Effects of High and Low Sugar Diets on Cardiovascular Disease Risk Factors

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Summary It has been proposed that a high sugar intake was associated with cardiovascular disease (CVD) risk and metabolic syndrome depending on the amount of carbohydrate (CHO), other nutrients in foods, and underlying metabolic disturbances. This study aimed to investigate the effects of high (HS) and low sugar (LS) diets on metabolic profiles in 25 middle-aged men at increased CVD risk in a 12-week randomised cross-over intervention study. An isocaloric dietary exchanged model consisted of HS (24% energy from sugar) and LS (6% energy from sugar) with comparable total CHO, fat and fibre composition in normal foods was used. Anthropometric, blood pressure and plasma lipid profile were measured pre- and post-intervention. Body weight, waist circumference and fat mass increased and decreased significantly after HS (by 0.7 ± 0.3 kg, 1.4 ± 1.0 cm and 0.5 ± 0.3 kg) and LS (by 2.1 ± 0.5 kg, 2.0 ± 0.8 cm and 1.4 ± 0.3 kg) ($p<0.05$), respectively. Plasma TG increased significantly after HS by 0.26 ± 0.07 mmol/L and decreased after LS by 0.35 ± 0.16 mmol/L. Plasma HDL decreased by 0.11 ± 0.03 mmol/L ($p<0.05$) after HS, whilst, plasma TC and LDL decreased significantly by 10% after LS. There was no significant change in other parameters after either diet. This study confirmed that a diet with a greater proportion of sugar increased CVD risk via negative changes in metabolic profiles including body weight, waist circumference and lipid parameters, whereas LS produced the positive effects. A restriction of sugar intake to lower than 10% energy intake is vital to reduce CVD risk.

Key Words high sugar, low sugar, cardiovascular disease, risk factors

A higher consumption of carbohydrate (CHO), mainly in the form of sugars, has been associated with increased CVD risk (*1*). In addition, the replacement of dietary fat with CHO of mixed quality has been linked to increased CVD risk, primarily through the elevation of plasma triglyceride (TG) and reduction of plasma high density lipoprotein (HDL) (*2*). The increase in plasma TG may occur as a result of increased synthesis and secretion of very low density lipoprotein-triglyceride (VLDL-TG) from the liver and/or through the impaired clearance of VLDL, whilst the reduction of plasma HDL is thought to occur with the altered TG metabolism and the remodelling of HDL into smaller particles (*2*).

Variations in the lipid response to dietary CHO have been attributed to the amount of CHO ingested, the presence of other nutrients in foods, and the existence of underlying metabolic disturbances, such as insulin resistance (*2*, *3*). Hyperinsulinaemia and insulin resistance have both been implicated in the pathophysiology of diabetes mellitus type 2 (T2DM), metabolic syndrome, and CVD, and more recently fatty liver disease (*4*, *5*). While a high intake of sugar, which is known to increase de novo lipogenesis (DNL) in the liver (*6*), the presence of hepatic insulin resistance, may be a potent combination for promoting the accumulation of fat in the liver leading to dyslipidaemia. Therefore, we aimed to perform a crossover intervention trial using a dietary exchange model consisting of high and low sugar diets in 25 middle-aged men at increased CVD risk, over 12 wk for each arm and to evaluate the metabolic markers including lipid profile, insulin and glucose.

MATERIALS AND METHODS

Participants' recruitment. The study participants were recruited through several approaches including an existing Clinical Research Network with GP surgeries in the county of Surrey, where by a letter of invitation to participate in the study, and a Participant Information Sheet was given to selected patients; direct contact with local authorities, companies, schools and colleges via email and letters; plus direct approaches to offices and shops to distribute leaflets, and display posters on local community notice boards, and leaflet drops in local residential areas. The health status and screening test results of all participants were checked and approved by the medical consultant prior to their entry into the study.

Study design. The study was of a controlled, randomised cross-over design with two 12-wk dietary interventions, high and low in sugar. Participants who expressed interest in taking part in the study were pre-

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screened by a telephone interview. Suitable participants were screened at the Royal Surrey County Hospital (RSCH) to determine their suitability according to cardio-metabolic risk score, as described in the previous study (3). Before commencing the dietary intervention, participants underwent four weeks of a run-in diet, during which they consumed their habitual diet and maintained their habitual level of physical activity. At the end of the four weeks, participants were randomised to receive either the high or low sugar diet for 12 wk using a dietary exchange model (DEM) as previously published (7). The DEM was developed based on National Dietary & Nutrition Survey (NDNS) data to test the influence of sugars on atherogenic lipoprotein phenotype (ALP). It focused on the carbohydrate exchanges to meet the starch to sugar ratio. At the end of the first intervention, participants returned to their habitual diet for 4 wk of wash-out, at the end of which they crossed-over to the alternative diet. Five home visits were conducted, at fortnightly intervals, on both dietary interventions, to deliver study foods, measure body weight and to check-on dietary compliance.

Study procedures. Participants were required to fast overnight before attending the screening visit, and all subsequent visits to the research centre. At screening visit, participants completed a socio-demographic questionnaire form and provided written consent to take part in the study. Anthropometry and blood pressure were measured and fasting blood samples were taken by the research nurse. The screening blood samples were promptly dispatched to the pathology department at the Royal Surrey County Hospital for analysis. Detailed information about the study objectives and procedures were explained to the participants, and a study handbook was provided. To avoid any changes in behaviour, participants were advised that any results from the study would not be revealed to them until completion of both dietary interventions.

For the intervention study, participants attended an initial visit at the research centre after an overnight fast. Anthropometry and blood pressure were measured, and fasting blood samples were taken. Participants were supplied with a quantity of study foods that was sufficient for two weeks, after which, foods were delivered to their homes at fortnightly intervals. Participants were given food portion sheets to complete on a daily basis for two weeks to monitor compliance. During the interventions, participants were instructed to maintain their habitual level of physical activity. Similar procedures were followed at the start of second intervention.

Anthropometrics and measurement of blood pressure. Height was measured using a wall-mounted stadiometer with shoes off and feet precisely in the designated area on the device. Body weight, BMI and body composition (including body fat, fat mass and free fat mass) were measured by electrical bio-impedance, using a body composition scale. Waist circumference was measured using a circumference measuring tape to the nearest 0.1 cm at the narrowest point between the iliac crest and the lowest rib. Blood pressure was measured using an electronic sphygonometer with the participants resting in a seated position for five minutes; the average of three readings obtained on the participant's left arm constituted the examination blood pressure. A mobile body weight scale was used to measure body weight at home visits.

Biochemistry analysis. Blood samples were taken by venipuncture of an antecubital vein in the forearm, from all participants after they had fasted overnight for a minimum of 12 h. The blood was collected into several different tubes (vacutainers) containing the following anticoagulants; K2EDTA for the determination of TC, TG and HDL-C; fluoride oxalate for plasma glucose, and lithium heparin for plasma insulin. Blood samples were immediately centrifuged at 2,500 rpm for 10 min at 4°C in a low speed centrifuge for the separation of plasma. Aliquots of plasma (0.5 mL) were dispensed into appropriately labelled cryovials, and stored at −80°C before analysis. All analyses were completed within 6 wk.

Measurement of plasma lipids, lipoproteins, insulin and glucose. Assays for plasma TG, TC, HDL-C, and glucose were performed on an ABX Mira auto-analyser (Roche Diagnostics, USA). Pre-calibrations for each assay and low- and high-quality controls (QCs) were included within each test. Assays were only performed if the QCs values were within acceptable confidence limits, as defined by the manufacturer. All samples (pre and post-dietary interventions were analysed within a single batch. Intra-assay CVs (within-run precision) were calculated using six duplicates. Plasma LDL-C was not measured directly on isolated LDL, but was calculated by the Friedewald Equation (8). Plasma insulin was measured by radioimmunoassay (RIA) using the Millipore Human Insulin assay (Merck Millipore, MA, USA).

Ethical clearance. This study complied with the code of ethics of the World Medical Association (Declaration of Helsinki) and was approved by the Surrey Research Ethics Committee (reference number: 08/H1109/227).

Statistical analyses. The mean daily intake of energy and nutrients were calculated from an average of the 3-d diet diaries collected in the run-in, and during both intervention phases.

All analyses were performed using SPSS package version 22 (SPSS for Windows, Chicago, USA). All data were checked for the normality. A paired Student t-test or non-parametric alternative Wilcoxon Signed Rank Test were used to examine the difference in variables before and after each diet. Differences between diets over time were examined by a two-way Analysis of Variance (ANOVA) or Kruskal Wallis (non-parametric alternative) was used for determining differences between diets over time. A non-parametric correlation test (Spearman's rho) test was used to evaluate associations between variables. Analysis of variance (ANOVA) with a Pos-hoc Tukey test for pair-wise differences, was used to analyse the difference between three or more groups. Analysis of covariates (ANCOVA) was used to determine the difference between diets in adjustment for signifi-

Table 1. Characteristics of participants at screening visit.

Characteristics	Mean [SEM]
Age (y)	56.8 [1.3]
Body weight (kg)	89.6 [1.7]
Body mass index (kg/m^2)	28.6 [0.3]
Waist circumference (cm)	103.9 [1.1]
Systolic blood pressure (mmHg)	132.9 [3.1]
Diastolic blood pressure (mmHg)	83.7 [2.1]
Body fat (%)	25.4 [0.6]
Fat mass (kg)	23.0 [0.8]
Free fat mass (kg)	67.1 [1.1]
Triacylglycerol (mmol/L)	1.74 [0.2]
Total cholesterol (mmol/L)	5.68 [0.2]
HDL-Cholesterol (mmol/L)	1.34 [0.1]
LDL-Cholesterol (mmol/L)	3.54 [0.1]
Fasting blood glucose (mmol/L)	5.58 [0.1]
Insulin (pmol/L)	56.1 [4.8]

Table 2. Composition of the habitual, high and low sugar diet intakes.

Nutrient	Habitual diet	High sugar diet	Low sugar diet
Energy (MJ/d)	9.8 [0.5]	10.6 [0.5]	9.9 [0.4]
CHO (g/d)	264 [15.1]	329 [14.9]	257 [12.0]
%Total energy	45 [1.1]	53 [1.3][ac]	44 [1.3][c]
Sugar (g/d)	120 [10.1]	173 [10.0]	56 [4.0]
%Total energy	21 [1.2]	28 [1.3][ac]	9 [0.5][bc]
Protein (g/d)	90 [5.3]	92 [4.5]	97 [4.8]
%Total energy	16 [0.7]	15 [0.5]	16 [0.5]
Fat (g/d)	87 [6.2]	77 [6.4]	89 [5.0]
%Total energy	33 [1.3]	27 [1.5][ac]	34 [1.2][c]
Saturated fat (g/d)	33 [2.8]	30 [3.0]	35 [2.8]
%Total energy	13 [0.7]	10 [0.8][c]	13 [0.8]
Fibre (g/d)	26 [1.4]	21 [1.2][a]	24 [1.5]
Sodium (g/d)	3.0 [0.2]	2.9 [0.2][c]	3.7 [0.2][b]

Data are mean [SEM]. [a] Habitual vs HS, [b] Habitual vs LS, [c] HS vs LS; $p<0.05$ (ANOVA with post-hoc Tukey test).

Table 3. Anthropometric variables and blood pressures at baseline and after the high and low sugar diets.

Variable	Time-point	High sugar diet	Low sugar diet
Body weight (kg)	Baseline	88.5 [1.7]	89.3 [1.8]
	12 wk	89.2 [1.8]	87.2 [1.9]
	Changes	+0.7 [0.3][a]	−2.1 [0.5][b]
BMI (kg/m^2)	Baseline	28.3 [0.3]	28.5 [0.4]
	12 wk	28.5 [0.3]	27.8 [0.4]
	Changes	+0.2 [0.1][c]	−0.7 [0.2][d]
Waist (cm)	Baseline	102.6 [1.1]	103.9 [1.4]
	12 wk	104.0 [1.3]	101.9 [1.2]
	Changes	+1.4 [1.0][e]	−2.0 [0.8][ae]
Body fat (%)	Baseline	25.7 [0.5]	25.9 [0.7]
	12 wk	26.1 [0.5]	25.0 [0.7]
	Changes	+0.4 [0.3]	−1.0 [0.2][b]
Fat mass (kg)	Baseline	22.8 [0.7]	23.3 [0.9]
	12 wk	23.3 [0.7]	21.9 [0.9]
	Changes	+0.5 [0.3][ae]	−1.4 [0.3][be]
Free fat mass (kg)	Baseline	65.7 [1.2]	66.2 [1.3]
	12 wk	65.9 [1.2]	65.2 [1.3]
	Changes	+0.2 [0.2]	−0.9 [0.3][ae]
Systolic BP (mmHg)	Baseline	127.2 [3.1]	129.0 [2.8]
	12 wk	133.5 [3.3]	127.6 [3.6]
	Changes	+4.0 [2.3]	+0.3 [2.3]
Diastolic BP (mmHg)	Baseline	81.7 [2.2]	82.3 [2.0]
	12 wk	83.7 [2.1]	80.2 [2.3]
	Changes	+1.2 [1.8]	−1.7 [1.5]

Values are mean [SEM]. Changes=12 wk–baseline, [a] $p<0.05$, [b] $p<0.01$ (Wilcoxon Signed Rank Test); Correlation with change in BMI, [c] $p<0.05$ (Spearman's Rho).

cantly associated covariates. Statistical significance was defined as $p<0.05$.

RESULTS

The anthropometric characteristics and blood biochemistry of the participants at screening are shown in Table 1. Participants included were controlled for age, BMI, physical activity level and medical history including smoking status and metabolic profile. All participants' were categorised as overweight/obese with BMI ranged between 26–32 kg/m^2.

Intake of energy and macronutrients

Table 2 shows the habitual intake, and the actual intake on each diet. There was no difference in total energy and protein intakes between the intervention diets. There were significant differences in the intake of dietary CHO, sugars, fat, saturated fat and sodium intakes between the high and low sugar diets.

Effects of high and low sugar diets on anthropometric variables and blood pressure

Body weight and BMI both increased (0.8% and 0.7%, $p<0.05$) after the high sugar diet, both and decreased (2.4%, $p<0.05$ and 2.5%, $p<0.01$) after the low sugar diets, respectively (Table 3). There was no relationship between body weight or BMI and total energy intake on either the high or low sugar diets. Waist circumference, percentage body fat, fat mass, and free fat mass all increased relative to baseline, after the high sugar diet, but all three of these variables decreased after the low sugar diet. There were significant positive associations between changes in BMI, waist circumference and fat mass on the high sugar diet and negative associations on the low sugar diet, and also an inverse relationship between the changes in BMI and free fat mass on the low sugar diet ($p<0.05$). There was no significant change in systolic or diastolic BP after either diet.

Effects of the high and low sugar diets on plasma lipid, lipoproteins, glucose and insulin

Plasma TG increased significantly after the high sugar diet (mean change±SEM=+0.26±0.07; $p=0.001$) and decreased after the low sugar diet (mean change±

Table 4.   Plasma concentrations of lipids, lipoproteins, glucose and insulin before and after the high and low sugar diets.

		High sugar diet			Low sugar diet		
		Mean	SEM	Paired *t*-test	Mean	SEM	Paired *t*-test
Plasma TG[a]	Pre	1.49	0.14		1.83	0.25	
(mmol/L)	Post	1.74	0.15		1.48*	0.13	
	Changes	0.26	0.07	*p*=0.001	−0.35	0.16	n.s.
Plasma TC	Pre	5.47	0.17		5.57	0.19	
(mmol/L)	Post	5.31	0.20		5.01*	0.20	
	Changes	−0.16	0.12	n.s.	−0.56	0.13	*p*=0.04
Plasma HDL[a]	Pre	1.30	0.06		1.30	0.05	
(mmol/L)	Post	1.19	0.06		1.16	0.05	
	Changes	−0.11	0.03	*p*=0.002	−0.14	0.02	*p*=0.000
Plasma LDL	Pre	3.49	0.14		3.44	0.16	
(mmol/L)	Post	3.33	0.15		3.18	0.17	
	Changes	−0.16	0.11	n.s.	−0.26	0.11	*p*=0.035
Plasma glucose	Pre	5.67	0.15		5.73	0.13	
(mmol/L)	Post	5.91	0.11		5.85	0.12	
	Changes	0.25	0.09	*p*=0.014	0.12	0.11	n.s.
Plasma insulin	Pre	126.89	8.26		113.35	6.17	
(pmol/L)	Post	115.94	6.20		115.92	8.52	
	Changes	−10.94	7.35	n.s.	2.57	8.10	n.s.

[a] Data were tested using log transformed values. n.s., not significant. * Difference between diets adjusted for baseline values ($p<0.05$) (ANCOVA).

SEM$=-0.35\pm0.16$; n.s.) (Table 4). There were strong associations between the concentrations of plasma TG at baseline and after the intervention on both diets ($p<0.001$). After normalisation and adjustment of the data for baseline values, there was a significant difference in plasma TG following the high and low sugar diets, that was independent of BMI, percentage of body fat, and plasma TC ($p<0.001$).

Mean plasma HDL-C decreased after the high and low sugar diets, by 8.5% ($p<0.01$) and 10.8% ($p<0.01$), respectively. In contrast to plasma TG, plasma HDL-C decreased consistently with an increasing contribution of energy from sugar, on the high sugar diet, whereas HDL-C increased in response to a decreasing contribution of energy from sugar on the low sugar diet. Plasma TC was significantly lower after the low sugar diet relative to the high diet (by 0.3 mmol/L, $p<0.05$) after adjustment for baseline values. Plasma TC decreased significantly after the low sugar diet by 10% ($p<0.05$) relative to baseline. Plasma LDL-C also decreased significantly after the high and low sugar diets by 4.6% ($p<0.05$). There was evidence of positive associations between changes in plasma TG, TC and plasma insulin after the high sugar diet, and a positive association between the changes in plasma TG and plasma glucose after the low sugar diet ($p<0.05$).

Plasma glucose increased by 4.4% relative to baseline after the high sugar. There was no significant difference in plasma glucose between the high and low sugar diets after 12 wk, and no change in plasma insulin after the high and low sugar diets.

DISCUSSION

This study had tested the effects of 12-week of high versus low sugar diets on anthropometric and cardio-metabolic profiles among 25 Caucasian men at increased cardio-metabolic risk and had observed several significant findings. While body weight should be maintained by a balance between energy intake and expenditure (9), paradoxically, there was a significant increase and decrease in body weight after the high and low sugar diets, respectively, without any significant change in energy intake. It is possible that the observed changes in body weight were influenced by a lower and higher energy expenditure on the high and low sugar diets, respectively. Unfortunately, energy expenditure was not measured. Another obvious explanation would be because of the underreporting of energy intake. While it is possible that the changes in body weight confounded the effects on plasma lipids and glycaemic control, a weight loss of between 5–10% has been shown to be required to influence plasma lipids and glycaemic control (10, 11), and the present study only reported changes of around 2%.

The reciprocal changes in body weight after each diet were paralleled by an increase in waist circumference, body fat and fat mass after the high sugar diet, and a decrease in the same variables after the low sugar diets. This might suggest that the changes in adiposity were regionalised in the intra-abdominal area, which is more relevant with respect to the generation of cardio-metabolic risk (12). Increased abdominal obesity has been frequently associated with insulin resistance, increased intracellular lipolysis and elevated postprandial plasma

non-esterified fatty acids (NEFA), which, in theory, may increase lipid substrate availability for TG synthesis in the liver (13). Conversely, a reduction in abdominal adiposity has been associated with beneficial effects on plasma lipids and insulin (14, 15). Abdominal obesity has also been linked to high blood pressure, whilst a decrease in waist circumference has been associated with reduced blood pressure (16). Previous studies have indicated that an increased intake of sugar might raise blood pressure (17), though there was no evidence for this in the present study. It is possible that an increase in both systolic and diastolic blood pressures after the high sugar diet was associated with the increased BMI and waist circumference. It is also possible that any favourable effect of the weight loss and reduced waist circumference on blood pressure (diastolic BP only) on the low sugar diet was counter affected by the increased intake of sodium on this diet (18). An increase in salt intake of 1 g/d (0.4 g/d of sodium) has been previously associated with an increase of 0.4 mmHg in systolic blood pressure (19).

Effects of high and low sugar diets on plasma lipids, lipoproteins, glucose and insulin

Elevated plasma TG is the most common lipid abnormality in participants at increased cardio-metabolic risk, and may exert direct and indirect effects on CVD risk. After adjusting for the interaction between diet and body weight change, the difference between plasma TG remained significant after both diets. This finding further supports the idea that the weight changes achieved in the participants were insufficient to affect plasma TG. There is a convincing body of evidence to suggest that an increased intake of dietary sugar intakes (>20% of energy) raises plasma TG by ≈60% (20). Previous studies concluded that dietary sugars, especially in the form of fructose increased plasma TG significantly. The present study produced results which corroborated these findings. The effect of dietary sugars on plasma TG were mostly significant in short and intermediate term studies (≤2 wk), whereas evidence from long-term studies was still lacking. Saris et al. observed a non-significant increase in plasma TG after six months of ≈29%E sugar intake (21). This non-significant increase may have been related to metabolic adaptation to a high intake of sugar in same participants, and a metabolic effect of dietary sugar that is dissipated over time (20, 22). It is reasonable to speculate that metabolic compensation to the high sugar diet, results in the reduction of fat oxidation in the liver, which promotes the accumulation of fat in this organ (23). Another possible explanation may be through reduced compliance on the long-term dietary intervention in some participants. The present study, which employed a high sugar intake for 12 wk, produced a significant increase in plasma TG, suggesting that the effect of dietary sugars on plasma TG becomes evident within three months.

In contrast to the potentially adverse effect of a high sugar diet on plasma TG, a diet low in dietary sugars produced the opposite, and potentially beneficial, effect on CVD risk by lowering plasma TG. This finding may be explained by the fact that a low sugar diet (9%E from sugar) decreased the production of TG and glucose in the liver via increased insulin sensitivity, together with a limitation on the availability of plasma NEFA from the periphery for TG production (22).

Correlation analysis also revealed associations between baseline and post-intervention plasma TG, suggesting an order-effect on plasma TG, though further analysis indicated that the post-diet plasma TG was significantly different between the high and low sugar diets, and were independent of baseline values. Consideration has also been given to other dietary confounding factors, such as dietary fat ($p<0.01$) and fibre (n.s.), since these were significantly different between the two diets. However, there was no significant relationship between plasma TG and energy derived from fat or dietary fibre intake in both diets.

Plasma HDL is well known to be inversely associated with CVD risk, and a low HDL concentration has been established as an independent negative risk factor of CVD (24). Findings from the present study are consistent with previous studies (25, 26), in showing a non-significant decrease in HDL-C after a high sugar diet. While a decrease in plasma HDL after the high sugar diet was an expected outcome, HDL also decreased after the low sugar diet. The HDL-lowering effect of a high sugar diet is believed to be mediated through the increase in plasma TG and the subsequent remodelling of HDL into smaller particles, which are then cleared from the plasma more rapidly (27). The effect of the low sugar diet on HDL is more difficult to interpret, and may be linked to changes in body weight or other diet and lifestyle factors that were not assessed. In contrast to a previous study (28), there was no association between age, BMI, physical activity level, smoking status and plasma HDL in the present study, perhaps suggesting a direct effect of the high sugar diet on the reduction on plasma HDL. A low intake of sugars has also been associated with raised HDL (29). However, despite a loss of body weight and reduction in TG and sugar intake in the present study, HDL was, unexpectedly, decreased after the low sugar diet. This finding is not unique, and has been reported previously (30). Possible explanations for the fall in HDL include; a decrease in apolipoproteins associated with HDL, and/or an increase in plasma insulin (31). This situation would decrease the synthesis and secretion of HDL, leading to a reduction in plasma HDL, though there was no evidence to support either of these effects.

Plasma TC and LDL is a classic biomarker of CVD risk. In the present study, the high sugar diet was accompanied by reductions in both plasma TC and LDL. This finding would be consistent with an LDL-lowering effect produced by a lower intake of saturated fat which was evident on the high sugar diet (−23%), and as previously reported (32). However, there was also evidence of a significant reduction in plasma LDL after the low sugar diet, but no change in the intake of saturated fat. It is possible that this finding was due to a high intake of dietary fibre on the low sugar diet, particularly in the

form of soluble fibre (*33*). In the present study, both diets produced unexpected, non-significant changes in plasma insulin. A high intake of sugar has also been shown to impair glucose tolerance, especially in population at increased cardio-metabolic risk (*34*, *35*). An increase in fasting glucose after the low sugar diet was an interesting finding. However, the change was small (2%) and insignificant, and may be not reflecting the actual effect.

CONCLUSION

The high sugar diet was associated with adverse effects on plasma TG, HDL, BMI, waist circumference and fasting glucose, leading to increased CVD risk, whilst the low sugar diet was associated with benefits on these risk factors. In conclusion, reducing sugar intake may be useful in managing CVD risks, especially in people with increased cardio-metabolic risk, who stand to gain greater benefits from this dietary modification.

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