



Neurobehavioural Profiles of Individuals with Fetal Alcohol Spectrum Disorders

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Topic

[Fetal alcohol spectrum disorders](#)

Introduction

Prenatal exposure to alcohol is the leading preventable cause of birth defects, developmental disorders and mental retardation in children.¹ The prevalence of fetal alcohol spectrum disorders (FASD) is estimated to be at least 9.1 per 1,000 live births,² or about 1% of the population, 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, deficits in learning, memory and executive functioning, and problem behaviours including hyperactivity, impulsivity, poor socialization and communication skills, and development of substance use problems.

The term “fetal alcohol syndrome” (FAS) was introduced in 1973⁴ and is defined by three criteria: central nervous system dysfunction, pre- and postnatal growth deficiencies, and specific craniofacial features (short *palpebral fissures*, indistinct *philtrum*, and thin *vermillion*).^{5,6} More recently, researchers have acknowledged that children with confirmed prenatal exposure to alcohol may not meet all diagnostic criteria while still exhibiting significant neurobehavioural impairments and neuroanatomical abnormalities.⁷ The non-diagnostic umbrella term fetal alcohol spectrum disorders has been adopted to capture the spectrum of consequences of alcohol exposure.⁸

Subject

Difficulty identifying children affected by prenatal alcohol exposure could be improved by the identification of a specific neurobehavioural profile within this population.

Problems

Identification of children with heavy prenatal alcohol exposure is hampered by the fact that along the continuum of FASD, only FAS is characterized by physical features. 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. While research over the last three decades has documented diverse and significant neurobehavioural deficits in children with FASD,^{7,9} 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 exposure to alcohol, researchers typically compare the performance of children with FASD to non-exposed controls on a wide range of neuropsychological tasks. Although few comparisons between children with FASD and other developmental disabilities exist, literature documenting similarities and differences between FASD and attention-deficit/hyperactivity disorder (ADHD) is emerging.

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 diagnosis of children with FASD and inform interventions for all children affected by heavy prenatal exposure to alcohol.

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 language, memory, visual-spatial skills, problem solving, cognitive flexibility and nonverbal learning.^{9,10} Additionally, children with heavy prenatal exposure to alcohol are at high risk for maladaptive and problem behaviours.^{11,12} Children with and without the facial dysmorphia associated with FAS display similar deficits in many of these domains, as described below.^{7,10}

Overall intelligence. Compared to their non-exposed peers, children with heavy prenatal alcohol exposure have diminished intellectual functioning. IQ scores typically fall in the borderline to low average range, with reductions in both verbal and performance IQ.¹³ Such deficits are persistent and stable.¹⁴⁻¹⁶ In addition, FAS is thought to be the leading cause of mental retardation in the United States although the majority of children with FAS are not mentally retarded.⁷

Learning and memory. Children with FASD have deficits in learning and memory, including deficits in the acquisition of both verbal and non-verbal information.^{17,18} 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.^{18,19}

Language. Children with heavy prenatal exposure to alcohol have been shown to have language deficits including speech production errors and phonological processing deficits (e.g., pseudoword reading).²⁰ Impairment in both expressive and receptive language has been reported^{10,20,21} although expressive abilities may be affected to a greater degree and both types of deficits may be secondary to diminished intellectual function.²²

Visual-spatial function. Children with heavy prenatal exposure to alcohol 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,²¹ spatial learning,¹⁵ spatial working memory,²³ spatial recall,²⁴ visual-spatial reasoning,²⁵ visual-perceptual matching (e.g., matching complex geometric shapes)²⁶ and sustained visual attention.²⁷

Executive function. Fetal alcohol spectrum disorders also are associated with deficits in executive functioning abilities such as problem solving, planning and flexibility in thinking. Children with FASD have been shown to have deficits on measures of mental manipulation of information,²⁸ conceptual set shifting,²⁹ concept formation,³⁰ rapid generation of verbal and nonverbal responses,^{31,32} and planning.²⁹ Results have been inconsistent regarding deficits on working memory and response inhibition.^{29,33}

Attention. Attention deficits are well established in children with FASD, with deficits documented in impulsivity,¹⁵ focused attention,³⁴ and commission and omission errors.³⁵ Impairments in sustained attention also have been consistently reported in children with FASD, with visual attention more impaired than auditory attention.^{27,34}

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. Children with FASD are at an increased risk for psychiatric disorders,³⁶ trouble with the law, alcohol and drug abuse, and other maladaptive behaviours.³⁷ Additionally, they are more likely than non-exposed children to be rated as hyperactive, impulsive or delinquent^{11,38} and frequently meet criteria for ADHD.^{39,40} Children prenatally exposed to alcohol exhibit poor adaptive skills and are less likely to live independently.^{12,41,42}

Specificity of neurobehavioural deficits in children with heavy prenatal alcohol exposure. The few studies comparing children with FASD to non-exposed children with ADHD and non-exposed children with low IQ scores lend support for a specific neurobehavioural profile associated with prenatal exposure to alcohol. 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.³² Other studies comparing children with FASD and ADHD demonstrate that alcohol-exposed children have greater difficulty on tasks of visual-spatial reasoning, problem solving, flexibility, and encoding and shift aspects of attention³³ as well as weaker social cognition and facial emotion processing ability.⁴³ On measures of communication and socialization skills, children with FASD display an arrest in development of adaptive ability, whereas non-exposed children with ADHD are delayed in adaptive skills and improve with age.¹² Thus, existing research suggests that children with FASD and children with ADHD have overlapping profiles of deficits and more research is needed to clarify the similarities and differences between these two populations. Compared to IQ-matched, non-alcohol-exposed peers, children with FASD have more externalizing behaviour problems,¹¹ impaired adaptive skills,⁴² and verbal learning deficits.⁴⁴ Children with FASD are similar to IQ-matched non-exposed peers on measures

of internalizing behaviour,¹¹ sustained attention and retention of verbal material.⁴³ Thus, common co-morbidities, 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.

Research Gaps

While a formidable literature exists documenting extensive deficits in children with heavy prenatal alcohol exposure, questions remain as to whether children with FASD display a distinct neurobehavioural profile that can be specifically attributed to prenatal alcohol exposure. The specificity of deficits in the domains of attention, executive function, visual-spatial abilities, and learning/memory require further investigation. Future research initiatives comparing children with FASD to non-exposed children with similar clinical features, including but not limited to ADHD and low IQ, may elucidate the unique behavioural phenotype of FASD.

Conclusions

Prenatal exposure to alcohol affects nearly 1 in every 100 children and leads to a spectrum of neurobehavioural consequences, including decreased overall intelligence and specific deficits in learning, memory, language, visual-spatial skills, executive functioning 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 dysmorphism required for a diagnosis of FAS. Few studies exist comparing children with FASD to children with similar neurobehavioural features. The extant research suggests that while there is some overlap in the profile of non-alcohol-exposed children with ADHD or low IQ, children with FASD have specific deficits in problem behaviours, adaptive skills and some neuropsychological domains.

Implications

The identification of a syndrome-specific profile would improve diagnosis of children with heavy prenatal exposure to alcohol, especially the majority of children with FASD who do not display facial dysmorphism, and would inform treatments and interventions for children along the spectrum of effects. 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 for FASD include behavioural, speech, occupational and physiological therapies, early intervention programs, and psychosocial and educational interventions.⁴⁵ Existing interventions are not necessarily specific to FASD, thus greater clarity of the neurobehavioural profile exhibited by affected children could direct clinicians in the development of rational treatments that are specific to the disorder. Successful treatments may incorporate approaches used for children with other developmental disorders, but ultimately must take into consideration the specific deficits associated with FASD.

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