Asking difficult questions about fetal alcohol spectrum disorder in the context of the child, the mother, and the systems in which they live



Sabrina H Y Eliason, Anton R Miller, W Ben Gibbard, Gurpreet Salh, Nancy Lanphear

Alcohol is a known teratogen and prenatal alcohol exposure remains a major ongoing public health concern. Fetal alcohol spectrum disorder has become the diagnosis for describing individuals who have been affected by prenatal alcohol exposure. In this Viewpoint, we raise major concerns about its continued use as a diagnostic term in how it perpetuates a misleading and outdated narrative about child development and maternal health. We argue that the term fetal alcohol spectrum disorder has contributed to a culture of racism and discrimination for many who are diagnosed with it. The term fetal alcohol spectrum disorder fails to capture the progress made in our collective understanding of neurodevelopment through advancements in the field of genetics and in understanding the effects of trauma and adversity. We call for urgent international collaborative action to review the use of it as a diagnostic term and, more broadly, to reconsider the practice of diagnosing disabilities as medical illnesses. We suggest that this practice fails to recognise that outcomes of functioning and participation in individuals are not only the results of health conditions, but are also the products of complex interactions and experiences of individuals within the families and societies in which they live.

Introduction

Alcohol is an established teratogen and prenatal alcohol exposure can have substantial and lifelong adverse effects on an individual's health, neurodevelopment, and functioning. ¹⁻³ Effects of prenatal alcohol exposure are a major ongoing public health concern globally. ⁴⁻⁵ Our concern is that the use of fetal alcohol spectrum disorder as a medical diagnostic entity has restricted underlying conceptual validity and its use is flawed in practice at the time of writing. From our perspectives, the harms of using a medical diagnostic term that identifies maternal alcohol use as the primary cause of a person's functional limitations outweigh the benefits.

This Viewpoint outlines a series of scenarios that we have regularly encountered through combined decades of clinical and academic experience as developmental paediatricians working with children and families living with fetal alcohol spectrum disorder. The child, family, caregiver, clinician, and service provider narratives included are combined experiences from many that we have encountered asking similarly themed questions in our clinical practices. We present these scenarios in three major contexts: the child, the mother, and the systems in which they live.

These scenarios highlight opportunities for progress, innovation, and improved health outcomes. Collaborative engagement from clinicians, researchers, policy makers, and people with disabilities and their care and support systems is needed across all areas of neurodevelopmental medicine, mental health, and maternal and child health to improve the practice of supporting individuals with neurodevelopmental differences. Fetal alcohol spectrum disorder as a diagnostic term requires urgent attention because it risks harm to those who receive it by perpetuating a misleading and outdated discourse of maternal and child health that fails to integrate the progress made

Key messages

- It is increasingly difficult to confirm prenatal alcohol exposure as a singular or primary cause of an individual's functional difficulties, especially in the context of what is known about the concurrence of adverse childhood experiences, genetics, and other health factors that can affect neurodevelopment
- Fetal alcohol spectrum disorder as a diagnostic term
 perpetuates racism and discrimination among
 marginalised groups that experience oppression within
 existing systems of health care, education, and social
 support, and might prevent the recognition of other
 factors contributing to a child's health or developmental
 presentations
- Fetal alcohol spectrum disorder-specific supports or funding that require a diagnosis can create barriers to care, especially when prenatal alcohol exposure cannot be confirmed
- Fetal alcohol spectrum disorder as a diagnostic term is due for revision in the context of evolving concepts of inclusion and acknowledgment that a person's health and functional outcome can be modified by personal, family, and societal factors
- Moving towards a non-categorical approach by use of a broader diagnostic term, such as neurodevelopmental disorder with qualifiers identifying areas of functioning affected might be a solution
- Ongoing documentation of prenatal alcohol exposure in confidential health records should continue to provide support for parents to minimise effects on future pregnancies and to continue to support research and policy decisions regarding the effects of prenatal alcohol exposure

Lancet Child Adolesc Health 2024; 8: 835–42

Published Online September 16, 2024 https://doi.org/10.1016/ S2352-4642(24)00188-3

Division of Developmental Pediatrics, Department of Pediatrics, University of Alberta, Edmonton, AB, Canada (S H Y Eliason MD): Glenrose Rehabilitation Hospital, Edmonton, AB. Canada (S H Y Eliason); Division of Developmental Pediatrics, Department of Pediatrics University of British Columbia Vancouver, BC, Canada (A R Miller MD, G Salh MD, N Lanphear MD); Sunny Hill Health Centre at BC Children's Hospital, Vancouver, BC, Canada (A R Miller, G Salh, N Lanphear); Section of **Developmental Pediatrics**, Department of Pediatrics, University of Calgary, Calgary, AB, Canada (W B Gibbard MD); Alberta Children's Hospital, Calgary, AB, Canada (W B Gibbard)

Correspondence to: Dr Sabrina H Y Eliason, Glenrose Rehabilitation Hospital, Edmonton, AB T5G OB7, Canada sabrina.eliason@ualberta.ca in the past several decades in our collective understanding of neurodevelopment, intersectionality, and public policy.

The context of the child

From a birth mother: "Unless you can prove with 100% certainty that prenatal alcohol exposure is what caused my child's impairments, I will not accept this diagnosis. Can you even prove it? What impact does my trauma history have on my child's development? Will this diagnosis impact what treatment you'll provide my child?"

Determining causality

Correctly attributing a clinical presentation of neurodevelopmental differences solely to prenatal alcohol exposure is problematic. Many adverse prenatal and postnatal exposures are present in the context of prenatal alcohol exposure⁷ and not accounting for these exposures both compromises diagnostic validity and accuracy and neglects the findings of contemporary clinical research.8-11 The diagnostic methodology in fetal alcohol spectrum disorder as a clinical diagnosis is also inherently flawed. It is not acceptable to take a known risk factor from population health and assume it to be a causal factor in a specific individual.^{9,12} The effects of prenatal alcohol exposure on neurodevelopment can be influenced by the timing of prenatal exposure, dose, and duration, as well as by genetic factors in the birthing parent and infant that can alter the susceptibility of developing neurodevelopmental differences as a result of prenatal alcohol exposure.13

Among children diagnosed with fetal alcohol spectrum disorder, an estimated 13-14% also have chromosomal deletions and duplications that might be an additional or better explanation for neurodevelopmental differences.14,15 Although many diagnostic guidelines for fetal alcohol spectrum disorder suggest ruling out genetics and other causes of a child's presentation before confirming fetal alcohol spectrum disorder, explicit and routine recommendation for genetic investigations-such as a microarray—is not the standard recommendation to date in many diagnostic guidelines.16-21 This practice increases the risk of misattributing prenatal alcohol exposure as the sole causal factor of neurodevelopmental differences in some individuals where genetic factors might also be present. It also increases the risk of underdiagnosing genetic disorders in those affected by prenatal alcohol exposure by attributing all health and neurodevelopmental differences to prenatal alcohol exposure.

Adversity and neurodevelopment

Fetal alcohol spectrum disorder is disproportionately diagnosed in children from marginalised and oppressed living situations and has been identified as being more prevalent in children from foster and adoptive homes, orphanages, and institutions.^{22–25} Children living in these settings often have adverse childhood experiences, such as poverty, neglect, and maltreatment, which can also

have negative neurodevelopmental consequences independently of prenatal alcohol exposure. ^{26–28} Childhood adversity, maternal adversity, and poor maternal nutrition during pregnancy can also have implications for epigenetic changes and lasting effects on health. ³³

Adverse childhood experiences alone are associated with neurodevelopmental and mental health conditions, such as learning difficulties, sleep disturbances, deficits in cognitive and executive functioning, memory difficulties, anxiety, sensory processing differences, attention problems, substance use disorders, and depression.^{29,31} As a result, assumed characteristics of fetal alcohol spectrum disorder might be misattributed solely to prenatal alcohol exposure, rather than from the cumulative effects of genetics and other prenatal or postnatal adverse exposures. It could be argued that a diagnosis, such as fetal alcohol spectrum disorder, might not have been accepted from its inception if the evidence that exists to date about the cumulative effects of genetics, trauma, and adversity had existed 50 years ago.

Evidence for fetal alcohol spectrum disorder-specific interventions

Identifying a specific cause of a child's functional differences often has little to no practical implications for the individual's management or care plans. A child's functional presentation is a better predictor of their need for services and outcomes, rather than their specific diagnosis. We suggest that the substantial time and resource-intensive efforts needed to confirm prenatal alcohol exposure as the primary cause of a child's neurodevelopmental presentation outweighs potential therapeutic benefits.

There is, however, value in asking about prenatal alcohol exposure for the surveillance of children who might have an increased possibility of neurodevelopmental differences, to support early recognition and access to support. If a parent is available to discuss the history of prenatal alcohol exposure, these conversations can be opportunities to mitigate risks for future pregnancies. Asking about prenatal alcohol exposure is similar in importance to asking questions about the presence or absence of medications taken during pregnancy, gestational age at birth, and other complications of the pregnancy and delivery to understand factors and contexts that could affect the presentation of the child.

In clinical practice, confirming prenatal alcohol exposure as the primary cause of a child's developmental presentation is difficult, if not impossible, because of the co-occurrence of other prenatal and postnatal risk factors, medical factors, and family and social factors that can affect brain development. We have two hypothetical clinical scenarios, informed by our clinical experiences, to illustrate this.

In the first scenario, a school-aged child aged 8–12 years presented for a fetal alcohol spectrum disorder assessment with substantial challenges in attention, affect

regulation, executive functioning, academics, and communication. Their history included birth at 29 weeks gestational age, poorly controlled gestational diabetes, poor maternal nutrition, inconsistent access to a healthcare provider, inter-partner violence during pregnancy, and prenatal alcohol exposure. The neonatal period was complicated by prolonged requirement of mechanical ventilation, prolonged sedation, and recurrent episodes of hypoxia. The child spent the first 2.5 months of life in the neonatal intensive care unit. The health history of both parents included challenges with academics, attention regulation, affect regulation, communication, and executive functioning. Even with prenatal alcohol exposure confirmed, it is clinically impractical to distinguish which factor is the primary cause of the child's challenges. Furthermore, whether the cause of the child's functional challenges is prenatal alcohol exposure, rather than any other aspect of their history, should not affect which supports would be beneficial.

In a second scenario, a child was born at term with an uncomplicated birth history, aside from little prenatal care and a confirmed prenatal alcohol exposure history. After birth, the child was in the care of a parent who had difficulty providing a safe and secure attachment relationship due to their own trauma history, unstable health, no family supports, unstable housing, and low income. The birthing parent had a history of school noncompletion, anxiety, depression, cognitive challenges, and executive functioning difficulties. In the child's early life, they had unstable housing, food insecurity, substantial emotional and physical abuse, and systemic barriers restricting access to health-care services, mental health care, and educational supports. The child also had a history of recurrent ear infections and conductive hearing loss that was not identified until the fetal alcohol spectrum disorder assessment. The child had never been to a dentist or optometrist for routine surveillance or screening. In this scenario, it is again difficult to confirm prenatal alcohol exposure as the primary cause of a child's developmental and behavioural presentations.

When considering both scenarios, it is important to highlight that as developmental paediatricians, we as the authors frequently encounter children with similar functional presentations who do not have prenatal alcohol exposure. These scenarios illustrate how a focus on prenatal alcohol exposure and its over-attribution as the primary cause of a child's challenges can distract from relevant and potentially modifiable health and public policy factors that could also contribute to, or more positively affect, a child's functional presentation.

Many specific and informed approaches to fetal alcohol spectrum disorder highlight the importance of trauma-informed care, the need for individualised accommodations for functional differences, and awareness of the effects of social factors on health outcomes.³² In addition, interventions studied in individuals with

fetal alcohol spectrum disorder are also relevant to other neurodevelopmental presentations and highlight that these treatments are not specific to fetal alcohol spectrum disorder alone. Overall, evidence from high-quality research to support specific interventions for fetal alcohol spectrum disorder continues to be scarce.33-35 Access to trauma-informed and culturally safe health care that provides support by meeting the individual needs of children and their families within the context of their histories and communities should be a standard of care for all children, not only children with a history of prenatal alcohol exposure. The types and intensity of support for an individual with prenatal alcohol exposure or any other neurodevelopmental difference are likely to change over the lifespan, whereas a diagnosis might not. Adjusting supports on the basis of functional needs, rather than on a diagnosis alone, could allow for a more individualised approach of providing support over the lifespan.

The context of the mother

From birth mothers: "When I tell people that my child has fetal alcohol spectrum disorder I feel like I am disclosing my personal and confidential history to strangers. Not everyone at our school needs to know my story. I'm trying to move on from my past, but now I also have the knowledge that I caused my child's condition, and this will follow us both for life. Why do I have to re-live my past to get the help my child needs?" From adoptive parents: "I feel like I must disclose that I'm an adoptive parent because otherwise teachers ask questions and I feel judged for things I didn't do. It also makes me worry about whether this diagnosis will impact how my child sees their birth mother."

Pregnancy, fetal health, infant health, and prenatal alcohol exposure have traditionally been considered the responsibilities of the mother. This view absolves the role of partners, communities, social policies, health systems, and society from supporting the mother throughout pregnancy and places the determination of eligibility of a child for a fetal alcohol spectrum disorder diagnosis on them. Fetal alcohol spectrum disorder clinics face additional barriers to diagnosis when mothers are missing, deceased, or unable to confirm prenatal alcohol exposure.

Lyall and colleagues³⁷ identified five major themes that affect a mother's ability to modify alcohol use during pregnancy: social relationships and norms, stigma, trauma and other stressors, alcohol information and social messaging, and access to trusted equitable care and essential resources. Studies often focus on alcohol use as an individual's choice and do not consider the effects of structural or systemic factors on prenatal alcohol use. Lyall and colleagues highlight that a woman's social identity in terms of race, socioeconomic status, and identity as someone with a disability influences their relationship with alcohol

and access to help through systems of care. These factors cumulatively contribute to the circumstances for prenatal alcohol use.

Furthermore, McGuire and colleagues³⁸ outlined the web of risk factors and conditions that can contribute to the occurrence of prenatal alcohol exposure, such as nutrition, maternal socioeconomic status, maternal age, marital status, other substance use, religion, parity, pregnancy complications, antenatal care, access to contraception, prenatal stress, maternal mental health, intimate partner violence, alcohol use of partners and family, and genetics, all of which affect presentations of fetal alcohol spectrum disorder. This degree of complexity is aligned with previous research that identified the multidimensional context that contributes to fetal alcohol spectrum disorder.³⁹

Use of the term fetal alcohol spectrum disorder as a public health prevention tool has limitations because the diagnostic label does not capture the full scope of causal factors that also require attention for prevention to occur. The alcohol part of fetal alcohol spectrum disorder is only part of the story, yet it remains the primary focus of diagnosis and prevention strategies. This practice continues to contribute to blame, shame, and stigma of individuals, despite causal factors that are often systemic.

In the context of health, education, and social sectors within systems

From service providers, such as teachers, community service providers, or policy makers: "In our publicly funded system, diagnoses are required to access supports. I worry that if we do not have a fetal alcohol spectrum disorder diagnosis we will lose access to funding and intervention supports that children can only access with a diagnosis. I don't really care what they call it if the child gets what they need." From clinicians: "Why are marginalised Indigenous children living in the care of children's services seen disproportionately in our fetal alcohol spectrum disorder clinic?" Two major systemic factors stand out as barriers to supporting children and families affected by prenatal alcohol exposure: racism and the use of categorical developmental diagnoses to describe variations in functioning.

Racism

Higher rates of alcohol use during pregnancy increase the odds of giving birth to a child with differences in brain development, regardless of race.⁴⁰ This is consistent with data from the southwestern USA that suggests there is no significant difference in the prevalence of fetal alcohol spectrum disorder by race, ethnicity, or socioeconomic status.⁴¹ In fact, White women are reportedly more likely to drink before and during pregnancy than Black, African American, or Hispanic women, but they are less likely than other groups to have a child identified

with birth-related outcomes related to their alcohol consumption. 42,43

May and colleagues" suggested that factors such as BMI, lifelong nutrition, and nutrition during pregnancy could have substantial effects on the relative risk of a child developing fetal alcohol spectrum disorder and that this risk is further influenced by environmental, community, and family factors that vary between individuals. These differential health outcomes should be considered in the context of systemic and structural racism and other social and systemic factors that can influence health outcomes.⁴⁵ Examples of these social and systemic factors could include, but are not limited to, unstable housing or homelessness, poverty, food insecurity, little access to child care, growing up in a household with domestic violence, little access to a primary care provider.

In Canada, there is an over-representation of fetal alcohol spectrum disorder research in Indigenous children and prevalence studies have historically suggested that Indigenous populations have a higher occurrence of fetal alcohol spectrum disorder than other populations. However, there is also less research of other neurodevelopmental conditions that occur in childhood within Indigenous groups, such as autism and cerebral palsy. These differences can contribute to bias in how resources are allocated within Indigenous communities, further clinical referral biases, and promote under-recognition of other non-prenatal alcohol exposure health and neurodevelopmental differences in Indigenous children, which can have implications on health and developmental outcomes.

We have encountered situations in which Indigenous children with intellectual developmental disorder have been referred for fetal alcohol spectrum disorder assessment without being offered genetic testing to investigate other causes of their neurodevelopmental differences. Offering a chromosomal microarray is a standard of care for children with intellectual developmental disorder and global developmental delay in Canada.²⁰ In the combined experience of the authors, when a chromosomal microarray has been completed, pathogenic genetic variances have sometimes been able to explain the child's physical and neurodevelopmental presentation in addition to, and sometimes better than, their history of prenatal alcohol exposure. Evolving evidence would support the clinical utility of also offering exome sequencing for children with global developmental delay or intellectual developmental disorder, although this testing is not readily available without referral to geneticists at our sites currently.48 In the meantime, we have offered referral to genetics for further investigation of causes of global developmental delays, even when prenatal alcohol exposure is present. Referral bias that assumes an increased likelihood that prenatal alcohol exposure is a cause of neurodevelopmental differences in Indigenous children could contribute to an increased risk of harm from undiagnosed genetic disorders.

There is also the potential of underdiagnosis of autism or other neurodevelopmental diagnoses by fetal alcohol spectrum disorder assessment teams. In Canada, these teams are generally not trained to complete autism assessments and diagnoses, unless they are embedded in developmental or health centres with other expertise. Furthermore, in jurisdictions where there might be disproportionately more social acceptance of specific funding for autism, under-recognition of autism in Indigenous populations could cumulatively contribute to an inequity of access to supports and acceptance as well as increased stigma and discrimination for Indigenous children with neurodevelopmental differences.

We have observed that in some communities with high Indigenous representation, multidisciplinary developmental assessment teams and comprehensive health or educational supports are designated for people with suspected or confirmed fetal alcohol spectrum disorder and there are fewer supports for individuals with other neurodevelopmental differences. In these situations, if a child with neurodevelopmental differences does not have a confirmed prenatal alcohol exposure history, they might not have access to the same quality of assessment or supports. Increasing fetal alcohol spectrum disorderspecific diagnostic assessment services and supports in Indigenous populations without also increasing access to more broad neurodevelopmental assessments and supports risks restricting access to assessment and support for Indigenous children without prenatal alcohol exposure. Instead of diagnosis-specific approaches, using non-categorical approaches to assess and support functioning and participation for all children could provide a more equitable and responsive way forward.

Categorical approaches

The practice of diagnosing mental health conditions or neurodevelopmental disabilities dates to the 1980s.⁴⁹ However, the diagnostic categories outlined in classification systems, such as the Diagnostic and Statistical Manual of Mental Disorders, are not distinct categories. Individuals grouped within these diagnoses are heterogeneous in both genotype and phenotype.⁵⁰ Fetal alcohol spectrum disorder has evolved in parallel to this process, although there are many similar considerations. As clinicians and researchers, we must ask whether the diagnosis of fetal alcohol spectrum disorder has served to reduce the prevalence of the condition, or increase public awareness or acceptance of it.

Using categorical diagnostic terms, such as fetal alcohol spectrum disorder, to establish eligibility for supports and services is problematic because children with the same diagnoses can have different functional needs. Non-categorical approaches to neurodevelopmental conditions, such as the WHO International Classification of Functioning, Disability, and Health consider individual

factors of the child within the context of their living situation and are evolving to become preferred approaches in developmental paediatrics.^{31,51}

Developing a culture that supports children for their areas of functioning and goals for participation, rather than for their diagnosis, could increase accessibility of supports by allowing clinicians and therapists to directly assess for and support the functional needs of individuals, without having to wait for a physician to confirm a diagnosis. This approach might also increase accessibility to supports across the lifespan and allow for supports to be adjusted, as different environmental supports or adaptations are required in different contexts over time as the individual's goals evolve.⁵²

The creation of non-categorical approaches could include a broad diagnosis, such as a neurodevelopmental disorder, with secondary and modifiable specifiers highlighting areas of the child's functioning that benefit from support. An example would be neurodevelopmental disorder benefiting from support for attention regulation, motor skills, and communication. In this example, the neurodevelopmental disorder would be the diagnosis and the areas benefiting from support could be specifiers that change over the course of the individual's lifespan. The option of adding a specifier for the cause of the condition could be considered, although the benefits of this—eg, from a research or population and public health perspective—must be weighed against the potential individual and societal harms as outlined above.

Removal of all neurodevelopmental diagnoses and a shift towards functional descriptions could be a future strategy. However, to date, many systems and policies are rooted in biomedical models of diagnoses for the provision of supports. We warn that at the time of writing this, the complete removal of diagnoses aimed to identify and validate the lived experiences of people with neurodevelopmental differences could cause more harms than benefit. This could remain true until there is an international and collaborative commitment across all existing neurodevelopmental diagnoses to evolve towards systems based on the universal acceptance of equity across the full spectrum of diverse presentations of functioning. Before this happens, if the diagnosis of fetal alcohol spectrum disorder is removed completely and not replaced, any or all supports and resources for individuals affected by prenatal alcohol exposure could be put at risk. Discussions about changes to diagnostic approaches must anticipate the effects of any proposed changes on individuals and systems and seek to mitigate negative effects. A suggestion would be to continue to encourage the universal practice of asking about prenatal alcohol exposure prenatally and during birth histories and to encourage the continued documentation of prenatal alcohol exposure confidentially in health records. These steps should be for the purpose of potential risk-management strategies to support parents, ongoing research, and policy-making decisions. Identification of prenatal alcohol exposure should not be

Search strategy and selection criteria

References for this Viewpoint were identified through searches of PubMed with the search terms "FASD", "Neurodevelopmental Disabilities," "disability", "inclusion", "stigma", "functioning", "prenatal alcohol exposure", "genetics", "trauma", "adversity", and "Indigenous" from Jan 1, 1990, to April 1, 2024. Articles were also identified through searches of the author's own files. Only full-text articles and abstracts in English were reviewed. The final reference list was generated on the basis of relevance to the scope of this Viewpoint.

a requirement for determining eligibility for assessment or supports.

A diagnosis implying prenatal alcohol exposure as a primary cause of functional challenges could be considered if a child presents with no other diagnosis that captures their substantial functional difficulties, if prenatal alcohol exposure is substantial, if no other major contributing factors are likely, and only after genetic testing-such as a microarray and potentially exome sequencing—is completed to rule out other contributing genetic factors. If prenatal alcohol exposure is not confirmed directly by the parent or through readily available birth records, until further evidence is available we would suggest no further investigation or resource allocation into confirming the presence or absence of prenatal alcohol exposure until it is warranted. The presence of a child's substantial functional difficulties should be sufficient evidence to provide further support and developmental assessment, regardless of the cause. Collective reflection is needed to reconsider the social and ethical implications of a lifelong disability diagnosis that hinges on birthing parents disclosing an otherwise confidential, sensitive, and sometimes traumatic portion of their own history.

Opportunities

International attention is urgently needed to address the harms of using fetal alcohol spectrum disorder as a diagnostic term. Fetal alcohol spectrum disorder implies that prenatal alcohol exposure is the known and primary cause of a person's disability, which is an implication we argue is no longer tenable for most individuals. Furthermore, as a diagnosis it perpetuates stigma and outdated narratives of pregnancy, infant health, and child health as the sole responsibility of the mother, rather than a shared social responsibility with their partners, communities, and society.

We strongly recommend that the term fetal alcohol spectrum disorder be revised to consider the complex interactions and cumulative factors that contribute to neurodevelopment. Any future terminology or assessment practices should strongly consider the effects of these factors on the experiences of individuals living

with the condition. There should be deliberation on who bears the cost of any future terminology and who benefits. Decisions of whether a child receives supports for functional challenges should not hinge on the requirement of a diagnosis, especially one that requires disclosure of sensitive and potentially traumatic events in the lives of their birthing parent.

This discussion on fetal alcohol spectrum disorder highlights the need to realign our collective understanding of neurodevelopment as a continuum and draw attention to the barriers that arise within existing clinical practice of assigning categorical diagnoses to differentiate ability and disability to portions of this continuum. A more person-centred, neurodiversity-affirming strategy could be to develop common terminology to describe individuals' function and required support. Preferences and priorities of individuals with a history of prenatal alcohol exposure, biological parents (especially mothers), and caregiving families must be central to informing diagnostic terminology and any changes to or discontinuation of fetal alcohol spectrum disorder as a diagnostic term.

Conclusion

A child's functioning is not static or predetermined by their biomedical conditions. Outcomes of functioning and health are influenced by factors outside of the child, such as their family, community, and the social and political systems in which they live. The difficulties with using fetal alcohol spectrum disorder as a diagnostic term are embedded in how systems of medical diagnosis, intervention, and societal approaches focus on prenatal alcohol exposure as a summative explanatory framework. This approach has become restrictive. Changing diagnostic and intervention approaches will be difficult, but if change can be achieved through broad discussion and consensus then much needed advancements in medicine, research. equity, and health policies are possible. Asking difficult questions about issues, such as fetal alcohol spectrum disorder, are necessary steps towards providing more equitable medical services and improving health and developmental outcomes for all children.

Contributors

SHYE completed writing the original drafts and major revisions, with sub-sections written by ARM, WBG, and GS. All authors contributed to the conceptualisation of the content of the Viewpoint and the reviewing and editing of the manuscript. All authors had full access to the references and data included in this manuscript and accept responsibility for the submission of this publication.

Declaration of interests

ARM has a service contract with the Provincial Health Services Authority of British Columbia to attend conferences and receives support from Sunny Hill Foundation for academic activities. WBG has a contract with Elsevier to edit a book and write a chapter regarding the harms of assessment and diagnosis for neurodevelopmental disorders. He received payment for this work. All other authors declare no competing interests.

${\bf Acknowledgments}$

As the authors of this Viewpoint, we recognise that we write from a position of privilege as developmental paediatricians practicing in academic, urban centres, and as individuals who have not had lived

experience of fetal alcohol spectrum disorder. We also recognise that we have written from settler perspectives about Indigenous issues, oppression, and racism. We thank the many children, families, and caregivers who have shared their lived experience with us through our clinical work over our combined careers. We have prioritised a commitment to representing their experiences cumulatively and honestly throughout this Viewpoint.

References

- Jones KL, Smith DW. Recognition of the fetal alcohol syndrome in early infancy. Lancet 1973; 302: 999–1001.
- Clarren SK, Smith DW. The fetal alcohol syndrome. N Engl J Med 1978; 298: 1063–67.
- 3 Little RE, Streissguth AP. Effects of alcohol on the fetus: impact and prevention. Can Med Assoc J 1981; 125: 159–64.
- 4 Popova S, Lange S, Probst C, Gmel G, Rehm J. Global prevalence of alcohol use and binge drinking during pregnancy, and fetal alcohol spectrum disorder. *Biochem Cell Biol* 2018; 96: 237–40.
- 5 Lange S, Probst C, Gmel G, Rehm J, Burd L, Popova S. Global prevalence of fetal alcohol spectrum disorder among children and youth: a systematic review and meta-analysis. *JAMA Pediatr* 2017; 171: 948–56.
- 6 Racine E, Bell E, Di Pietro NC, Wade L, Illes J. Evidence-based neuroethics for neurodevelopmental disorders. Semin Pediatr Neurol 2011: 18: 21–25.
- 7 Streissguth AP, Bookstein FL, Barr HM, Sampson PD, O'Malley K, Young JK. Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. J Dev Behav Pediatr 2004; 25: 228–38.
- 8 Astley SJ. Profile of the first 1,400 patients receiving diagnostic evaluations for fetal alcohol spectrum disorder at the Washington State Fetal Alcohol Syndrome Diagnostic & Prevention Network. Can J Clin Pharmacol 2010; 17: e132–64.
- 9 Price A, Cook PA, Norgate S, Mukherjee R. Prenatal alcohol exposure and traumatic childhood experiences: a systematic review. *Neurosci Biobehav Rev* 2017; 80: 89–98.
- Lebel CA, McMorris CA, Kar P, et al. Characterizing adverse prenatal and postnatal experiences in children. Birth Defects Res 2019; 111: 848–58.
- 11 Flannigan K, Kapasi A, Pei J, Murdoch I, Andrew G, Rasmussen C. Characterizing adverse childhood experiences among children and adolescents with prenatal alcohol exposure and fetal alcohol spectrum disorder. *Child Abuse Negl* 2021; 112: 104888.
- Miller AR. Diagnostic nomenclature for foetal alcohol spectrum disorders: the continuing challenge of causality. *Child Care Health Dev* 2013; 39: 810–15.
- 13 Sambo D, Goldman D. Genetic influences on fetal alcohol spectrum disorder. Genes. 2023; 14: 195.
- 14 Zarrei M, Hicks GG, Reynolds JN, et al. Copy number variation in fetal alcohol spectrum disorder. Biochem Cell Biol 2018; 96: 161–66.
- 15 Jamuar SS, Picker JD, Stoler JM. Utility of genetic testing in fetal alcohol spectrum disorder. J Pediatr 2018; 196: 270–74.
- 16 Bower C, Elliott EJ 2016, on behalf of the Steering Group. Report to the Australian Government Department of Health: Australian guide to the diagnosis of FASD. February, 2020. https://www.fasdhub.org. au/siteassets/pdfs/australian-guide-to-diagnosis-of-fasd_allappendices.pdf (accessed Aug 2, 2024).
- 17 Cook JL, Green CR, Lilley CM, et al. Fetal alcohol spectrum disorder: a guideline for diagnosis across the lifespan. CMAJ 2016; 188: 191–97.
- Scottish Intercollegiate Guidelines Network. SIGN 156: children and young people exposed prenatally to alcohol: a national clinical guideline. Scottish Intercollegiate Guidelines Network. 2019. https://www.sign.ac.uk/media/1092/sign156.pdf (accessed July 7, 2024).
- Hoyme HE, Kalberg WO, Elliott AJ, et al. Updated clinical guidelines for diagnosing fetal alcohol spectrum disorders. *Pediatrics* 2016; 138: e20154256.
- 20 Carter MT, Srour M, Au PB, et al. Genetic and metabolic investigations for neurodevelopmental disorders: position statement of the Canadian College of Medical Geneticists (CCMG). J Med Genet 2023: 60: 523–32
- 21 Srivastava S, Love-Nichols JA, Dies KA, et al. Meta-analysis and multidisciplinary consensus statement: exome sequencing is a firsttier clinical diagnostic test for individuals with neurodevelopmental disorders. Genet Med 2019; 21: 2413–21.

- 22 Abel EL, Hannigan JH. Maternal risk factors in fetal alcohol syndrome: provocative and permissive influences. *Neurotoxicol Teratol* 1995; 17: 445–62.
- 23 Kambeitz C, Klug MG, Greenmyer J, Popova S, Burd L. Association of adverse childhood experiences and neurodevelopmental disorders in people with fetal alcohol spectrum disorders (FASD) and non-FASD controls. BMC Pediatr 2019; 19: 498.
- 24 Popova S, Lange S, Shield K, Burd L, Rehm J. Prevalence of fetal alcohol spectrum disorder among special subpopulations: a systematic review and meta-analysis. *Addiction* 2019; 114: 1150–72.
- 25 Bright MA, Thompson LA. Association of adverse childhood experiences with co-occurring health conditions in early childhood. *J Dev Behav Pediatr* 2018; 39: 37–45.
- 26 Luby JL, Barch D, Whalen D, Tillman R, Belden A. Association between early life adversity and risk for poor emotional and physical health in adolescence: a putative mechanistic neurodevelopmental pathway. JAMA Pediatr 2017; 171: 1168–75.
- 27 Su Y, D'Arcy C, Yuan S, Meng X. How does childhood maltreatment influence ensuing cognitive functioning among people with the exposure of childhood maltreatment? A systematic review of prospective cohort studies. J Affect Disord 2019; 252: 278–93.
- 28 Park M, Kobor MS. The potential of social epigenetics for child health policy. Can Public Policy 2015; 41 (suppl 2): S89–96.
- 29 Zarei K, Xu G, Zimmerman B, Giannotti M, Strathearn L. Adverse childhood experiences predict common neurodevelopmental and behavioral health conditions among US children. *Children* 2021; 8: 761.
- 30 Gregorowski C, Seedat S. Addressing childhood trauma in a developmental context. *J Child Adolesc Ment Health* 2013; 25: 105–18.
- 31 Miller AR, Gardiner E, Rosenbuam PL. A non-categorical approach to childhood neurodisability: concepts, evidence, and implications for clinical practice, organization of services, teaching, and research. In: Eisenstat DD, Goldowitz E, Oberlander TF, Yager JY, eds. Neurodevelopmental pediatrics: genetic and environmental influences. Cham: Springer Nature, 2023: 685–95.
- 32 Rutman D. Becoming FASD informed: strengthening practice and programs working with women with FASD. Subst Abuse 2016; 10 (suppl 1): 13–20.
- 33 Reid N, Dawe S, Shelton D, et al. Systematic review of fetal alcohol spectrum disorder interventions across the life span. Alcohol Clin Exp Res 2015; 39: 2283–95.
- 34 Ordenewitz LK, Weinmann T, Schlüter JA, et al. Evidence-based interventions for children and adolescents with fetal alcohol spectrum disorders – a systematic review. Eur J Paediatr Neurol 2021: 33: 50–60.
- 35 Popova S, Charness ME, Burd L, et al. Fetal alcohol spectrum disorders. Nat Rev Dis Primers 2023; 9: 11.
- 36 McBride N, Johnson S. Fathers' role in alcohol-exposed pregnancies: systematic review of human studies. Am J Prev Med 2016; 51: 240–48.
- 37 Lyall V, Wolfson L, Reid N, et al. "The problem is that we hear a bit of everything...": a qualitative systematic review of factors associated with alcohol use, reduction, and abstinence in pregnancy. Int J Environ Res Public Health 2021; 18: 3445.
- 38 McQuire C, Daniel R, Hurt L, Kemp A, Paranjothy S. The causal web of foetal alcohol spectrum disorders: a review and causal diagram. Eur Child Adolesc Psychiatry 2020; 29: 575–94.
- 39 May PA, Gossage JP. Maternal risk factors for fetal alcohol spectrum disorders: not as simple as it might seem. Alcohol Res Health 2011; 34: 15–26.
- 40 Oh SS, Kang B, Park J, et al. Racial/ethnic disparity in association between fetal alcohol syndrome and alcohol intake during pregnancy: multisite retrospective cohort study. JMIR Public Health Surveill 2023; 9: e45358.
- 41 May PA, Hasken JM, Baete A, et al. Fetal alcohol spectrum disorders in a midwestern city: child characteristics, maternal risk traits, and prevalence. Alcohol Clin Exp Res 2020; 44: 919–38.
- 42 Elek E, Harris SL, Squire CM, et al. Women's knowledge, views, and experiences regarding alcohol use and pregnancy: opportunities to improve health messages. *Am J Health Educ* 2013; 44: 177–90.
- 43 Pruitt SM, Hoyert DL, Anderson KN, et al. Racial and ethnic disparities in fetal deaths - United States, 2015–2017. MMWR Morb Mortal Wkly Rep 2020; 69: 1277–82.

- 44 May PA, Gossage JP, White-Country M, et al. Alcohol consumption and other maternal risk factors for fetal alcohol syndrome among three distinct samples of women before, during, and after pregnancy: the risk is relative. Am J Med Genet C Semin Med Genet 2004; 127C: 10–20.
- 45 Braveman PA, Arkin E, Proctor D, Kauh T, Holm N. Systemic and structural racism: definitions, examples, health damages, and approaches to dismantling. *Health Aff* 2022; 41: 171–78.
- 46 Di Pietro NC, Illes J. Disparities in Canadian indigenous health research on neurodevelopmental disorders. J Dev Behav Pediatr 2014; 35: 74–81.
- 47 Bruno G, Chan TA, Zwaigenbaum L, Coombs E. Indigenous Relations Circle; Nicholas D. Indigenous autism in Canada: a scoping review. J Autism Dev Disord 2023; published online July 22. https://doi.org/10.1007/s10803-023-06045-z.
- 48 Manickam K, McClain MR, Demmer LA, et al. Exome and genome sequencing for pediatric patients with congenital anomalies or intellectual disability: an evidence-based clinical guideline of the American College of Medical Genetics and Genomics (ACMG). Genet Med 2021; 23: 2029–37.

- 49 London EB. Categorical diagnosis: a fatal flaw for autism research? Trends Neurosci 2014; 37: 683–86.
- 50 Lombardo MV, Mandelli V. Rethinking our concepts and assumptions about autism. Front Psychiatry 2022; 13: 903489.
- 51 Chipeur S, Zwicker J. Children with neurodisabilities and public policy: universal design for function rather than diagnosis. In: Gibbard WB, ed. Developments in neuroethics and bioethics: neuroethics and neurodevelopment. Amsterdam: Elsevier; 2023: 247–75.
- Miller AR, Rosenbaum P. Perspectives on "disease" and "disability" in child health: the case of childhood neurodisability. Front Public Health 2016; 4: 226.

Copyright o 2024 Elsevier Ltd. All rights reserved, including those for text and data mining, AI training, and similar technologies.