

Post-traumatic stress disorder and birthweight: methodological challenges

Authors' Reply

Sir,

Sampling, trauma history assessment, and symptom reporting all play a role in determining the prevalence of post-traumatic stress disorder (PTSD). The prevalence of prenatal PTSD ranges from 3.5% in a sample with a 29% trauma rate to 16% in a sample with a 60% rate of abuse.¹ We found an 8% prevalence overall, but defined a range from 2.9% in the private sector to 13.9% in the public sector. The highly sensitive trauma measure we used did not seem to inflate PTSD rates, as less severe trauma exposures were rarely antecedents to PTSD diagnosis (e.g. divorce was the 'worst' trauma in 1% of cases). Childhood maltreatment conveyed the greatest risk (OR 11.9, 95% CI 3.6–39.9) for PTSD in pregnancy.¹ We chose the National Women's Study PTSD module, a gold-standard structured diagnostic interview used in the largest study of PTSD in women from the USA, so that we could compare symptom profiles of our pregnant sample with 2000 women of childbearing age from the nationally representative sample. We concluded that pregnant women reported symptoms that were specific to traumatic stress.²

Dr Reed's point that PTSD alone did not account for the entire 200 g decrement is well taken and warrants nuancing.³ A more precise wording of our finding would be that 'the PTSD-affected cohort experienced a decrement of more than 200 g in birthweight'. Our PTSD cohort differed, as expected, in its levels of socio-economic disadvantage and risk behaviours. Disadvantage and substance use are both vulnerabilities for and outcomes of PTSD.⁴

To echo Dr Reed's point empirically, we modelled just three variables: PTSD, socio-economic status (SES) and substance use, predicting birthweight. With the whole sample, the relationship of PTSD to birthweight was mediated by SES, and substance use was not independently associated. The group not maltreated followed this pattern. In the childhood maltreatment group, the associations of PTSD and SES were additive, and both were mediated by substance use in the final step.

The combination of early developmental trauma and prenatal PTSD seems potent in human childbearing, as has been shown in animal models.⁵ We should not diminish the potential import of PTSD as a risk factor for adverse perinatal outcomes because it co-occurs with other intractable problems. Rather, we should consider how perceiving maltreatment and PTSD as potential root causes for some young women's disadvantages and stress might provide

opportunities for novel approaches to addressing these issues.

Finally, we had provided correlations addressing a conceptual issue about the extent to which mechanisms for trauma or PTSD-associated adverse outcomes might differentially affect growth and parturition. Due to the post hoc and conceptual nature of this analysis, we had not provided tests of significance. The difference between the birthweight and gestational age correlation of the PTSD-affected ($n = 255$, $r = 0.699$) and non-trauma-exposed ($n = 277$, $r = 0.765$) cohorts was not significant ($P = 0.102$). The difference between birthweight and gestational age correlation in infants born to mothers maltreated in childhood ($n = 174$, $r = 0.608$) and those of infants born to non-maltreated mothers ($n = 665$, $r = 0.760$) was significant ($P < 0.001$).

We hope this exchange facilitates the work of other research teams studying the impact of trauma and traumatic stress on women's health and childbearing. ■

References

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Accepted 26 September 2011.

DOI: 10.1111/j.1471-0528.2011.03200.x

Placenta accreta is associated with IVF pregnancies: a retrospective chart review

Sir,

I read with interest the article by Esh-Broder and colleagues¹ and noted that one of the triggers for performing placental histology was 'in caesarean sections, cases with heavy bleeding from the vaginal bed'. As the incidence of caesarean section was greater in the *in vitro* fertilisation (IVF) group, their findings could partly be explained by an increase in the percentage of placentas examined in the IVF