Under the Influence: Fetal Alcohol Spectrum Disorders and the Biology of Trauma

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The past two decades have seen revolutionary advances in understanding the neurodevelopmental consequences of childhood trauma (Nemeroff, 2016; Teicher & Samson, 2016). As awareness has spread, trauma-informed practices have been urged on medical practitioners (Oral et al., 2016) and child welfare agencies (Hanson & Lang, 2016). Evidence-based, trauma-specific therapies have been widely promoted and adopted (Blaustein & Kinniburgh, 2010; Cohen et al., 2016; Lanktree & Briere, 2013).

Childhood maltreatment often co-occurs with parental substance abuse and prenatal alcohol exposure, which are also known to affect children's neurodevelopment (Coggins et al., 2007; Walsh et al., 2003). Understanding of the global effects of prenatal alcohol exposure lags behind that of toxic stress, and little research has explored the relationship between the two. However, there are considerable and often confusing similarities between the clinical manifestations of toxic stress and prenatal alcohol exposure, and their interactions can be significant. This paper evaluates the similarities, differences, and interactions between the two conditions and the concomitant implications for diagnosis and intervention.

Trauma and Neurodevelopment

Traumatic experience, especially when chronic or repeated, alters neurophysiology. Changes are actuated primarily through the release of stress hormones at various sensitive periods in a child's development (Shonkoff et al., 2012; Teicher & Samson, 2016). Faced with an acute threat, the brain's neuroendocrine stress response system (hypopthalamic-pituitary-adrenal axis, or HPAA) prioritizes safety by temporarily activating brain areas and physiologic systems that help the organism react to danger. When the threat passes and the environment returns to normal, these previously useful adaptations need to be reversed. Such plasticity, or resilience, is one of the hallmarks of a healthy brain.

Chronic or repeated ("toxic") stress puts a strain on this system, especially when adult caregivers do not provide soothing interactions that help regulate the child's psychophysiological state. The brain's adaptation to chronic or repeated stress can produce hypervigilance, constant hyperarousal, and hyperreactivity. The chronically aroused brain, prioritizing quick and decisive responses, devotes less attention to developing cognitive functions and controls (Marusak et al., 2015). Input from sensory systems may be muted, presumably to decrease distraction and distress (Shimada et al. 2015; Teicher & Samson, 2016). Such learned stress responses, however, often prove maladaptive in classrooms or

other normative environments. While it is tempting to think about these changes as evidence of damage, it may be more useful to recognize them as protective adaptations (Elbers et al., 2017; Teicher & Samson, 2016).

Numerous studies examine how the experience of childhood maltreatment and resultant toxic stress appear to impact the growth and structure of the growing brain (Hart & Rubia, 2012; Marusak et al., 2015; Kavanuagh et al., 2017; Paquola et al., 2016; Teicher & Samson, 2016). When alterations in brain structure arise, they consistently appear to reduce the size, connectivity, and functioning of a few prime areas (see Table 1): the prefrontal cortex, anterior cortex, hippocampus, amygdala, corpus callosum, and cerebellum. These regions control executive functions, working memory, attention, inhibition, and the processing of emotions.

Findings from clinical studies of neurocognitive functioning map closely with data gathered through brain imaging of trauma victims (Hart & Rubia, 2012; Herringa et al., 2013; Paquola et al., 2016; Teicher & Samson, 2016) (see Table 2). Identified difficulties include lowered IQ and deficits in attention, language, abstract reasoning, visual-spatial skills, and inhibition. Memory can be diminished, as can the ability to regulate emotion and attention. Robust findings suggest that childhood trauma interferes with the development of executive functions (Ford, 2009; Gabowitz et al., 2008; McCrory et al., 2010). Hyperaroused, inattentive, and impulsive children tend to miscue social and other situations, contributing to relational difficulties. Reduced connectivity between neurons has been linked with anxiety, depression, and low IQ (Teicher & Samson, 2016).

Trauma is clinically associated with difficulties with mood and behavior (see Table 2) (Ford, 2009; Gabowitz et al., 2008; Goslin et al, 2013). In young children, trauma adaptations may manifest as symptoms of regression and anxiety. As children age and cognitive and behavioral demands increase, anxiety, depression, aggression, withdrawal, dissociation, learning problems, hyperactivity, social difficulties, and somatic complaints become prominent. High incidences of ADHD, PTSD, and bipolar disorder occur in adulthood (Felitti et al., 1998; Ford, 2009; Sugaya et al., 2012).

Fetal Alcohol and Neurodevelopment

Only in recent decades have the toxic effects of prenatal alcohol exposure (PAE) been studied with scientific rigor. Fetal alcohol syndrome (FAS), a combination of distinctive phenotypical traits including growth inhibition and neurodevelopmental impairments, was first described by Jones and Smith (1973). Children with FAS exhibit characteristic facial dysmorphologies, growth deficits, and congenital anomalies involving other organ systems. Cognitive, emotional, and behavioral impairments are common (Mattson et al., 2019).

It has subsequently been recognized that individuals exposed *in utero* to alcohol, even those who do not display the distinctive facial features and poor growth seen with FAS, tend to suffer similar, persisting neurodevelopmental impairments (Murawski et al., 2015; Mattson et al., 2001). When neurodevelopmental deficits are the only manifestation of toxic exposure, which is the case in an estimated nine out of 10 children with FASD, the disorder is harder to recognize (Bakhireva et al., 2018; Green et al., 2009; Mattson et al., 2019; May et al., 2009).

Though the broad term FASD is still commonly used in the literature to refer to all disorders related to PAE, FASDs with neurodevelopmental features appear in the most recent *Diagnostic and Statistical Manual, 5th Edition*, or DSM–5 (APA, 2013) under the more specific sobriquet "Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE)." The new diagnosis focuses on the importance of alcohol-associated neurologic impairments. Diagnosis of ND-PAE requires demonstration of neurocognitive, self-regulatory, and executive function deficits in a child with known exposure to alcohol in utero. Though including children with FAS, diagnosis of ND-PAE does not depend on demonstrating any phenotypic criteria.

Ethyl alcohol is a neurotoxin, which can affect neuroreceptors and neurohormonal modulation at

Table 1. Comparison of Brain Anomalies.

	Trauma	FASD/PAE
Affected brain regions	• Reduced size or thickness of o Prefrontal cortex (PFC) o Anterior cortex (ACC) o Hippocampus o Amygdala o Corpus callosum (CC) o Cerebellum • Alterations in sensory systems: o Visual cortex o Occipital pole o Auditory cortex o Insula • Fiber tracts linking different areas of the brain show reduced integrity • Different cortical organization: Reduced centrality of left CC and temporal pole, and increased centrality of right precuneus and right anterior insula	• Reduced size or thickness of o Overall brain o Prefrontal cortex o Amygdala o Basal ganglia/Caudate nucleus o Left temporal mode of hippocampus o Corpus callosum o Cerebellum o Grey matter • Volume asymmetries in hippocampus greater than in controls • Abnormalities in corpus callosum, including thinning, displacement, and sometimes absence • Reduced myelination of sensory and motor pathways, and prefrontal cortex • Atypical activity and disorganization of network connectivity
Associated neurocognitive difficulties	• Executive functioning (PFC, ACC, cerebellum) • Memory (hippocampus, PFC) • Regulation of emotions (amygdala, PFC, ACC, cortical network organization, cerebellum) • Regulation of attention (PFC, ACC, CC) • Impulsivity (PFC) • Lack of inhibition (PFC) • Difficulty with learning, problem-solving and complex tasks (CC) • Difficulty accurately detecting emotions and social cues (amygdala, CC, ACC, visual cortex, occipital pole) • Language deficits (fiber tracks) • IQ deficits (fiber tracks, CC) • Visual memory and spatial deficits (fiber tracks, visual cortex, occipital lobe, cerebellum) • Self-awareness (anterior insula)	• Executive functioning (basal ganglia, PFC, CC, cerebellum) • Memory (basal ganglia, CC, hippocampus) • Regulation of emotions (amygdala, PFC, basal ganglia, cerebellum) • Regulation of attention (PFC, CC) • Impulsivity (PFC) • Lack of inhibition (basal ganglia, PFC) • Difficulty with learning, problem-solving, and complex tasks (CC, cerebellum) • Difficulty understanding emotions and social cues (CC) • Language deficits (CC, temporal lobe) • Lowered IQ (myelination, PFC, grey matter) • Motor difficulties (cerebellum, motor pathways, parietal lobe)

Table 2. Comparison of Symptoms and Common Comorbidities.

	Trauma	FASD/PAE
Cognition	• Language deficits • Lowered IQ and learning difficulties • Memory difficulties • Difficulties with attention • Understanding social interactions • Rigid problem-solving styles • Difficulties with abstract reasoning	• Speech and language delays • Intellectual and learning disabilities • Memory difficulties • Difficulties with attention • Theory of Mind difficulties • Difficulty with reasoning, problem solving, and understanding consequences of actions • Problems following directions leads to rule breaking
Self-regulation/ Behavior	• ADHD symptoms • Reactive to stress • Difficulty controlling impulses • Difficulty regulating emotions and behavior • Aggression associated with physical abuse • Dissociation	• ADHD in approximately 50%–90% of children • Reactive to stress • Difficulty controlling impulses • Difficulty regulating emotions and behavior • Rigidity and resistance to change • Behavioral problems and rule-breaking
Emotion	• Comorbid mood and anxiety disorders • Emotional overarousal • Guilt or shame	• Comorbid mood and anxiety disorders • May not share emotions easily • Difficulty recognizing others' emotions
Social	• Miscue social interactions • Difficulty managing social relationships	• Difficulty with social cuing • Difficulty with reciprocal social relationships • May not show affection • Lacks understanding of others' thoughts and feelings
Sensory/ Physical	• Hypersensitive to stimuli or decreased body awareness • Somatic complaints • Physical health problems: heart, respiratory, digestive, arthritis, diabetes	• Sensory sensitivities • Sensory integration difficulties • Facial dysmorphology • Growth deficits • Vision or hearing problems • Heart, kidney, or bone problems • Poor coordination/motor difficulties

every stage of the brain's development (Boschen & Klintsova, 2017, Fidalgo et al., 2017). Brain imaging conducted on individuals with ND-PAE illuminates widespread alterations to neural anatomy (see Table 1). Researchers studying both animal and human subjects conclude that fetal alcohol exposure decreases the brain's overall size, with alterations in the basal ganglia, caudate nucleus, prefrontal cortex, temporal and parietal lobes, and cerebellum (Henry et al., 2007; Mattson et al., 2019; Wilhoit et al., 2017). Functionally, widespread atypical activity and disorganization of network connectivity have been noted (Hoyme et al., 2016; Mattson et al., 2019). Reduction in grey matter and stunted myelination of sensory and motor pathways also occur (Hoyme et al., 2016). Abnormalities have been observed in the corpus callosum, including decrease in size and alterations in shape and volume (Mattson et al., 2001; Wilhoit et al., 2017). In the human hippocampus, volume asymmetries exceed those in control children (Mattson et al., 2001).

Corresponding behavioral and performance deficits are observed clinically (compare Tables 1 and 2). As might be expected from the DSM-5 criteria, deficits have been described in cognitive function (memory, intellect, reasoning, information processing), self-regulation (sensory integration, regulating behavior, inhibiting responses), and executive function (following directions, social skills, learning, attention) as well as in speech and language, vision and hearing problems, and motor function (Chasnoff et al., 2015; Hoyme et al., 2016; Mattson et al., 2019; Wilhoit et al., 2017). Approximately 70%–90% of children with FASD exhibit problems with attention, hyperactivity, and impulse control (Green et al., 2009; Greenbaum et al., 2009; Mattson et al., 2001; Wilhoit et al., 2017). It has been suggested that the damage done to the prefrontal cortex underlies ADHD seen after alcohol exposure (Louth et al., 2016).

Children with ND-PAE may miss social cues and struggle to show affection to caretakers (Wilhoit, et al., 2017), behavior which can lead to the misdiagnosis of an autistic spectrum disorder (ASD; Mukherjee et al., 2011; Stevens et al., 2013). As social learning requires both the pattern recognition skills of the right cerebral hemisphere and the analytic talent of the left, it may be hypothesized that children with inadequate development of the corpus callosum would find social learning challenging.

Because of their many challenges, children with ND-PAE are at risk for secondary pathology. Acquired psychiatric problems include depression and anxiety (Mattson, et al., 2001; Wilhoit et al., 2017). Impulsivity, difficulty following directions, and diminished understanding of consequences can lead to rule-breaking and trouble with the law (Mattson et al., 2001). Limited adaptive living skills may make achieving independence difficult.

Combined Effects of FASD and Trauma

Only a few studies try to probe the symptoms of children with PAE who have also experienced trauma and neglect, and their results must be interpreted cautiously (see Price et al., 2017, for a review). The most extensive study by Henry et al. (2007) compared 274 children aged 6–16 years old, 97% of whom had suffered severe trauma and 40% who had also been diagnosed with FASD and found that the combined group scored statistically lower on intelligence, attention, memory, receptive, and expressive language. Parents and teachers also rated the trauma/FASD group as showing more oppositional, social, impulsive, and inattentive symptoms than the trauma alone group. Coggins et al. (2007) studied 573 children aged 6–12 diagnosed with FASD and found that a high percentage had experienced abuse and neglect. They evaluated children's social communication abilities and concluded that the combination of FASD and maltreatment conspire to robustly compromise children's abilities. Koponen et al. (2009) studied 38 children in foster care with a mean age of 10 who had been exposed in utero to alcohol and found that traumatic experiences aggravated social, emotional, and neurocognitive problems. Mukherjee et al., in 2019, compared data on 99 subjects and concluded that prenatal alcohol exposure inflicted more neurodevelopmental damage than neglect. The authors cautioned against misattributing children's symptoms to neglect and poor parenting quality when the primary damage may arise instead from prenatal alcohol exposure.

The limited research conducted thus far suggests that PAE and trauma, when combined, lead to worse symptoms than would be expected in either condition alone. A child whose stress response system and executive functioning abilities are diminished possesses fewer tools with which to organize and regulate behavioral responses to stress. Resilience is compromised by both conditions.

Challenges in Applying Research Findings

Although researchers have attempted to delineate the independent and combined effects of both prenatal toxins and postnatal trauma, results of the many studies and meta-analyses must be viewed cautiously. Studies are often troubled by small sample sizes, differences in populations, lack of consistent measures, and other problems.

Controlling for confounding variables, though of crucial importance, proves especially difficult. One of the greatest challenges researchers face is separating the effects of pre-existing neurodevelopmental difficulties from those of subsequent maltreatment. Parental alcohol abuse is both a diagnostic necessity for ND-PAE and a major association with child maltreatment (Walsh et al., 2003). Yet when Kavanaugh et al. (2018) reviewed studies investigating neurocognitive impairments in maltreated youth, only three out of 24 published reports specifically mention excluding individuals with FASD, leaving open the possibility that a proportion of subjects in the studies of childhood trauma suffered from undetected ND-PAE, potentially confounding results. Conversely, traumatic postnatal experiences can be a significant confounding factor in studies purporting to demonstrate effects of early alcohol exposure.

Fortunately, animal models provide independent evidence for the neurobiological effects of both prenatal alcohol and of analogues to childhood maltreatment. Rodents exposed to alcohol in a controlled environment predictably develop altered neural architecture, endocrine dysfunction, and behavioral changes analogous to those seen in humans with ND-PAE (Mattson et al., 2019; Fish et al., 2018; Hellemans, et al., 2008). Laboratory animals, known not to have been exposed to alcohol in utero, also show alterations in response systems and brain regions affecting learning and executive functioning when subjected to early stress (Flandreau & Toth, 2018, Teicher et al., 2006), producing behaviors analogous to PTSD. Animal studies help give confidence that, despite the much greater difficulties in drawing valid conclusions from human studies, fetal alcohol exposure does target many of the same structures affected by traumatic stress, potentially impairing resilience.

Diagnostic Issues

Given their similar target organs and presentations, the effects of trauma can be difficult to separate from those associated with ND-PAE. As always, awareness is the first step, as differential diagnosis is only as good as the variety of conditions considered. Before attributing a given dysfunction to the exclusive effects of trauma, it is important to consider the role played by coexisting neurodevelopmental disorders, especially ND-PAE. In seeking to understand the effects of childhood trauma, assessors should find out as much as possible about the child's baseline level of function before the trauma and view behaviors in this context.

Both alterations in brain structure wrought by trauma and those seen after PAE affect parts of the brain that regulate stress, but underlying mechanisms differ. Alcohol affects the developing fetus as a teratogen, damaging structures and neural networks. Alcohol's effects appear to be more widespread and severe than trauma's (Henry et al., 2007; Price et al., 2017; Mukherjee et al., 2019). Trauma's alterations typically begin *after* birth, as the brain reorganizes itself to contend with environmental circumstances. The brain's innate ability to learn and change in response to new experiences and stimuli remains intact (Belsky & Pluess, 2009; Hart, 2011). In fact, some evidence suggests that children who are most susceptible to brain changes as a result of adversity may also be more amenable to the reparative effects of positive experience (Belsky & Pluess, 2009). Therapeutic interventions depend on such ongoing plasticity.

Evidence suggests that the damage inflicted by alcohol is more permanent and less responsive to treatment than are the changes wrought by early trauma

(Murawski et al., 2015; Young et al., 2016). When the brain's ability to learn and change is organically impaired, response to standard therapies is affected. Effective interventions cannot be implemented without considering the independent and combined consequences of trauma and ND-PAE.

Evidence-based recommendations for assessment of trauma's effects cast a wide net, but often not wide enough to catch the interplay with alcohol-related neurodevelopmental disorders. Time and attention are given to determining symptoms common to trauma along cognitive, relational, affective, behavioral, and somatic domains. They include examining inaccurate and maladaptive thinking, social difficulties, anxiety, depression, anger, and self-regulation (as well as their severe manifestations in suicidality, substance abuse, or psychosis), dissociation, flashbacks, traumatic triggers, and avoidance (Cohen et al., 2016; Ford, 2009; Wherry, 2014). In the few instances that published recommendations urge assessment of executive functioning and neurocognitive skills, impairments are generally viewed as sequelae of maltreatment. Gabowitz et al. (2008), in describing the neurocognitive assessment of a 10-year-old boy with a history of severe emotional and physical neglect, who presents with dissociation and impairments in executive functioning, organizing information, integrating details, inhibition, and inflexibility, conclude that

> If a trauma framework were not applied to this case, it is likely that Zachary would be labeled with a diagnosis that captured his specific behavioral manifestations (e.g., Attention Deficit Disorder, Oppositional Defiant Disorder), and treatment would be targeted to his isolated symptoms (aggression/impulsivity, difficulty concentrating, not following directions), without attention to their etiologies or functions…. Zachary's early traumatic experiences have resulted in neuropsychological deficits in his executive functioning. (p. 172)

As cogent as these comments are, it is perhaps ironic that the authors do not describe attempts to screen for prenatal alcohol exposure, which is also well known to impair executive function.

One of the difficult diagnostic questions concerns whether and to what degree observed symptoms precede or result from trauma. Trauma on its own certainly contributes to neurocognitive difficulties. At the same time, neurodevelopmental impairments such as ND-PAE are seen both to intensify the risk of maltreatment and to heighten sensitivity to stress, increasing incidence and severity of PTSD (Finzi-Dottan et al., 2006). Conversely, high IQ, executive functioning, and verbal ability tend to boost resilience, decreasing behavior problems after traumatic experiences (Goslin et al., 2013; Horn et al., 2018). Given this bidirectional interaction, it can be difficult to recognize whether a given symptom arises from trauma-related maladaptation, preexisting neurocognitive difficulties, or a combination of both. Signs of neurodevelopmental difficulties, including those associated with alcohol exposure, may be mistaken for trauma or the existence of trauma overlooked in children with significant neurocognitive impairments.

Research on, and recognition of, FASDs is still evolving, and many professionals have difficulty with diagnosis and treatment. Studies indicate that missed and misdiagnoses are common (Chasnoff et al., 2015; May et al., 2018; Woolgar & Baldock, 2015) and that families perceive lack of support from their medical providers (Coons et al., 2018; Domeij et al., 2018; Helgesson et al., 2018). This lack is all the more surprising as FASDs are common: It is estimated that from 3% to 10% of the general United States population may qualify for the diagnosis. In the child welfare population, the incidence rises to 17%, reflecting the interaction between ND-PAE and trauma (May et al., 2018; Young et al., 2016; Zarnegar et al., 2016). Among the population presenting for mental health treatment, percentages of FASDs are likely to be greater. Given both the frequent absence of distinctive physical characteristics and the difficulties obtaining a reliable history of maternal alcohol consumption, as well as the frequent co-occurrence of alcohol exposure with subsequent abuse and neglect, it is likely that even these high numbers are underestimations. The condition likely remains undetected in many children.

Diagnosis is further complicated by the fact that ND-PAE is a heterogeneous disorder. Damage and symptomatology vary widely, depending on timing, duration, and severity of exposure as well as genetic vulnerabilities. The DSM triad of neurocognitive impairment, poor self-regulation, and lack of executive function may also manifest differently through the lifespan and result in other medical and psychiatric diagnoses. In a meta-analysis of behavioral symptoms in children with FASD diagnoses, Popova et al. (2016) identified 428 additional diagnoses describing medical, mental, neurocognitive, and behavioral disorders. The most prevalent neurocognitive and behavioral conditions included impulsivity (90.7% pooled prevalence), receptive language disorder (81.8%), and expressive language disorder (76.2%).

What is not clear from Popova's meta-analysis is how many subjects in the pooled studies also experienced in utero exposure to other toxic substances, or whether the children experienced subsequent childhood adversity and to what degree. Nicotine, opiates, cocaine, and methamphetamines have been associated with decreased fetal growth and later with children's impulsivity, attention, learning, and executive functioning difficulties (Behnke et al., 2013). Concomitant use of drugs and alcohol, which occurs in many instances, complicates attribution of a specific problem to one toxin or another. Also unclear is whether subsequent exposure to trauma might have affected the range or severity of symptoms noted in the studies.

Confidently diagnosing FASDs is complicated as there are at present no laboratory tests that could objectively confirm alcohol exposure in utero, and parents fearing stigma and guilt may not provide an accurate history of alcohol intake (Murawski et al., 2015). Especially for children in the child welfare system, prenatal histories may not be known (Bakhireva et al., 2018; Murawski, et al., 2015). As a result, as Young et al. (2016) note,

> When children with ADHD and associated FASD are separated from their birth mothers and moved through the care system, they are often inaccurately identified as having insecure or disorganized attachment disorders, instead of being accurately identified as having developmental, emotional, and behavioral difficulties attributed to PAE. (p. 9)

Interpretation of symptoms can be biased by clinicians' familiarity with some disorders (notably attachment and PTSD) and not others (Coons et al., 2018; Domeij et al., 2018; Woolgar & Baldock, 2015; Young et al., 2016). The combined and cascading effects of FASD with maltreatment make it particularly hard to recognize FASD as an underlying impairment and identify it as a factor in treatment (Zarnegar et al., 2016).

Assessment involves piecing together the diagnosis through evaluation of symptoms and signs, taking a careful history of prenatal exposures, and ruling out other disorders that might cause similar symptoms.

Treatment

A healthy brain that has adapted to a stressful environment can be expected to be more resilient than one whose coping mechanisms have been compromised by prenatal toxins. The extent to which traumatized children with comorbid FASD possess the neurocognitive capacity to partake in trauma treatment remains under-researched. As neurocognitive difficulties influence how well children understand, retain, and apply interventions, trauma-informed treatments need to consider children's neurocognitive abilities and the types of interventions in which they can best engage. In their study of foster children with FASD, Koponen et al. (2009) found that children whose diagnoses of FASD had been missed exhibited more behavior problems than diagnosed children, perhaps because their symptoms were misunderstood and appropriate interventions not offered.

The current clinical emphasis on trauma has led some clinicians to recommend that, when potential comorbidities exist, trauma should be treated first (Griffin et al., 2011). However, impaired resilience associated with an FASD can mean slower progress and more challenges in therapy, and increased stress and frustration for caregivers whose expectations do not take the child's limitations into account (Koponen et al., 2009; Paintner et al., 2012). When two conditions are so closely related, it would be a mistake to treat either preferentially.

Behavioral medications prescribed in FASDs or trauma tend to target presenting symptoms, demonstrating significant, if quite variable, success in controlling ADHD, anxiety, and depression. Specific evidence for medications' clinical efficacy in the presence of ND-PAE remains rudimentary. While one literature review found that stimulants worked to decrease symptoms in 88% of studied FASD patients (Paintner et al., 2012), another systematic review found little evidence to support the use of psychotropic medications in FASDs (Mela et al., 2018). Indeed, increased behavioral disturbances have been reported after medication (Murawski et al., 2015; Young et al., 2016). Neurobiological differences seen after prenatal exposure to alcohol may make those individuals respond differently to symptom-directed medication.

As individuals with FASDs tend to break rules and can find themselves in legal trouble (Mattson et al., 2001), child welfare and legal professionals would benefit from increased knowledge of ND-PAE. When youth's underlying disabilities go unrecognized, they are expected to understand and perform better than they are able. They may be given punitive or impractical sentences and service plans that set them up for failure. Institutions and professionals serving maltreated youngsters should become informed about the types of supportive and ameliorative interventions FASD youth require.

Conclusions

Because prenatal alcohol exposure alters the same parts of the brain as trauma, its presence is often obscured and overshadowed by a history of adversity. Yet overlooking its effects on a child's presentation and symptoms would be a mistake. Alcohol damages tissues and brain structures more widely and permanently than does trauma, affecting how a child learns, grows, and reacts to stress (Mattson et al., 2019; Murawski et al., 2015; Wihoit et al., 2017). As alcohol diminishes structures in the brain that confer resilience, children with FASDs who are subsequently exposed to traumatic experiences will be less prepared to deal with them and suffer greater and longer-lasting consequences.

Since children subject to maltreatment show high degrees of comorbid FASDs (Coggins et al., 2007; Koponen et al., 2009), professionals who specialize in trauma treatment and evaluation should become aware of FASDs and routinely screen for them in children presenting with neurocognitive deficits. By recognizing and assessing for alcohol exposure, professionals will gain useful information to guide and improve clinical, legal, and child welfare services.

Much remains unknown about the combined effects of FASD and trauma, as well as the types of interventions best suited to support and treat individuals with dual exposure. Longitudinal research is needed that can track many aspects of neurodevelopment over time, beginning in the prenatal period. Studies should include controls as well as children affected singly and doubly by FASDs and trauma. Research is also needed to determine how maltreated children with FASDs respond to current interventions in clinical, legal, and child welfare arenas, and what further interventions are needed to improve their functioning.

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