Letters to the Editor

Re: Moderate alcohol intake during pregnancy and risk of fetal death

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The report on alcohol intake and fetal loss by Andersen et al.1 addresses an unresolved question of great public interest using a large, high-quality data resource and appropriate analytic methods, and concludes that modest amounts of alcohol are associated with increased risk of fetal loss before the 16th week of pregnancy. They infer that this association is likely to be causal and therefore advise pregnant women to abstain from drinking alcohol in the first 16 weeks of pregnancy.

Although there is little reason to question whether the reported associations are present, there is serious doubt about whether that association is likely to be causal. An alternative explanation for their results is reverse causality, in which correlates or markers of the pregnancy loss allow for increased alcohol intake. Little is known about the causes and manifestations of pregnancy loss, but we do know the following. (i) Lack of nausea is strongly associated with increased risk of miscarriage, in the order of a 2-to-3-fold difference between women who do not versus those who do experience nausea.2–4 It is not clear whether the lack of nausea reflects impending loss, owing to the absence of the hormonal surge typical of early pregnancy, or an actual loss of the pregnancy that has not yet become apparent.5

(ii) There is a lag of variable length, but in the order of days to several weeks between loss of fetal viability and recognition of the fetal loss, based on such symptoms as bleeding or pain.6,7 (iii) Nausea in early pregnancy is thought to commonly lead to aversion to caffeine, selected foods and, to some extent, alcohol.8

With these premises, it is likely that women who experience nausea in early pregnancy will spontaneously choose to abstain from alcohol to a greater extent than women not experiencing nausea, not with the intent of protecting their pregnancy but based on preferences. Women without nausea will have an increased risk of a later fetal loss, either because the pregnancy has already ended without their knowledge or is at greater risk of ending. This is a different concern than biased reporting of alcohol use as a result of being interviewed after a known miscarriage, which was considered and addressed to the extent possible in the article. The phenomenon of concern was described insightfully >20 years ago pertaining to caffeine,9 and analogous to the argument about physical activity and its consequences, also likely to be discouraged by nausea in early pregnancy.9

The analytic solutions to this problem are not obvious, as the nausea (or lack of nausea), the behavioural responses to the nausea (avoidance of alcohol) and the pregnancy loss itself (whether it has been recognized) are virtually concurrent. Perhaps serial ultrasounds to assess fetal viability would help disentangle the cause and effect sequence, at least to provide assurance that the behaviours are occurring while the fetus remains viable. Despite the wealth of high-quality data collected in the Danish National Birth Cohort or any other large pregnancy cohort, it is not clear what can be done to address the hypothesis of reverse causality and therefore, at minimum, more tempered inferences and advice would be warranted.

References


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