Dietary fat intake is associated with insulin resistance and an adverse vascular profile in patients with T1D: a pooled analysis

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What was done
Authors performed a pooled cross-sectional analysis of the baseline pre-treatment data from 3 RCTs (n=107 aged 18-50). Estimated glucose disposal rate (eGDR) was used as an Insulin Resistance (IR) marker. eGDR was calculated using BMI, presence of hypertension and HbA1C using a validated formula. A lower eGDR correlated with greater insulin resistance (IR)
With the pooled data, authors created a model to predict the effect of different macronutrient substitutions (eg: replacing an % energy from fat with an equal % energy from carbohydrate) on eGDR and vascular risk. Vascular risk was determined through measurement of vascular markers (Tumour necrosis factor alpha (TNFα), fibrinogen, tissue factor (TF) and plasminogen activator inhibitor-1 (PAI-1))

What this research tells us
Patients with lowest eGDR (highest IR) reported higher fat and lower carbohydrate intakes. This relationship remained after adjustment for energy intake. Unsaturated fat and fibre levels were similar across all eGDR tertiles.
Greater total energy intake, which was mainly driven by increased fat consumption, was associated with greater IR with those with the highest energy intakes, having the lowest eGDR.

The model showed that increasing amounts of dietary carbohydrate (and subsequent reduction in fat) resulted in greater insulin sensitivity, and a lower vascular risk profile, whereas increased dietary fat at the expense of carbohydrate or protein was associated with increased IR.

Limitations or discussion points
This cross-sectional analysis looked at a single time point and cannot prove causality. Participants with greater IR could have already chosen to follow a higher fat, lower carbohydrate diet in an attempt to improve glycemic control or lose weight. This is likely considering the widely held view in the type 1 community that carbohydrates worsen blood glucose management and consequently restrict carbohydrate to some degree. The authors describe plausible mechanisms by which dietary fat intake could influence IR, which have been demonstrated in a number of other studies.

There is a body of evidence looking beyond macronutrients, at type of fat and carbohydrate quality, and the level of food processing on insulin resistance and cardiovascular risk. This study could not go into that detail, and dietitians would probably want to be able to advise patients on those aspects.

Self-reported dietary intake assessments have inherent limitations, but a strength of this study was that 2 different validated methods were used, including one weighed, and results were consistent between methods.
eGDR is not a direct measure of IR. It is a calculation of other markers. However, eGDR has been validated against other ways of measuring IR like HOMA-IR and the impractical hyperinsulinemic-euglycemic glucose clamp technique.

Areas for future research
It would be useful to confirm these results with similar studies that used other, more direct, ways of measuring IR. Furthermore, longitudinal studies looking at the same variables including children with type 1 diabetes and randomized cross-over intervention studies would be useful in examining the relationship between total fat intake and IR. It would also be useful if future studies could separate any effect of substituting different types of fat on IR.

Impact on dietetic practice
Many people with T1D limit their portions of starchy carbohydrate foods, replacing them with high fat alternatives, with the aim of reducing post prandial rise in blood glucose and the size of their insulin doses. By doing this their typical diet will higher in fat, which is associated with insulin resistance. Dietetic education could include discussions around the impact of higher fat diets on insulin resistance and cardiovascular health.