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Preliminary report: the effect of a 6-month dietary glycemic index manipulation in addition to healthy eating advice and weight loss on arterial compliance and 24-hour ambulatory blood pressure in men: a pilot study

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Abstract

We aimed to determine whether altering dietary glycemic index (GI) in addition to healthy eating and weight loss advice affects arterial compliance and 24-hour blood pressure (BP), both coronary heart disease (CHD) risk factors. Middle-aged men with at least 1 CHD risk were randomized to a 6-month low-GI (LGI) or high-GI (HGI) diet. All were advised on healthy eating and weight loss. They were seen monthly to assess dietary compliance and anthropometrics. Carotid-femoral pulse wave velocity (PWV), fasting blood lipid profile, and glucose and insulin concentrations were measured at baseline and at months 3 and 6. Six-hour postprandial glucose and insulin responses and 24-hour ambulatory BP were also assessed at baseline and month 6. Thirty-eight subjects (HGI group, n = 16; LGI group, n = 22) completed the study. At month 6, groups differed in dietary GI, glycemic load, and carbohydrate intake ($P < .001$). Fasting insulin concentration and insulin resistance (calculated by homeostatic model assessment) were lower in the LGI than the HGI group ($P < .01$). The reduction in total cholesterol and 24-hour BP was bigger in the LGI than the HGI group ($P < .05$); and only the LGI group had significant reductions ($P < .05$) in PWV, low-density lipoprotein cholesterol, and triacylglycerol concentration. There were no differences in postprandial glucose or insulin responses between the groups. The results suggest that an LGI diet may be more beneficial in reducing CHD risk, including PWV and 24-hour BP, even in the setting of healthy eating and weight loss; and thus, further study is warranted.

1. Introduction

The risk of coronary heart disease (CHD) is determined by a number of factors including arterial compliance [1], that is, the capacity of the arterial system to accommodate volume at a specific pressure [2]. The mechanisms linking arterial compliance to CHD are not completely understood, but may involve insulin resistance promoting endothelial dysfunction, oxidative stress, vascular smooth muscle cell growth, and stimulation of the sympathetic nervous system [3]. Improvement in arterial compliance reduces cardiac morbidity and mortality [4]. Pulse wave velocity (PWV), a surrogate measure of arterial compliance, is inversely associated with compliance. Pulse wave velocity represents the propagation velocity of the pulse wave transit between 2 major arteries; for example, carotid-femoral is a measure of the compliance of the aorta (also referred to as *aortic PWV*) and correlates well with cardiovascular mortality and morbidity [5].

Glycemic index (GI) is a numeric classification of carbohydrate (CHO) foods based on their glycemic response, that is, the rate of postprandial glycemia [6]. Lowering the GI of CHO consumed leads to a reduction in CHD risk [7]. In the present investigation, we hypothesized that in the setting of a healthy eating diet, a 6-month low-GI (LGI) diet compared with a high-GI (HGI) diet would improve PWV and 24-hour blood pressure (BP) in men at risk of CHD. To our knowledge, no previous study examined these particular CHD risk factors in relation to dietary GI. However, a previous study found that a 6-month intensive lifestyle modification (which did not include dietary GI manipulation) in type 2 diabetes mellitus patients improved glycemic

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control and decreased progression of carotid intima-media thickness, a surrogate marker of atherosclerosis [8].

2. Methods

The study was ethically approved by Hammersmith, Queen Charlotte's and Chelsea Hospitals Research Ethics Committee (04/Q0406/111). It had a randomized parallel design. Subjects included in the study after a medical and dietetic screening were men aged 35 to 65 years with at least 1 recognized CHD risk factor (eg, body mass index [BMI] 27-35 kg/m^2, waist ≥94 cm, total cholesterol to high-density lipoprotein ratio ≥5.0, raised BP up to a maximum of 140/90 mm Hg) but otherwise were in good health and not on medication. They gave written informed consent before participation.

All subjects were advised on healthy eating for CHD prevention [9] and weight loss if BMI was greater than 25 kg/m^2. The weight management advice was based on previously published methodology [10] that aims to reduce energy intake by 500 kcal less than the estimated needs and to support this change through behavioral techniques. All volunteers were randomized to an LGI or HGI diet for 6 months and asked to include at least 1 CHO-containing food with meals and snacks according to randomization aiming to achieve a difference in GI between groups of at least 12 points. Examples of HGI foods are white or wholemeal bread, cornflakes, Weetabix (Kettering, Northamptonshire, UK), potatoes (baked, mashed, roasted), couscous, risotto rice, melon, pineapple, and rice cakes. Examples of LGI foods are seeded bread, brown pita bread, muesli, porridge, sweet potatoes, pasta, noodles, basmati slow-cook rice, beans, lentils, apples, dried fruit, and unsalted nuts. Both groups were asked to avoid foods of the opposite GI. Dietetic consultations and anthropometric measurements were carried out monthly. Dietary compliance was assessed using monthly semiquantitative 3-day diaries analyzed using Dietplan6 (Forrest Hill Software, Sussex, United Kingdom). Diet GI calculation was based on available CHO as proposed by Wolever and Jenkins [11]. A GI value (GI glucose = 100) was assigned to all CHO-containing foods containing at least 5 g CHO/100 g from published sources [12-14]. The GI of mixed meals was calculated as proposed by Wolever and Jenkins [15]. If the GI was not known, it was estimated based on similar foods. Diet glycemic load (GL) was calculated as the product of diet GI and CHO intake divided by 100.

Fasting blood samples (after a 12-hour fast and avoiding alcohol and exercise for 24 hours) were taken at baseline and at months 3 and 6 to assess the lipid profile and glucose and insulin concentrations. The homeostatic assessment model [16] was used to measure fasting insulin sensitivity (%S), insulin resistance (the reciprocal of %S), and β-cell function using the mean of 2 fasting insulin (in picomoles per liter) and glucose (in millimoles per liter) plasma samples. At baseline and month 6, a 6-hour postprandial metabolic assessment was carried out to assess glucose and insulin responses. A high-fat test meal consisting of 60 g white bread, 10 g margarine, 60 g cornflakes, 100 mL double cream (mixed with 150 mL water to have a milky consistency), 10 g sugar, and decaffeinated tea or coffee that provided 946 kcal, 93.6 g CHO, 61 g fat, and 11.1 g protein (expressed as percentage of energy: 47% CHO, 33% fat, 15% protein) was provided at time 0 (t_0) minute for consumption. No food or drink was consumed during the 6-hour postprandial period. Blood samples were taken at −30, −15, 0, 30, 60, 120, 180, 240, 300, and 360 minutes.

Pulse wave velocity carotid-femoral was assessed at baseline and at months 3 and 6 using the previously validated Complior SP (Version 1.1.9r; Artech Medical, Pantin, France) data system [17]. All measurements were performed by the same 2 observers (EP and CBT) on the right side of the body in the supine position, in the rested and fasted state. Brachial BP was measured twice, and the mean was recorded. The distance between the carotid and femoral arteries was determined with a nonelastic tape applied at a straight line between the 2 arteries. Pulse wave velocity was estimated by acquiring 2 simultaneous tracings using pulse sensors applied at the carotid and femoral arteries. Ten consecutive pressure waveforms were recorded to cover a complete respiratory cycle [18].

2.1. 24-Hour BP

24-Hour ambulatory BP monitoring (ABPM) was carried out using the validated Diasys Integra II ABPM system (Novacor, Rueil, France) [19-20]. The monitor was programmed to measure BP every 30 minutes between 7:00 AM and 10:00 PM and every 60 minutes between 10:00 PM and 7:00 AM, thus providing a total of 39 readings in a 24-hour period. Subjects were instructed not to consume alcohol or exercise while wearing the monitor.

2.2. Statistical analysis

Data were checked for normality using the Shapiro-Wilks test. Data are presented as mean ± SD when normally distributed or median [interquartile range] when nonnormally distributed. Anthropometric data were compared using mixed models after log transforming nonnormal data. The rest of the variables were compared using unpaired Student *t* tests for normally distributed data or Mann-Whitney *U* tests for nonnormal data. Within-group comparisons of baseline and month 6 data were also carried out using paired *t* tests for normally distributed data and Wilcoxon tests for nonnormal data. A *P* value not exceeding .05 (2-sided) was considered statistically significant. Analysis of the 6-hour postprandial glucose and insulin responses was carried out by comparing the incremental area under the curve (IAUC) [15]. Repeated-measures analysis of variance (ANOVA) was used to compare glucose and insulin responses with time, group, and visit as independent factors. Analysis was carried out using SPSS for Windows (Version 14.0; SPSS, Chicago, IL).

3. Results

Ninety-one subjects were screened for the study, of which 56 were recruited and 38 completed the study (HGI group, n = 16; LGI group, n = 22). Groups did not differ at baseline.

3.1. Dietary intake

All volunteers received healthy eating advice. Twenty of 22 subjects in the HGI group and all subjects in the LGI group received weight management advice because their BMI was greater than 25 kg/m^2. Diet composition did not differ between groups at baseline. Both groups reduced their energy intake during the study, the HGI group by 236 ± 632 kcal/d and the LGI group by 447 ± 499 kcal/d (P = .3). The nonsignificantly bigger reduction in energy consumption by the LGI group may be due to more subjects in the LGI group receiving weight loss advice or a higher satiety after consumption of LGI foods compared with HGI foods by preventing marked postprandial glucose oscillations [21].

Table 1
Fasting blood lipid profile, fasting glucose and insulin concentration, BP, and carotid-femoral PWV results of the HGI and LGI groups at baseline and month 6 and changes from baseline (mean ± SD or median [interquartile range])

	n of paired data	HGI group	n of paired data	LGI group
Total cholesterol (mmol/L)	16		22	
Baseline		5.19 ± 0.91		5.61 ± 0.79
Month 6		5.21 ± 1.20		5.16 ± 0.95§
Δ from baseline		0.02 ± 0.56*		−0.45 ± 0.62*
LDL cholesterol (mmol/L)	16		22	
Baseline		3.34 ± 0.80		3.62 ± 0.63
Month 6		3.23 ± 1.33		3.40 ± 0.75‡
Δ from baseline		−0.11 ± 0.73		−0.22 ± 0.49
HDL cholesterol (mmol/L)	16		22	
Baseline		1.10 [0.96-1.37]		1.10 [1.01-1.26]
Month 6		1.07 [0.98-1.17]		1.12 [0.99-1.24]
Δ from baseline		−0.01 [−0.18 to 0.12]		0.00 [−0.08 to 0.04]
Triacylglycerols (mmol/L)	16		22	
Baseline		1.29 [1.06-1.78]		1.63 [1.23-2.83]
Month 6		1.46 [0.95-1.78]		1.35 [0.83-1.77]‡
Δ from baseline		−0.02 [−0.23 to 0.11]		−0.39 [−1.11 to 0.05]
Fasting glucose (mmol/L)	16		22	
Baseline		5.14 ± 0.36		5.20 ± 0.47
Month 6		5.04 ± 0.35		4.99 ± 0.47
Δ from baseline		−0.10 ± 0.35		−0.18 ± 0.45
Fasting insulin (pmol/L)	14		19	
Baseline		56.7 ± 29.8		44.3 ± 19.2
Month 6		48.9 ± 21.8		32.6 ± 13.1†§
Δ from baseline		−7.8 ± 28.3		−11.7 ± 16.8
Brachial SBP (mm Hg)	16		22	
Baseline		132 ± 15		130 ± 13
Month 6		122 ± 13§		126 ± 12§
Δ from baseline		−10 ± 10		−5 ± 10
Brachial DBP (mm Hg)	16		22	
Baseline		81 ± 10		81 ± 11
Month 6		76 ± 8§		78 ± 9§
Δ from baseline		−5 ± 7		−2 ± 9
Carotid-femoral PWV (m/s)	14		18	
Baseline		9.9 [9.2-10.6]		10.3 [10.0-10.9]
Month 6		9.4 [9.0-10.5]		9.7 [9.3-10.3]‡
Δ from baseline		−0.3 [−0.6 to 0.5]		−0.4 [−1.4 to 0.0]
24-h SBP (mm Hg)	11		20	
Baseline		118 ± 18		128 ± 14
Month 6		121 ± 17		115 ± 12§
Δ from baseline		3 ± 18		−13 ± 17*
24-h DBP (mm Hg)	11		20	
Baseline		79 ± 8		83 ± 7
Month 6		79 ± 5		77 ± 7§
Δ from baseline		−1 ± 5		−5 ± 5†

Δ indicates change.
*$P \le .05$ and $^{\dagger}P \le .01$; between-group comparison by t test for normally distributed data or by Mann-Whitney U tests for nonnormally distributed data. $^{\ddagger}P \le .05$ and $^{\S}P \le .01$; within-group comparison of baseline and month 6 results by paired t test for normally distributed data or by Wilcoxon test for nonnormally distributed data.

Dietary GI (HGI group: 63.2 ± 5.6, LGI group: 50.6 ± 4.6; $P < .001$), diet GL (HGI group: 175.0 ± 45.6, LGI group: 114.4 ± 31.5; $P < .001$), and CHO intake (HGI group: 278 ± 7 g/d, LGI group: 224 ± 50 g/d; $P < .001$) differed between groups at month 6. There were no other differences in dietary composition between the 2 groups. A GI value was assigned to 96.9% of CHO consumed, of which 80.4% was from published sources and 16.5% was estimated.

3.2. Anthropometrics

Anthropometric measurements were significantly reduced within groups over time ($P < .001$ for weight, BMI, waist, and hip measurements), but groups did not differ (results not shown). The HGI group lost 3.0 ± 4.2 kg, and the LGI group lost 2.2 ± 3.6 kg ($P = .3$).

3.3. Results of fasting blood tests, homeostatic assessment model, and postprandial response

No differences between groups were seen at baseline. Over the 6-month period, there was a bigger reduction in total cholesterol in the LGI group compared with the HGI group and also within the LGI group. Low-density lipoprotein (LDL) cholesterol and triacylglycerol concentration fell only within the LGI group (Table 1). At month 6, fasting insulin (HGI group: 48.9 ± 21.8 pmol/L vs LGI group: 32.6 ± 13.1 pmol/L, $P < .01$) and percentage insulin resistance (HGI group: 0.88 ± 0.39 vs LGI group: 0.61 ± 0.24, $P = .02$) were lower in the LGI than the HGI group, whereas there was a trend for %S to be higher in the LGI group (HGI group: median = 126.5 [interquartile range: 103.6-145.2] pmol/L vs LGI group: 170.3 [131.0-250.5] pmol/L, $P = .06$). A within-group analysis showed an increase in %S of the LGI group ($P < .01$), whereas β-cell function fell ($P < .01$) possibly because of the increase in %S ($P < .01$) allowing β-cells to function at a lower rate.

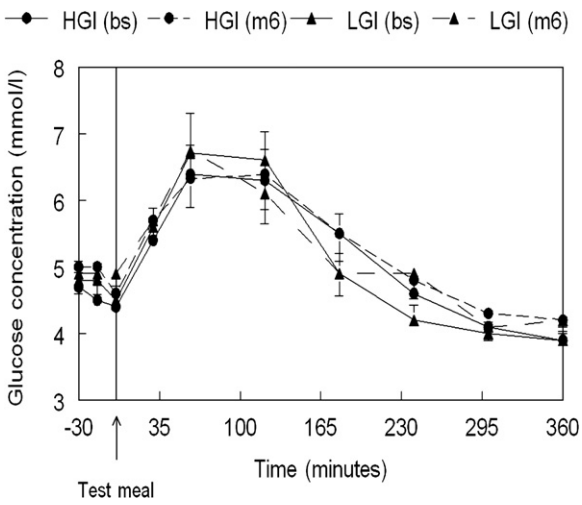

Fig. 1. Fasting and 6-hour postprandial glucose response of the HGI and the LGI groups to a standard meal at baseline and at month 6 (values are mean ± SEM). Test meal consumed at 0 minute (see text for composition). Repeated-measures ANOVA revealed a significant effect of time ($P < .001$), no differences between groups ($P = 1.0$) or visits ($P = .5$), and no interaction effects. Bs indicates baseline; m6, month 6.

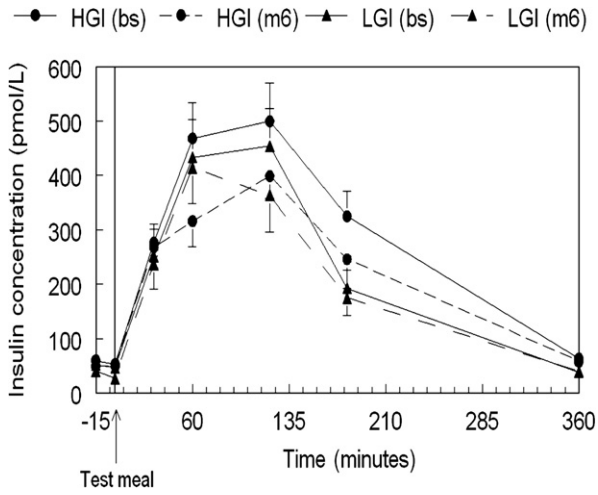

Fig. 2. Fasting and 6-hour postprandial insulin response of the HGI and the LGI groups to a standard meal at baseline and at month 6 (values are mean ± SEM). Test meal consumed at 0 minute (see text for composition). Repeated-measures ANOVA revealed a significant effect of time ($P < .001$), no differences between groups ($P = .3$) or visits ($P = .1$), and no interaction effects.

Postprandial glucose results were available for 15 of 16 subjects in the HGI group and 18 of 22 subjects in the LGI group and postprandial insulin results for 14 of 16 subjects in the HGI group and 19 of 22 subjects in the LGI group because of either hemolysis or cannula failure during the postprandial assessment. Analysis of IAUC glucose was based on plasma samples taken at all time points, whereas analysis of IAUC insulin was based on plasma samples taken at −15, 0, 30, 60, 120, 180, and 360 minutes because of cost constraints. Repeated ANOVA of glucose and insulin IAUC showed a significant effect of time ($P < .001$) but no differences between groups or visits and no interaction effects (Figs. 1 and 2).

3.4. BP and arterial compliance

Groups did not differ in brachial systolic BP (SBP), diastolic BP (DBP), and PWV at baseline or month 6 (Table 1). However, a within-group analysis showed that both groups had reductions in SBP and DBP, whereas PWV fell only in the LGI group. Valid results for 24-hour ABPM were obtained from 11 subjects in the HGI group and 20 subjects in the LGI group. The LGI group had significant reductions in 24-hour SBP and 24-hour DBP, both compared with the HGI group and also within the group (Table 1).

4. Discussion

The present study showed that dietary GI can be manipulated over a 6-month period. Although all subjects

lost weight, the reduction in total cholesterol was bigger in the LGI group, a finding that agrees with a *Cochrane Review* reporting that an LGI diet leads to a small reduction in cholesterol [22]. In addition, only the LGI group had reductions in LDL cholesterol and triacylglycerol concentration possibly because of a faster clearance in intestinally derived triacylglycerol remnants [23]. Although measurement of apolipoprotein B-48 and apolipoprotein B-100 lipoproteins was outside the scope of this investigation, this information could be obtained by further study. The LGI group also showed a reduction in fasting insulin concentration and insulin resistance and a tendency for improvement in insulin sensitivity. This is in support of previous studies showing that LGI foods improved both in vivo insulin sensitivity and in vitro insulin responsiveness of adipocytes [24], increased first-phase insulin release [25], and improved insulin secretion [26]. Furthermore, a recent meta-analysis of 37 prospective observational studies found that diets with an HGI or high GL independently increased the risk of heart disease (relative risk: 1.25) and that the associations were more positive and of more magnitude between GI and chronic disease than between GL and disease [7]. However, it should be noted that 90% of the participants in the studies were female.

Brachial SBP and DBP significantly fell in both groups over the study period, probably because of weight loss. However, only the LGI group had a significant reduction in 24-hour BP that may be related to the improvement in insulin sensitivity. Moreover, only the LGI had a reduction in aortic PWV over the 6-month period, an important finding because PWV predicts cardiovascular disease in healthy subjects above and beyond traditional risk factors [1]. The reduction may be related to improvements in insulin resistance, BP, total cholesterol, and weight loss that act either directly on the arterial wall or through reducing inflammation and/or affecting the nitric oxide system [27-28].

This study is the first to examine the effect of dietary GI manipulation on PWV and 24-hour BP. However, further data need to be collected before reaching definitive conclusions. A power calculation using data from this study and a difference in PWV of 1.5 m/s between groups (SD = 2 m/s, 0.8 power level, 2-sided) suggests a sample size of 28 subjects in each group.

In conclusion, results of this pilot investigation suggest that an LGI diet may be of more benefit on PWV, 24-hour BP, lipid profile, fasting insulin, and insulin sensitivity even in the setting of healthy eating and weight loss. These data support the need for further research in this area.

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