Much of the distress linked with COVID-19 will be clearly associated with psychosocial problems (isolation, unemployment, bereavement), but some will be firmly biological, following from COVID-19 infection itself. Managing these kinds of complex, biopsychosocial problems is precisely what psychiatry has done for decades. Psychiatry has never been as purely biological as the biologists would like, or as purely psychosocial as others would wish. It is a unique mix, and COVID-19 is our greatest challenge yet.

Response to the article 'The role of prenatal stress as a pathway to personality disorder: longitudinal birth cohort study'

We have discussed the paper by Brannigan et al1 at our journal club, and we wish to raise some of the points that arose from a critical appraisal of this paper, and the panel discussion that ensued.

First, the figures presented in the abstract appear to be inaccurate. The figure 3.28 (odds ratio for any stress exposure) has been taken from the unadjusted results, and the figure 3.29 (odds ratio for moderate stress) has been taken from the adjusted results, and the figure 7.02 (odds ratio for those exposed to severe stress) does not appear at all in the results. There appears to be an error in the figure 7.02 (odds ratio for those exposed to severe stress).

Second, the figures presented in the abstract are inaccurate. The figure 3.28 (odds ratio for any stress exposure) has been taken from the unadjusted results, and the figure 3.29 (odds ratio for moderate stress) has been taken from the adjusted results, and the figure 7.02 (odds ratio for those exposed to severe stress) does not appear at all in the results. There appears to be an error in the figure 7.02 (odds ratio for those exposed to severe stress).

However, the main concerns raised were around the statistical methods used. There was some variability in the number of prenatal questionnaires returned. Therefore, we adjusted for the numbers of questionnaires returned to account for this. This provides no information on how many questionnaires were returned by each participant and how this was adjusted for. Does this not mean that, for some participants, ‘prenatal stress’ could actually refer to ‘self-reported stress during a single month of pregnancy’?

A modal measure of stress was used as it best represented the individual scores when compared with the mean, which was less accurate due to variability in the number of returned prenatal questionnaires. From this we were struggling to understand the reasoning behind use of the mode; we felt it possible that single highly stressful events would not have been captured in the final data.

Using diagnosis of personality disorder on a hospital discharge register as the primary outcome of interest, there were only 40 positive cases, and only 9 with no comorbid psychiatric diagnosis. This is a very small sample to compare with the 3586 without a diagnosis. Moreover, some of the covariates that the authors controlled for had groups of participants as small as one individual, which made the overall results too unstable to interpret.

Various psychosocial mechanisms were suggested, including ‘early life separation from parents, childhood trauma and parenting styles’, however, there was no suggestion that these variables were related directly to levels of prenatal stress.

Mental health support in the perinatal period is clearly of huge importance for the well-being of mother, baby and the wider family, and research into this area is needed. However, there is a responsibility to ensure that the statistics are robust, and conclusions justified, particularly in view of the extensive media coverage generated by this paper.

Re: 'The role of prenatal stress as a pathway to personality disorder'

In their recent paper, Brannigan et al (2020) describe their examination of the relationship between self-reported maternal stress during pregnancy, and the subsequent development of personality disorder in the offspring. We would like to raise several points about the study and the conclusions drawn by the authors.

The study is based on the results of a subjective, single-item, three-point Likert scale completed at unspecified point(s) during pregnancy. Women were asked whether they had experienced ‘no stress’, ‘some stress’, or ‘notable stress’ since their last antenatal appointment. Of the entire cohort of 6468 women, 3626 completed the Likert scale questionnaire at least once during their pregnancy; these women were included in the analysis. A further 2842 women were not included in the analysis, presumably because they did not complete the questionnaire. We question whether this is a valid or meaningful measure of stress during the antenatal period.

A wide range of biological, psychological and social stressors may contribute to subjective feelings of stress. Perhaps it is the ongoing impact of these stressors, rather than prenatal maternal stress per se, that lead to the increased odds of a subsequent diagnosis of personality disorder. Furthermore, in many cases stress does not resolve immediately after giving birth: is it possible that ongoing maternal stress in the postnatal period (and beyond) might have a more significant impact on parenting practices, and thus on a child’s development?

Maternal well-being and mental health is an important area of study, and the authors rightly note that there is a strong evidence base to support the use of interventions to reduce maternal stress. However, this paper implies a causal link between antenatal maternal stress and subsequent diagnosis of personality disorder that is not evidenced by the method and results presented here. We feel that research in this area should be reported carefully, to avoid contributing to a potentially harmful culture of mother-blaming.

Declaration of interest

None declared.


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