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ARTICLE

The relation of low glycaemic index fruit consumption to glycaemic control and risk factors for coronary heart disease in type 2 diabetes

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Abstract
Aims/hypothesis Sugar has been suggested to promote obesity, diabetes and coronary heart disease (CHD), yet fruit, despite containing sugars, may also have a low glycaemic index (GI) and all fruits are generally recommended for good health. We therefore assessed the effect of fruit with special emphasis on low GI fruit intake in type 2 diabetes.
Methods This secondary analysis involved 152 type 2 diabetic participants treated with glucose-lowering agents who completed either 6 months of high fibre or low GI dietary advice, including fruit advice, in a parallel design.
Results Change in low GI fruit intake ranged from −3.1 to 2.7 servings/day. The increase in low GI fruit intake significantly predicted reductions in HbA_{1c} ($r=-0.206$, $p=0.011$), systolic blood pressure ($r=-0.183$, $p=0.024$) and CHD risk ($r=-0.213$, $p=0.008$). Change in total fruit intake ranged from −3.7 to 3.2 servings/day and was not related to study outcomes. In a regression analysis including the eight major carbohydrate foods or classes of foods emphasised in the low GI diet, only low GI fruit and bread contributed independently and significantly to predicting change in HbA_{1c}. Furthermore, comparing the highest with the lowest quartile of low GI fruit intake, the percentage change in HbA_{1c} was reduced by −0.5% HbA_{1c} units (95% CI 0.2–0.8 HbA_{1c} units, $p<0.001$).
Conclusions/interpretation Low GI fruit consumption as part of a low GI diet was associated with lower HbA_{1c},

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blood pressure and CHD risk and supports a role for low GI fruit consumption in the management of type 2 diabetes.
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Keywords CHD risk · Dietary guidelines · Dietary sugars · Fructose · Fruit · GI · HbA_{1c} · HDL-cholesterol · Low glycaemic index · Type 2 diabetes

Abbreviations

CHD	Coronary heart disease
GI	Glycaemic index
TG	Triacylglycerol

Introduction

Increased sugar intake and, more recently, high fructose consumption, especially from high fructose corn syrup, has attracted attention for its potential negative impact on health. Concerns have been raised especially in respect to body weight control and increased risk of diabetes and coronary heart disease (CHD) [1–7]. On the other hand, sugars in fruit are viewed in a very different light and the public are recommended to eat more fruit, together with vegetables and wholegrain cereals, as part of general dietary advice in order to maintain health and avoid specific diseases such as diabetes, cardiovascular disease and cancer [8–12].

This apparent contradiction in relation to the effect of sugars may be due in part to the fibre and cell wall structure of fruit, which limits the rate of sugar absorption in the gastrointestinal tract resulting in flatter glycaemic responses [13].Thus, a flatter glycaemic response has been seen after consumption of whole fruit when compared with fruit puree and even more so when compared with drinking fruit juice [13, 14].

Fruit in general have a glycaemic index (GI) that ranges from 56 to 103 GI units (on the bread scale). We hypothesised that selection of those at the lower end of the range may provide the greatest benefit in reducing the overall glycaemic response. As a result we emphasised the use of low GI fruit in a previously published study examining the role of a low GI diet in type 2 diabetes. This study has now provided us with the opportunity to assess the relation of low GI fruit intake with the metabolic changes observed as part of the overall low GI diet [15].

Methods

Participants Details of the study protocol have been reported previously [15]. Recruitment took place from May 2004 to December 2006, with the last follow-up visit at the end of May 2007. Of those recruited, 210 were found to be eligible and were randomised. Eligible participants were men or postmenopausal women with type 2 diabetes who were taking oral agents to control their diabetes, with medications stable for the previous 3 months and who had HbA_{1c} values at screening between 6.5% and 8.0% (Table 1). None had clinically significant cardiovascular, renal or liver disease (alanine transaminase > three times the upper limit of normal) and none was undergoing treatment for cancer. Individuals were accepted after surgery or myocardial infarction providing an event-free 6 month period had elapsed prior to the study. This study is a secondary analysis, but differs from the original study [15] in that it focuses on the 152 participants who completed the study and also provided 7 day food records, which were used to determine fruit intake.

The study was approved by the research ethics board of St Michael's Hospital and the University of Toronto, and written consent was obtained from all participants.

Protocol In this secondary analysis of completer data from a previously published study [15], participants were randomised to one of two parallel 6 month treatments: a low GI diet or a high cereal fibre diet. During the study, equally strong emphasis was placed by dietitians on the potential value of both treatments.

Participants were seen at the Clinical Nutrition and Risk Factor Modification Center of St Michael's Hospital, a University of Toronto Teaching Hospital, at baseline, weeks 2 and 4, and thereafter at monthly intervals until the end of the 6 month period. During the first month, participants received instructions on the diet to which they were allocated. At all centre visits, participants were weighed in indoor clothing without shoes and a fasting blood sample was taken. Blood pressure was measured seated on three occasions at 1 min intervals using an Omron automatic sphygmomanometer (OMRON Healthcare, Burlington, ON, Canada) and the mean of the three measurements was taken. In addition, participants brought with them their 7 day food records covering the week prior to the visit and these were discussed with the dietitians.

During the study, participants were asked to maintain their exercise pattern and keep their glucose-lowering agents constant throughout the study.

Dietary interventions General dietary advice conformed to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) and the ADA guidelines [16] to reduce saturated fat and cholesterol intakes [17]. Of all the participants, 84.9% were overweight (BMI ≥25 kg/m^2) and 50.7% were obese (BMI ≥30 kg/m^2) and wished to lose weight. They were informed that this was not a weight loss

Table 1 Baseline characteristics of study participants

Characteristic	High cereal fibre (n=73)	Low GI (n=79)	Significance of difference p value
Age (years)	62±9	61±10	0.669
Sex (male/female, n/n)	45/28	51/28	0.305/0.553
Weight (kg)	86±16	87±21	0.885
BMI (kg/m^2)	31±5	30±6	0.732
Ethnicity, n (%)			
European	46 (63)	57 (72)	0.162
Indian	14 (19)	11 (14)	0.345
Far Eastern	3 (4)	6 (8)	0.254
African	8 (11)	3 (4)	0.113
Hispanic	1 (1)	2 (3)	0.500
Native American	1 (1)	0 (0)	0.500
Smokers, n (%)	2 (3)	8 (10)	0.055
Glucose (mmol/l)	7.56±1.52	7.64±1.75	0.767
HbA_{1c} (%)	7.03±0.46	7.17±0.57	0.100
No. participants <7	36	29	0.229
No. participants ≥7	37	50	0.099
Total cholesterol (mmol/l)	4.24±0.75	4.18±0.95	0.661
LDL-cholesterol (mmol/l)	2.48±0.64	2.47±0.89	0.934
HDL-cholesterol (mmol/l)	1.14±0.28	1.06±0.31	0.103
TG (mmol/l)	1.36±0.71	1.42±0.79	0.643
Systolic blood pressure (mmHg)	128±14	127±16	0.727
Diastolic blood pressure (mmHg)	74±9	73±10	0.846
Duration of diabetes (years)	7±6	9±7	0.062
Hypoglycaemic agents, n (%)	73 (100)	77 (97)	0.403
Thiazolidinedione	25 (34)	25 (32)	0.556
Biguanide	58 (79)	63 (80)	0.358
Sulfonylurea	29 (40)	49 (62)	0.015
Meglitinides (non-sulfonylurea)	2 (3)	1 (1)	0.500
Alpha glucosidase inhibitors	2 (3)	3 (4)	0.500
Cholesterol-lowering medications, n (%)	46 (63)	55 (70)	0.539
Blood pressure medications, n (%)	52 (71)	52 (66)	0.213

Values are mean ± SD unless stated otherwise

Differences in categorical variables were assessed by binomial tests of equality

Differences in continuous variables were assessed by two-sample t test

study but appropriate advice was given on portion size and fat intake to help them meet their body weight objectives. Participants were also provided with a checklist with either low GI or high cereal fibre food options as approximately 15 g carbohydrate servings. The number of carbohydrate servings prescribed covered 42–43% of total dietary energy. Three servings of fruit and five servings of vegetables were encouraged on both treatments. On the low GI treatment, the carbohydrate intakes emphasised were low GI breads, breakfast cereals, parboiled rice, pasta, beans, barley, bulgar and low GI fruit. Temperate climate fruit, which are generally low GI, were the focus and included apples, pears, citrus fruit (oranges, tangerines and grapefruit), berries (strawberries, raspberries, cranberries, blackberries and blueberries) and the *Prunus* family (nectarines, peaches and plums). On the cereal fibre treatment, the focus was on wholewheat breads, breakfast cereals and tropical fruit with glycaemic indices that were closer to that of the average diet, such as bananas, mangoes, guavas, grapes, raisins, watermelon and cantaloupe. Low GI or temperate climate fruit had GI values of <70 GI units (bread scale), with the exception of blueberries, with a value of 76 GI units, based on recent values for individuals without diabetes [18]. Higher GI fruit, predominantly tropical fruit, had values >70 GI units [18]. Participants were also advised against eating fruit recommended on the alternative treatment. Checklists were completed by participants on a daily basis throughout the study and 7 day diet records were completed prior to each visit. Adherence was assessed from the 7 day diet records. The overall aim was to achieve a 10–20% reduction in GI on the low GI diet while keeping dietary fibre similar between treatments.

Biochemical analyses Blood glucose was measured in the hospital routine analytical laboratory by a glucose oxidase method using a Random Access Analyzer and reagents (SYNCHRON LX Systems, Beckman Coulter, Brea, CA, USA) (CV 1.9%). HbA_{1c} was analysed by a designated HPLC method (Tosoh G7 Automated HPLC Analyzer, Grove City, OH, USA) (CV 1.7%). Serum was analysed for total cholesterol, triacylglycerol (TG) and HDL-cholesterol, also using a Random Access Analyzer (CV 1.5–2.4%). Diets were assessed for available carbohydrate (total carbohydrate–fibre) using a computer program based on US Department of Agriculture data [19].

Statistical analyses The primary outcome was HbA_{1c}, with glucose, total cholesterol, LDL-cholesterol, HDL-cholesterol, TG, blood pressure, body weight and CHD risk as secondary measures. Analyses were undertaken on individuals who completed the study and also provided diet records at the start of and during the study (n=152).

Baseline data are expressed as means ± SDs. All other data are expressed as means (95% CI). All analyses were carried out using SAS software, version 9.2 [20].

Pearson correlations as well as partial correlations controlling for body weight change and change in fibre intake were undertaken to determine the relation of low GI fruit to measures of glycaemic control and CHD risk. The data from the two treatments were pooled and both the absolute differences in servings of fruit, and the carbohydrate from fruit expressed as a percentage of the total carbohydrate, were related to the percentage changes from baseline in the outcome measures. Overall 10 year CHD risk was calculated according to the Framingham cardiovascular risk equation [21]. In our current analyses, only raw and frozen fruit were included. We excluded processed fruit products such as juices, canned fruit and jams as unmodified fruit was the focus of our assessment. Two-sample Student's t test was used to assess differences between treatments at baseline and between changes across treatments. Binomial tests of equality were used to assess differences at baseline for categorical variables.

Participants were also divided into four equal groups based on the magnitude of the change they made in low GI fruit intake, expressed as a percentage of daily available carbohydrate from fruit [(available carbohydrate from fruit ÷ total available carbohydrate in the diet) × 100]. The significance of differences between those in the upper quartile of change in low GI fruit intake vs those in the lowest quartile was assessed using an ANOVA model (Proc GLM in SAS version 9.2) [20], with percentage change in measurements as the response variable.

Finally, to assess the contribution of low GI fruit to the absolute change in HbA_{1c}, as the primary outcome in the context of the other major low GI food components, a regression analysis was undertaken in SAS using an ANOVA model. In this analysis, the assessment of each dietary component was carried out in a model adjusted for change in fibre (g/kJ or kcal) and total fruit intake (% of available carbohydrate). The eight individual low GI dietary components were fruit, bread, breakfast cereals, pasta, beans, parboiled rice, barley and bulgar, each expressed as a percentage of total carbohydrate.

Results

Of the 210 individuals randomised, 155 completed the study [15] and dietary records for both pretreatment and end of treatment were available for 152 participants. At baseline, individuals taking either high cereal fibre or low GI diets were similar in terms of physical characteristics, ethnicity, smoking status, glycaemic and lipid control and medication use, with the exception of higher sulfonylurea use by the low GI diet group (Table 1).

Fruit consumption At baseline, participants were consuming 1.4 servings (95% CI 1.2–1.6) of raw and frozen fruit daily. The most commonly consumed fruits were bananas followed, in descending order, by apples, oranges, pears, tangerines and berries (Fig. 1). Low GI fruit accounted for 0.7 servings/day (95% CI 0.5–0.9) and 0.7 servings/day (95% CI 0.5–0.9) came from higher GI fruit.

At the end of 6 months, participants following the low GI diet increased their low GI fruit consumption from 0.7 (95% CI 0.6–0.9) to 1.3 (95% CI 1.1–1.5) servings/day (p<0.001). On the high cereal fibre diet low GI fruit consumption was reduced from 0.8 (95% CI 0.6–0.9) to 0.3 (95% CI 0.2–0.4) servings/day (p<0.001). Total fruit intake remained similar on both the low GI and high fibre diets (2.0 servings/day for both).

On the low GI diet, the change in total fruit and low GI fruit ranged from −3.5 to 3.0 servings/day and −1.9 to 2.7 servings/day, respectively. On the high fibre diet, the respective figures were total fruit −3.7 to 3.2 servings/day and low GI fruit −3.1 to 0.5 servings/day. As this is a secondary analysis, pooling the data from both treatments was undertaken. The changes in total fruit and low GI fruit intake ranged from −3.7 to 3.2 servings per day and −3.1 to 2.7 servings per day, respectively, a substantial span of approximately six servings of low GI fruit per day.

Relation of changes in total and low GI fruit intake to changes in measures of glycaemic control and CHD risk factors No significant associations were seen between total fruit intake and changes in measures of glycaemic control and CHD risk factors.

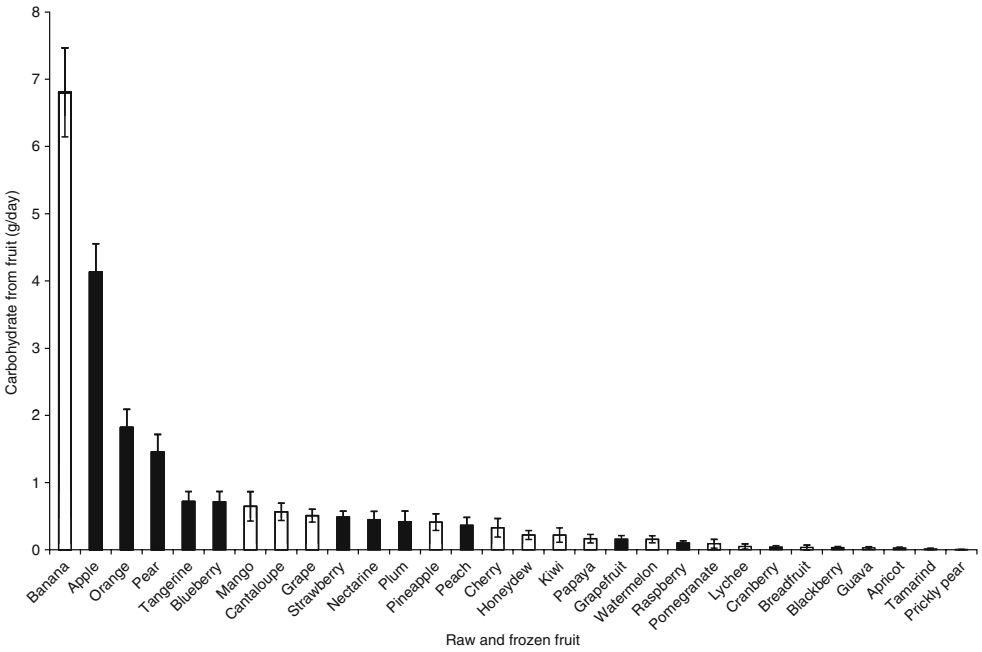

Fig. 1 The distribution of average daily carbohydrate (g/day) from consumption of individual fruit for all participants at baseline (n=152). Black bars, low GI fruit; white bars, higher GI fruit

Low GI fruit intake, expressed as servings, was related to a reduction in HbA_{1c} (%) (r=−0.206, p=0.011), systolic blood pressure (r=−0.183, p=0.024) and CHD risk (r=−0.213, p=0.008). Expressed as a percentage of total carbohydrate intake there was again a negative relation with HbA_{1c} (r=−0.218, p=0.007) and calculated CHD risk (r=−0.192, p=0.018), but a positive relation with HDL-cholesterol (r=0.216, p=0.008, Table 2).

Controlling for the effect of change in body weight in a partial correlation analysis did not alter the associations between the change in low GI fruit as percentage of total carbohydrate consumed and the reduction in HbA_{1c} (r=−0.269, r=0.001). This assessment suggested that the effect of low GI fruit on HbA_{1c} was largely independent of weight loss.

Relation of changes in individual fruit intake to changes in measures of glycaemic control and CHD risk factors Individually, citrus fruit and berry consumption as a percentage of total carbohydrate intake was related to a reduction in the primary outcome, HbA_{1c} (Table 2). Apple consumption correlated negatively with TG, the total cholesterol/HDL-cholesterol ratio and CHD risk, and positively with HDL-cholesterol. Berry intake was related negatively to glucose and blood pressure but positively to TG. No significant associations were seen with pear and prunus fruit consumption.

Glycaemic control and body weight in the highest and lowest quartiles Participants were divided into quartiles according to the magnitude of their change in low GI fruit intake across the 6 month treatment period (Electronic supplementary material [ESM] Tables 1–3). There was a relative difference of 2.2 servings/day (95% CI 1.9–2.4, p<0.001) between the changes in low GI fruit intake for the highest and lowest quartile (ESM Table 3), and an absolute difference of 1.6 servings/day (95% CI 1.2–1.9, p<0.001) at the end of the study.

The highest quartile of low GI fruit showed a significantly greater reduction from baseline in HbA_{1c} (7.2% to 6.5% or a mean reduction of −0.8% HbA_{1c} units, 95% CI −1, −0.5, p<0.001) compared with the lowest quartile (7.0% to 6.7% or a mean reduction of −0.3% HbA_{1c} units, 95% CI −0.5, −0.1, p=0.01). These two changes were significantly different from each other, with a mean difference of 0.5% HbA_{1c} units, 95% CI 0.2–0.8, p<0.001 (equivalent to a −6% percentage reduction in HbA_{1c}; Fig. 2).

No significant differences were seen in the changes in body weight or BMI. Even after adjusting the quartiles of low GI fruit intake according to change in BMI, the significance of the treatment difference previously observed in HbA_{1c} remained unaltered. Also, the increase in fibre intake was greatest in the fourth quartile of low GI fruit intake. Furthermore, after adjustment for the change in fibre intake, the increase in low GI fruit was still associated with an improvement in HbA_{1c} (r=−0.21, p=0.009).

Serum lipids, blood pressure and CHD risk in highest vs lowest quartiles HDL-cholesterol was significantly increased by 7.3% (95% CI 1.2–13.3%, p=0.019; ESM Table 3) on the highest quartile of low GI fruit (a change of 0.05 mmol/l, 95% CI 0.03–0.08, p=0.045) compared with

Table 2 Association of low GI fruit intake with study measurements in 152 completers

Study outcomes %Δ week 24–0	Value	Change in fruit intake (% of total available carbohydrate intake)					
		Apples	Citrus (oranges, tangerines, grapefruits	Berries (strawberries, raspberries, blueberries, blackberries, cranberries)	Pears	Prunus family (plum, peaches, nectarines)	Total low GI fruit
HbA_{1c}	*r*	−0.135	−0.219	−0.228	0.121	−0.073	−0.218
	p	0.096	0.007	0.005	0.136	0.372	0.007
Glucose	*r*	−0.125	−0.008	−0.167	−0.014	−0.030	−0.141
	p	0.124	0.918	0.040	0.863	0.715	0.083
Weight	*r*	−0.016	0.112	−0.096	0.123	−0.136	−0.014
	p	0.846	0.170	0.239	0.132	0.095	0.865
Total cholesterol	*r*	−0.098	−0.001	0.019	−0.052	0.103	−0.020
	p	0.228	0.990	0.813	0.522	0.208	0.804
LDL-cholesterol	*r*	0.013	−0.007	−0.070	−0.009	0.059	0.007
	p	0.872	0.928	0.395	0.911	0.473	0.930
HDL-cholesterol	*r*	0.223	0.156	−0.105	0.098	0.060	0.216
	p	0.006	0.055	0.199	0.231	0.459	0.008
TG	*r*	−0.210	−0.069	0.233	−0.090	0.103	−0.070
	p	0.009	0.396	0.004	0.268	0.208	0.394
C-reactive protein	*r*	0.031	−0.004	−0.071	0.151	0.050	0.065
	p	0.716	0.960	0.403	0.075	0.559	0.443
Systolic blood pressure	*r*	−0.017	−0.006	−0.302	−0.035	−0.034	−0.122
	p	0.839	0.940	0.000	0.666	0.682	0.134
Diastolic blood pressure	*r*	−0.017	−0.140	−0.162	0.035	0.069	−0.067
	p	0.833	0.086	0.046	0.667	0.400	0.410
CHD risk	*r*	−0.211	−0.089	−0.067	−0.099	0.039	−0.192
	p	0.009	0.274	0.409	0.223	0.635	0.018

the lowest quartile (a change of −0.03 mmol/l, 95% CI −0.15, 0.11, p=0.217). Similarly, the relative reduction in systolic blood pressure between quartiles was −4% (95% CI −8, 0%, p=0.044; ESM Table 3), as was the calculated 10 year CHD risk (−13%, 95% CI −23, −3%, p=0.010).

Contribution of low GI fruit to the overall low GI intervention Assessment of the contribution of the components of the low GI diet to the change in HbA_{1c} was carried out using a regression model, controlling for change in fibre and total fruit intake. Only low GI fruit and low GI bread intake were significant predictors (r=−0.233, p=0.0017 and r=−0.228, p=0.002, respectively; Table 3). When both low GI fruit and low GI bread were assessed in the same regression model, both independently predicted the change in HbA_{1c} (p=0.031 and p=0.038, respectively).

Discussion

In this secondary analysis of a low GI study, consumption of two additional daily servings of low GI fruit (the difference between the lowest and highest quartiles of intake) was associated with a significant benefit in glycaemic control, blood lipids and blood pressure. The effect of altering the nature of the fruit eaten has not previously been assessed in diabetes to our knowledge, but may have benefits for both micro- and macrovascular disease, the treatment of which is the major therapeutic goal for type 2 diabetes.

Despite dietary advice to the general public to eat more fruit and vegetables and encouraging data from cohort studies indicating less cardiovascular and cerebrovascular disease [12, 22–25], the results of the few randomised controlled trials of the impact on cardiovascular disease and cancer have been disappointing [26–29]. However, fruit advice has been general and has not focused on low GI fruit [26–29].

On the other hand, very small increases in fructose intake of 7–10 g (a ‘catalytic’ amount) have been shown to prime glucose metabolism, reducing postprandial glucose concentrations [30–33] and increasing liver glycogen synthesis threefold by increasing flux through glycogen synthase, assessed by magnetic resonance spectroscopy [34]. At the

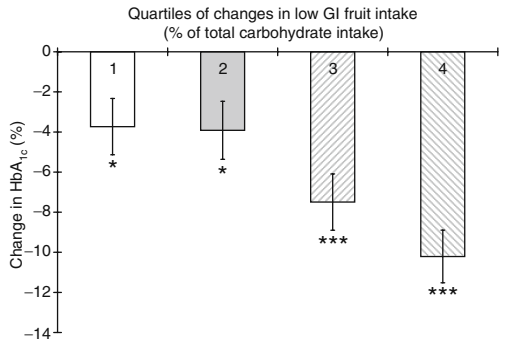

Fig. 2 Changes in HbA_{1c} (%) for four quartiles of change in low GI fruit intake. The difference between extremes of quartiles (quartile 1 vs quartile 4) was significant ($p=0.001$). Within-treatment differences were assessed by paired t test (*$p\leq0.05$ and ***$p\leq0.001$). Different coloured bars with quartile numbers at the top of the bars represent different quartiles of change in low GI fruit intake

same time, it has also been demonstrated that low-dose fructose infusion restores the inhibitory effect of hyperglycaemia in reducing net hepatic glucose output in type 2 diabetes, possibly by increasing fructose-1-phosphate. In turn, fructose-1-phosphate displaces glucokinase from its nuclear regulatory protein and allows its translocation to the cell surface to facilitate portal glucose uptake and its retention within hepatocytes [35]. It may be, therefore, that the increase in low GI fruit, by releasing an additional 6 g or more of fructose from the small intestine into the circulation over an extended period of time, has a disproportionately large effect in reducing postprandial blood glucose excursions.

The situation is very different for large amounts of fructose (17–25% of dietary energy intake) incorporated into sweetened beverages, baked goods and breakfast cereals [6, 36–40]. Early on, high fructose intakes were associated with increased TG levels [36]. Later studies noted increases in LDL-cholesterol [6, 37, 38]. Most recently, raised postprandial TG responses have been reported after high fructose consumption, especially in men, together with increased remnant particle concentrations, more visceral fat and impaired carbohydrate tolerance [6]. These effects of high fructose intake over time would be expected to increase the risk of diabetes and cardiovascular disease. At more modest intake levels, sucrose and fructose intake have not been associated with increased CHD risk [41–43].

Fruits in general are also sources of fibre, minerals, antioxidants and phenolics, which may reduce serum lipids and oxidative damage, lower blood pressure, improve diabetes control and, over time, decrease CHD outcomes. However, definitive roles for all these components remain to be established [44–48]. Furthermore their relevance to the present study is less clear as it was the nature (GI) rather than the quantity of fruit eaten that was altered. On the other hand, cohort studies have assessed the effect of dietary GI on diabetes incidence and CHD [41–43, 49, 50] and significant positive associations have been found in the larger studies [41, 49, 50]. Nevertheless, the nature of the individual fruit consumed was not reported in these studies [41–43, 49, 50].

A weakness of the present study may be seen as singling out low GI fruit for detailed assessment when low GI fruit consumption was only one of the strategies used to reduce the overall GI of the diet. Nevertheless, in regression analysis involving all eight components of the low GI diet, low GI fruit intake was one of only two independent determinants of change in HbA_{1c}. This association remained even after adjustment for fibre and total fruit intake. In addition, weight loss was also present on both the low GI and high fibre treatments. However, correction for body weight change in a partial regression analysis did not alter the significance of associations previously seen with simple Pearson correlations between low GI fruit intake and HbA_{1c} and calculated CHD risk. Finally, although fruits are of special interest for a number of reasons, including their role as a natural source of fructose in the diet, there has been great difficulty in increasing fruit intake, despite universal advice to the public.

The strengths of the study included the first attempt to define the health benefits of individual fruit in type 2 diabetes, the detailed dietary recording—which has allowed the type of fruit consumed in the diet to be clearly identified and the amounts determined—and the substantial participant numbers, which enabled statistical significance to be established.

In conclusion, the data suggest that selection of low GI fruit is associated with improvement in HbA_{1c}. Such changes may also favourably affect HDL-cholesterol, blood pressure and overall CHD risk. Further studies are required to confirm these findings and determine optimal levels of fruit consumption to maximise glycaemic control.

Table 3 Regression analysis of total fruit, fibre and key low GI diet components against change in HbA_{1c}

Key diet components[a]	Adjusted r	Regression model, p value	Key component, p value
Low GI fruit	−0.233	0.011	0.0017
Low GI bread	−0.228	0.012	0.002
Parboiled rice	−0.069	0.299	0.124
Legumes	−0.047	0.346	0.157
Low GI cereal	−0.032	0.372	0.176
Pasta	−0.050	0.456	0.252
Bulgar	−0.052	0.462	0.258
Barley	−0.101	0.689	0.672

[a] All models were adjusted for change in fibre (g/kJ or kcal) and total fruit (% of available carbohydrate) intake

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