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LETTER TO THE EDITOR

Persisting Concerns Regarding the New Canadian FASD Guidelines

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We appreciate Cook et al.'s (2018) response to our recent critique of the new Canadian FASD guidelines (McLennan & Braunberger, 2017) and welcome the opportunity for further discussion.

With respect to the proposition to include Diagnostic and Statistical Manual of Mental Disorders (DSM) affect regulation related diagnoses as FASD criteria, we found the assertion that it was not their intent "…to advance the pathophysiological relationship of FASD and mental disorders…" (Cook et al., 2018, p. 84) puzzling. Stipulating certain mental disorders as criteria for an FASD diagnosis *does* advance an etiological relationship between prenatal alcohol exposure (PAE) and certain mental disorders. We would also argue that diagnosis is more than "…merely an identification process…" (Cooke et al., 2018, p. 84); rather it is a significant organizing approach in health and society which can have substantial implications and consequences (including harms). Also concerning is the interpretation of cited references used to support the claim of an established correlation between PAE and affect regulation related disorders. The cited reference with the strongest research design actually found NO significant association between binge drinking and mood and anxiety disorders (Barr et al., 2006). Further, the effect size relationship between FASD and mental health problems was less than the effect size between not being breastfed or prenatal marijuana exposure and mental health problems (Barr et al., 2016). The interpretation of another cited reference is compromised by including referrals from an inpatient child psychiatric ward where high rates of affective disorders are anticipated regardless of PAE (O'Connor et al., 2002). It is problematic if these references are reflective of the human evidence being used to support the inclusion of affect regulation related disorders as criteria for an FASD diagnosis.

We are confused by the authors' mixed use of DSM in their argumentation. On the one hand, they find it useful for operationalizing affect regulation domains for FASD diagnostic criteria, however, they also find it problematic as "…earlier versions of the DSM did not recognize the influence of PAE, and FASD manifestations were diagnosed as other DSM conditions" (Cook et al., 2018, p. 84). So are the authors proposing that if someone were to meet criteria for an affection regulation related disorder in the context of PAE with some other characteristics attributed to FASD, that that person does not actually have a DSM disorder but the affect regulation abnormalities should be understood as a part of FASD? What about comorbidity? How do the authors propose determining when affect regulation abnormalities are a function of PAE or FASD, or a function of other potential contributors to affect regulation abnormalities (e.g., genetics)? Without clarifying this issue, it is not clear on what basis the authors' advance the proposition that persons with FASD have been misdiagnosed with DSM conditions.

We have no particular disagreement with the suggestion that neurodevelopmental testing at two standard deviations below mean values will likely correlate with impairment, nor that these measures might be correlated with brain imaging abnormalities. The concern is that there is a lack of

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evidence that these abnormal findings can be reliably attributed to PAE in individual patients, even when PAE is verified, given the multiple potential contributors to neurodevelopmental abnormalities. The default response to this complexity appears to be that this will be determined by the black box of clinical judgment and multidisciplinary teams with no clear operationalization of processes that can be systematically scrutinized.

The authors defend the "at-risk" category as "… justified by the importance of emphasizing consistent care and early intervention services given the social instability that many such children are facing" (Cook et al., 2018, p. 85). We agree that children facing social instability should receive consistent care and early intervention services. However, this should be a societal goal regardless of PAE status. There is not a clear rationale, nor supporting evidence, for carving out a unique service approach for children with PAE (McLennan, 2010). A surveillance and intervention package could be proposed for all children facing social instability (not just those with PAE), particularly given the lack of evidence for the effectiveness of any *unique* early intervention for children with PAE versus early interventions understood effective for children with a diversity of vulnerabilities. The authors seem to indirectly support this perspective by referencing the general literature on early childhood interventions.

Finally, we find the statement "Traditional psychiatric interventions may be ineffective if the underlying brain dysfunction is not recognized" (Cook et al., 2018, p. 84) problematic given that there is as of yet no convincing evidence of any *unique* effects of special FASD interventions as summarized and concluded in a recent systematic review (SBU [Swedish Agency for Health Technology Assessment and Assessment of Social Services], 2016). Indeed, our concerns with the new guidelines are not just academic or pragmatic, but foundational.

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