Authors' reply

We thank Tavares & McAlpine and Brown & Ginestet for their interest in our study. We also wish to highlight that our paper 'The role of prenatal stress as a pathway to personality disorder: longitudinal birth cohort study'[1] cites our previous work with this cohort[2] in the introduction as more detailed information about the cohort and the methodology can be found there, which was not included in this paper because of space constraints and minimising repeated information across publications.

Tavares & McAlpine query the validity of the stress measure that we used in our study. As stated in our paper we used a subjective rather than an objective measure of stress. There is evidence that the subjective rating of the impact of a stressor by an individual is more strongly associated with both health outcomes and the biological embedding of the experience compared with objective measures of stress.[3] We highlight in the paper that our measure of stress was based on a repeated single item in a self-report questionnaire and acknowledge that 'there is evidence to suggest that the correlation between questionnaire measures of stress and the biological stress response is only moderate. It is possible that our measure of stress is a marker of heightened risk for psychiatric disorder that is unrelated to a direct biological stress response. However, the feasibility of using a simple stress question to identify women who could benefit from psychosocial support and intervention during pregnancy warrants further investigation.'

Tavares & McAlpine also query whether this association is driven by the impact of the stressors rather than the impact of the stress. We did not specifically examine individual stressors and cannot comment on this possibility but we have stated in the discussion that the association that we find may not be a direct impact of stress but may be mediated through indirect mechanisms.

Tavares & McAlpine also query whether postnatal stress could be the reason for the association that we found. In our discussion, we explicitly state that this is very possible. 'It is likely that a woman who is stressed during the antenatal period will also be stressed during the postnatal period. It is possible that any continuation of stress into the postnatal period could have an impact on the parent–child relationship, and on early parenting style and warmth, thus affecting the child's development.'

Tavares & McAlpine suggest that our paper implies causal links between prenatal stress and subsequent offspring psychiatric disorders. We explicitly state in our discussion that this association may be mediated through a number of different pathways and that prenatal stress may be a proxy measure of other factors. We always talk about associations in the paper and not causation and have very carefully highlighted the limitations of our work.

The authors state 'We feel that research in this area should be reported carefully, to avoid contributing to a potentially harmful culture of mother-blaming.' We wholeheartedly agree with you, and hope that our paper helps to show the importance of having perinatal mental healthcare made easily accessible. It is vital for the health of this and the next generation that we prioritise the emotional and social support needs of families.

We thank Brown & Ginestet for highlighting that we have inadvertently used adjusted odds ratio in the abstract instead of the unadjusted figure and that two of the numbers in Table 1 are the wrong way round. A corrigendum has been published addressing these comments, available at https://doi.org/10.1192/bjp.2020.50.

Brown & Ginestet query our adjustment for total number of questionnaires and question the numbers submitted per person. The median number of questionnaires returned per person was six, with first and third quartiles of four and eight, respectively. As suggested by Brown & Ginestet, our measure could comprise 'self-reported stress during a single month during pregnancy', however, there were only 233 out of 3626 cases within the sample where this occurred.

Brown & Ginestet query our adjustment for the number of questionnaires returned by women and our use of a modal model. We used the modal over a mean or median as those figures may not be representative of the overall reported stress throughout pregnancy. For example, for a women who returned seven questionnaires throughout pregnancy, two reporting no stress, two reporting some stress and three reporting severe stress, the median would suggest this woman was slightly stressed, whereas based on the total number of questionnaires returned, the woman spent more time during pregnancy being severely stressed. Also, because of the variation in the number of prenatal questionnaires returned across participants, the mode was more representative of experienced stress when compared with the mean.

Brown & Ginestet query our sample size. We discuss this in the strengths and limitations section stating that because of the use of the hospital discharge register, the outcomes captured here only include those severe enough to be admitted to hospital, which may underrepresent the total levels of personality disorder within the population. We acknowledge that some of the confounder groups had small numbers, such as antenatal depression. This was adjusted for as separating subjective stress and depression may give a clearer view of the impact of stress, which is often intertwined with depression as part of a single definition along with feelings of anxiousness.[4] However, we also specifically reported stepwise additions of confounders because of the low numbers within some confounding variables.

Finally, as part of the discussion we were attempting to show some of the potential direct or indirect pathways from prenatal stress to psychiatric disorder that could be examined in future research. The mention of 'early life separation from parents, childhood trauma and parenting styles' are factors that have been shown to be associated with psychiatric disorder development, and thus we suggested that they warrant further research in conjunction with prenatal stress.

We agree that it is important to ensure statistics are robust and conclusions are justified. We have carefully delineated the limitations of our work in the discussion. We were careful not to infer causality from the association that we have shown and highlight the need for replication of these results.

Declaration of interest

none declared.

1 Brannigan R, Tanskanen A, Huttunen MO, Cannon M, Leacy FP, Clarke MC. The role of prenatal stress as a pathway to personality disorder: longitudinal birth cohort study. *Br J Psychiatry* 2020; **216**: 85–9.

2 Brannigan R, Cannon M, Tanskanen A, Huttunen MO, Leacy FP, Clarke MC. The association between subjective maternal stress during pregnancy and offspring clinically diagnosed psychiatric disorders. *Acta Psychiatr Scand* 2019; **139**: 304–10.

3 Rutter M. Why is the topic of the biological embedding of experiences important for translation? *Develop Psychopathol* 2016; **28**: 1245–58.

4 Dunkel Schetter C, Glynn LM. Stress in pregnancy: empirical evidence and theoretical issues to guide interdisciplinary research. In *The Handbook of Stress Science Biology, Psychology and Health* (eds Contrada RJ and Baum A): 321–44. Springer Publishing Company, 2011.

Ross Brannigan, PhD candidate, Royal College of Surgeons in Ireland, Ireland; **Mary Clarke**, Senior Lecturer, Royal College of Surgeons in Ireland, Ireland. Email: rossbrannigan@rcsi.com

doi:10.1192/bjp.2020.180

https://doi.org/10.1192/bjp.2020.180 Published online by Cambridge University Press