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Mechanisms contributing to lactose and sucrose-induced postprandial lipaemia

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Fructose-containing sugars can exaggerate postprandial lipaemia and stimulate hepatic de novo lipogenesis (DNL) when compared to glucose-based carbohydrates(1). Galactose has recently been shown to increase postprandial lipaemia compared to glucose(2), but mechanisms remain uncharacterised. The aim of this study was to assess the effect and mechanisms of lactose-induced lipaemia.

Twenty-four non-obese adults (12 male and 12 female) completed three trials in a randomised, crossover design (28 ± 7-day washout). During trials, participants consumed test drinks containing 50 g fat with 100 g of carbohydrate. The control carbohydrate was a glucose polymer (maltodextrin), the experimental carbohydrate was galactose-containing carbohydrate (lactose) and the active comparator was fructose-containing carbohydrate (sucrose). Hepatic DNL was assessed by the ^2H_2O method and [U-^{13}C]-palmitate was added to the test drink to trace the fate of the ingested fat. Blood and breath samples were taken to determine plasma metabolite and hormone concentrations, in addition to plasma and breath ^{2}H and ^{13}C enrichments. Data were converted into incremental under the curve (iAUC) and were checked for normality by visual inspection of residuals. Differences between trials were assessed by one-way ANOVA. Where a main effect of trial was detected, post- hoc t-tests were performed to determine which trials differed from lactose according to the principle of closed-loop testing.

The plasma triacylglycerol iAUC (mean ± SD) in response to maltodextrin was 51 ± 68 mmol/L*360 min. Following lactose ingestion, plasma triacylglycerol iAUC increased to 98 ± 88 mmol/L*360 min ($p<0.001$ vs maltodextrin), which was comparable to sucrose [90 ± 95 mmol/L*360 min ($p=0.41$ vs lactose)]. Hepatic DNL in response to maltodextrin was 6.6 ± 3.0%. Following ingestion of lactose, hepatic DNL increased to 12.4 ± 6.9% ($p=0.02$ vs maltodextrin), which was comparable to sucrose [12.2 ± 6.9% ($p=0.96$ vs lactose)]. Exhaled $^{13}CO_2$ in response to maltodextrin was 10.4 ± 4.1 mmol/kgFFM*360 min. Following ingestion of lactose, exhaled $^{13}CO_2$ was 8.8 ± 4.9 mmol/kgFFM*360 min ($p=0.09$ vs maltodextrin), which was lower than sucrose [11.1 ± 3.9 mmol/kgFFM*360 min ($p=0.01$ vs lactose)].

These data are consistent with the hypothesis that hepatic *de novo* lipogenesis contributes to both lactose and sucrose-induced lipaemia and provide a rationale to investigate the longer-term effects of lactose and sucrose on metabolism.

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References

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