Alcohol consumption and risk for type 2 diabetes: Enhancing causal inference with a Mendelian Randomisation approach

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Introduction / Issues: Observational evidence often suggests a J-shaped relationship between alcohol consumption and risk for type 2 diabetes, but it is unclear whether these findings reflect genuine causal relationships. Genetic-based methods can enhance our ability to make causal inferences, but existing studies employing these approaches are limited and sparse. Using genetic methods, we aimed to evaluate the functional form and strength of the alcohol–type 2 diabetes relationship.

Method / Approach: A secondary analysis of the UK Biobank was performed (recruitment 2006-10; follow-up until 2016). Linear and non-linear Mendelian Randomisation analyses of the relationship between drinks per week and HbA1C levels/risk for type 2 diabetes were conducted.

Key Findings OR Results: For the primary analyses, the sample size was 305,614 (52% female; mean baseline age: 56.8). Non-linear Mendelian Randomisation suggested a J-shaped relationship between alcohol and incident type 2 diabetes risk, but the small protective effect was largely limited to women, and not robust to all sensitivity analyses. Observational results produced protective effects for both sexes, even for very large weekly drinking volumes. Alcohol was associated with reduced baseline HbA1C levels in both sexes – a finding robust to sensitivity analyses.

Discussions and Conclusions: Low-level alcohol consumption may have causal benefits for average glucose levels, but a protective effect against risk for developing diabetes is more ambiguous and at best is confined to women consuming small volumes. Observational evidence substantially overestimates the benefits, and underestimates the harms, of alcohol for diabetes risk.

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