Objective: To investigate perinatal risk factors for testicular cancer in a Northern Ireland population cohort.

Methods: Perinatal data have been routinely recorded in Northern Ireland for all births in the period 1971–1986 (n=447, 665). Testicular cancer status was ascertained in this cohort by identifying 249 individuals diagnosed from 1971 to 2008 and date of birth in the period 1971–1986.

Results: Increased testicular cancer risk was associated with higher maternal age (>55 years), lower birth weight (<2500 g), caesarean delivery, decreased birth order and lower socio-economic status. Following multivariable analyses, the association between higher maternal age and testicular cancer risk remained significant (OR 1.55; 95% CI 1.07 to 2.25). Caesarean delivery was also associated with a significant increase in the risk of testicular cancer (OR 1.65; 95% CI 1.11 to 2.44). Increasing birth order was associated with a significant decrease in the testicular cancer risk (OR 0.59; 95% CI 0.41 to 0.83) comparing birth order three or more with the firstborn.

Conclusion: These findings demonstrate that maternal age at delivery, birth order and mode of delivery are significantly associated with testicular cancer risk. These associations may be due to earlier exposure to infectious agents or increased immune modulation.

Introduction: Despite alcohol being an established carcinogen and recent prospective studies showing increased prostate cancer risk among heavy drinkers, alcohol is not an established risk factor for prostate cancer.

We aimed to investigate the causal role of alcohol on prostate cancer through conventional observational epidemiology techniques and Mendelian randomisation, by using genetic variants influencing the propensity to drink or modifying the physiological response to alcohol.

Methods: A case-control study was nested in the case identification phase of a large British population-based RCT for treatment of localised prostate cancer (ProtecT). 2400 prostate-specific antigen detected prostate cancer cases of white ethnicity and 12,700 controls matched on age and general practice provided data on alcohol consumption. Eighteen SNPs in alcohol-metabolising genes (ADH, ALDH2) were genotyped in a sub-sample of cases and controls.

Results: There was some evidence of a modest decrease in low Gleason-grade (RR 0.96; 95% CI 0.93 to 0.99) and increase in high-grade (RR 1.04; 95% CI 1.00 to 1.08; p difference=0.004) prostate cancer per 10 alcohol units/week increase in consumption, not explained by current BMI, blood pressure, co-morbidities, or reverse causation.

Results from genetic association analyses including an interaction between an ADH1B functional variant and alcohol consumption suggested that alcohol causally increases prostate cancer risk. However, this study was underpowered to detect a difference between results from instrumental variable analyses and conventional observational epidemiology models.

Conclusion: These results support small increases in high-grade prostate cancer risk caused by heavy alcohol drinking. The required independent replication in populations with incident (non prostate-specific antigen-detected) prostate cancer is currently under-way.