisms (a component of social responsiveness) and significant progress toward individualized goals in the areas of sensory processing and regulation, social–emotional function, and [fine motor] skills. No significant differences were found in the scores on the [Sensory Processing Measure] or the [Quick

Neurological Screening Test, 2<sup>nd</sup> Edition]. A subsequent analysis did identify that significantly more children could complete or partially complete the QNST–II after intervention. Results identified significant post-intervention differences in social responsiveness between the SI and FM groups, as determined by [Social Responsiveness Scale] scores in the area of autistic mannerisms. Children in the SI group had significantly fewer autistic mannerisms after interventions. Previous studies found similar outcomes when assessing the reduction of stereotyped or self-stimulatory behaviors in children with [pervasive developmental disorder] after sensory-based or SI interventions. Autistic mannerisms ‘include stereotypical behaviors or highly restricted interests characteristic of autism’” (Pfeiffer, 2011).

### Supplementation

It is possible that certain supplements may help symptoms of ASD. “The dopamine (DA) transporter (DAT) tunes DA neurotransmission by active reuptake of DA from the synapse. [One] laboratory has recently characterized the first de novo mutation in the human dopamine transporter (hDAT) reported in a patient diagnosed with autism spectrum disorder, which results in a Thr to Met substitution at site 356 (hDAT T356M). Clinical data have previously established that mean serum Zn<sup>2+</sup> levels are significantly lower in children diagnosed with ASD compared to unaffected children and that there exist disturbances in Zn<sup>2+</sup> metabolism in patients diagnosed with ASD. hDAT T356M is the first de novo DAT mutation found in a patient with ASD, and hDAT T356M functional deficits can partially be rescued by Zn<sup>2+</sup>.” (Hamilton, 2015). While no direct link between Zn<sup>2+</sup> regulation of hDAT and development of ASD has been found, it is possible that the addition of it later in life may improve symptoms. (Hamilton, 2015). As with all medication/supplementation, caution must be taken, but in the future this type of treatment may become more and more supported.

### Outcomes

It is typically understood that while there is no cure for autism, symptoms can be improved to increase function with daily activities. However, new research suggests that a very small percentage of people originally diagnosed with autism no longer have the disorder, or, at the very least, no longer meet the diagnostic criteria. (Autism Speaks, 2016) Though it is possible that the person in question may have just been misdiagnosed originally, others theorize that it may be possible to “grow out” of certain forms of autism, or, that with enough skilled treatment, individuals can progress past the diagnosis. It is important to

remember that this outcome is not typical, though more research is being done daily on best treatment practices. At the very least, through skilled therapy, most people with autism are generally able to improve their quality of life and participation in daily activities, even though they may still present with symptoms. Many adults with ASD live independently, work typical jobs, and lead very fulfilling lives. Continued research and treatment will hopefully afford this opportunity to everyone with this diagnosis. (Autism Speaks, 2016)

## CONCLUSION

People with autism face many challenges, not least of which includes persistent social difficulty. No current theory can fully explain the cause or complications of ASD, but combined together they can begin to paint a picture of the condition. (Silva, 2013). For the time-being, occupational therapists can work with the knowledge they do have to enrich and improve the lives of individuals with this condition.

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