

## **A review of the evolution of diagnostic practices for Fetal Alcohol Spectrum Disorder**

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*Fetal Alcohol Spectrum Disorders (FASD) are characterized by cognitive, emotional, behavioural, and social disabilities. FASD are complex and pose many challenges for clinicians and researchers in the assessment, diagnosis, and intervention process. The variations in amount, timing, and frequency of alcohol that is consumed during pregnancy can produce a wide spectrum of deficits ranging from extremely debilitating impairments to subtle problems that can be easily misdiagnosed. This spectrum often creates confusion as symptoms may be very different between different children, thus making appropriate intervention elusive as well. Failure to accurately identify this population also reduces the opportunities to implement preventative programs. By informing the psychological community of the unique features of this population it is possible that this disorder may become more widely recognized and understood resulting in more accurate diagnoses and creative interventions to prenatal alcohol-related problems. More importantly, children diagnosed with FASD, and their caregivers, are more likely to be provided with the extra support and understanding their condition requires.*

### **Introduction**

Fetal Alcohol Spectrum Disorders (FASD) are described as the leading causes of preventable emotional, behavioural, cognitive, and social dysfunction (Abel & Sokol, 1986, 1987; Streissguth, 1997; Streissguth & O'Malley, 2000). Until recently, however, FASD has received relatively little recognition, and, in fact, has been described as one of the most under-treated of the lifelong developmental disabilities within the mental health disciplines (Streissguth & O'Malley, 2000). Spurred on by recent high profile reports of crimes committed against or by those who have been diagnosed with FASD, discussion of this syndrome has spanned several disciplines and research has flourished. Teachers, social workers, psychologists, and physicians are just a few of the professionals finding that knowledge and understanding of FASD would be an asset to their work.

Fortunately, there are positive outcomes for prenatally exposed children who are diagnosed and treated early (Streissguth, 1997), and thus the need and urgency for global understanding and consistent diagnostic and assessment approaches within and across disciplines grows. Accurate assessment is crucial for appropriate diagnosis and intervention. In fact, Whaley, O'Connor, and Gunderson (2001) argue that early intervention based on accurate diagnoses "could prevent the social skills deficits evidenced in these children as they grow older. For this reason, there is a need for clinicians to screen patients for prenatal alcohol exposure when making decisions about treatment options" (p. 1022). Moreover, Clarren, Carmichael Olson, Clarren, and Astley (2000) emphasize the importance of accurately determining etiology of observed behaviour for two key reasons. First, they state that etiological awareness alerts clinicians and others who work with children with FASD to the complex nature of the profile, and allows for an attitude change from seeing a child as disobedient to seeing a child as disabled. Secondly, they argue that accurate identification may help prevent prenatal alcohol exposure in the biological mother's future pregnancies as she will be identified and may therefore receive the necessary support within a prevention program. As such, appropriate diagnosis is essential so that purposeful intervention may follow. Unfortunately, three key problems plague the FASD field: 1) problems with diagnostic consistency, 2) lack of knowledge of FASD and appropriate interventions for FASD, and 3) public policy and funding initiatives that fail to effectively address the diverse needs of this population.

As with many disorders, current knowledge about FASD is tautological. Because there is no definitive test for FASD, researchers and clinicians diagnose FASD symptomatically and then look back to these abnormalities to better refine diagnosis. Consequently, our knowledge base continues to grow and evolve, and it can be difficult for practitioners to remain abreast of all developments. Thus, the purpose of this review is to recap the changes in the diagnosis of FASD over the last thirty years, provide clarity into the changing terminology, identify some of the unique difficulties confronting the diagnosis of FASD, and, finally, to describe the most current diagnostic model being used in FASD as well as best practices that can be applied in working with this population.

*The Early Years*

As with any disorder, definitional issues are at the heart of diagnosis. The features of what was to become known as Fetal Alcohol Syndrome (FAS) were originally described in 1968 by Lemoine, Haronsseau, Borteyru, and Menuet, who identified a consistent set of physical features in infants of mothers with alcoholism. Then in 1973, Jones and Smith, dysmorphologists at the University of Washington Medical School, identified a "similar pattern of craniofacial, limb, and cardiovascular defects associated with prenatal onset growth deficiency and developmental delay" in eight unrelated children of three ethnic groups born to alcoholic mothers (Jones, Smith, Ulleland, & Streissguth, 1973, p. 1267). The authors thought the prenatal alcohol exposure might be responsible for some of the functional abnormalities and joint malpositions comprising this syndrome. In a separate paper, Jones and Smith (1973) named this pattern or cluster of anomalies in children born to alcoholic mothers Fetal Alcohol Syndrome (FAS) and identified diagnostic criteria based on three features: 1) pre- and/or postnatal growth deficiency; 2) a distinct pattern of craniofacial malformations; and 3) central nervous system (CNS) dysfunction.

Diagnostic development did not move forward directly from this point. Instead, there was a resistance within the scientific and clinical community to the idea of alcohol as a teratogen (Randall, 2001), despite the clinical evidence. Consequently, the next step required providing the necessary evidence to confirm the teratogenicity of alcohol, and to better understand the effects of alcohol in utero. Animal studies in particular in this area allowed researchers to confirm that alcohol is, on its own, clearly a teratogen (Chernoff, 1977; Randall, 1987; Sulik, Johnson, & Webb, 1981) since potentially confounding effects, such as nutrition, other drug exposures, and postnatal rearing conditions, were controlled to help rule out alternative explanations. In fact, prenatal alcohol exposure has been demonstrated in many species to cause all four teratogenic endpoints (death, malformations, growth deficiency, and functional deficits) depending on the dose, timing, and conditions of exposure (Randall, Ekblad, & Anton, 1990; Schenker et al., 1990; West, 1986). As such, the teratogenicity of alcohol was firmly established.

As animal studies began to establish credibility for the conclusion that alcohol is a teratogen, researchers worked to better define the three

diagnostic criteria for FAS as first described in 1973. The first criteria, necessary for a diagnosis of FAS, are the facial features as this is the only component of FAS that cannot be explained by any other disorder. The anomalies that appear to create the FAS appearance are localized to the central facial region and form a sort of "T" (Institute of Medicine, 1996). While precise assessment of these characteristics through consistent interpretation of measurements has yet to be universally implemented, the issue has been addressed by researchers such as Astley and Clarren (1995, 1997), who have developed criteria for the facial phenotype of FASD, within which the diagnosis of FAS falls. Diagnosis using these criteria, however, becomes more difficult as the individual ages, since the facial malformations often become less pronounced or even disappear in adulthood (Conner, Streissguth, Sampson, Bookstein, & Barr, 1999). Moreover, while the "FAS face" helps to establish the presence of FASD, absence of the face does not rule out FASD (Mattson, Riley, Gramling, Delis, & Jones, 1998).

The second criterion, evidence of growth retardation, must be present in at least one of the following areas: low birth weight for gestational age, decelerating weight over time not due to nutrition, and disproportional low weight to height compared to medical norms (Institute of Medicine, 1996). However, as with the facial phenotype, this feature is not a necessary component since the unique FASD facial characteristics as well as brain dysfunction have been described in the absence of any growth deficiencies (Institute of Medicine, 1996).

The final criterion for a diagnosis of FAS is Central Nervous System (CNS) dysfunction. Research has revealed that there is no question that prenatal alcohol exposure affects the developing brain (Mattson & Riley, 1998). As early as 1973, at which time the first brain of an FAS infant was studied in an autopsy, clear structural abnormalities were observed by researchers (Jones & Smith, 1973). However, a consistent pattern of brain anomalies has yet to be confirmed, and some researchers have even questioned whether a specific pattern of impairment even exists (Clarren, 1986; Peiffer, Majewski, Fischbach, Bierich, & Volk, 1979). While current medical techniques (e.g., MRI) that allow for a more systematic measure of alcohol's teratogenicity have provided support for the possibility that some patterns of deficits may be present due to the varying levels of susceptibility to the effects of alcohol during development (e.g., Mattson et al., 1992; Riley et al., 1995; Roebuck,

Mattson, & Riley, 2002), diagnosis currently relies on identification of a pattern of abnormalities that make up the syndrome in conjunction with reports of alcohol use by the mother during pregnancy.

As knowledge about prenatal alcohol exposure began to grow, other diagnoses parallel to FAS emerged to account for effects that may have some but not all of the characteristics of FAS. Since the CNS develops during the entire pregnancy, as opposed to the craniofacial features that only develop between the nineteenth and twenty-second days of pregnancy, the teratogenic effects of alcohol can affect the CNS without being evident in the facial features. Thus researchers have suggested that children without the characteristic facial features of FAS, but who have been exposed to alcohol prenatally, may still have incurred damage to the brain that could be evident in their significant cognitive deficits. Mattson and colleague (1998) indicate that "children with histories of heavy prenatal alcohol exposure display neuropsychological deficits and, furthermore, that these deficits persist in the absence of the pattern of physical features associated with FAS" (p. 152). Both animal and human studies have revealed that hyperactivity, problems with response inhibition, attention deficits, poor habituation, poor coordination, and poor state regulation are associated with alcohol use during pregnancy (Mattson & Riley, 1998), and many of these deficits have also been found in children who do not necessarily meet the criteria for a diagnosis of FAS (Brown et al., 1991; Coles et al., 1997; Goldschmidt, Richardson, Stoffer, Geva, & Day, 1996; Jacobson, Jacobson, Sokol, Martier, & Ager, 1993). As a result, a variety of other diagnostic terms such as Fetal Alcohol Effects (FAE) and the more current Alcohol Related Neurodevelopmental Disorder (ARND), the term that has now replaced FAE, were introduced within the broad classification that came to be named Fetal Alcohol Spectrum Disorder, or FASD. In general, regardless of diagnostic label, accuracy and precision in diagnosis could not be assured because of the lack of diagnostic specificity (Astley & Clarren, 2000). Consequently, researchers and clinicians were forced to re-examine the original diagnostic criteria.

The Institute of Medicine's report on FAS (IOM; 1996) addressed the lack of diagnostic clarity by identifying five categories of alcohol-related disability drawn from the original three diagnostic criteria, but incorporating increased detail and specificity. Within the IOM framework, the categories rank characteristics as indicating level of

severity, in which diminishing physical indications were thought to coincide with fewer cognitive deficits, although behaviourally both disorders are marked by increased impulsivity, aggression, and social problems (Jacobson & Jacobson, 1999). This diagnostic approach has been described as a Gestalt approach to diagnosis as it entails a look at the whole clinical picture to form a judgment regarding whether the pattern that presents is consistent with a diagnosis of FAS or ARND.

#### *Problems with the Gestalt Approach*

The Gestalt approach, even after being enhanced by the IOM criteria, has still been plagued by criticism for failing to establish a means for providing valid and reliable diagnoses. While more detail was provided, problems remained. Two areas in particular emerged as problematic: 1) the multidisciplinary nature of FASD diagnosis, and 2) the complexity of FASD.

#### *A Multidisciplinary Diagnosis*

The first area of concern relates to the variety of disciplines (e.g., medical, psychological) that play a role in diagnosis. Yet, each of these disciplines currently lacks the full range of expertise required to provide a comprehensive diagnosis. For instance, the Institute of Medicine (1996) stated in their publication that while a trained clinician (such as a psychologist) may diagnose ARND for the purposes of screening and referral, the medical diagnosis of FAS must be left to dysmorphologists and clinical geneticists. This separation of the two presentations of prenatal alcohol exposure, while appropriate, mimics to some degree a division within the literature. While the medical researchers explore the medical abnormalities present in children prenatally exposed to alcohol, the psychological community is focusing their research on the neurological damage incurred through early alcohol exposure, and the subsequent cognitive and behavioural impairment. This division, while useful in research, creates some practical concerns for diagnosis and day-to-day work because the combination of both medical and psychological elements may not exist naturally in many current clinical settings. Consequently, FAS is vastly misdiagnosed (Codero, Floyd, Martin, Davis, & Hymbaugh, 1994). Contributing to this problem has been the lack of universal standards for diagnosis (O'Leary, 2004). While some areas offer training for physicians or other practitioners, and provide

guidelines for best practices, they are not required to receive this training and many remain uninformed on this issue. Similarly for psychologists, while training exists, no universal criteria for diagnosis have been accepted. As will be discussed shortly, some steps are being taken to rectify this absence of clearly defined standards but, to date, use of this new material is far from universal and remains unknown to many.

CNS deficits are at the center of understanding FASD. As a result, an interdisciplinary approach is warranted. Streissguth, Barr, Kogan, and Bookstein (1996) have identified the consequences of many of these deficits as Secondary Disabilities. In particular, they have reported that, of those diagnosed with FAS, over 90% have mental health problems, 60% have been expelled from school, 60% have been in trouble with the law, 50% have been or are in jail, 50% have engaged in inappropriate sexual activity, and 30% have alcohol or drug problems (Streissguth et al., 1996). Further support is provided in research by Fast, Conry, and Lock (1999) in which they found that children with FAS were disproportionately represented in the juvenile justice system. Clearly, the severity of these problems will require the involvement of many disciplines to effectively identify the needs of each child. But with so many professionals involved, an accurate diagnosis can be difficult to establish without effective communication networks and a comprehensive understanding of FASD by all parties involved. Thus, one key aspect of intervention needs to be the establishment of appropriately integrated structures to adequately identify and ultimately address the needs of these individuals. In addition, researchers have noted that a standardized interdisciplinary approach to early diagnosis is essential for more accurate monitoring of FASD and, consequently, more appropriate allocation of resources (Astley, 2004).

#### *A Complex Diagnosis*

The second area of difficulty involves the complexity in making a diagnosis. Given the diverse features present in FASD, in addition to often dubious maternal reports of alcohol consumption and lack of familiarity with the disorder (Ernhart et al., 1995), accurate diagnosis is frequently identified as a concern. Unfortunately, the CNS dysfunction associated with FAS, while more prevalent as a prenatal alcohol effect, has not been considered to be as specific or unique as the facial dysmorphology, thus making diagnosis more dependent on the Gestalt

approach. As noted earlier, this CNS dysfunction is neurological in origin and spans all areas of cognitive and behavioural functioning, resulting in the dysfunction presenting differently in different individuals depending on factors such as the timing or extent of alcohol exposure. The Institute of Medicine (1996) states that these problems include disordered motor development, diminished intellectual functioning, delayed and disordered speech and language development, problems in social perception, memory deficits, and deficits in response inhibition and attention. Consequently, knowledge of the entire spectrum of impairment is necessary. Even then, accurate diagnosis can be elusive since psychologists might still diagnose symptoms without truly understanding etiology.

While researchers and clinicians have studied FASD in an effort to identify a consistent pattern of deficits, instead what they have found for the most part in many areas is a lack of consistent deficits. As yet, no single type of CNS damage or pattern of dysfunction has been identified that characterizes all children who have been prenatally affected by alcohol (Streissguth, 1997). Research examining developmental outcomes has yielded variable results (Institute of Medicine, 1996) that could have many origins; different patterns of maternal alcohol consumption and different levels of individual susceptibility are just two possible factors. At the same time it may be more likely that general trends could be identified if all studies were identifying FASD using consistent diagnostic criteria. This is particularly true in the case of ARND where the more objective physical characteristics are absent. Thus, there are concerns that ARND is either being over- or under-diagnosed, which poses a considerable hurdle for researchers. Furthermore, the population varies within the research—some studies focus on FAS, some on ARND and some mix the groups. The Gestalt approach has had its drawbacks as identification is nonspecific and, with so much variability, misconceptions abound. While this is a reality for this disorder, it certainly seems that achieving increased consensus and clarity would not only assist with diagnosis and research but also with the vital interventions that follow.

*Best Practices and The Four Digit Code*

In 1997, Astley and Clarren introduced the 4-Digit Diagnostic Code for diagnosis within the broad classification of FASD. This new method was



created partly in response to the Institute of Medicine's recommendations that a more reliable and valid set of diagnostic definitions be adopted. Accordingly, this system is designed to determine the degree to which the facial dysmorphic features, growth retardation, and CNS involvement exist as a consequence of prenatal alcohol exposure, rather than defining severity. An added advantage to this system is that it uses a team approach that allows for a comprehensive review of function in all areas, thus ensuring communication between medical and psychological diagnosticians as well as other key mental health professionals. Briefly, this system documents the magnitude of expression of the four key components of the syndrome, specifically: 1) growth impairment, 2) the FAS facial phenotype, 3) evidence of brain damage, and 4) prenatal alcohol exposure, on separate four-point Likert Scales (Astley & Clarren, 2000). A rank of "1" on any scale means a finding within the normal range. A "4" on any scale indicates a finding that corresponds with accepted cases of FAS. A score of "2" or "3" specifically defines intermediate steps between typical and atypical presentation of FAS characteristics. These scales do not necessarily measure increasing severity, rather they are scales of greater confidence that FAS is present and, as such, a diagnosis of FAS requires ranks of 3 or 4 in all categories. There are, however, many other possible alcohol-related diagnoses provided depending on the code obtained, in which case there is much more room for consideration of the entire spectrum of this disorder. It is with this in mind that the most recent and comprehensive term to be used has been Fetal Alcohol Spectrum Disorders (FASD) as it allows for the continuum of deficits to be considered for discussion and research purposes. That said, categorical terms such as FAS and ARND, which establish artificial boundaries within the spectrum, are still required at this time because they provide clear diagnostic terms that permit access to funding and supports.

#### *Into the Future*

Diagnosis of FASD has evolved greatly over the last thirty years, from its original description in 1973 based on medical observation to its current form in which diagnostic criteria span the domains of medical, psychological, and educational functioning. Current thought reflects the knowledge that FASD is not a dichotomous condition. Rather its clinical features and even the history of alcohol exposure itself range along

separate continuums from normal to clearly impaired. As a result, clinicians and researchers have been challenged with the task of diagnostic consistency. Nonetheless, despite the variability within this population in terms of specific deficits, there is still a consistent pattern in the presentation of these deficits that includes increased social problems, aggression, inattention, and delinquency (Mattson & Riley, 2000). In addition, this impairment has been identified in many areas of functioning, regardless of whether or not the full criteria for FAS are met. In fact, it has been suggested by researchers that the facial dysmorphology characteristic of FAS is often absent and may even be of little importance in truly understanding the impact of alcohol on the brain (Chudley, Conry, Cook, Loock, & LeBlanc, 2005).

As well, we are beginning to understand that FASD can affect individuals in different ways. In other words, while many of the secondary disabilities, or consequences, of the deficits may be similar, the path to these disabilities may be varied. The apparent incongruity will likely continue to be a focus of research as we continue to seek a clear understanding of diagnoses of FASD, and subsequently implement interventions and apply appropriate policy. It seems likely that the diagnosis of FASD will never be as simple as a single test or characteristic. Instead, children and adults will need to be considered on an individual basis to establish their specific needs as reflected in the 4-Digit Code style of diagnosis. Researchers and clinicians will need to continue to hone the focus towards specific CNS deficits as this may, over time, reveal unique neuropsychological characteristics of these individuals, which will, in turn, increase diagnostic precision. However, in order to see this implemented, clinicians and researchers need to be informed about the existence of FASD as well as its importance in the diagnostic process, both for intervention and prevention purposes. As long as clinicians continue to make diagnoses or researchers continue to conduct studies based on outdated criteria, misdiagnosis will likely ensue.

While also increasing diagnostic accuracy, increasing awareness of FASD will allow for an expanded understanding of the unique deficits and appropriate interventions that may be implemented as a consequence. Moreover, with further elucidation of the deficits present, researchers and practitioners may be in a better position to evaluate the usefulness of existing interventions that are effective with other groups. Rasmussen

(2005) makes this point when she notes that “much can be learned if the intervention research with other populations can be applied to individuals with FASD” (p. 1365). One such program did just this when they incorporated FASD women into an existing community intervention model and consequently saw improved outcomes including decreased alcohol and drug use, increased use of contraceptives and medical and mental health care services, and stable housing (Grant et al., 2004). Researchers involved in this study noted that “by combining education with follow-up hands-on experience, we demystified the FASD disability for the providers, who were then able to deliver services appropriately tailored to the specific needs of FASD patients” (Grant et al., 2004, p. 507).

Finally, policymakers will need to recognize the extent of the resources required by children with FASD and their families, and provide the financial support necessary for the individualized intervention that is required. At this time the diagnosis of FAS has not been monitored consistently on a provincial or national basis, resulting in significant under-reporting and inadequate allocation of funds (Astley, 2004). Therefore, increased awareness and communication are necessary so that consistent practices can be implemented, evaluated and monitored. This is not a hopeless problem without solution but, rather, one that requires special consideration and attention to its unique and varied presentation. Researchers have revealed that high levels of support both externally and internally are required for families raising children with FASD (Brown, 2003), which will only be attained through increased knowledge and awareness of the unique needs of FASD within community agencies. Through implementation of consistent models of diagnosis and best practice models, early identification of this population will likely facilitate implementation of programs that will provide this increased level of awareness. This will hopefully result in the increased support necessary to promote optimal functioning for these individuals with FASD, and those involved in their care, at the earliest age possible.

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