Chapter 2
Fetal Alcohol Spectrum Disorders: Academic and Psychosocial Outcomes

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Definitional Issues and Prevalence

Prenatal exposure to alcohol is the leading cause of preventable birth defects, developmental disorders, and intellectual disability (American Academy of Pediatrics Committee on Substance Abuse and Committee on Children with Disabilities, 2000). However, the precise nomenclature and criteria for diagnostic categories used to define the population of affected individuals remains contested. The effects of intrauterine alcohol exposure result in a continuum of behavioral, cognitive, neurological, and physical symptoms. As a consequence of the heterogeneity of clinical presentation, the categorization of affected children has been defined differently across a variety of classification systems (see Table 2.1).

Although some variability exists in the details, there is relative consensus regarding the medical diagnosis of fetal alcohol syndrome (FAS), which relies on a triad of symptoms: (1) evidence of two or more characteristic facial features, such as short palpebral fissures, smooth philtrum, and a thin vermillion border of upper lip; (2) evidence of prenatal or postnatal growth deficiency with a height or weight of below the 10th percentile at any point of the child's life (corrected for racial norms, if possible); and (3) evidence of deficient brain growth or abnormal morphogenesis (Astley, 2013; Hoyme et al., 2005; Jones et al., 2006). See Fig. 2.1. The third criterion can be satisfied by the presence of structural brain abnormalities or microcephaly (head circumference $\leq$10th percentile). Receiving a diagnosis of FAS is often recognized as a qualifying disorder to provide access to referrals and services (Bertrand et al., 2004). See Fig. 2.2.

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Table 2.1 Summary and comparison of the various diagnostic schemas for prenatal alcohol related disorders

	4-Digit Code	Revised Institute of Medicine	Canadian	National Task Force on FAS/FAE
FAS				
Facial characteristics	Simultaneous presentation of short palpebral fissures (≤2 SDs), thin vermillion border, smooth philtrum[a]	Two of the following: short palpebral fissures (≤10th percentile), thin vermillion border, smooth philtrum	Simultaneous presentation of short palpebral fissures (≤2 SDs), thin vermillion border, smooth philtrum	Simultaneous presentation of short palpebral fissures (≤10th percentile), thin vermillion border, smooth philtrum
Growth retardation	Height or weight ≤10th percentile	Height or weight ≤10th percentile	Height or weight or disproportionately low weight-to-height ratio (≤10th percentile)	Height or weight ≤10th percentile
Central nervous system (CNS) involvement	Head circumference (occipital-frontal circumference [OFC]) ≥2 SDs below norm or significant abnormalities in brain structure or evidence of hard neurological findings or significant impairment in three or more domains of brain function (≥2 SDs below the mean) as assessed by validated and standardized tools	Head circumference (OFC) ≤10th percentile or structural brain abnormality	Evidence of three or more impairments in the following CNS domains: hard and soft neurologic signs; brain structure; cognition; communication; academic achievement; memory; executive functioning and abstract reasoning; attention deficit/hyperactivity; adaptive behavior, social skills, social communication	Head circumference (OFC) ≤10th percentile or structural brain abnormality or neurological problems or other soft neurological signs outside normal limits or functional impairment as evidenced by global cognitive or intellectual deficits, below the 3rd percentile (2 SDs) below the mean or functional deficits below the 16th percentile (1 SD) below the mean in at least three domains: cognitive or developmental markers, executive functioning, motor, social skills, attention/hyperactivity, and other (i.e., sensory, memory, language)

Alcohol exposure	Confirmed or not confirmed	Confirmed or not confirmed	Confirmed or not confirmed	Confirmed or not confirmed
Partial FAS				Not proposed
Facial characteristics	Short palpebral fissures (≤2 SDs) and either a smooth philtrum or thin vermillion border, with the other being normal OR palpebral fissure (≤1 SD) and both a smooth philtrum and thin vermillion	Two or more of the following: short palpebral fissures (≤10th percentile), thin vermillion border, smooth philtrum	Two or more of the following: short palpebral fissures, thin vermillion border, smooth philtrum	Not applicable
Growth retardation	Not required	Either height or weight ≤10th percentile OR (see CNS involvement)	Not required	Not applicable
Central nervous system (CNS) involvement	Same as for FAS	Head circumference ≤10th percentile or structural brain abnormality or behavioral and cognitive abnormalities inconsistent with developmental level	Same as for FAS	Not applicable

(continued)

Table 2.1 (continued)

	4-Digit Code	Revised Institute of Medicine	Canadian	National Task Force on FAS/FAE
Alcohol exposure	Confirmed	Confirmed or not confirmed	Confirmed	Insufficient data to provide guidance for this diagnosis. Formed group to discuss
Alcohol-related neurodevelopmental disorder	Does not propose this diagnostic category, but rather has several categories assessing functional deficits			Not applicable
Central nervous system involvement	Same as for FAS	Either (1) structural brain anomaly or OFC ≤10th percentile or (2) evidence of a complex pattern of behavioral or cognitive abnormalities inconsistent with developmental level that cannot be explained by genetics, family background, or environment alone	Same as for FAS	Not applicable

Alcohol exposure	Confirmed	Confirmed	Confirmed	Not applicable
Notes	The 4-Digit Code provides an assessment of effects in four areas (growth, face, CNS, and alcohol exposure) that results in 256 different codes and 22 diagnostic categories A specific pattern or level of alcohol exposure is not required, just that alcohol exposure is confirmed or not	Alcohol exposure is defined as a pattern of excessive intake or heavy episodic drinking	Alcohol exposure is defined as a pattern of excessive intake or heavy episodic drinking A domain is considered "impaired" when on a standardized measure: Scores are ≥2 SDs below the mean, or there is a discrepancy of at least 1 SD between subdomains or there is a discrepancy of at least 1.5–2 SD among subtests on a measure	Alcohol exposure levels are not defined, but the authors cite evidence of alcohol exposure based upon clinical observation; self-report; reports of heavy alcohol use during pregnancy by a reliable informant; medical records documenting positive blood alcohol levels, or alcohol treatment; or other social, legal, or medical problems related to drinking during pregnancy

Table from Warren KR, Hewitt BG, Thomas JD (2011) Fetal alcohol spectrum disorders: Research challenges and opportunities. Alcohol Research and Health 34:4–14

All of the diagnostic schemes assume that genetic or medical causes have been ruled out and that appropriate norms are used when available

[a]For palpebral fissure norms, the 4-Digit Code uses Hall et al. 1989, Hoyme utilizes Thomas et al. 1987, and Chudley provides both the Thomas and Hall charts; the National Task Force guidelines do not mention which chart to use. Hall recently wrote that her charts underrepresented normal palpebral fissure length (Hall 2010) and should be replaced by those from Clarren et al. (2010)

[b]All of the diagnostic schemes use the University of Washington Lip-Philtrum Guide (http://depts.washington.edu/fasdpn/htmls/lip-philtrum-guides.htm), Astley (2004)

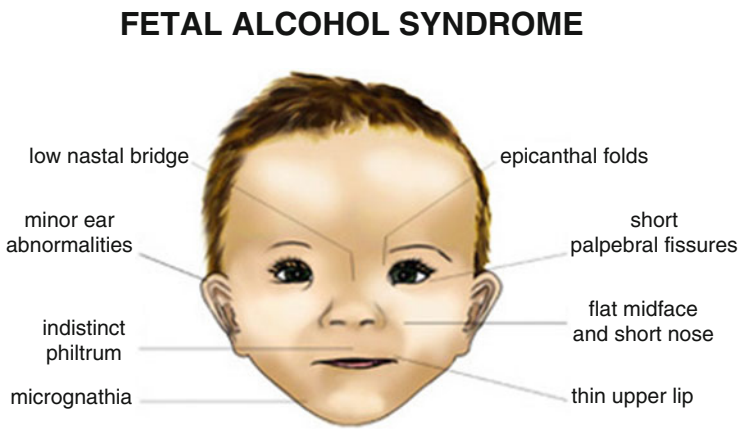

Fig. 2.1 Facial characteristics associated with fetal alcohol exposure

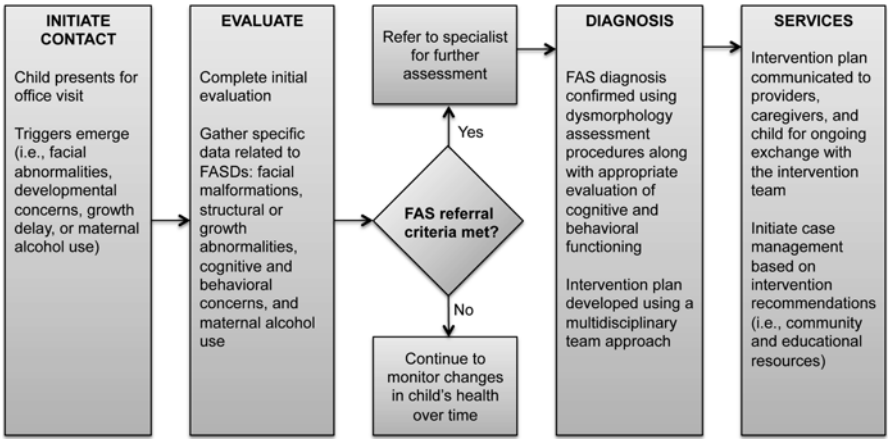

Fig. 2.2 Framework for FAS Diagnosis and Services. Adapted from Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis. National Center on Birth Defects and Developmental Disabilities, Center for Disease Control and Prevention, Department of Health and Human Services in coordination with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect. 2004. http://www.cdc.gov/ncbddd/fasd/documents/FAS_guidelines_accessible.pdf

The majority of children affected by alcohol do not meet all of the physical criteria required for an FAS diagnosis (May et al., 2014). For example, children who present with facial dysmorphology, but do not have growth deficiency or structural brain abnormalities may only meet criteria for partial FAS (pFAS). Most importantly, the majority of children affected by prenatal alcohol exposure do not demonstrate clear facial dysmorphology, which can greatly hinder identification of alcohol-affected individuals. Fetal alcohol spectrum disorders (FASDs) encompass the continuum of effects that result from prenatal alcohol exposure, including FAS (see Table 2.1, Fig. 2.3).

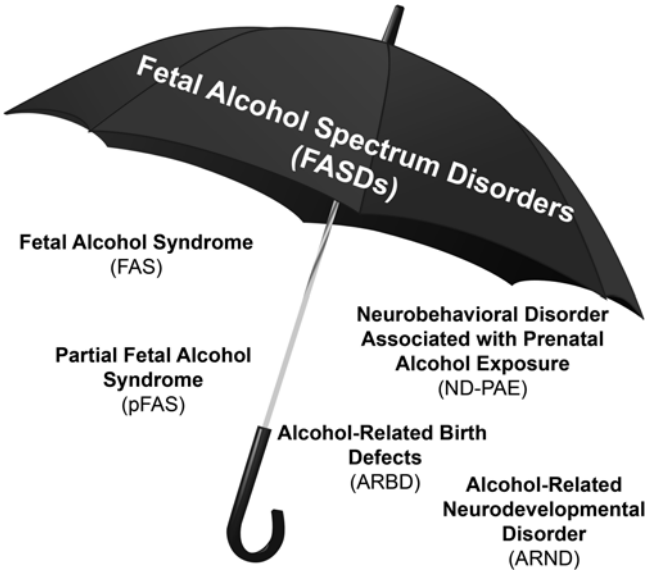

Fig. 2.3 Fetal Alcohol Spectrum Disorders encompass the continuum of potential effects of prenatal alcohol exposure

Recently, the effects of prenatal alcohol exposure have been incorporated into the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) as a condition requiring further study, referred to as Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE) (American Psychiatric Association, 2013). A similar term, Neurodevelopmental Disorder Associated with Prenatal Alcohol Exposure, is listed as a billable diagnosis as a prototypical example under Other Specified Neurodevelopmental Disorder (American Psychiatric Association, 2013). This difference in terminology (neurobehavioral vs. neurodevelopmental) is likely to cause confusion, especially since the latter diagnosis can be given without attending to the proposed criteria for ND-PAE. The proposed criteria for ND-PAE are listed in Fig. 2.4 and require indication that the individual was exposed to alcohol at some point during gestation (including prior to pregnancy recognition) and that the exposure was more than "minimal." The precise dosage is not specific and relies on clinical judgment, although minimal exposure is defined as 1–13 drinks per month (and never more than two drinks per occasion) prior to pregnancy recognition and/or following pregnancy recognition (American Psychiatric Association, 2013). In addition to exceeding a minimal level of prenatal alcohol exposure, the patient must also have impaired neurocognition, self-regulation, and adaptive functioning. As the location of the disorder in the DSM-5 suggests, there is ongoing research to determine the feasibility, sensitivity, and specificity of the criteria to identify those affected by prenatal alcohol exposure.

Guidelines for alcohol-related diagnoses along the spectrum are developed to ensure valid and reliable identification of those affected by alcohol exposure (Farag, 2014). The greatest consequence of the ongoing debate over the diagnostic criteria

A. More than minimal exposure to alcohol during gestation, including prior to pregnancy recognition. Confirmation of gestational exposure to alcohol may be obtained from maternal self-report of alcohol use during pregnancy, medical or other records, or clinical observation.
B. Impaired neurocognitive functioning as manifested by one or more of the following:
1. Impairment in global intellectual performance (i.e., IQ of 70 or below, or a standard score of 70 or below on a comprehensive developmental assessment).
2. Impairment in executive functioning (e.g., poor planning and organization; inflexibility; difficulty with behavioral inhibition).
3. Impairment in learning (e.g., lower academic achievement than expected for intellectual level; specific learning disability).
4. Memory impairment (e.g., problems remembering information learned recently; repeatedly making the same mistakes; difficulty remembering lengthy verbal instructions).
5. Impairment in visual-spatial reasoning (e.g., disorganized or poorly planned drawings or constructions; problem differentiating left from right).

C. Impaired self-regulation as manifested by one or more of the following:
1. Impairment in mood or behavioral regulation (e.g., mood lability; negative affect or irritability; frequent behavioral outbursts).
2. Attention deficit (e.g., difficulty shifting attention; difficulty sustaining mental effort).
3. Impairment in impulse control (e.g., difficulty waiting turn; difficulty complying with rules).

D. Impairments in adaptive functioning manifested by two or more of the following, one of which must be (1) or (2):
1. Communication deficit (e.g., delayed acquisition of language; difficulty understanding spoken language).
2. Impairment in social communication and interaction (e.g., overly friendly with strangers; difficulty reading social cues; difficulty understanding social consequences).
3. Impairment in daily living skills (e.g., delayed toileting, feeding, or bathing; difficulty managing daily schedule).
4. Impairment in motor skills (e.g., poor fine motor skills development; delayed attainment of gross motor milestones or ongoing deficits in gross motor function; deficits in coordination and balance).

E. The onset of the disorder (symptoms in Criteria B, C, and D) occurs in childhood.
F. The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.
G. The disorder is not better explained by the direct physiological effects associated with postnatal use of a substance (e.g., a medication, alcohol or other drugs), a general medical condition (e.g., traumatic brain injury, delirium, dementia), another known teratogen (e.g., fetal hydantoin syndrome), a genetic condition (e.g., Williams syndrome, Down syndrome, Cornelia de Lange syndrome), or environmental neglect.

Fig. 2.4 Criteria for Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE) listed as a condition for further study in the Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition (DSM-5). Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (Copyright ©2013). American Psychiatric Association. All Rights Reserved.

is the risk that affected children and adults may be under-identified or misdiagnosed and therefore underserved. The current diagnostic schemas suffer from various shortcomings, including lack of consensus, lack of validation studies, difficulties in balancing sensitivity and specificity given the variation in clinical presentation, and hardships in educating physicians, dissemination, and implementation (Astley, 2014; Larcher & Brierley, 2014; Salmon & Clarren, 2011).

New objective screening tools, including neonatal testing and the development of potential biomarkers, can assist in the identification of alcohol-exposed children at birth (Koren et al., 2014; Zelner et al., 2010, 2012). Screening techniques are

inherently plagued with potential ethical and moral issues. These include the risk of disproportionately targeting specific groups, inaccurate screening, and the concern of stigma and judgment associated with maternal drinking during pregnancy. The controversy regarding the demarcation of viable life and determination of personhood has also led to issues concerning the criminalization of drinking while pregnant (Drabble, Thomas, O'Connor, & Roberts, 2014; Yan, Bell, & Racine, 2014). A recent review of state responses to alcohol use and pregnancy found that there is great variability in the characteristics of policies, ranging from primarily supportive to primarily punitive (Drabble et al., 2014). One issue that arises in testing for alcohol exposure at birth is that even if it is possible to accurately determine prenatal alcohol exposure with adequate sensitivity and specificity, it is not certain that an individual will be negatively affected. Both a false positive and a false negative diagnosis may have detrimental effects throughout a child's development.

While it is difficult to accurately assess drinking, when compared to antenatal or prospective reports, retrospective reports identify 10.8 times as many women as risk drinkers (Hannigan et al., 2010). While retrospective reports were originally considered to be less accurate, changes in motivation and other factors lead them to be a more effective indicator of prenatal exposure that is validated in the prediction of additional behavioral problems compared to antenatal reports (Hannigan et al., 2010). However, despite the validity of retrospective reports, relying on maternal reports alone is not an effective way to identify rates of individuals with prenatal alcohol exposure. A study comparing the prevalence rates of prenatal alcohol exposure based on maternal self-reporting versus objective meconium screening (the earliest stool of the infant) found that the meconium testing yielded over four times as many cases as would have been identified by self-reporting alone (Lange, Shield, Koren, Rehm, & Popova, 2014). Therefore, additional methods of assessing prenatal alcohol exposure may be important to avoid missing affected children.

A pilot study found that ultrasound parameters may allow for early detection of alcohol-mediated negative outcomes resulting from central nervous system dysfunction, which may be more predictive of later dysfunction compared to other screening measures that do not distinguish alcohol-exposed from alcohol-affected outcomes (Kfir et al., 2009). This study found that there were significant differences between alcohol-exposed fetuses and controls in somatic and brain measurements in utero. If these prenatal measurements correlate with subsequent neurodevelopmental outcomes, they may serve as early biomarkers of alcohol exposure as well as early detectors of alcohol-related deficits.

In an effort to increase the identification of children affected by prenatal exposure history, researchers have focused on defining the neurobehavioral patterns associated with FASDs. A variety of screening checklists have been created to facilitate diagnosing FASDs, though with only moderate success (Burd, Klug, Li, Kerbeshian, & Martsolf, 2010; Fitzpatrick et al., 2013). Parent report measures have also been used to help differentiate alcohol-affected individuals from other clinical groups. For example, the Neurobehavioral Screening Test (NST) consists of ten items from a commonly used behavioral scale (the Child Behavior Checklist) (Nash et al., 2006). The NST was tested in a small group of alcohol-exposed children, children with attention-deficit/hyperactivity disorder (ADHD), and controls, with

86 % sensitivity and 82 % specificity for detecting alcohol-exposed children. Certain items from the NST were also able to differentiate children with FASDs from children with oppositional defiant disorder (ODD) and conduct disorder (CD) (Nash, Koren, & Rovet, 2011).

In order to move beyond subjective checklists and parent reporting measures, researchers have attempted to define a sensitive and specific neurobehavioral profile of alcohol-exposed children using objective neuropsychological measures (Jacobson, 1998; Kodituwakku et al., 2006; Mattson et al., 2013; Mattson & Riley, 2011). While classification accuracies have reached adequate levels when comparing alcohol-exposed children to typically developing controls, the classification rates are lower when comparing exposed children to non-exposed children with other diagnoses, such as ADHD (Mattson et al., 2013). Currently, in cases where alcohol exposure is suspected but dysmorphology is not present, the neurobehavioral effects may become the primary tool for identification, acting as a potential diagnostic phenotype.

Individuals with dysmorphology may be identified regardless of the neurobehavioral profile. Therefore, it is important to create a profile that is able to accurately categorize non-dysmorphic children affected by prenatal alcohol exposure. This has been codified in the DSM-5 and is the basis for ND-PAE. A large multi-site study using a standardized neuropsychological battery compared controls, non-exposed children with ADHD, and children who had heavy exposure to alcohol but were not dysmorphic (Mattson et al., 2013). The classification rates were modest when comparing the clinical groups. However, these studies have considerable clinical relevance, as the differential diagnosis between non-dysmorphic alcohol-exposed children and other clinical groups are common within clinical settings.

The combination of a dysmorphology examination and a neurobehavioral assessment may facilitate classification of children where exposure history is unknown. The classification rates for the current brief screeners and the neurobehavioral profile indicate that a more accurate screening tool is needed to help with identification and access to services (Koren et al., 2014; LaFrance et al., 2014). A tiered system of screening tools (dysmorphology, parent report measures, and direct child measures) can assist in identifying children at high risk (Goh et al., 2015). Children who are screened as positive could be referred for a slightly longer testing battery, similar to the FAS referral process (see Fig. 2.2). This type of system would balance the need for feasibility, sensitivity, and specificity. The collaboration of primary care providers, screening tools, and the creation of an effective neurobehavioral profile is imperative, as all are critical steps for getting a child or adult affected by alcohol exposure connected to a system of services.

Prevalence

Although prevalence estimates vary dramatically depending on the sample and diagnostic schema utilized, there is a consensus that heavy prenatal alcohol

exposure is a major public health concern and large economic burden (Popova et al., 2013; Stade et al., 2009). Recent estimates using active case ascertainment among first grade children in an American middle class community found the prevalence of FAS was between 6 and 9 children per 1000 (May et al., 2014). The prevalence of FASDs (including FAS) was estimated between 24 and 48 children per 1000. Estimates in other countries and in specific ethnic populations are substantially higher (May et al., 2007a, 2007b, 2009). Further, prevalence rates are extremely high within the foster care system (Burd, Cohen, Shah, & Norris, 2011; Chasnoff, Wells, & King, 2015). Approximately 70 % of children diagnosed with FASDs are currently in or have previously been involved in the foster care system (Burd et al., 2011). As many as 80 % of children within foster or adoptive care who are affected by prenatal alcohol exposure are misdiagnosed or missed completely (Chasnoff et al., 2015).

Impact on the Developing Child

Due to the diffuse teratogenicity of alcohol on the brain, the impact of alcohol exposure on the developing child can occur across physical, cognitive, behavioral, and psychosocial domains (Mattson, Crocker, & Nguyen, 2011). The most severe outcomes of alcohol use during pregnancy are those that result in fetal or newborn death, including miscarriage, stillbirth, and sudden infant death syndrome (Alm et al., 1999; Iyasu et al., 2002). In the surviving child, the possible effects are highly variable, ranging from facial dysmorphology or intellectual disability, to mild behavioral or cognitive deficits. The effects of prenatal alcohol exposure may vary based on timing and amount of exposure in addition to other confounding factors such as individual genetic vulnerability. Based on these facts, the National Institute of Alcohol Abuse and Alcoholism (NIAAA) and the Surgeon General recommend complete abstinence from alcohol during pregnancy (NIAAA, 2013; US Surgeon General, 2005).

Intellectual Functioning

Prenatal exposure to alcohol leads to global cognitive impairment (Mattson et al., 2011; Mattson & Riley, 1998). The heterogeneity of clinical presentation is clear in the range of intelligence scores seen in children on the fetal alcohol spectrum disorder continuum. Full scale intelligence scores are standardized on a scale with a mean of 100 and standard deviation of 15. Scores for children with prenatal alcohol exposure vary widely—from below 50 to over 110, spanning the range from intellectually deficient to above average. Children with FAS have an average IQ of approximately 70, two standard deviations below the mean. However, intellectual

deficits can be present with and without dysmorphology (Mattson, Riley, Gramling, Delis, & Jones, 1998) and are apparent across both verbal and nonverbal domains (Mattson & Riley, 1998). The median IQ of over 400 individuals with FASDs (ranging from 6 to 51 years old) was 86 (Streissguth et al., 2004). This finding has been replicated in 8–16 year olds in a recent multisite study (Glass et al., 2013), suggesting the average individual with heavy prenatal alcohol exposure has an intelligence score approximately 1 standard deviation lower than the average non-exposed individual. This suggests that in general, having FAS is related to more severe cognitive deficits overall, however there is a great deal of variability in functioning across the fetal alcohol spectrum.

Importantly, most children with FASDs will not meet the criteria for intellectual disability despite having significant neurobehavioral deficits. Studies found that 24 % of children with FAS and 7–16 % of children with fetal alcohol effects[1] met the basic criteria of having an IQ of below 70 (Streissguth et al., 2004; Streissguth, Barr, Kogan, & Bookstein, 1996). A prospective longitudinal study found that low intellectual functioning coupled with low adaptive functioning leads to a much higher rate of secondary disabilities and adverse life outcomes for people with FASDs (Streissguth, 2007). This may be especially true for individuals who have diminished intellectual capacity but do not meet the diagnosis for intellectual disability and therefore may not qualify for services or interventions.

Even relatively low maternal consumption of alcohol during pregnancy can lead to reductions in global intelligence—consumption of two drinks per day on average was related to a seven point decrement in IQ after accounting for other covariates (Streissguth, Barr, & Sampson, 1990). Mothers with this rate of alcohol consumption during pregnancy are over three times more likely to have a child with subnormal (<85) IQ scores (Streissguth et al., 1990). However, not all studies have found that light alcohol exposure leads to impaired global intellectual functioning (Kelly et al., 2013; Skogerbo et al., 2013). Yet, these results should be reviewed with caution as the methodologies used have been questioned (Powell, 2012) and the results are inconsistent with the majority of the literature, which emphasizes that low-moderate prenatal alcohol exposure should not be considered safe (Jacobson & Jacobson, 2014; O'Leary, Taylor, Zubrick, Kurinczuk, & Bower, 2013).

There are discrepancies on both ends of the spectrum in terms of the relationship between amount of alcohol exposure and outcomes. A recent meta-analysis found a significant, albeit small, association between mild-moderate exposure and behavior and cognition (Flak et al., 2014). On the other hand, children who have histories of heavy prenatal alcohol exposure, even those with facial dysmorphology, may have average IQ scores despite struggling in other domains. This heterogeneity supports the importance of a comprehensive assessment of functioning.

[1] Fetal alcohol effects is an outdated term that refers to individuals with prenatal alcohol exposure who have some, but not sufficient features to warrant a diagnosis of FAS.

Academic Functioning

The combination of cognitive and behavioral effects of prenatal alcohol exposure often leads to poor performance in academic achievement (Burd, Cotsonas-Hassler, Martsolf, & Kerbeshian, 2003; Burd, Klug, Martsolf, & Kerbeshian, 2003; Church & Kaltenbach, 1997; Streissguth et al., 1990, 2004). As many as 60 % of clinically referred adolescents over the age of 12 were found to have disrupted school experiences (e.g., suspended, expelled, dropping out from school), and 65 % received some remedial help with reading and math (Streissguth et al., 1996). Impairment in school functioning persists throughout development and occurs in all three major academic domains (math, reading, and writing) to varying degrees (Alati et al., 2013; Goldschmidt, Richardson, Stoffer, Geva, & Day, 1996; Howell, Lynch, Platzman, Smith, & Coles, 2006).

The precise association between amount of alcohol exposure, timing of alcohol exposure, and specific effects on academic functioning has not been fully elucidated. A longitudinal prospective study found a linear dose-response relationship between prenatal alcohol exposure and math (Goldschmidt et al., 1996). This finding remained significant even when controlling for IQ. Interestingly, the relation between alcohol dose, timing of alcohol exposure, and verbal academic domains (reading and spelling) was modeled more accurately using a threshold model of one drink/day during the second trimester.

For children with prenatal exposure to alcohol, deficits in academic achievement exceed what would be expected by intellectual functioning (Kerns, Don, Mateer, & Streissguth, 1997). This exacerbation could be due to the combination of behavioral and cognitive impairments. There are complex interactions between cognitive ability, behavioral functioning, socio-emotional functioning, attention deficits, concomitant psychopathology, environmental supports, school environment, and access to services, all of which can influence academic outcomes (Kable, Taddeo, Strickland, & Coles, 2015).

Mathematics has been the most extensively studied academic area within the realm of prenatal alcohol exposure, as it is thought to be particularly vulnerable to the teratogenic effects of alcohol exposure (Goldschmidt et al., 1996; Howell et al., 2006; Jacobson, Dodge, Burden, Klorman, & Jacobson, 2011; Streissguth et al., 1994a; Streissguth, Barr, Sampson, & Bookstein, 1994b). Children with prenatal exposure to alcohol demonstrate impairments on higher order mathematical reasoning as well as lower order functions such as basic numerical processing, proximity judgment, and cognitive estimation (Coles, Kable, & Taddeo, 2009; Jacobson et al., 2011; Kopera-Frye, Dehaene, & Streissguth, 1996; Meintjes et al., 2010). For example, a longitudinal study compared children with prenatal alcohol exposure (with and without dysmorphology) to other populations that are also at risk for academic and school functioning issues, such as adolescents with low socioeconomic status and students in special education placements (Howell et al., 2006). This study found that children with alcohol exposure and dysmorphology demonstrated lower IQ than all other groups. The non-exposed special education group performed worse

on basic reading and spelling, however alcohol-exposed children with dysmorphology performed lowest on mathematics. It appears this effect was the result of difficulties with mathematical reasoning, as there was no difference between groups on a simpler numerical operations task. This supports the suggestion that alcohol-exposed children have greater difficultly on tasks with higher cognitive demands (Kodituwakku et al., 2006).

Recent investigations have begun to identify the underlying cognitive mechanisms of mathematical functioning and the neural correlates of mathematical deficits. One study found that immediate memory and attention contribute to mathematical functioning (Rasmussen & Bisanz, 2009), while another study found that spatial processing had a significant effect on mathematics achievement (Crocker, Riley, & Mattson, 2015). Weak visual-spatial skills have also been related to poor performance on math tests (Kable, Coles, & Taddeo, 2007). In addition to deficits in underlying cognitive mechanisms, structural and functional abnormalities in the brain have been associated with impaired mathematics achievement in alcohol-exposed children (Lebel, Rasmussen, Wyper, Andrew, & Beaulieu, 2010; Santhanam, Li, Hu, Lynch, & Coles, 2009).

While math remains the most studied academic domain in children who have histories of prenatal alcohol exposure, these children are also at risk for impairment in other clinically significant domains. Unlike math, reading abilities have not been the focus of much research, possibly because alcohol-exposed children are not as severely impaired in this domain. As reading is incredibly important for daily functioning, even a lesser impairment may still result in greater functional deficits. Though less work has been done in this area, studies consistently report that children with prenatal alcohol exposure demonstrate deficits in reading and spelling (Adnams et al., 2007; Glass, Graham, Akshoomoff, & Mattson, 2015; Howell et al., 2006; Kodituwakku, 2007; O'Leary et al., 2013; Streissguth, Barr, Carmichael Olson, et al., 1994a). School-age children with FASDs score approximately 1.0–1.5 standard deviations below the mean of typically developing children on basic reading and spelling tasks (Howell et al., 2006). Alcohol-exposed children also demonstrate impairments in the underlying components of literacy skills, including phonological processing and rapid automatized naming (Adnams et al., 2007; Glass et al., 2015; Streissguth, Barr, Carmichael Olson, et al., 1994a). A recent study found that in addition to the expected contributions of phonological processing for spelling, working memory may also be an important consideration in developing interventions for this population (Glass et al., 2015).

Additionally, legible penmanship is a necessary component of academic success, both within the classroom and for standardized testing required in schools. Children with histories of heavy prenatal alcohol exposure have a variety of impairments that might lead to poor handwriting, including motor and visuospatial deficits (Mattson et al., 2011). While there have been anecdotal reports of poor handwriting, only recently has it been addressed in a more systematic manner. In one study, mean handwriting scores for children with FASDs were below average compared to the norms provided by the measure (Duval-White, Jirikowic, Rios, Deitz, & Olson, 2013). However, this study was conducted using a fairly small number of children and no control group.

Neuropsychological Functioning

For children with histories of prenatal alcohol exposure, difficulties with academics may be in part the result of deficits in numerous neuropsychological domains. One of the most prominent areas affected by prenatal alcohol exposure is in the domain of executive functioning. Executive functioning is highly clinically relevant, as it predicts adaptive behavior, social behavior, and theory of mind (Rasmussen, Wyper, & Talwar, 2009; Schonfeld, Paley, Frankel, & O'Connor, 2006; Ware et al., 2012). Children with FASDs struggle with higher order processes in planning, attention, organization, and cognitive flexibility (Green et al., 2009; Rasmussen, Soleimani, & Pei, 2011; Vaurio, Riley, & Mattson, 2008). Alcohol-exposed children demonstrate significant deficits in inhibitory control (Nguyen et al., 2014), which may be related to aberrant neural processes (Fryer et al., 2007b). In addition, alcohol-exposed children often demonstrate deficits in attention, a finding which has been confirmed by both parent reporting and objective measures (Glass et al., 2014). Impaired executive functioning may be substantively related to the high frequency of ADHD in this population (Fryer, McGee, Matt, & Mattson, 2007a). There are some quantitative differences between alcohol-exposed children with and without ADHD, however these distinctions are more apparent in the behavioral domain than in the cognitive domain (Glass et al., 2013; Ware et al., 2014). Deficits have also been reported in working memory, the ability to maintain and manipulate information, which can have a negative impact on both academic and social success (Burden, Jacobson, Sokol, & Jacobson, 2005; Rasmussen et al., 2011; Schonfeld et al., 2006). Poor processing speed and sluggish cognitive tempo (Graham et al., 2013) may present in the classroom as difficulty with interpreting and synthesizing information, inconsistent production of work, difficulty following directions, struggling with classroom routines, and problems keeping up with the class.

In addition to these executive function and processing speed deficits, alcohol-exposed children also demonstrate a delay in verbal skills, both in terms of language and memory. They have delayed language acquisition (Church & Kaltenbach, 1997; O'Leary, Zubrick, Taylor, Dixon, & Bower, 2009) and poor verbal fluency (Schonfeld, Mattson, Lang, Delis, & Riley, 2001). As they get older, these children often demonstrate deficits in both receptive and expressive language (McGee, Bjorkquist, Riley, & Mattson, 2009b). Research on verbal memory in FASDs has found that alcohol-exposed children struggle with encoding information more than retaining information (Kaemingk, Mulvaney, & Halverson, 2003; Mattson, Riley, Delis, Stern, & Jones, 1996; Mattson & Roebuck, 2002), which is different from the pattern seen in children with ADHD (Crocker, Vaurio, Riley, & Mattson, 2011). Alcohol-exposed children, despite poorer learning, demonstrate equivalent relative retention as non-exposed controls, suggesting that once the information is encoded, it can be retained. These deficits persist throughout adolescence; one study found that at the age of 14, initial deficits in encoding and acquisition continue to be responsible for deficits in both immediate and delayed conditions (Willford, Richardson, Leech, & Day, 2004).

Although deficits in verbal learning and memory are well researched and consistently reported, studies on nonverbal learning and memory have yielded less straightforward results (Mattson et al., 1996; Mattson, Autti-Rämö, May, Konovalova, & the CIFASD, 2006; Streissguth et al., 1990; Willoughby, Sheard, Nash, & Rovet, 2008). Children with FASDs have problems in basic visuospatial skills (Mattson & Calarco, 2002; Schonfeld et al., 2001), which can contribute to deficits in more complex tasks required for academic performance (Crocker et al., 2015). Children with heavy prenatal alcohol exposure demonstrate deficits in visual learning and fluency, although these deficits appear to be of the same severity or magnitude as the verbal deficits in these areas (Kaemingk et al., 2003; Schonfeld et al., 2001). There are some differential patterns for visual and verbal information. Once one accounts for initial verbal learning, there are no longer differences in delayed recall of verbal material. For visual information, children with heavy prenatal alcohol exposure recalled less information after a delay even after accounting for initial learning (Mattson & Roebuck, 2002), although these differences are not found universally (Kaemingk et al., 2003).

Emotional and Behavioral Functioning

In many cases, the emotional or behavioral problems associated with prenatal alcohol exposure are the presenting problem that leads a family to seek services. Rates of concomitant psychopathology are high in FASDs, particularly for externalizing disorders such as ADHD (Fryer, McGee, et al., 2007a; Paley, O'Connor, Kogan, & Findlay, 2005; Walthall, O'Connor, & Paley, 2008; Ware et al., 2013). These emotional and behavioral issues may affect academic functioning and psychosocial abilities beyond the effects of lower cognition. High rates of externalizing problems are consistently reported in alcohol-exposed children (Paley et al., 2005; Sood et al., 2001). As children become older, these behaviors can become the basis for psychiatric diagnoses of oppositional defiant disorder, conduct disorder, and delinquent behavior (Fryer, McGee, et al., 2007a; O'Connor & Paley, 2009; Roebuck, Mattson, & Riley, 1999; Schonfeld, Mattson, & Riley, 2005). Alcohol-exposed children also demonstrate higher rates of internalizing behaviors, which in turn can result in other psychiatric diagnoses such as depression and anxiety (Hellemans, Sliwowska, Verma, & Weinberg, 2010; Mattson & Riley, 2000; O'Connor & Paley, 2006). The severity of anxiety, depression, and withdrawal lead to increased rates of self-harm and suicide attempts (Baldwin, 2007). While there are some shared characteristics in social and communication domains with autism spectrum disorders, children with FASDs do not demonstrate the classic repetitive or restrictive behaviors (Stevens, Nash, Koren, & Rovet, 2013).

Other aspects of functioning can also impact a child's academic and social behaviors. For example, children with histories of alcohol exposure also demonstrate impaired sleep habits (Chen, Olson, Picciano, Starr, & Owens, 2012; Jan et al., 2010). Findings indicate that alcohol-exposed children have significantly

more sleep disturbances, shortened sleep duration, and an increased rate of night awakenings (Wengel, Hanlon-Dearman, & Fjeldsted, 2011). Sleep is a critical contributor to school performance, with poor sleep quality and sleepiness being related to learning, memory, and academic achievement in a meta-analysis of typically developing children (Dewald, Meijer, Oort, Kerkhof, & Bogels, 2010).

Psychosocial Outcomes

There is strong evidence of impaired social skills in children with prenatal alcohol exposure, which can result in reduced social competence, clinginess, not getting along with others, being teased or bullied, poor social judgment, and issues responding to social cues (Freunscht & Feldmann, 2010; Kully-Martens, Denys, Treit, Tamana, & Rasmussen, 2012; McGee, Bjorkquist, Price, Mattson, & Riley, 2009a; Way & Rojahn, 2012). These social deficits fail to improve with age; as social demands increase, children with FASDs fall even further behind (Crocker, Vaurio, Riley, & Mattson, 2009). Although verbal abilities are sometimes thought to be a relative strength, impairments in these areas can cause difficulties in social communication and conversational exchange (O'Connor et al. 2006, 2012). Alcohol-exposed children have difficulty providing appropriate information in conversations (Coggins, Timler, & Olswang, 2007) and processing social information (McGee, Bjorkquist, Price, et al., 2009a). Further, children with prenatal alcohol exposure struggle to balance linguistic and social-cognitive task demands during conversations (Coggins, Olswang, Carmichael Olson, & Timler, 2003). For example, these children often use ambiguous references and do not appropriately distinguish concepts in their stories (Thorne, Coggins, Carmichael Olson, & Astley, 2007). Inadequate theory of mind is also apparent as alcohol-exposed children fail to consider the perspective of the listener during social interactions (Coggins et al., 2007).

There are many factors that contribute to poor social functioning. Many of these children must contend with the effects of both prenatal alcohol exposure and living in adverse environments (Coggins et al., 2007). The combination of these factors can exacerbate social deficits. In turn, these impairments may affect school performance and academic achievement as well as contribute to the high rates of psychopathology in this population.

As alcohol-exposed children enter their adolescent and teenage years, they are forced to navigate the journey towards independence with additional factors that make the transition even more difficult. Alcohol-exposed adolescents, whether or not they qualify for a diagnosis of intellectual disability, are significantly impaired in their abilities to solve social problems in everyday life (McGee, Fryer, Bjorkquist, Mattson, & Riley, 2008). Even without physical dysmorphology, alcohol-exposed youth demonstrate significant deficits in psychosocial functioning (Roebuck et al., 1999). When matched on IQ, children with FAS still demonstrate social deficits. Therefore, social deficits are beyond what can be expected in low IQ, and findings suggest that these skills may be arrested rather than simply delayed (Thomas, Kelly, Mattson, & Riley 1998).

Impact on Other Areas of Functioning

In addition to the cognitive and behavioral deficits presented above, motor functioning and sensory perception are sensitive to the effects of alcohol. A recent meta-analysis found a significant association between moderate to heavy prenatal alcohol exposure and general motor deficits, particularly in balance and coordination (Lucas et al., 2014). Balance issues in this population are attributed to central core problems and longer latency responses and not entirely the result of vestibular disturbances (Roebuck, Simmons, Richardson, Mattson, & Riley, 1998). A study using a parent reporting measure for children ages 3–6 found that the majority of children with FAS demonstrated clinically relevant delays in general motor development (Kalberg et al., 2006). Fine motor skills were significantly more delayed than gross motor abilities. In a large study, the majority of individuals with FAS or other FASDs demonstrated deficits in motor function (Connor et al., 2006). Children with FASDs demonstrate more variability in performance and more frequently score low average or below on sensory motor measures, particularly in tasks that require visual-motor speed and precision (Jirikowic, Olson, & Kartin, 2008). Unfortunately, these are skills that are required frequently in school settings. These researchers also found that IQ scores may serve as a mediating factor for motor performance or a "surrogate" for neurocognitive deficits. Further, structural and functional differences in neural regions such as the cerebellum (Bookstein, Streissguth, Connor, & Sampson, 2006) and corpus callosum (Wozniak et al., 2009) may contribute to poor motor skills in individuals with FASDs.

Sensory perception deficits are seen at very young ages, with reduced visual acuity noted as early as 6.5 months after birth in a South African cohort even after considering gestational age, birth size, or other neurocognitive deficits (Carter et al., 2005). A diagnosis of FAS is also associated with various measures of hearing, including a developmental delay in auditory maturation, sensorineural hearing loss, and repeated otitis media resulting in conductive hearing loss and central hearing loss (Church, Eldis, Blakley, & Bawle, 1997; Church & Kaltenbach, 1997). One study found very high rates of hearing related issues in children with FAS (Church et al., 1997), however another study looking at children with FASDs found the rates of hearing loss to be no greater than expected (Cohen-Kerem, Bar-Oz, Nulman, Papaioannou, & Koren, 2007). As children with FASDs have visual and auditory sensoriperceptual deficits, which can directly contribute to socio-emotional and school functioning (Jirikowic et al., 2008), they should be screened for early evaluation, detection, and intervention.

Specific Diagnostic Outcomes

In the federal Individuals with Disabilities Education Act (IDEA) of 2007, FAS is listed as a presumptive eligibility diagnosis allowing individuals to obtain services. Unfortunately, the children who are affected by prenatal alcohol exposure but do not

meet the criteria for FAS or an intellectual disability (or other eligible diagnosis) may not qualify for these services. For example, children with an alcohol-related neurodevelopmental disorder (the prototypical example of Other Specified Neurodevelopmental Disorder) or the proposed ND-PAE diagnosis may not meet the strict eligibility criteria even though they have similar neurobehavioral impairments as children with FAS (Mattson et al., 1998). The ability to retrospectively and definitively identify alcohol-affected children without the characteristic facial features of FAS remains elusive. Special education and access to services vary by county and state, and therefore the present discussion focuses on nationwide services.

Currently, the most common and feasible method of receiving services for an alcohol-related neurodevelopmental disorder is to qualify for services under a different diagnosis, such as intellectual disability, Other Specified Neurodevelopmental Disorder, or ADHD, or to qualify under a specific catch all category based on functioning and symptomology such as the Other Health Impairment (OHI) category of IDEA. Legal precedents providing services for individuals with intellectual disability, or those requiring similar services, have facilitated access to services in some locations. These services may include a unique type of parenting class, a structure for collaboration between parents, teachers, and counselors, and potentially creating an individualized education plan (IEP). While not all children with FASDs will qualify for an IEP, they may meet criteria under the Section 504 of the Federal Rehabilitation Act of 1973 (*Section 504 of the Federal Rehabilitation Act of 1973*, 29 U.S.C.794, 1973). This can facilitate implementation of individualized classroom accommodations, yet falls short of creating an individualized plan for the student (Senturias, 2014).

While ND-PAE has been added to the DSM-5, it is currently listed as a "condition for further review," making it difficult for children and adolescents to access services as they may not be billable to insurance. In the interim, the DSM includes "Neurodevelopmental Disorder Associated with Prenatal Alcohol Exposure" as an example of Other Specified Neurodevelopmental Disorder (315.8, page 86). Note that the usage of "neurodevelopmental disorder" in the body of the DSM versus "neurobehavioral disorder" in the appendix may cause confusion. Further, the allowed usage of code 315.8 to indicate an alcohol-related disorder can be done without consideration of the criteria of ND-PAE. Individuals with FASDs can require the services of numerous providers, including primary care, specialist centers, occupational therapy, physical therapy, psychosocial skills training, and educational specialists (Rogers-Adkinson & Stuart, 2007). There are several FASD service centers (McFarlane & Rajani, 2007) that provide models for the continued development of resources. However, there is no easy or practical way to standardize the service needs for children with FASDs because each child will have unique patterns of deficits and may require a more individualized approach. Potentially, a modular system of interventions targeted at areas of high risk of deficits may be most effective. Semi-structured interviews revealed that there were no standardized special education classes that were appropriate for all alcohol-affected children (Autti-Rämö, 2000). Instead, each child needed individual supports based on their own patterns of functioning.

Prognosis and Moderating Factors

Until recently, it has not been possible to thoroughly research the developmental trajectory of alcohol exposure. The first cohort of children with FAS were diagnosed in the early 1970s (Jones & Smith, 1973) and are now reaching middle adulthood. There are several large prospective longitudinal studies that are attempting to characterize the effects of alcohol exposure over time, however more research is necessary to fully understand the developmental trajectory (Covington, Nordstrom-Klee, Ager, Sokol, & Delaney-Black, 2002; Elliott, Payne, Morris, Haan, & Bower, 2007; Kuehn et al., 2012; O'Callaghan, O'Callaghan, Najman, Williams, & Bor, 2007; Robinson et al., 2010; Streissguth, 1994c). A recent study of 22-year-olds found that prenatal alcohol exposure was associated with elevated rates of internalizing and externalizing behaviors in adulthood (Day, Helsel, Sonon, & Goldschmidt, 2013).

Adults with FASDs may continue to need support in many areas of their lives due to poor adaptive functioning, even though they may present clinically as cognitively intact. Adults are found to benefit from case management, however many individuals with FAS or another FASD may need continued support in housing, vocational rehabilitation, transportation assistance, and activities of daily living. Adults with prenatal exposure to alcohol have high rates of involvement in the legal system, high rates of substance abuse, and negative long-term outcomes. In one study, over 40 % of adults with FASDs had been incarcerated at some point, 20 % been confined for substance abuse treatment, and 30 % been confined to a mental institution (Streissguth et al., 1996). In spite of these reports, very little is known about the long-term effects of prenatal alcohol exposure as very little research has been conducted to empirically assess adult functioning in this population.

Many factors contribute to poor outcomes in alcohol-exposed children and therefore their deficits may be considered multi-determined (Streissguth et al., 2004). Risk factors commonly associated with alcohol exposure include: poor prenatal care, prenatal exposure to other teratogens (e.g., environmental, other drug exposures), parental psychopathology, neglect, abuse, frequent interactions with the foster care system, and changes in custody (Fagerlund, Autti-Rämö, Hoyme, Mattson, & Korkman, 2011). A longer time spent in residential care is the strongest risk factor associated with behavioral problems for children with prenatal alcohol exposure (Fagerlund et al., 2011). Alcohol-exposed children were also at greater risk of behavioral problems if they were not visibly alcohol affected and thus may not have received the services they needed during critical developmental periods (Fagerlund et al., 2011). Unfortunately, there are multiple systems-level barriers in place that hinder the ability for children and adults with FASDs to receive both diagnostic clarification and adequate services (Petrenko, Tahir, Mahoney, & Chin, 2014a). Children who receive an early diagnosis and are raised in a stable environment are 2–4 times more likely to avoid adverse life outcomes (e.g., disrupted school experiences, trouble with the law, inappropriate sexual behavior, or alcohol and drug problems) (Streissguth et al., 2004). There are also certain protective factors that can help attenuate the secondary disabilities often associated with FASDs, including

early identification and diagnostic clarification, early access to effective services, a stable and nurturing home, and not having a history of maltreatment or abuse (Petrenko et al., 2014a; Streissguth et al., 1996, 2004).

Considerations for Prevention/Intervention/School Accommodations and School Reintegration

Prevention

Early identification is a crucial step to improve outcomes for affected individuals and can facilitate the development and implementation of effective treatment (Carmichael et al., 2009). However, identification at any age remains difficult due the heterogeneity in clinical presentation, lack of objective and accurate biomarkers, lack of public awareness, and continued debate regarding the best classification system (Elliot, Payne, Haan, & Bower, 2006; Payne et al., 2011a, 2011b). A survey conducted by the American Academy of Pediatrics and the Centers for Disease Control and Prevention found that "pediatricians are knowledgeable about fetal alcohol syndrome but do not feel adequately trained to integrate the management of this diagnosis or prevention efforts into everyday practice" (Gahagan et al., 2006). Although government services such as the NIAAA warn that there is no safe amount to drink during pregnancy, the effects of light alcohol exposure remain strongly contested in public media (Drabble et al., 2014; NIAAA, 2013). As a result, pregnant women may be receiving mixed messages, which can also hinder prevention efforts.

It is common for women to underreport the amount of alcohol consumed during pregnancy, especially when the amounts are significant (Stockwell et al., 2004). This may be particularly problematic in situations where a woman may feel stigmatized. Women who drink during pregnancy may also not seek proper prenatal care. Even if they attend all prenatal appointments, questions regarding alcohol exposure may not be asked (Morleo et al., 2011). Many practitioners do not feel sufficiently trained in the diagnosis of fetal alcohol spectrum disorders, nor comfortable in talking about alcohol use with pregnant patients (Elliot et al., 2006; Payne et al., 2011b). Thus, it is rarely possible to confirm the accuracy, frequency, and timing of prenatal alcohol exposure.

While it remains difficult to categorize or classify individuals affected by alcohol exposure, there is a consensus regarding the known detrimental effects of alcohol on the developing fetus. Since 1973 when FAS was first identified, the negative effects of heavy prenatal alcohol exposure have been widely studied and accepted by the scientific community, with continued refinement of the neurobehavioral profile (Mattson et al., 2013). This information has resulted in widespread public health campaigns supporting abstinence from alcohol while pregnant. Unfortunately, these methods have not resulted in a significant decrease of drinking during pregnancy over the last decade. In a recent report, between 7.6 and 14.3 % of women who are aware that they are pregnant in the USA report alcohol consumption during their pregnancy, and 1.4 % report binge drinking (Grant et al., 2004; Thomas, Gonneau, Poole, & Cook, 2014).

However, this is likely an underreport of alcohol consumption during pregnancy more generally, as many women may not realize they are pregnant for 5–7 weeks and may continue to drink at high levels prior to pregnancy recognition. Specific maternal risk factors are associated with the development of fetal alcohol spectrum disorders. These include a maternal age of greater than 30 years, specific ethnic and racial backgrounds, lower socioeconomic status, having a previously affected child, undernutrition, and specific genetic backgrounds (Jones, 2011). A greater understanding of risk factors can facilitate more successful and well-informed interventions.

Currently, there are several behavioral interventions aimed at preventing alcohol-exposed pregnancies (Floyd et al., 2007; Hanson & Jensen, 2015; Mengel, Searight, & Cook, 2006). Some programs focus primarily on psychoeducation, as medical professionals at all levels of care have expressed a weakness in knowledge of this area (Payne et al., 2011a, 2011b). Other programs target women through community outreach, counseling sessions, social support, and screening (Floyd et al., 2007; Hanson & Jensen, 2015; Mengel et al., 2006).

As almost half of all pregnancies (49 %) are unplanned, many intervention programs attempt to target women prior to the conception (Finer & Henshaw, 2006). While the rates of unintended pregnancies have declined for college graduates and women of higher socioeconomic status, the rates of unintended pregnancies for unmarried and low-income women between the age of 18–24 remain above the national average (Finer & Henshaw, 2006). CHOICES, an integrated behavioral intervention funded by the CDC, has targeted these high-risk women through randomized controlled trials in a variety of clinical settings including primary care, university hospitals, jails, substance abuse treatment settings, and community settings (Floyd, Ebrahim, Boyle, & Gould, 1999; Hanson & Jensen, 2015; Velasquez, von Sternberg, & Parrish, 2013). Motivational interviewing, personalized feedback, contraceptive counseling, and reducing risky behaviors are the main tenets of the program, which has been demonstrated to be feasible and efficacious.

Another issue complicating the identification of FASDs is the lack of a standardized assessment protocol. One limiting factor of diagnostic clarification in the community setting is access to a trained dysmorphologist or a physician with dysmorphology training needed to make a diagnosis of FAS. Many physicians, including pediatricians, in the USA and abroad lack specific training on identifying FASDs. However, one study found that after a relatively short training session (2 days) many pediatricians were able to accurately diagnose FAS based on physical features—pediatricians' diagnoses were confirmed by the dysmorphologists in 83.5 % of the cases (Jones et al., 2006). Educating our front line medical practitioners is critical to increasing the number of children correctly diagnosed with prenatal alcohol exposure.

Intervention

Key areas of need in the realm of FASDs include a lack of appropriate services, lack of access to existing services, and lack of effective prevention strategies (Carmichael et al., 2009; Ryan, Bonnett, & Gass, 2006). Parents and service providers in focus

groups request interventions that are consistent with a positive behavior support network. Specifically, these would include central coordination, early diagnosis, support across the life span, and a strengths-based approach (Petrenko, Tahir, Mahoney, & Chin, 2014b). Caregivers express the need for support from professionals and extended family in the context of a structured environment and greater appreciation and understanding of a specific child's needs (Brown, Sigvaldason, & Bednar, 2005; Grant, Ernst, Streissguth, Phipps, & Gendler, 1996).

There have been several reviews of interventions for children and adolescents with fetal alcohol spectrum disorders within the past decade (Burd, 2006; Kodituwakku, 2010; Paley & O'Connor, 2011; Peadon, Rhys-Jones, Bower, & Elliott, 2009). Interventions must consider the cognitive and behavioral impairments that may affect a child's academic achievement, as both may contribute to deficits in overall functioning (Jirikowic, Gelo, & Astley, 2010). For example, the selective vulnerability of mathematical functioning to prenatal alcohol exposure has led to development of promising math interventions (Coles et al., 2009; Kable et al., 2007). The Math Interactive Learning Experience (MILE) program for children with FASDs focuses on core skill deficits as well as issues that may contribute to poor performance. The program includes lessons on meta-cognitive problem solving skills ("plan-do-review") and learning readiness. In this context, learning readiness involves preparing the child's environment and considering behavioral modifications to ensure that learning can be most successful. The MILE program is individualized to the child's pace, and involves more feedback and active interaction regarding error patterns. In initial studies assessing MILE, the intervention group of 3- to 10-year-olds demonstrated significant improvement in mathematical knowledge, both directly after a 6-week intervention, and 6 months after the intervention was completed (Coles et al., 2009; Kable et al., 2007). A 15-week, randomized clinical trial found that those in the program demonstrated significant gains in math that persisted over six months (Kable et al., 2015). The MILE program was also recently successfully disseminated into community settings, which is an important aspect of all interventions (Kable et al., 2015).

A language and literacy intervention conducted in South Africa has also been successful (Adnams et al., 2007). The program produced significant improvement in certain preliteracy and literacy variables. Despite these gains, alcohol-exposed children were still significantly different from controls, demonstrating improvement but not remediation. For the underlying cognitive mechanisms related to language and literacy, rehearsal training and neurocognitive habilitation training demonstrated improvement, specifically in working memory span and executive functioning (Loomes, Rasmussen, Pei, Manji, & Andrew, 2008). However, it is not clear if these findings generalize beyond the task to school settings (Melby-Lervag & Hulme, 2013).

In addition to the cognitive targets, there have also been interventions focused on the socio-emotional and behavioral concerns in children with heavy prenatal alcohol exposure (Kully-Martens et al., 2012; O'Connor et al., 2012). Some of these interventions are administered to the child directly, such as children's friendship training and adaptive behavior programs. Friendship training demonstrated significant improvement in social skills in children with and without alcohol-exposure

(Keil, Paley, Frankel, & O'Connor, 2010; O'Connor et al., 2012; Paley & O'Connor, 2011). Attempts to improve adaptive behavior and safety behavior have also demonstrated success. A virtual reality intervention for fire and street safety resulted in significantly better knowledge of the intervention, and 72 % of the 32 children were able to generalize their knowledge to behavioral settings (Coles, Strickland, Padgett, & Bellmoff, 2007).

Interventions also include parent-child interaction therapy and family systems models. The Families Moving Forward program has successfully reduced child behavior problems and improved parenting efficacy by combining a developmental systems perspective with a family systems approach (Olson, Oti, Gelo, & Beck, 2009). Likewise, a one-on-one parent mentorship model called Step by Step was also effective in helping caregivers reduce their needs and achieve goals (Denys, Rasmussen, & Henneveld, 2011).

School Accommodations and School Reintegration

A review of FASDs in relation to academic achievement found that successful interventions need to balance the use of environmental modifications and the many additional factors at play with alcohol-affected children (Green, 2007). The heterogeneity of academic, behavioral, and cognitive function in children with FASDs makes it extremely difficult to create a one size fits all academic curriculum. For instance, the range of intellectual function in these children is quite broad, and therefore effective interventions must cater to a wide range of abilities. In addition, programs must understand and address the interplay between cognitive, academic, social, emotional, and behavioral challenges. For example, disruptive behavior may be due to behavioral impulsivity or executive dysfunction, both of which are common in FASDs.

Assessment of school-based services for children with FASDs is a burgeoning area of research. In the classroom, a combination of evidence-based interventions may be the most efficacious, as they can target various areas simultaneously. Since 60–95 % of alcohol-exposed children are diagnosed with ADHD (Fryer, McGee, et al., 2007a; Mattson et al., 2011), it may be worthwhile to investigate the feasibility of repurposing existing, empirically supported ADHD interventions for children with FASDs. Unfortunately, the availability of interventions has fallen far below the needs of alcohol-exposed children, and many of these programs are still being studied to assess generalizability, feasibility, and efficacy. Cognitive deficits such as those well documented in the domains of executive function, learning, and memory must be considered in all aspects of educational intervention, including giving instructions and assignments, arranging the environment, planning transitions, and overall interactions and expectations within the classroom. Other considerations must include the behavioral difficulties as well as auditory and visual perception deficits.

The high variability in IQ scores and differential profiles associated with heavy prenatal alcohol exposure requires a true individualized education plan. Unfortunately, many children with FASDs may not be able to receive services

because of strict interpretations of cutoffs, as the majority of affected children have IQ scores in the average or borderline range. In spite of a potentially adequate overall general cognitive ability level, many have a variety of behavioral and cognitive deficits that can dramatically affect performance. Children with average intelligence may benefit from assessment to determine whether specific issues with attention, verbal learning and memory, auditory memory and processing, and executive functioning exist and may be contributing to poor performance. In addition, alcohol-exposed children often present with adaptive functioning deficits and behavioral issues that can interact and limit academic success. Behavioral plans may also be a way to provide support for both the behavioral challenges and the cognitive deficits. Behavioral observations in the classroom are also recommended to understand how the children function when faced with higher cognitive demands and in a more distracting environment.

There continues to be a dearth of research to support the development, implementation, evaluation, and dissemination of interventions despite the known detrimental effects of prenatal alcohol exposure. However, there has been an increase in educational and cognitive interventions that have shown preliminary success. Increased structure, both at home and in the classroom, are useful behavioral tools for children who have low executive functioning.

Most studies of caregivers of children with FASDs focus on understanding caregiver and child interactions at home, but there is little research that has examined how caregivers address issues with schools. One study in a western Canadian province systematically assessed how caregivers behave in response to school difficulties (Swart, Hall, McKee, & Ford, 2014). Caregivers of children with FASDs were found to focus on two themes: orchestrating schooling (e.g., available to be in the classroom, building connections with teachers, anticipating difficulties) and keeping up appearances to avoid being viewed as inadequate parents (e.g., reframing behaviors, controlling access to information, trying to fit in with other parents). These are both areas that should be addressed through intervention.

Conclusion

Since the effects of alcohol exposure are extremely heterogeneous, children who fall under the umbrella of fetal alcohol spectrum disorders often go unrecognized. The lack of early identification can lead to reduced access to services for alcohol-affected individuals. Children with histories of prenatal alcohol exposure must be considered in a holistic manner, as they may present with medical, cognitive, behavioral, and adaptive functioning impairments. An educational plan should be developed with the guidance of a team of specialists that are able to help conceptualize all aspects of the individual's functioning. The interplay between behavior and cognition is incredibly important for both academic and social settings, as they can interact to determine success. Diagnostic clarification and the refinement of a neurobehavioral profile for fetal alcohol spectrum

disorders are at the forefront of research focus. While the neurobehavioral profile is not fully elucidated, it is abundantly clear that there are many distinct behavioral and cognitive domains that must be considered while developing and implementing interventions for this population.

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