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Neurobehavioural Profiles of Individuals with Fetal Alcohol Spectrum Disorders

Gemma Bernes, BA, Jessica O'Brien, PhD, Sarah N. Mattson, PhD

Center for Behavioral Teratology, San Diego State University, USA

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Introduction

Prenatal exposure to alcohol is the leading preventable cause of birth defects, developmental disorders, and intellectual disabilities in children.[1] The prevalence of fetal alcohol spectrum disorders (FASD) is estimated to range between 1.1 and 5.0% in the United States,[2] and has been identified in all racial and ethnic groups.[3] Children prenatally exposed to alcohol can suffer from serious cognitive deficits and behavioural problems as well as alcohol-related changes in brain structure. Heavy prenatal alcohol exposure is associated with decreased intellectual functioning and deficits in learning, memory, and executive functioning. Additionally, alcohol exposure is associated with problem behaviours including hyperactivity, impulsivity, poor socialization and communication skills, and the development of substance use problems.

The term "fetal alcohol syndrome" (FAS) was introduced in 1973[4] and is defined by three criteria: specific craniofacial features (short palpebral fissures, indistinct philtrum, and thin vermillion), pre- and/or postnatal growth deficits, and central nervous system (CNS) dysfunction.[5-7] Recent updates to the diagnostic criteria now recommend evidence of both abnormal structural CNS development

(head circumference ≤ 10th percentile, structural irregularities, and/or nonfebrile seizures) and functional CNS impairment (cognitive and/or behavioural).[5] While examination of the consensus between diagnostic criteria is at the forefront of current research,[8] it is clear that children with confirmed prenatal alcohol exposure may not meet all diagnostic criteria while still exhibiting significant neurobehavioural impairments and neuroanatomical abnormalities.[9-11] The non-diagnostic umbrella term fetal alcohol spectrum disorders has been adopted to capture the spectrum of consequences of alcohol exposure.[5]

Subject

The development of a neurobehavioural profile for FASD can aid in the identification of children affected by prenatal alcohol exposure that otherwise would be quite difficult to discern. In addition to improving the identification of affected individuals, a neurobehavioural profile also will aid in improving intervention tools and early treatment options, as well as increasing the accuracy of incidence rates of the disorder.

Problems

The identification of children with heavy prenatal alcohol exposure is impeded by the fact that along the continuum of FASD, only diagnoses on the severe end of the spectrum (i.e. FAS and partial FAS) are characterized by physical facial features.[5] The majority of alcohol-affected children lack some or all of these physical markers and are therefore more difficult to identify, especially if a clear history of alcohol exposure is unavailable. Despite the presence of significant neurobehavioural impairments, many children are missed (80.1%) or misdiagnosed (6.4%).[12] While research over the last four decades has documented diverse and significant neurobehavioural deficits in children with FASD,[13] it is not clear whether one or more profiles of neurocognitive function exist and if so, whether these profiles are specific to this population.

Research Context

In order to determine the deficits associated with heavy prenatal alcohol exposure, researchers typically compare the performance of children with FASD to non-exposed controls on a wide range of neuropsychological tasks. Additionally, in order to improve upon the specificity of the neurobehavioural profile, comparisons are made with other clinical groups of children who display an overlap in neuropsychological performance to alcohol-affected children.[14] The majority of these comparisons are focused on individuals with low IQ scores and those with attention-

deficit/hyperactivity disorder (ADHD).

Key Research Questions

Recent research has addressed whether children with FASD exhibit a unique neurobehavioural profile. The identification of a syndrome-specific profile would improve the diagnosis of children with FASD and inform interventions for all children affected by heavy prenatal alcohol exposure.

Recent Research Results

Summary of neurobehavioural deficits in children with heavy prenatal alcohol exposure. FASD is associated with a number of neurobehavioural impairments including lower overall intelligence and deficits in executive functioning, learning and memory, language, visual-spatial functioning, and attention.[13] Additionally, children with heavy prenatal alcohol exposure are at a high risk for developing maladaptive and problematic behaviours.[15,16] Children with and without the facial dysmorphia associated with FAS display similar deficits in many of these domains, as described below.

Overall intelligence. Compared to their non-exposed peers, children with heavy prenatal alcohol exposure have diminished intellectual functioning. The IQ scores for these individuals typically fall in the borderline to low average range, with reductions in both verbal and performance (nonverbal) IQ.[9] Such deficits are persistent and stable.[17-19] Among those with FASD, lower intellectual ability levels are reported for those on the severe end of the spectrum (i.e. FAS),[20] however, children with and without the facial dysmorphology display intellectual impairment.[9] In addition, FAS is thought to be the leading known cause of intellectual disability (i.e. IQ less than 70) in the United States, although the majority of children with FAS are not intellectually disabled.[1]

Executive function. Global executive functioning deficits are present across the spectrum of FASD.[21] Children with FASD display deficits on measures of verbal and nonverbal fluency,[22] problem solving and planning,[23] concept formation, and set-shifting.[24] Although deficits are also observed in working memory and response inhibition domains, results are inconsistent and further research is necessary.[25-27]

Learning and memory. Children with FASD display deficits in learning and memory, including deficits in the acquisition and recall of both verbal and non-verbal information.[28,29] These impairments are present even when compared to IQ-matched samples.[30] However, overall

memory function is complex and may not be globally affected by prenatal exposure to alcohol. For example, while learning and recall are impaired, retention of verbal (but not nonverbal) material appears to be spared in most studies.[29,31] Deficits are also found with spatial learning and memory, primarily using animal models.[32]

Language. Children with heavy prenatal alcohol exposure display deficits in various basic language skills including speech production,[33] phonological processing (e.g., pseudoword reading), articulation,[34] word ordering, and grammatical understanding.[35] Furthermore, children with FASD make more grammatical errors compared to controls; grammatical error rate measures could be a potential biomarker that can significantly aid in the identification of those with FASD.[36] Overall, receptive and expressive language skills are impaired in alcohol-exposed children, although expressive abilities may be affected to a greater degree, and both types of deficits may be secondary to diminished intellectual functioning.[37]

Visual-spatial function. Children with heavy prenatal alcohol exposure have also been shown to have a variety of visual-spatial deficits, although these are not as well studied. Deficits include problems with basic figure copying,[38] spatial learning,[18] spatial working memory,[23] spatial recall,[39] visual-spatial reasoning,[40] visual-perceptual matching (e.g., matching complex geometric shapes),[41] and sustained visual attention.[42]

Attention. Attention deficits are well documented in children with FASD, specifically in the establishing and sustaining aspects of attention.[43] Within the area of sustaining attention, increased omission errors,[42] decreased accuracy rates, and slower reaction times are observed.[44] Additionally, attending to visual information appears to be more severely impaired compared to auditory information.[42,44]

Behaviour problems and psychiatric disorders. Children prenatally exposed to alcohol are at a high risk for problem behaviours that can interfere with their home, school and social environments. This includes an increased risk for psychiatric disorders,[15,45,46] trouble with the law, alcohol and drug abuse, and other maladaptive behaviours.[16] Additionally, they are more likely than non-exposed children to be rated as hyperactive, impulsive, or delinquent,[11,47] and frequently meet the diagnostic criteria for ADHD.[15,46,48] Further, children prenatally exposed to alcohol exhibit poor adaptive skills and are less likely to live independently.[49,50]

Specificity of neurobehavioural deficits in children with heavy prenatal alcohol exposure. Studies comparing children with FASD to non-exposed children with ADHD or low IQ scores lend support for a specific neurobehavioural profile associated with prenatal alcohol exposure. On measures of executive functioning, both alcohol-exposed children and non-exposed children with ADHD demonstrate deficits on sorting tasks and letter vs. category fluency, but only the alcohol-exposed group display overall deficits on letter fluency and letter-number switching.[51] Other studies comparing children with FASD and ADHD demonstrate that alcohol-exposed children display greater difficulty on tasks of visual-spatial reasoning, problem solving, and encoding and shifting aspects of attention,[52] as well as weaker social cognition and facial emotion processing abilities.[53] Additionally, children with FASD display greater deficits in working memory, fluency, planning, and set-shifting compared to children with ADHD.[54] Both clinical groups have deficits in delayed recall of verbal information, but only children with FASD display deficits in the recognition of material, whereas children with ADHD are more impaired in the retention of learned material.[28] On measures of communication and socialization skills, children with FASD display an arrest in the development of adaptive abilities, whereas non-exposed children with ADHD are delayed in adaptive skills, as abilities tend to improve with age.[50] When comparing overall neuropsychological performance, children with alcohol exposure are more impaired compared to those with an ADHD diagnosis, however no differences are found between alcohol exposed children with and without ADHD.[55] Although the concurrent presence of FASD and ADHD does not appear to further affect cognitive functioning, the comorbidity does have an exacerbating effect on communication skills[56] and an increased rate of psychiatric disorders.[57] In summary, existing research suggests that children with FASD and children with ADHD have overlapping profiles of deficits, but with varying patterns and degrees of impairment, which are compounded by their comorbidity. Therefore, more research is needed to clarify the similarities and differences between these two diagnoses, and to further refine a neurobehavioural profile for FASD.

Children with FASD are similar to IQ-matched, non-exposed peers on measures of internalizing behaviour,[11] fine motor skills,[58] sustained attention, retention of verbal material, and expressive and receptive language skills.[36] However, children with FASD have more externalizing behaviour problems,[11] impaired adaptive skills,[59] and verbal learning deficits compared to IQ-matched controls.[58] Thus, common comorbidities, like low IQ and ADHD, do not entirely account for the neurobehavioural deficits reported in FASD, and other co-occurring factors need to be studied.

Several studies have demonstrated the potential usefulness of a neurobehavioral profile in the identification of children with FASD. Performance on various neurobehavioural measures were successful in distinguishing between children with prenatal alcohol exposure and non-exposed controls with overall classification accuracy rates ranging between 71.5% - 92%.[60-62] Furthermore, when differentiating between alcohol-exposed and ADHD children, the neurobehavioral profile had an overall classification accuracy rate of 73.9%.[61] Executive function and spatial processing measures,[60] as well as measures of attention[62] are particularly sensitive to the detection of prenatal alcohol exposure.

Research Gaps

The use of a neuropsychological profile in the aid of the diagnosis of FASD has been shown to be useful and increase success rates compared to relying on the presence of dysmorphological characteristics alone. Additional research is necessary to further define this neurobehavioural profile, as well as to continue to identify deficits in various areas including working memory, response inhibition, and visual-spatial function, as impairments in these areas are less well understood, as discussed above. Additionally, the identification of risk and resiliency factors is imperative in the development of preventative measures. Improving identification techniques will aid in earlier diagnosis, enhance targeted interventions, and ultimately lead toward better outcomes for affected individuals.[49]

Conclusions

Prenatal exposure to alcohol affects 1.1 to 5.0% of the population and leads to a spectrum of neurobehavioural consequences, including decreased overall intelligence and specific deficits in executive functioning, learning, memory, language, visual-spatial skills, and attention. Children with FASD display deficits in daily living skills and are at a higher risk for problem behaviours. These abnormalities have been documented in children both with and without the facial dysmorphology required for a diagnosis of FAS. Although there is some overlap in the deficits observed of non-alcohol exposed children with ADHD or low IQ, children with FASD display a specific pattern in areas including adaptive skills, problematic behaviours, and some neuropsychological domains. The potential clinical utility of a neurobehavioural profile in the aid of differential diagnosis has been shown, yet further research is necessary to continue to refine and increase classification accuracy.

Implications

The identification of a syndrome-specific profile would improve diagnosis of children with heavy prenatal exposure to alcohol, especially for the majority of children with FASD who do not display the facial dysmorphology. Additionally, further refinement of a neurobehavioural profile for FASD would inform treatments and interventions for children along the spectrum. The effects of alcohol on the developing central nervous system are permanent and irreversible. Treatments are currently symptom-based and are aimed at addressing the cognitive and behavioural consequences of prenatal exposure to alcohol. Current treatments include those targeted toward improving attention, self-regulation, social skills, and adaptive abilities.[63] Furthermore, given the array of cognitive impairments associated with prenatal alcohol exposure, many affected individuals experience trouble in academic and learning environments.[64] Interventions using modified teaching strategies that focus on improving language, literacy, and mathematic abilities have been successful.[65] Finally, parent-focused interventions that provide parents with effective parenting strategies not only show advancements for the child, but also reduce parent stress and improve upon the parent-child relationship.[63,65] Existing interventions have been shown to have a positive effect for children with FASD, thus greater clarity of the neurobehavioural profile exhibited by affected children could further direct clinicians in the development of rational treatments that are specific to the disorder.

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