

detect bias due to unmeasured confounding; and 3) to combine estimates from population-based and case-control studies in meta-analyses.

Methods This whole-of-population study of children born from 1999–2013 ($n=286,058$, aged <15 years) used de-identified linked administrative datasets from the South Australian Early Childhood Data Project. T1D was diagnosed for 557 children during hospitalization (ICD-10-AM codes, E10, E101-E109) from 2001–2014. Maternal smoking data was sourced from the South Australian Perinatal Statistics Collection, where information at birth is collected by midwives/neonatal nurses using a validated tool. Hospitalization for any injury occurring at school was used as a negative-control outcome. Adjusted Cox proportional hazard ratios (HR) were calculated in the main analysis and the negative-control outcome. Random-effects meta-analysis was used to summarize effects of prenatal smoking on childhood T1D.

Results Compared with non-smokers, smoking throughout pregnancy was associated with 23% lower childhood T1D risk (HR 0.77; 95% CI 0.60–1.00), with similar effects for smoking in first-half (HR 0.78; 95% CI 0.60–1.01) and second-half (HR 0.75; 95% CI 0.57–0.98) of pregnancy. The negative-control outcome analysis (HR 0.95; 95% CI 0.86–1.05) suggested the effect of prenatal smoking on T1D was not due to unmeasured confounding. These results were consistent with meta-analytic estimates of prenatal smoking and childhood T1D risk from population-based (HR 0.70; 95% CI 0.60–0.81) and case-control studies (OR 0.71; 95% CI 0.55–0.86).

Conclusion Maternal smoking in pregnancy was associated with lower risk of childhood T1D. The negative-control outcome analysis suggests this effect is unlikely to be due to unmeasured confounding. Our meta-analytic estimates also showed lower risk of T1D for children exposed to maternal smoking during pregnancy. Smoking causes irreversible harm to the fetus, however understanding the mechanism of protective effects (e.g. nicotine exposure, gene expression) might lead to new insights about T1D.

OP75

A SYSTEMATIC REVIEW AND META-ANALYSIS OF RISK FACTORS FOR PREGNANCY-ASSOCIATED STROKE

¹R Green*, ¹L Tata, ²N Sprigg. ¹Division of Epidemiology and Public Health, University of Nottingham, Nottingham, UK; ²Division of Clinical Neuroscience, University of Nottingham, Nottingham, UK

10.1136/jech-2019-SSMabstracts.78

Background Stroke in younger women is rare, however, pregnant women have a significantly increased risk around delivery and in early postpartum. Despite known risk factors, such as high blood pressure, the contribution of female-specific factors to women's stroke risk are poorly understood. Whilst there are pathophysiological reasons for increased stroke in pregnancy, it is of clinical and public health importance to determine the extent to which these strokes can be pre-identified by background risk and pregnancy-related factors. We conducted a systematic review to identify risk factors for pregnancy-associated stroke; this included risk factors pre-existing (to pregnancy) and those developing during pregnancy and labour.

Methods An electronic search of PubMed, MEDLINE and EMBASE databases, without language, study design or publication date restrictions, was performed in November 2018.

Study inclusion criteria were reported risk factors/characteristics for women with stroke during pregnancy or up to 12 months after delivery and for a comparison group of pregnant/postpartum women without stroke. Stroke timing (antenatal, perinatal, postnatal), diagnostic type and fatality were assessed. Data were extracted and, where possible, a random effects meta-analysis was conducted, heterogeneity quantified using I^2 . Methodological quality was assessed using an adapted Newcastle-Ottawa scale.

Results Of 3784 papers screened, 9 studies met the inclusion criteria comprising 11,398 women with stroke and >85 million comparison women across 4 countries. Eight studies reported effect measures for at least one risk factor. Of fourteen risk factors reported, 8 showed a statistically significant increase in pregnancy-associated stroke; pooled odds ratios with 95% confidence intervals: maternal age >35 years 2.66 (1.83–3.87), black ethnicity 1.56 (1.35–1.81), smoking 1.96 (1.64–2.34), alcohol use 2.32 (1.41–3.81), drug abuse 1.82 (1.19–2.77), hypertension 4.80 (3.26–7.06), pre-eclampsia 10.30 (8.26–12.84) and cesarean delivery 4.85 (2.22–10.59). Parity, body mass index, obesity, diabetes, infection, and migraine were not associated. Studies provided limited data to assess risk factors according to stroke timing, type and fatality.

Conclusion Our findings improve current understanding of the relative contributions of different risk factors for pregnancy-associated stroke. However, our work highlights the very few existing studies in this area. The available studies assessed a limited number of risk factors, and many were similar to those known to increase stroke regardless of pregnancy. Studies including detailed risk assessment in relation to pregnancy, delivery and postpartum complications as well as women's background risks are needed. Additionally, future research should establish whether risk factors differ according to stroke type and time-period in relation to pregnancy.

OP76

A SYSTEMATIC REVIEW AND META-ANALYSIS ON NEURODEVELOPMENTAL EFFECTS OF PRENATAL VITAMIN D IN HUMANS

E Morales*, AM García-Serna. *Pediatrics Research Group, IMIB-Arrixaca Biomedical Research Institute, Murcia, Spain*

10.1136/jech-2019-SSMabstracts.79

Background Vitamin D plays a key role in brain development and function; however, evidence in humans has never been systematically reviewed. Hence, we conducted a systematic review, accompanied by meta-analyses where possible, to summarize the existing evidence in humans on the relationship between prenatal 25-hydroxyvitamin D [25(OH)D] circulating levels and neurodevelopmental outcomes, including cognition, psychomotor performance, language development, behavior, ADHD, and autistic traits.

Methods PubMed, Web of Science and SCOPUS databases were systematically searched using keywords. Study eligibility criteria were: 1) original epidemiologic study performed in humans; 2) available information on circulating concentration of 25(OH)D in maternal or newborn blood as exposure; 3) outcome assessment included information on the offspring's neurodevelopment evaluated by standardized test scores; and 4) available data on the relevant estimates of effect size and the corresponding 95% confidence intervals (CIs). For all the