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RESEARCH FORUM

Fetal alcohol spectrum disorders: A literature review with screening recommendations

The authors examine intellectual and developmental disabilities caused by prenatal exposure of fetuses to alcohol and other drugs

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Prenatal exposure of fetuses to alcohol, as compared to prenatal exposure to other substances of abuse (including cocaine, heroin, and marijuana), produces the most deleterious neurobehavioral effects, making it the leading known cause of intellectual and developmental disabilities in the world (Astley, 2004). In 2003, Dr. Kenneth Warren, the director of the office of scientific affairs at the National Institute on Alcohol Abuse and Alcoholism, reported, “The consensus, I think, at this point, is that most of the adverse effects that had been reported due to cocaine and crack use were from alcohol use” (cited in Carroll, 2003, p. D4). This consensus has received strong support from subsequent research on the developmental consequences of prenatal drug exposure (primarily cocaine). In stark contrast to reports in the late 1980’s of dire consequences for so-called “crack babies,” subsequent research using much more methodologically sophisticated designs has in general concluded that the effects, which are not always found, are best characterized as “small” or “subtle” (Ackerman, Riggins, & Black, 2010; Betancourt et al., 2011; Lester, Legasse, Lin, & Pescosolido, 2008; Richardson et al., 2011). These findings stand in marked contrast to those for prenatal exposure to alcohol, as subsequently will be discussed, which can result in devastating consequences when the exposure is large enough (Bertrand et al., 2004; Kodituwaku, 2007; Riley, Mattson, & Thomas, 2009; Streissguth et al., 2004).

Although the deleterious effects of prenatal exposure to alcohol on the developing human have been known for centuries, these effects were not documented in the medical literature until 1968. The pattern of effects which has become known as Fetal Alcohol Syndrome (FAS) was not identified until 1973 (Calhoun & Warren, 2007). Since then it has become clear that these deleterious effects result in a spectrum of structural anomalies and behavioral and neurocognitive disabilities termed Fetal Alcohol Spectrum Disorders (FASD). FASD is a non-diagnostic umbrella term which encompasses the range of adverse outcomes that can occur in a person whose mother drank alcohol during pregnancy (Bertrand et al., 2004). FAS, which is a medical diagnosis within the designation FASD, represents the full Fetal Alcohol Syndrome caused by prenatal exposure to alcohol and results in the following characteristics: a) central nervous system (CNS) dysfunction, b) prenatal onset of growth deficiency that persists postnatally, and c) specific facial anomalies (Astley, 2006; Hoyme et al, 2005; Olson et al., 2009; Riley, Mattson, & Thomas, 2009). The term FASD was developed to acknowledge that individuals who failed to meet FAS criteria can still have severe negative effects caused by prenatal exposure to alcohol, though those with FAS are typically more impaired (Chasnoff, Wells, Telford, Schmidt, & Messer, 2010; Fryer et al. 2007; Olson, King, & Jirikowic, 2008; Olson et al., 2009).

Recent data from the Centers for Disease Control and Prevention (CDC) [2009a] found that 12.2% of pregnant women aged 18-44 reported consuming alcohol. This level of use in pregnancy results in a FASD prevalence of 2-5% among young children in both mixed racial and mixed socioeconomic school populations (May et al., 2009). Despite the long-standing knowledge of the serious adverse sequelae of prenatal exposure to alcohol, its prevalence, and the fact that there are thousands of articles and hundreds of books devoted to it (Abel, 2006), most children and adults with FASD probably go through life undiagnosed (Streissguth, 1997). For example, in a case ascertainment study of all elementary schools in two counties in Washington State, only 1 of the 7 students found to have FAS had been previously diagnosed (Clarren et al., 2001). Furthermore, if and when children are referred for a FASD evaluation, few are initiated by schools (e.g., 5%), but rather by medical, psychological or social service providers with the result that the average age of referral is about 9 ½ years and relatively late in a child’s life for this type of diagnosis (Olson et al., 2007).

Hence, given the importance of detecting FASD combined with the failure to identify such individuals, the goal of this article is to help advance the ‘call to action’ issued by National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect to set up effective strategies for screening, referral, and treatment planning in critical community settings such as special education (Olson et al., 2009). The referral process, which begins when a front line provider of services such as a school psychologist starts to suspect that a child might have an FASD, is predicated upon the clinician having a thorough knowledge of the

characteristics of FASD (CDC, 2009b; Bertrand et al., 2004). School psychologists need appropriate knowledge to enable them to make an informed referral to a multidisciplinary FASD diagnostic team which can then evaluate and provide recommendations for interventions (Astley, 2004, 2006; Bertrand et al., 2004; Hoyme et al., 2005; Olson, King, & Jirikowic, 2008; Olson et al., 2009).[1] (#footnotes) Such a referral to an expert FASD diagnostic team is essential because of the broad array of outcomes that define FASD (Astley, 2004, 2006; CDC, 2009b; May et al., 2009; Olson et al., 2009).

FASD characteristics

Several diagnostic guidelines have been developed to capture the spectrum of effects of prenatal exposure to alcohol, yielding a variety of designations such as: fetal alcohol syndrome (FAS), partial fetal alcohol syndrome (PFAS), fetal alcohol effects (FAE), alcohol-related birth defects (ARBD), alcohol-related neurodevelopmental disorder (ARND), static encephalopathy/alcohol exposed (SE/AE), and neurobehavioral disorder/ alcohol exposed (ND/AE) (Astley, 2004, 2006; Astley et al., 2009; Bertrand et al., 2004; Hoyme et al., 2005; Olson et al., 2009). Although a specific consensus diagnostic standard has yet to be reached, there is consensus that four major criteria must be considered in detecting a FASD: a) confirmed maternal alcohol exposure, b) facial anomalies, c) growth deficiency, d) central nervous system (CNS) damage/ dysfunction (Astley, 2004, 2006; Bertrand et al., 2004; Hoyme et al., 2005; Olson et al., 2009).

Confirmed prenatal alcohol exposure

The U.S. Surgeon General's 2005 advisory that women who are pregnant or considering becoming pregnant abstain from using alcohol (Olson et al., 2009) is based on findings that even low levels of prenatal exposure to alcohol can have adverse effects on development; adverse effects are dose dependent, with heavier maternal consumption associated with more severe outcomes (Olson, King, & Jirikowicz, 2008; Riley, Mattson, & Thomas, 2009).[2] (#footnotes) Note, however, that while there is a robust consensus on the adverse effects on the fetus of heavy maternal drinking over an extended period of time during pregnancy, the issue of whether or not adverse effects are associated with low to moderate drinking is one of ongoing dispute (Abel, 2009; Kelly et al., 2010). The difference in consumption patterns and timing of exposure, as well as other variables such as maternal health, genetic background of mother and child, and synergistic interactions with other substances helps explain the marked variability in FASDs outcomes, even among heavy drinkers (Abel, 2006; Guerri, Baziner, & Riley, 2009; Olson, King, & Jirikowicz, 2008).

For example, in the Seattle Prospective Longitudinal Study (Streissguth, 2007), of the 500 children who were exposed prenatally to a range of alcohol levels (22% of whom were exposed to maternal 'heavy drinking'), only 2 were diagnosed with FAS and 12 with alcohol-related neurodevelopmental disorder (ARND) which refers to central nervous system impairments caused by alcohol but without facial anomalies or growth deficiency. Therefore, prenatal exposure to alcohol alone is not sufficient to warrant a diagnosis of a FASD, as there must be evidence for the other criteria (Bertrand et al., 2004). Equally important to note, is that a lack of confirmation of alcohol use should not preclude a referral if evidence for the other three criteria are present (Astley, 2006; Bertrand et al., 2004). With these understandings, the following recommendations are offered for screening for prenatal alcohol exposure.

Facial anomalies

A variety of dysmorphic facial features have been associated with FAS since 1973 when the syndrome was first given its name (Abel, 1998). It is likely that these facial malformations are the product of alcohol exposure during the third week of gestation (Riley, Mattson, & Thomas, 2009). Although there are a host of congenital anomalies that can be alcohol-related, no single dysmorphic feature is unique to any particular syndrome (Bertrand et al.,2004). However, there is a cluster of three specific facial anomalies that are unique to FAS (Astley, 2004, 2006; Astley et al., 2009). They are: (a) a shorter distance between each end of the eye socket opening (called the palpebral fissure), (b) lack of skin fold indentation between the nose and upper lip (also known as smooth or indistinct philtrum), (3) a thin upper lip (called a thin vermillion border). Specific guidelines have been developed for dysmorphologists to enable precise measurement of these features (Astley, 2004, 2006). This uniqueness is critically important diagnostically for two reasons. First, unlike the criteria of growth deficiency and CNS damage/dysfunction which can have causes other than prenatal alcohol exposure, if this phenotype is present, it must have been caused (with the single exception of a very rare disorder, *toluene embryopathy*, Bertrand et al., 2004) by prenatal alcohol exposure. Hence, the individual must have FAS, even if maternal prenatal alcohol consumption cannot be confirmed (Astley, 2004, 2006). Second, since there is very little change in this unique facial phenotype with age (Astley & Clarren, 2001; Astley, personal communication, March 3, 2009), if these anomalies are present in adolescence or adulthood, they are diagnostic for FASDs.

Lastly, it is also very important to note that although most children who have an FASD do not have this triad of anomalies (Olson et al., 2007), these children can suffer CNS damage/dysfunction from prenatal alcohol exposure that is as severe as those with the anomalies (Astley et al., 2009; Olson, King, & Jirikowic, 2009). This is because the worst time to expose the fetal brain to alcohol is during the third trimester, after the facial features have formed, when the brain experiences a tremendous growth spurt (Fields, 2009).

Growth deficiency

Growth deficiencies have been consistently documented in FASD. This criterion is met if prenatal or postnatal height or weight are at or below the 10th percentile at any one point in time (Bertrand et al., 2004; Olson et al., 2007). Care must be taken that the child was not nutritionally deprived at the single point in time when the growth deficit was present (Bertrand et al., 2004). This criterion can be difficult to detect in adolescence/ adulthood, as the earlier deficits may no longer be evident because the child has caught up (Streissguth, 2007), though height deficits may still be present (Spohr, Wilms, & Stenhausen, 2007). However, even though growth deficiencies may be minimal or absent in adolescence/ adulthood, limiting its use as an indicator of a FASD (Streissguth, 2007), any prior history of growth deficiency is consistent with the criterion for diagnosing a FASD (Bertrand, Floyd, & Weber, 2005). Furthermore, it is critical to understand that individuals can suffer from severe CNS damage/dysfunction without demonstrating growth deficiencies (Olson, King, & Jirikowic, 2009).

Central nervous system abnormalities

CNS abnormalities represent the most devastating consequence of FASD (Astley, 2006; Guerri, Baziner, & Riley, 2009; Riley, Mattson, & Thomas, 2009) and generally persist throughout the life span (Bertrand et al., 2004). Furthermore, as previously discussed, these abnormalities can be present despite the absence of facial anomalies and/or growth deficiencies. Indeed, most children who suffer from CNS abnormalites caused by prenatal exposure to alcohol have neither facial anomalies nor growth deficiencies (Bertrand et al., 2004; Rasmussen & Bisanz, 2009a). Although the most common CNS abnormality is reduction in brain volume, the brain is not uniformly sensitive to prenatal exposure to alcohol (Guerri, Baziner, & Riley, 2009; Riley, Mattson, & Thomas, 2009). Various brain regions such as the cerebellum, corpus callosum, hippocampus, and basal ganglia are particularly sensitive to structural insults which, in turn, can be related to various neuropsychological impairments (Guerri, Baziner, & Riley, 2009; Norman et al., 2009; Riley, Mattson, & Thomas, 2009). Several potential mechanisms have been identified as mediators of the CNS abnormalities: a) alteration in the regulation of gene expression, b) interference with neural stem cell proliferation, migration and differentiation, c) disturbances in molecules that mediate cell-cell interactions, d) impairment in activation of molecular signaling controlling cell growth and death, e) derangements in glia proliferation, differentiation and functioning (Guerri, Baziner, & Riley, 2009).

The diagnostic criterion is met if one of three types of CNS abnormalities, or any combination thereof, are present: a) structural such as reduction in head circumference, or brain abnormalities which can be detected by various brain imaging methods such as reduction in size or shape of the corpus callo-sum, b) neurologic such as motor problems or seizures not resulting from postnatal insult, c) functional such as substandard cognitive functioning in various domains (Astley, 2006; Bertrand et al., 2004). Although the first two types of deficits are the purview of the medical profession, and hence will not be discussed, it is important that psychologists understand that such structural and neurologic abnormalities can be caused by prenatal exposure to alcohol and include them in their diagnostic formulations if they are present. The following discussion, apropos of the psychologist's role, will focus on the functional deficits which have been identified across a broad swath of cognitive abilities (Kodituwakku, 2007 Olson, King, & Jirikowic, 2009). These deficits are best understood as clustering into three distinct domains: a) reduced general mental ability, b) non-verbal learning disability, c) Attention-Deficit/ Hyperactivity Disorder (ADHD).

General mental ability

General mental ability or g represents a general proficiency in learning, reasoning, thinking abstractly and otherwise processing complex information efficiently and accurately (Gottfredson, 2008). Impaired general mental ability constitutes the central cognitive impairment (Kodituwaku, 2007) and the most devastating consequence of prenatal alcohol exposure (Riley, Mattson, & Thomas, 2009). The mean IQ in FAS is in the low 70's for those with facial anomalies and in the low 80's for those without facial anomalies (Riley, Mattson, & Thomas, 2009). Approximately 25% of individuals with a FASD have an intellectual disability (ID; Bertrand et al., 2004; Streissguth et al., 2004).

However, it is also most important to recognize that even though for 75% of those with a FASD, their cognitive impairment in g does not meet the typical cut-off of two standard deviations below the mean falling to the 3rd percentile or lower as required for ID (Spruill & Harrison, 2008), it is a grave mistake to describe these individuals, as some have done (e.g., O'Malley, 2007, p. 2), as having a "normal IQ." For example, in the best study to date for determining the average IQ associated with FASD, the median IQ of 415 individuals with a FASD (median age 14 years, range 6-51) was 86 (Streissguth et al., 2004). This means that approximately 50% of individuals with a FASD have a general mental ability level between the 3rd percentile (IQ=70) and the 16th percentile (IQ=86) and would be described as functioning in the 'borderline' or 'low average range' of intelligence. Such a level of general cognitive functioning however (even for those in the deceptively designated 'low average range' of 80-86) can be expected to cause significant problems in adaptation (Gottfredson, 2003). This expectation received impressive support from a puzzling finding in the Seattle prospective longitudinal study of FASDs (Streissguth, 2007). The 221 individuals with FAE[3] (#footnotes) had higher rates of adverse life outcomes than the 138 individuals with FAS. The authors speculated that this may have been due to the fact that school and community services are typically only provided for those with IQ's below 70; and since only 7% of those with a FASD had IQ's below 70, compared to 24% of those with FAS, those with a FAS were less likely to have had appropriate interventions which they clearly needed since they were not capable of 'normal' cognitive functioning.

Finally, it is also important to note that FASDs are fully compatible with average and above intellectual functioning, as IQ's as high as 126 have been found (Streissguth et al., 2004). This does not automatically mean however that there is no impairment in cognitive functioning. Since general mental ability does not capture the full spectrum of cognitive abilities (Gottfredson, 2008; McGrew, 2009), it cannot be expected to and does not capture the full spectrum of cognitive deficits caused by a FASD (Bertrand et al., 2004; Hoyme et al, 2005). Among the cognitive deficits not accounted for by g, there is a distinctive pattern best conceptualized as a non-verbal learning disability (NLD; CDC, 2009b; Don & Rourke, 1995; Rasmussen & Bisanz, 2009b; Rourke, 2009).

Non-verbal learning disability

An account of NLD was first presented in 1967 by researchers who indentified children impaired in their ability to make sense of the non-verbal aspects of day-to-day functioning with subsequent accounts identifying additional deficits in social interaction, visual-spatial, and mathematical ability (Tsatsanis & Rourke, 2008). Since then, the principal investigators of NLD, Byron Rourke and his colleagues (Rourke, 2009; Rourke et al., 2002; Tsatsanis & Rourke, 2008) have more fully fleshed out the cardinal clinical features of NLD in terms of a distinctive pattern of assets and deficits. Moreover, they have concluded that high functioning individuals with a FASD exhibit most of these features (Don & Rourke, 1995; Rourke, 2009).

The major deficits of NLD that are commonly found in individuals with FASD are: a) poor coordination and motor slowness, often more marked on the left side of the body, b) marked deficiencies in visual-spatial skills and visual spatial memory, c) marked relative deficiencies in mechanical arithmetic in contrast to adequate/good verbal skills such as word decoding, spelling, vocabulary, rote verbal memory, c) notable difficulty in adapting to novel/complex situations, d) difficulty in dealing with cause and effect relationships and marked deficiencies in appreciation of incongruities (e.g., humor), f) marked deficiencies in non-verbal problem solving, concept formation, and hypothesis testing, g) marked difficulty with more complex verbal material and written text usually because the material is abstract, inferential, or requires an appreciation of relevant vs. irrelevant detail, h) marked deficits in the capacity to benefit from feedback in novel/complex situations (Don & Rourke, 1995; Tsatsanis & Rourke, 2008). These impairments contribute to deficits in social perception, social judgment, and social interaction skills, and make it all but impossible for an individual with NLD to adapt to novel interpersonal situations (Rourke, 2009).

Perhaps the most important implication of NLD for understanding individuals with FASD is that because some basic verbal abilities, such as vocabulary, are relatively well developed (Rourke, 2009; Rourke et al., 2002; Tsatsanis & Rourke, 2008), damaging false expectations can be generated (Streissguth, 1997). For example, in his account of raising his son Adam who had FAS, Michael Dorris (1990) provided numerous instances of perplexing dysfunctional behavior in which Adam repeatedly "did not learn from his mistakes, inconvenient or maddening as they often were " (p.200) and certainly "didn't know what he was talking about" (p. 154).

Attention-deficit/hyperactivity disorder

Although individuals diagnosed with FASD account for about only 2 percent of individuals with ADHD (Nigg, 2006), it is the most frequent neuropsychiatric presentation of an individual with a FASD throughout the lifespan (O'Malley, 2007), with rates ranging from 60 to 95 percent (Astley et al., 2009; Burd et al., 2003; Fryer et al., 2007; Herman, Acosta, & Chang, 2008; Kodituwakku et al., 2006; Streissguth et al., 1996). The issue of whether deficits are the same in developmentallybased and alcohol induced ADHD is yet to be resolved (Riley, Mattson, & Thomas, 2009;Vaurio, Riley, & Mattson, 2008).

Executive functions

Most individual with ADHD have impaired executive functioning (EF) [Barkley, 2011]. Although the concept of executive functions (EF) has been difficult to define definitively, a generally accepted conception is that they represent a class of higher order cognitive abilities (such as response inhibition and working memory) that allow for strategic planning, impulse control, cognitive flexibility and goal-directed behavior (Weyandt, 2009) and thereby facilitate behavioral and affective regulation (Bar-kley, 2006; Brown, 2008; Nigg, 2006). EF impairment is invariably cited as contributing to major life problems (Riley, Mattson, & Thomas, 2009). Hence it is not surprising that impaired EF functioning is commonly found in those with a FASD since up to 95% of those with a FASD have ADHD (CDC, 2009b; (Fryer et al., 2007; Kodituwakku, 2007; Mattson & Vaurio, 2009; Riley, Mattson, & Thomas, 2009). Such impairment helps account for the problems in multiple domains (social, legal, alcohol use and mental health) that characterize many individuals with a FASD (Riley, Mattson, & Thomas, 2009).

Guidelines for referral to a team of FASD specialists

As previously discussed, the referral process is initiated at the point when a service provider, such as a school psychologist or other education personnel, starts to have suspicions of an alcohol related disorder for a child (Bertrand et al., 2004). This process is predicated on adequate knowledge of the domains affected by FASD, such as that provided by the present review. The following guidelines, informed by national guidelines (Floyd et al., 2004) and the present review, indicate that the following "triggers" should prompt consideration for a referral to a team of FASDs specialists.[4] (#footnotes) It should be noted that the national guidelines advise that when in doubt the preferable option is to refer (Bertrand et al., 2004).

Prenatal alcohol exposure

Given that denial and underreporting of alcohol consumption is common and particularly so when significant amounts of alcohol are consumed (Abel, 2006), it is rarely possible to confirm the accuracy, frequency and timing of prenatal alcohol exposure (Astley et al., 2009). An approach emphasizing sensitivity would dictate that any maternal report of consumption during pregnancy, especially in association with behavior problems and/or marked developmental delay in the first three years of life, should prompt serious consideration of a referral (Astley, 2006; Olson et al., 2007). Moreover, referral should be strongly considered if a) the mother was an alcoholic, b) the mother has at least one other child with a FASD, c) there is a history of social/medical/legal problems related to alcohol (Bertrand et al., 2004).

Facial anomalies

Since the facial phenotype is not simply present or absent, but varies in magnitude across FASD designations (Astley et al., 2009), if one or all of the three facial anomalies appear to be present to the untrained eye, a referral is warranted. This is especially true if, in addition to the apparent facial phenotype, there are indications of prenatal exposure to alcohol and either growth deficiency or CNS abnormality. Growth Deficiency A growth deficiency, especially an apparently small head size, which is clearly not due to a nutritional deficiency or other biological cause, and is associated with a CNS abnormality, may be indicative of a FASD.

Central nervous system abnormalities

Although the profile of cognitive dysfunction among individuals with FASDs is highly variable and hence there is no unique profile specific to alcohol teratogenicity (Astley et al., 2009), it is clear that deficiencies in general mental ability, NLD, and ADHD commonly occur.

These deficits, especially in interplay with the cumulative risk of adverse psychosocial circumstances, typically result in poor academic and occupational performance and a variety of other adverse life outcomes such as incarceration, substance abuse, psychiatric hospitalization, etc. (Olson, King, & Jirkiowic, 2008; Streissguth et al., 2004; Yumoto, Jacobson, & Jacobson, 2008). However, it is also equally clear that the triad of CNS deficits can be caused by conditions other than FASDs (Astley, 2004; Astley et al., 2009). This poses a diagnostic challenge which helps explain why there is no validated screening test for FASD (Goh et al., 2009) and why most of the individuals with a FASD have not been diagnosed. Indeed, absent a severe facial phenotype or obviously stunted growth which would be apparent to even an untrained eye, it would seem that the screening challenge is insuperable.

Perhaps the best advice is that once school psychologists understand that the CNS deficits commonly occurs in FASD, even in the apparent absence of the facial phenotype and growth deficiency, they should ‘take another look’ when individuals present with such characteristics (Benson, 2008). This might be especially true for ADHD, as O’Malley (2007) has suggested that the major reason FASD are so often unrecognized by professionals is that they ‘masquerade’ as developmental ADHD. Furthermore, particular attention should be given to those cases in which ADHD presents with a low IQ (e.g., 85 or below), since the average IQ deficit for children and adults with developmental ADHD is relatively small compared to the IQ deficit for those with a FASD and ADHD (Barkley, 2006; Bridgett & Walker, 2006). For example, the study of attention and EF deficits in children with either ADHD or heavy prenatal alcohol exposure (ALC) reported a 20 point difference between the average IQ’s of the two groups: 105 (ADHD), 85 (ALC; Vaurio, Riley, & Mattson, 2008).

Conclusion

Fetal alcohol spectrum disorders result in permanent abnormalities across physical, neurological, and cognitive domains and are present in an astonishingly high 2-5% of young children across school populations. Given the challenges in identification and the fact that most individuals with a Fetal Alcohol Spectrum Disorders: A Literature Review with Screening Recommendations FASD have not been diagnosed, school psychologists need to fully understand and carefully consider the possibility that the ruinous effects of prenatal exposure to alcohol may be a factor when they are conducting evaluations. If any of the requisite “triggers” are evident, strong consideration should be given for a referral to a specialist FASD team, with the rule of thumb being “when in doubt refer.” If diagnosis of a FASD is confirmed, an intervention plan is developed by the team in concert with education personnel, caregivers, and child (Bertrand et al., 2004). Note that the approach to developing individualized and targeted interventions specific to children with FASD is not predicated on the notion that children with FASD present a “unique” neuropsychological or behavioral profile (Astley et al., 2009) but upon the fact that these children as a group are very heterogeneous in the nature, severity, and multiplicity of their problems (Bertrand, 2009).

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1) Since a discussion of intervention and teaching recommendations is beyond the scope of the article, the reader can consult the following excellent resources for such information: National Organization on FAS (www.nofas.org/educator/teaching), Bertrand (2009), and Paley and O'Connor (2009).

2) Heavy or binge drinking for women is defined as 4 or more drinks on at least one occasion (Centers for Disease Control, 2009a).

3) The study's designation for individuals who had heavy prenatal alcohol exposure but did not manifest the full physical features of FAS.

4) If it is determined that a referral is warranted, the list of FASD regional training centers listed in the ap-pendix of the CDC competency-based curriculum development guide (CDC, 2009a) can be consulted to facilitate such a referral. http://www.cdc.gov/ncbddd /fasd/curriculum/FASDguide_web.pdf

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