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Title: Prenatal alcohol exposure and childhood atopic disease: A Mendelian randomisation approach

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Body: Background: Few epidemiological studies have investigated whether alcohol consumption during pregnancy increases the risk of childhood atopic disease. A difficulty with using reported alcohol intake to measure exposure is that under-reporting is common and associations are likely to be confounded. In contrast, a Mendelian randomisation approach should produce unbiased and unconfounded effect estimates and can strengthen causal inference. Methods: In a UK population based birth cohort, the Avon Longitudinal Study of Parents and Children (ALSPAC), we have analysed whether the maternal alcohol dehydrogenase (ADH)1B gene variant is associated with childhood atopic outcomes at 7 years of age. Carriers of the minor A allele drink less in pregnancy and metabolise alcohol faster, thus reducing adverse effects of alcohol on the fetus. We also analysed associations with reported alcohol consumption in pregnancy. Results: Maternal ADH1B genotype was strongly associated with childhood asthma. Mothers carrying the minor A allele were half as likely to have children with asthma as mothers who were homozygous for the G allele (odds ratio 0.50 (95% CI: 0.33 to 0.75), P=0.001, N=6,701). There were no significant associations with other atopic outcomes. In contrast, mothers who reported drinking once a week or more in the last two months of pregnancy were less likely to have children with asthma and hayfever than mothers who reported never drinking (OR 0.81 (0.65 to 0.99) and 0.75 (0.59 to 0.95), respectively). Conclusions: The genetic results suggest that prenatal alcohol exposure increases the risk of childhood asthma. The contradictory associations with reported alcohol intake are likely to be confounded.