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Fetal alcohol spectrum disorders are clearly brain-based

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Alcohol is a well-established teratogen that can result in a wide range of adverse reproductive and developmental outcomes. One of the most serious outcomes is the probability of fetal alcohol spectrum disorders (FASDs), a group of lifelong physical, behavioral, and intellectual impairments.[1] FASDs are most prevalent in countries or regions with severe alcohol abuse issues such as South Africa and Eastern Europe.[2] In recent years, high prevalence rates have been reported in an aboriginal community in the Fitzroy Valley region of Western Australia.[3]

As part of a larger effort in this region through the Lililwan Project, Lucas et al.[4] investigated the presence of soft neurological signs among 7- to 9-year-old children using the Quick Neurological Screening Test – Second Edition (QNST-2). Overall, the research team found that the cohort performed similarly to the norming sample, but children with prenatal exposure to alcohol displayed significantly more soft neurological signs, and children with an FASD had the greatest number of these signs. The results are consistent with other evidence of the full progression from prenatal alcohol exposure through morphological brain changes to neurological impairment to adverse outcomes, including FASDs. An associative process has been replicated in animal models and was described anecdotally in clinic samples.

This study provides systematic evidence and underscores how prenatal alcohol exposure changes brain function, resulting in poor neurological and developmental outcomes. It contributes information about the pathway from changes in brain morphology to altered brain function to observable skills or deficits. Further, it describes how children diagnosed with an FASD exhibit a higher number of alterations in brain function, which would likely be associated with greater risks of psycho-educational and adaptive impairment. While this study was not able to identify a consistent pattern of deficits – most likely reflecting the heterogeneic nature of FASDs – it still highlights the brain-based nature of impairment. It is recognized that FASDs are characterized by a complex interaction of dose, timing, and pattern of exposure in addition to environmental factors.[5]

A second major contribution is the demonstration of an increase in adverse neurological signs that are observable in the group of children prenatally exposed to alcohol, but who did not meet criteria for an FASD diagnosis. This finding fills a gap between imaging studies that document changes in brain morphology resulting from prenatal exposure to alcohol and functional deficits seen in definitively diagnosed FASDs. It further reinforces the understanding that harm can occur at any level and timing of alcohol exposure even if that harm cannot be codified with an FASD diagnosis.

As with any behavioral research, there are limitations that must be addressed in future replications and extensions. Most notable is the use of a retrospective assessment of alcohol use during pregnancy after a long lag time (7–9y), which could introduce recall bias. Knowing more about the functional status of the children with deficits would have been useful. Finally, the authors may be overreaching to promote the QNST-2 as a diagnostic tool for FASDs at this time (as it describes functional rather than diagnostic issues), but its potential for early identification and referral are clear. Based on findings from this study, poor performance on the QNST-2 should certainly trigger consideration of possible in utero exposure to alcohol. Future prospective studies are strongly encouraged to address this point. Finally, findings from this study (and the Lililwan Project as a whole) underscore and reinforce current recommendations that females who may be pregnant abstain from alcohol to prevent an alcohol-exposed pregnancy, which can lead to lifelong neurological, neurodevelopmental, and functional harm.

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