



Coffee drinking lowers diabetes risk: Observational proof or confounding?

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As most researchers recognise, randomised trials of different diets, dietary factors or drinks are very difficult to maintain for long enough periods to be able to link such interventions to hard outcomes. Another issue that also makes the interpretation of these studies difficult is that of food displacement, whereby adding a new dietary factor will often require a reduction of something else to enable calorie intake to remain balanced. This is why the nutrition field has relied for many years upon observational data. However, this approach has its own difficulties, in particular the issue of residual confounding, whereby individuals who have certain dietary habits also differ in other ways, which are not always easy to adjust for, and, of course, one can never fully adjust for confounders.

Let's take the example of coffee intake, which apparently "reduces" diabetes risk perhaps by virtue of the associated caffeine. There have been multiple observational studies addressing this potential association, and even meta-analyses have largely shown that the more one drinks of coffee, the lower the risk of diabetes (Huxley et al, 2009). However, to date, no one had adequately estimated whether the association between coffee and diabetes differed between caffeinated and decaffeinated drinks. This gap was addressed by Ding et al in a systematic review and meta-analysis (summarised alongside). The authors did a very careful analysis of all available data, including assessing each study's quality. Their overall findings suggested a 9% lower risk of diabetes per one extra cup of caffeinated coffee drunk; however, there also appeared a 6% lower risk of diabetes per extra cup of decaffeinated coffee drunk, with no statistical difference between these two estimates.

The authors of this report suggest that caffeine *per se* may not be the "magic" coffee ingredient to lower diabetes risk but point to other ingredients. Of course, an alternative conclusion is that both estimates reflect residual confounding and that coffee intake, whether caffeinated or decaffeinated, does not lower diabetes risk.

So how does the average clinician or person with diabetes approach these results? In reality, the data cannot prove a real effect, and so we are still left guessing. The most sensible advice remains to eat and drink a balanced diet – one that is enriched in fibre and protein but limits refined sugars and saturated fats. Yet, surely there must be a way beyond this dietary observational merry-go-round short of trials?

Recently, genetics has come to the fore and could help. As we all know, people can be genetically prone to differing behaviours (e.g. alcohol intake), and, if the relevant genes can be identified, and they capture a reasonable level of variation in intake of certain foods or drinks, this information may help to better differentiate causal and non-causal associations. Such work, including research on genes linked to caffeine intake, is in its infancy in the diet and drink field but should mature with time. For now, to lessen the risks of diabetes, the key and dominant message remains focused on behaviours associated with favourable weight and weight trajectories, and, currently, most other modifiable interventions, short of drugs, pale into insignificance. ■

Huxley R, Lee CM, Barzi F et al (2009) Coffee, decaffeinated coffee, and tea consumption in relation to incident type 2 diabetes mellitus: a systematic review with metaanalysis. *Arch Intern Med* 169: 2053–63

Diabetes Care

Does coffee consumption lead to a lower risk of T2D?

Readability ////

Applicability to practice ///

WOW! Factor ///

1 An extensive systematic review and meta-analysis was carried out to investigate the trend between drinking coffee and a lowered T2D risk; specifically in this study, the authors compared the difference between caffeinated and decaffeinated coffee.

2 A search of PubMed and EMBASE for articles published between 1966 to February 2013 found 28 articles that fitted the inclusion criteria: published in English; a prospective cohort or nested case-control design; categorised coffee consumption; and the measured outcome was T2D risk.

3 Overall, there were 1 109 272 participants included and 45 335 reported cases of T2D. The follow-up for incident T2D ranged from 10 months to 20 years and the median follow-up was 11 years.

4 Compared with no or rare coffee consumption, the relative risk (RR) for diabetes was 0.92 (95% confidence interval [CI], 0.90–0.94) for one cup/day, and decreased with each increase in cup consumed.

5 The RR of T2D for a one cup/day increase was 0.91 (95% CI, 0.89–0.94) for caffeinated coffee consumption and 0.94 (95% CI, 0.91–0.98) for decaffeinated coffee consumption. There was no significant difference between drinking caffeinated and decaffeinated coffee in lowering the risk of T2D.

6 Coffee consumption was inversely associated with the risk of T2D in a dose–response manner.

Ding M, Bhupathiraju SN, Chen M et al (2014) Caffeinated and decaffeinated coffee consumption and risk of type 2 diabetes: a systematic review and a dose-response meta-analysis. *Diabetes Care* 37: 569–86

Diabetologia

Adherence to predefined dietary patterns and incident cases of T2D

Readability ✓✓✓
 Applicability to practice ✓✓
 WOW! Factor ✓✓

1 The authors aimed to assess the association between predefined dietary patterns and T2D risk in a European population using data from the EPIC (European Prospective Investigation into Cancer and Nutrition) study.

2 For this analysis (named the EPIC-InterAct study), data from 9682 verified incident T2D cases and 12 595 randomly selected participants for the sub-cohort from the EPIC database were used. There were 661 verified incident T2D cases in the sub-cohort.

3 Habitual dietary intake was assessed at baseline with country-specific questionnaires.

4 Two diet-quality scores (alternative Healthy Eating Index [aHEI] and Dietary Approaches to Stop Hypertension [DASH]) and three reduced rank regression (RRR)-derived dietary pattern scores were constructed.

5 After multivariable adjustment, including body size, the aHEI and DASH scores were not significantly associated with diabetes, although for the aHEI, there was a tendency towards an inverse association in countries with higher mean age.

6 Adherence to specific RRR-derived dietary patterns commonly characterised by high intake of fruit and vegetables and low intake of processed meat, sugar sweetened-drinks and refined grains may lower T2D risk.

InterAct Consortium (2014) Adherence to predefined dietary patterns and incident type 2 diabetes in European populations: EPIC-InterAct Study. *Diabetologia* **57**: 321–33

Diabetes Care

Screening for T2D: Chinese risk score

Readability ✓✓✓
 Applicability to practice ✓✓✓
 WOW! Factor ✓

1 The authors developed a New Chinese Diabetes Risk Score for screening undiagnosed T2D in China.

2 Data comprising 16 525 men and 25 284 women aged 20–74 were analysed and took into account age, sex,

waist circumference, BMI, systolic blood pressure and family history of diabetes.

3 The point totals ranged from 0 to 51. The optimal cutoff point for previously undiagnosed diabetes was 25.

4 Increased age, male sex, BMI, waist circumference, systolic blood pressure and positive family history were significantly associated with the presence of undiagnosed T2D.

5 The new risk score provides a reliable tool to detect undiagnosed T2D among the Chinese population.

Zhou X, Qiao Q, Ji L, Ning F et al (2013) Nonlaboratory-based risk assessment algorithm for undiagnosed type 2 diabetes developed on a nation-wide diabetes survey. *Diabetes Care* **36**: 3944–52

Diabetes Care

Youth with T2D: Treatment patterns

Readability ✓✓✓
 Applicability to practice ✓✓✓
 WOW! Factor ✓✓

1 This study aimed to describe the treatment regimens among youth with T2D. In total, 474 participants (mean age 16.3 years, diabetes duration 24.2 months) completed a “SEARCH for Diabetes in Youth” study visit in the US.

2 The regimens were defined as lifestyle alone, metformin

monotherapy, any other oral hypoglycaemia agent(s) (OHA[s]) other than metformin, insulin monotherapy and insulin plus any OHA(s).

3 Over 50% of participants reported treatment with lifestyle or metformin alone. Participants on metformin alone had lower HbA_{1c} than those on insulin alone or insulin plus OHA(s) ($P < 0.001$). These differences remained significant after adjustment.

4 Over 50% of those on therapies containing insulin still experienced treatment failure, along with those with longer diabetes duration.

Badaru A, Klingensmith GJ, Dabelea D et al (2014) Correlates of treatment patterns among youth with type 2 diabetes. *Diabetes Care* **37**: 64–72

Diabetes Care

Effect of bariatric surgery on beta-cell function

Readability ✓✓✓
 Applicability to practice ✓✓✓
 WOW! Factor ✓✓✓

1 The effect of biliopancreatic diversion (BPD) surgery on beta-cell function was investigated in grade I and grade II obese people with T2D.

2 In a female study population of 19 lean-controls, 18 obese-controls and 31 obese women with T2D, 64% of the people with T2D underwent BPD surgery.

3 Oral glucose tolerance tests and hyperglycaemic clamps were performed 1 month after surgery, and mathematical models were used to estimate beta-cell function and other metabolic outcomes.

4 After BPD surgery, restoration of the basal disposition index ($P < 0.001$) and improvement of the stimulated disposition indices in oral and intravenous glucose stimulation of the beta-cells was observed ($P < 0.05$).

5 Beta-cell function, insulin sensitivity, and hepatic extraction of insulin improved after BPD, as did glycaemic control.

Junqueira Vasques AC, Pareja JC, de Oliveira Mda S et al (2013) Beta-cell function improvements in grade I/II obese subjects with type 2 diabetes 1 month after biliopancreatic diversion. *Diabetes Care* **36**: 4117–24

“Beta-cell function and insulin sensitivity, as well as hepatic extraction of insulin, improved after biliopancreatic division surgery in women with T2D.”