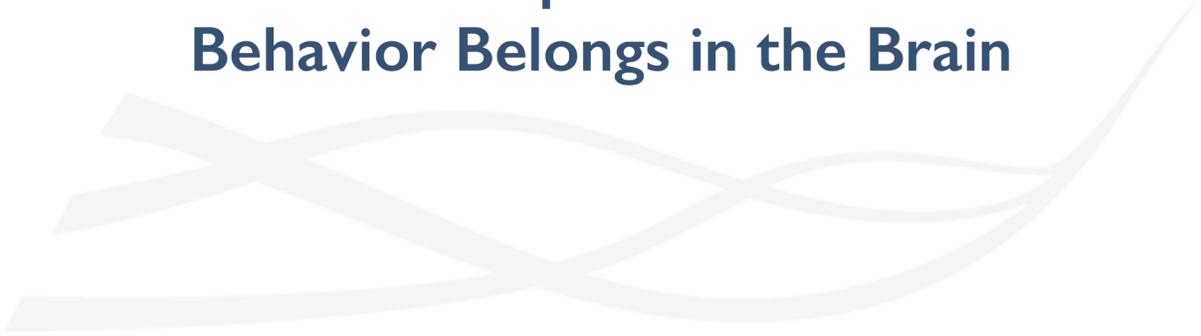


Guided Growth: *Educational Interventions for Children With Fetal Alcohol Spectrum Disorders*

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Chapter 4:

Fetal Alcohol Spectrum Disorders: Behavior Belongs in the Brain



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Fetal Alcohol Spectrum Disorders: Behavior Belongs in the Brain

Fetal alcohol syndrome (FAS) is the original name given to a cluster of physical and mental defects present from birth that is the direct result of a woman's consumption of alcoholic beverages while pregnant. Infants with FAS have signs in three categories: (1) growth deficiencies (2) central nervous system impairment and (3) facial dysmorphism. The mother's confirmed use of alcohol is not necessary to make a diagnosis of FAS if the child meets criteria in all three categories. However, to ensure accuracy and completeness in the child's medical records, physicians usually note when the diagnosis is based solely on these physical and developmental parameters and without confirmation of the mother's drinking.

As we begin this discussion, it is important to realize that the impact of prenatal alcohol exposure is not determined only by the cumulative "dose" of alcohol to which the child was exposed. Many reports demonstrate that a mother's binge drinking, with high peak blood alcohol levels, is actually more dangerous than chronic drinking. Additionally, recent studies regarding adolescents have noted that even "light drinking" during pregnancy has a significant detrimental impact on the adolescent's neurodevelopment.

Criteria for diagnosis

In the initial conceptualization of damage caused by prenatal alcohol exposure, a child was diagnosed with Fetal Alcohol Syndrome if a woman drank alcohol during pregnancy and the child demonstrated poor growth, the classic facial features, and developmental delays. However, we now know that many children prenatally exposed to alcohol have partial or no apparent

expression of these physical features but may have significant structural and functional changes in the brain that cause problems with intellectual, behavioral, or emotional development.

Guidelines published by the Centers for Disease Control and Prevention (CDC) and the Institute of Medicine have attempted to lay out diagnostic criteria that can be applied to the varied signs and symptoms with which children prenatally exposed to alcohol can present. Through a comprehensive evaluation by a multidisciplinary team, children can be assigned an alcohol exposure-related diagnosis based on the following criteria:

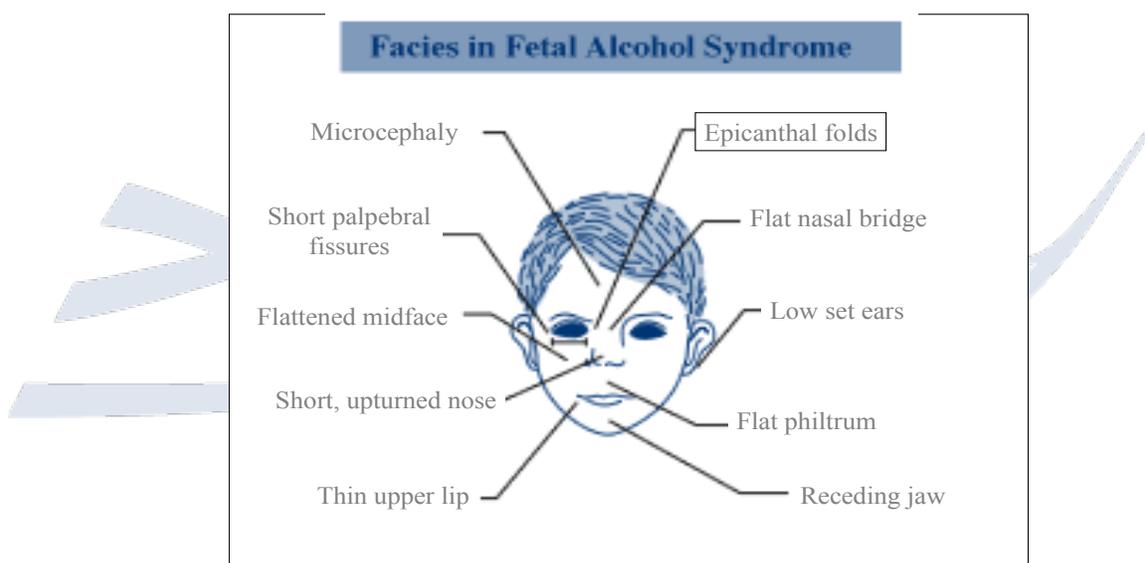
- **Growth retardation:** *Current or past weight and/or height below the 10th percentile, adjusted for age and gender.*

In the United States, the average birth weight of babies born at term (38 to 42 weeks gestation) is 7 pounds 8 ounces, with the low end of normal range down to 5 pounds 8 ounces. Babies born to mothers who use alcohol have an average birth weight of around 6 pounds and are more likely than babies born to mothers who abstained to weigh fewer than 5 pounds 8 ounces. As children with fetal alcohol syndrome grow older, they tend to continue to be small for their age—that is, both short and underweight. To meet the FAS diagnostic guidelines set for growth criteria, a child must have either reduced weight *or* height (at or below 10th percentile on standard growth charts) at birth *or* at any point in time after birth.

- **Facial dysmorphism:** *A thin upper lip, flattened philtrum, and small eyes, according to analysis of facial features utilizing the Lip-Philtrum Guide and digital facial photograph based on the criteria of Astley and Clarren.*

Facial features associated with prenatal alcohol exposure are consistent with an undergrowth of the midface that results in an overall flattening of the middle portion of the face. As a result, children with FAS exhibit,

- Epicanthal folds (extra skin folds coming down around the inner angle of the eye)
- Short palpebral fissures (small eye openings)
- A flattened elongated philtrum (no groove or crease running from the bottom of the nose to the top of the lip)
- Thin upper lip
- Small mouth with high arched palate (roof of the mouth)
- Small teeth with poor enamel coating
- Low set ears



These changes can vary in severity, but usually persist over the lifespan of the child.

Most people will not recognize any differences when they see the child, but physicians and other practitioners with experience working with children prenatally exposed to alcohol will be able to detect the changes.

A problem arises when clinicians rely too heavily on changes in facial structure to recognize the child affected by prenatal alcohol exposure. In animal studies, pregnant rats given alcohol on days 7 or 8 after conception had newborns with facial features typical of FAS.

However, giving the pregnant rats alcohol on days 1 through 6, or on or after day 9, did not affect the facial features in any way. Thus, the window of alcohol exposure that can affect children's facial features appears to be very narrow.

In addition to the facial dysmorphology, children with fetal alcohol syndrome also may have a variety of malformations of major organs, especially the heart, kidneys, eyes, and ears. Children with prenatal alcohol exposure frequently have vision problems; many have an eye that turns inward (i.e., esotropia, or a “lazy eye”). Also, the children can have a predisposition to ear infections and a high rate of partial or complete hearing loss (i.e., eighth nerve deafness), so a thorough hearing exam is recommended in the first year of life and should be repeated annually based on the child's speech and language development.

- **Central nervous system (CNS) abnormalities:** *Demonstration of structural, neurological, or functional CNS deficits^{1,2} as documented by the presence of microcephaly (current head circumference below 3rd percentile for age and gender) and/or functional deficits demonstrated as global cognitive delays with performance below the 3rd percentile on standardized testing or three or more domains of neurodevelopmental functioning more than 2SD below the normed mean on standardized measures of neurocognitive, self-regulatory, or adaptive functioning.*

Problems in the central nervous system can be manifest through structural, neurological, or functional changes. Structurally, a small head circumference (at or below 10th percentile) at birth or at any time thereafter indicates poor brain growth. For example, the average head size of term infants at birth is 35 centimeters, while the head size of a baby with FAS often is fewer than 33 centimeters. Neurological damage can manifest as seizures, problems in coordination,

difficulty with motor control, or a number of “soft” neurological deficits. Functionally, the average IQ in children with FAS is about 68, as compared to the general population’s average IQ of 100. Alcohol-exposed children, with or without the characteristic facial features or growth retardation, have consistently lower IQ scores than non-exposed children.

Importantly, even alcohol-exposed children with a “normal IQ” demonstrate difficulty with behavioral regulation, impulsivity, social deficits, and poor judgment, causing problems in day-to-day management in the classroom and home. In fact, children with FAS exhibit a wide range of functional difficulties much more commonly than global intellectual impairment; these difficulties include learning disabilities, poor school performance, diminished executive functioning (e.g., organization of tasks, understanding cause and effect, following several steps of directions), clumsiness, poor balance, and problems with writing or drawing. Behaviorally, many of the children have a short attention span, and often are described as impulsive and hyperactive.

From a brain structure perspective, prenatal alcohol exposure not only can cause the child to have a small brain overall, but also can stunt the growth of individual parts of the brain.⁹ This damaged growth may be present regardless of the child’s facial features. Problems with the formation and development of different parts of the brain can result in a wide range of behavioral and learning deficits. Many children and adolescents with prenatal alcohol exposure have trouble moving information between different brain regions; they cannot effectively use information to self-direct their behavior or to think in the abstract. They may have trouble learning new information and recording it in the brain—and then have even more difficulty retrieving the information they’ve already learned. A child may learn his multiplication tables one day but forget them the next.

Other parts of the brain also can be affected, impairing the child’s ability to coordinate planned motor movements and resulting in impulsive movement and clumsiness. Reduction in the size of the cerebellum in the back part of the brain, for example, produces difficulties with balance and arousal and may be a source of sleep problems. Again, remembering that such problems occur not only in children with the abnormal facial features associated with full expression of FAS, but also in alcohol-exposed children who “look normal” is important.

Following these criteria, individuals who meet all physical parameters for growth impairment and facial dysmorphology as well as neurodevelopmental deficits receive a diagnosis of fetal alcohol syndrome (*FAS*). Individuals with confirmed prenatal alcohol exposure, facial dysmorphology, and neurodevelopmental deficits, but with normal growth (height and weight) patterns, are diagnosed as partial FAS (*pFAS*). Individuals who have confirmed exposure and meet criteria for neurodevelopmental deficits, but do not meet criteria for facial dysmorphology, are classified as alcohol related neurodevelopmental disorder (*ARND*), and individuals with confirmed prenatal exposure and with malformations, including dysmorphic facial changes, but normal growth and normal neurodevelopment, fall into the category of alcohol related birth defects (*ARBD*).

In 2015, the American Psychiatric Association published the fifth edition of its *Diagnostic and Statistical Manual (DSM 5)*, which now includes in its nomenclature “Neurobehavioral Disorder associated with Prenatal Alcohol Exposure (*ND-PAE*).” *ND-PAE* was listed in the Conditions for Further Study and also given as an example under “Other Specified Neurodevelopmental Disorder (315.8).” The emphasis of the diagnostic approach to *ND-PAE* lies in its focus on the neurodevelopmental deficits in children with prenatal alcohol exposure. It is possible that, over time, *ND-PAE* will replace use of the term *ARND*.

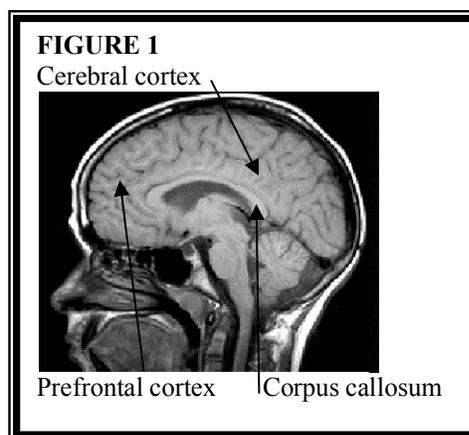
In an attempt to simplify the terminology and to develop an overarching approach to referring to all individuals affected by prenatal alcohol exposure, in April 2004, a group of federal agencies developed a consensus definition of fetal alcohol spectrum disorders (*FASD*):

...an umbrella term describing the range of effects that can occur in an individual whose mother drank during pregnancy. These effects may include physical, mental, behavioral, and/or learning disabilities with possible lifelong implications (Bertrand, 2004).

FASD is not meant to serve as a diagnostic term, but rather a unifying one to help us appreciate the many ways in which prenatal alcohol exposure can present in the affected individual. For our purposes, we will use the term *FASD* when the information applies to all alcohol-exposed children, including those with a diagnosis of *FAS*, *ARND*, *ND-PAE*, or *ARBD*. When the information refers to children with a specific diagnosis, such as *FAS*, we will use the appropriate term.

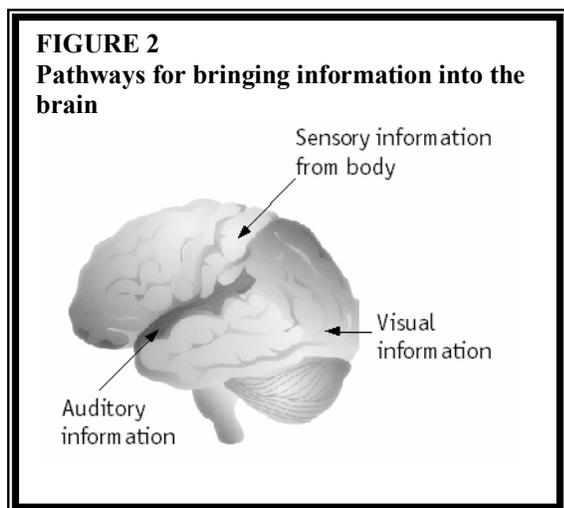
Information processing

The behavioral, emotional, and learning difficulties of children with prenatal alcohol exposure can best be understood as a deficit in the ability to process information. More specifically, children have difficulty recording information (bringing it into the brain); interpreting information; storing information in memory for later use; and using information to guide actions, behavior, emotions, language, and movement.



The normal MRI shown in Figure 1 demonstrates some key brain structures and areas affected by drug and alcohol exposure. The cerebral cortex is the outer shell of the brain; the

prefrontal cortex is a component of the overall cortex, the area that contains dopamine and serves as the regulatory center of the brain; the corpus callosum is a part of the limbic system, located in the exact midline of the brain.



Damage from drinking in the first trimester—that is, the first three months of pregnancy—mainly occurs in the midline structures of the brain, where the limbic system is located. The limbic system guides information processing: the way we bring information into the brain and use it to manage our behaviors, emotions, and thoughts. As

seen in Figure 2, data retrieved from our senses enter the brain through different pathways.

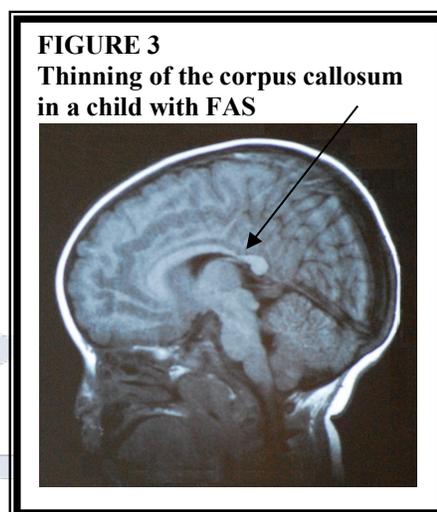
Visual information enters through the back portion of the brain, a region known as the occipital lobe. Touch, taste, and smell enter through the parietal lobe, located in the upper, posterior half of the brain. Auditory information enters through the ear, and the eighth cranial nerve carries the information from the ear to the inner midline section of the brain.

A primary job of the brain is to bring these disparate bits of sensory input together and conduct the information to the prefrontal cortex in the front of the brain. There, dopamine, a key neurotransmitter, is fired off at intervals to guide the individual in using and responding appropriately to the information via motor activity, behavior, emotion, speech and language. In other words, by regulating the amount and frequency of dopamine release, the individual is able to use information *from* the environment to manage a response *to* the environment.

Alcohol’s damage to the limbic system is what produces many of the functional difficulties we see in children, teens and adults exposed prenatally to alcohol. For example, the

hippocampus, situated in the posterior aspect of the limbic system, plays a role in consolidating new memories and applying information in novel situations. If the hippocampus is damaged, the child has difficulty transferring neurologically generated maps of information and experience to long-term memory storehouses in the temporal lobes. The child may know, cognitively, not to run out into a particular street in front of his house, but cannot retrieve that knowledge when approaching a different street. As a result, he runs out into the street, appearing to be “impulsive” or “hyperactive.”

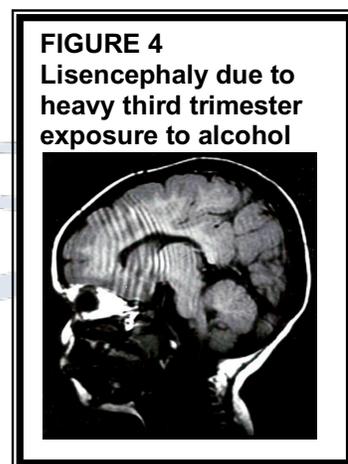
Other alcohol-induced structural changes in the brain can occur in the corpus callosum, the portion of the brain that permits the two major halves to share information. When compared to the normal MRI seen previously (Figure 1), the corpus callosum in the MRI of the child with FAS (Figure 3) is narrower at its posterior segment. This structural thinning effect disrupts



communication within the brain so that certain types of information can never reach consciousness. For example, an alcohol-exposed child may be able to recite the rules for good behavior in the school lunchroom, but be unable to regulate his behavior in accordance with those rules. As a result, he is described as disobedient or labeled with a diagnosis of oppositional defiant disorder (ODD): “He knows what he’s supposed to do,” exclaims his teacher. “He just won’t do it!” Although a child with FAS may meet diagnostic criteria for oppositional defiant disorder, diagnostic and therapeutic approaches must consider the nature of the structural brain defects that are producing the behavior before such a determination is made. We must look beyond the behavior we see to identify the root cause of that behavior.

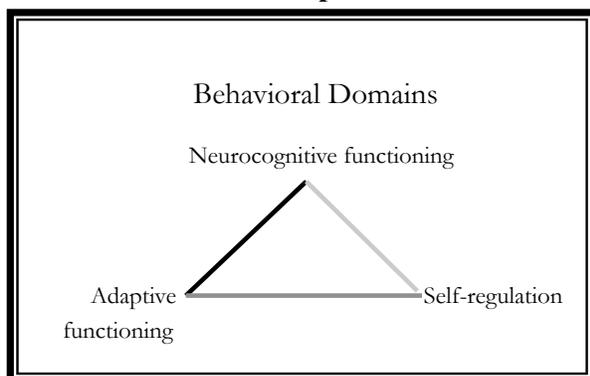
Finally, the thalamus (also part of the limbic system) receives input from all over the body and sends information to the cerebral cortex, the area of the brain responsible for cognition and learning. The thalamus also helps organize behavior related to survival: fighting, feeding, and fleeing. That is why young people with FASD often get a panicked look in their eyes when faced with a sudden change or threat, or overloaded with information. When parents describe the child as being “stubborn,” they are recognizing, perhaps, that the child diagnosed within the fetal alcohol spectrum does not learn from experience in the same way other children do. This is not willful behavior on the part of the child; rather, the connections between past instructions or experience and current behavior just don’t exist.

Alcohol exposure in the third trimester—the final three months of pregnancy—causes damage to the cerebral cortex, the outer shell of the brain. In the normal MRI shown previously (Figure 1), the cortex is folded in upon itself, forming the *gyri* and *sulci*, or valleys and ridges, in the brain. This folding occurs in the third trimester, producing increased brain surface area. In general, the more brain surface present in the cortex, the higher the level of cognitive functioning.



When a pregnant woman uses alcohol during the third trimester, however, brain cell migration is disrupted, interfering with the development of the gyri and sulci and significantly reducing brain surface area. As seen in Figure 4, also an MRI of a child with FAS, the brain is small, there are very few folds in the cortex of the brain, and the surface of the brain is quite smooth (known as lissencephaly). These changes may be among the major factors producing the intellectual disabilities seen in many children with FAS.

FASD and neurodevelopment



Based on an understanding of how prenatal alcohol exposure can cause problems related to a child's educational experience, as well as our previous discussion of information processing, we can best understand the alcohol-

induced neurodevelopmental deficits and their impact by viewing three domains of behavior: neurocognitive functioning, self-regulation, and adaptive functioning.

Neurocognitive Functioning

Neurocognitive functioning reflects the use of information at the highest level of thinking. Global intellectual deficits, as documented through a low IQ, or significant developmental delay among young children, are neurocognitive outcomes of prenatal alcohol exposure. However, several studies have shown that most individuals with FASD do not meet criteria for intellectual disability and have IQs well into the normal range. The actual neurocognitive difficulties can be much more subtle but play an important role in daily living and learning.

- **Executive functioning**

A deficit in executive functioning is one of the most frequently documented difficulties in children, teens, and adults with prenatal alcohol exposure. Essentially, executive functioning is the ability to plan and complete a task. Executive functioning difficulties translate into problems with planning, organizing, and problem-solving skills. In school, a child with prenatal alcohol exposure will make repetitive errors and demonstrate increasing deficits in overall performance on progressive planning tasks, suggesting difficulties incorporating feedback and shifting from unsuccessful strategies during problem-solving tasks. From a learning perspective, the individual with prenatal alcohol

exposure has trouble thinking about things in more than one way, so understanding and using more advanced concepts or strategies while approaching a task is more difficult.

- **Memory deficits** Difficulties with memory, including verbal and visuo-spatial memory, persist over the long term for children and youth with prenatal alcohol exposure. Of special importance, active working memory—the ability to store and process meaningful bits of information at a given moment—and the ability to manage goals in working memory interfere with the exposed individual’s ability to perform tasks that involve multiple-step instructions. Thus, the child appears to be inattentive when in reality he or she is having difficulty remembering lengthy verbal instructions.

Working memory also includes mental processes that enable temporary retention and manipulation of information about other people (“social working memory”). This particular arena of working memory has not been specifically studied in young people with FASD, but it stands to reason this may be another factor that impedes their social skills.

- **Visuospatial processing** Children and adolescents with prenatal alcohol exposure may appear disorganized at home or in the classroom, producing poorly planned constructions, having problems telling right from left, and experiencing problems using spatial cues. This disorganization can be the result of deficits in visual/spatial perception, visual memory, visual perceptual skills, visual-motor integration, visuo-motor integration, visual perception, and spatial memory.

Self-regulation Children and adolescents affected by prenatal alcohol exposure have difficulties regulating their behavior and emotions. These problems with self-regulation, often evident in early infancy, have an impact on physiologic processes and cause problems with sleeping and eating patterns as well as toilet habits.

- **Emotional regulation** Children and adolescents with FASD

frequently have difficulty regulating their reactions to even relatively minor stressful situations. They are described by their parents as having sudden outbursts and rages for no obvious reasons and moving across a wide range of emotions over a short period of time. They often are perceived as having a negative affect with internalized feelings of distress and anger. This impedes their ability to engage with others. Because these young children can be easily overwhelmed by ordinary daily experiences, they can often struggle to learn a new task and have difficulty responding to instruction or feedback.

- **Behavioral regulation** Difficulties with behavioral regulation

frequently are expressed through impulsivity, and children and adolescents with FASD often are perceived as breaking rules, cheating, fibbing, and stealing. However, this most often is due to an inability to regulate their behaviors and inhibit their impulses rather than having a “conduct disorder.” Misdiagnoses such as attention deficit disorder (ADD) or attention deficit hyperactivity disorder (ADHD) also are frequently applied to the young people affected by prenatal alcohol exposure, although such difficulties are related to problems in shifting attention and sustaining the mental effort required in order to complete tasks.

- **Sensory integration and processing** Jean Ayres, an occupational

therapist, first introduced the concept of sensory integration as the process by which the brain receives, organizes and interprets information from the environment. The information is received by sensory receptors in the eyes, nose, ears, fingers, mouth and skin. Sensations such as movement, body awareness, touch, sight, sound and the pull of gravity make up the overall sensory experience. The information that is received from the environment is then sent to the

corresponding regions of the brain where it is interpreted and organized. This process is what gives us our perception of the world and what is happening to us and around us.

But prenatal alcohol exposure's damage to the limbic system (the central substructure of the brain) interferes with the way the child interprets and uses all this information. Some children with prenatal alcohol exposure may require high levels of sensory input to understand what they are experiencing (sensory seeking), and others may have lower sensory thresholds (sensory avoidant). When the child is sensory seeking, he requires greater amounts of sensory stimulation for the input to register. Sensory-avoidant young people may exhibit distractible and irritable behaviors that interfere with academics, play, and social relationships. Because of this sensitivity, the child may be bothered by noises so quiet they do not even register for other children. The child not only may take note of background noises others filter out but may drift off task due to noises that occupy her attention, disrupting her ability to perform academically. Whether sensory-avoidant or sensory-seeking, individuals with sensory processing difficulties will appear disorganized and distractible, constantly trying to either decrease or increase their sensory input.

Adaptive Functioning

Adaptive functioning relates to the ability to accomplish tasks of daily living, such as following schedules, managing money, and understanding the concept of time. Extensive research has documented a significant gap between intellectual or cognitive capabilities and adaptive capabilities in alcohol-exposed individuals, and this factor helps to differentiate individuals with FASD from those with other diagnoses. Adaptive functioning deficits persist throughout the life span, most often becoming more and more evident as the child grows older. This requires ongoing and increasing support from parents and teachers.

- **Communication** Although the impact of alcohol exposure on global cognitive functioning (IQ) could explain the multiple deficits in all areas of language and communication seen in individuals affected by prenatal alcohol exposure, this is not always the case, and the nature of language and communication deficits can be highly variable. An individual affected by prenatal alcohol exposure may have trouble understanding figurative language, using abstract language, and keeping complex messages straight. Because of these difficulties, children or adolescents may be seen to talk excessively, have a tendency to make inappropriate comments, and have a disorganized conversational style that is difficult to follow.

- **Social skills** A lack of social skills often becomes evident as the child with FASD enters the school setting. Although children and adolescents affected by prenatal alcohol exposure want to be sociable and, in fact, may *appear* to be quite sociable, they have trouble making and keeping friends, usually have only superficial friendships, and end up with unsuitable peers as friends. When they are with people they perceive as friends, they often get lost in the conversation, unable to follow the nuances of body language, vocal inflections, and facial expression. As one young lady told me, “We’ll all be talking and everyone will laugh and I’ll say, ‘What’s so funny?’ and they’ll all say, ‘Oh, it’s an inside joke.’” This kind of impairment in social cognitive processes impede the young person’s ability to understand how and when to interact appropriately. Their tendency to be overly friendly with strangers, socially immature, have difficulty understanding and reading social consequences, and to be naive and gullible can lead to dangerous situations.

- **Daily living skills** As children grow older and mature, they are expected to become responsible for the basic tasks of daily living: e.g., dressing, bathing, toileting and feeding. By adolescence, when such daily living skills become much more

complex, the young people have trouble telling time, organizing their daily schedule, and managing money. They do not necessarily understand rules for personal safety and can be too trusting of strangers, finding themselves in unsafe situations. As the young people grow into adulthood, they frequently require ongoing adult supervision and adaptive support, making it difficult to find and keep a job.

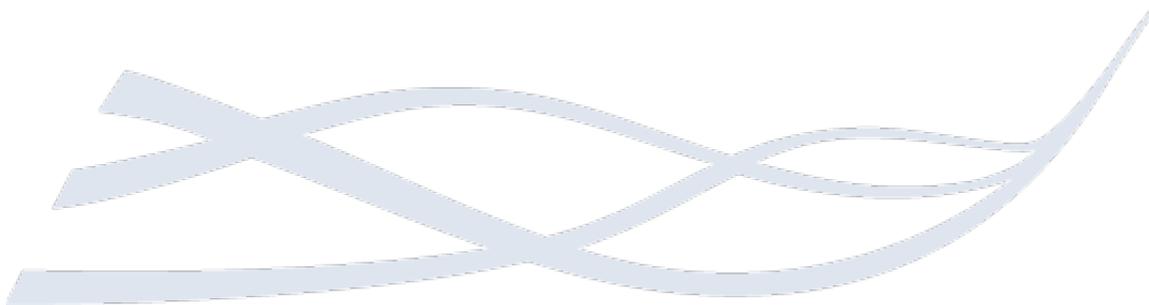
Given these multiple domains of neurodevelopmental deficits and problems, it is not surprising that difficulties with academic achievement occur significantly more frequently among children and adolescents affected by prenatal alcohol exposure. These problems exist even among those individuals whose overall level of cognitive functioning is within the normal or even higher than normal range.

Where does this leave the teacher?

The reality is that most children affected by prenatal alcohol exposure are undiagnosed or misdiagnosed. The key barrier to diagnosis is the lack of information regarding maternal alcohol use during pregnancy. Recent studies in general populations of pregnant women report that anywhere from 16% to 35% of the women have drunk some amount of alcohol during gestation, with the highest risk population frequently comprising middle class, well-educated women. However, physicians often are reluctant to address their patients' drinking, and as a result, alcohol use continues to lead to FASDs. It is not surprising, then, that many children come to school with significant difficulties that require different learning strategies and approaches, but these needs are unknown to the teacher. Unfortunately, this is a missed opportunity to change the life of a child. Streissguth and colleagues have noted that early diagnosis, especially prior to six years of age, coupled with earliest intervention is one of the strongest correlates with an improved outcome for the child long term. Delayed or incorrect diagnosis along with lack of

appropriate behavioral and educational interventions may lead to a higher incidence of secondary disabilities and greater need for special education services.

What does all this mean? It means that children with prenatal alcohol exposure are born with biologically based developmental risk factors that can be influenced, for better or worse, by the environment in which the child and adolescent is raised. There may be limits to how well the child can fare due to severity of damage induced by the alcohol exposure, but for each individual child those limits are unknown. This is where a teacher can step in and advocate for the best educational opportunities so that all children can reach their full potential.



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