## ORIGINAL ARTICLE

# Does the DASH diet lower blood pressure by altering peripheral vascular function?

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We tested whether lowering of blood pressure (BP) on the dietary approaches to stop hypertension (DASH) diet was associated with changes in peripheral vascular function: endothelial function, assessed by flow-mediated vasodilatation (FMD) of the brachial artery, and subcutaneous adipose tissue blood flow (ATBF). We also assessed effects on heart rate variability (HRV) as a measure of autonomic control of the heart. We allocated 27 men and women to DASH diet and control groups. We measured FMD, ATBF and HRV on fasting and after ingestion of 75 g glucose, before and after 30 days on dietary intervention, aiming for weight maintenance. The control group did not change their diet.

The DASH-diet group complied with the diet as shown by significant reductions in systolic ($P < 0.001$) and diastolic ($P = 0.005$) BP, and in plasma C-reactive protein ($P < 0.01$), LDL-cholesterol ($P < 0.01$) and apolipoprotein B ($P = 0.001$), a novel finding. Body weight changed by $<1$ kg. There were no changes in the control group. We found no changes in FMD, or in ATBF, in the DASH-diet group, although heart rate fell ($P < 0.05$). Glucose and insulin concentrations did not change. In this small-scale study, the DASH diet lowered BP independently of peripheral mechanisms.
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## Introduction

Elevated blood pressure (BP), particularly systolic hypertension, is a common and powerful contributor to cardiovascular disease.[1–3] It makes a major contribution to the global burden of disease.[4] Drug treatment of hypertension is effective and reduces cardiovascular end points.[5,6] However, lifestyle changes, mainly based on dietary changes with an increase in physical activity, can produce BP-lowering effects similar to those seen with drug treatment and can be sustained over several years.[7]

The dietary approaches to stop hypertension (DASH) diet[8] is an effective dietary means of reducing BP in people with hypertension, as well as in normotensives. It is high in fruits, vegetables, low-fat dairy products and nuts, and low in saturated fat and sugar and refined carbohydrate. It lowers BP within 2 weeks,[8] but the mechanisms involved are unclear. The DASH diet is effective irrespective of lowering of dietary sodium intake.[9] If mechanisms were identified, it might be possible to improve the effectiveness of the diet or to understand better how it might be complemented by pharmacological treatment; for instance, the DASH diet enhances the blood-pressure-lowering effect of losartan.[10] The DASH diet also improves measures of cardiovascular disease risk, including LDL cholesterol and insulin sensitivity.[11–13]

Peripheral vascular function is an important contributor to BP. In established hypertension, the major abnormality is increased total peripheral resistance.[14] We hypothesised that the DASH diet would lead to improvement of aspects of peripheral vascular function that can be measured and which might in turn be associated with metabolic improvements. One such aspect is endothelial function, dependent on nitric oxide generation. To our knowledge, the effects of the DASH diet on functional measures of endothelial function such as flow-mediated vasodilatation (FMD) have not been assessed earlier, even though functional or biochemical markers of endothelial function are improved by various dietary manipulations.[15,16] The regulation of blood flow through metabolically important tissues is another potential link between vascular and metabolic function. In particular, adipose tissue blood flow (ATBF) and its rapid response to nutrient intake seem to be markers of metabolic health. Impairment of the responsiveness of ATBF to nutrients is seen in obesity[17,18] and in insulin resistance,[19] and reduced ATBF responsiveness correlates with cardiovascular disease risk markers.[19,20]

Therefore, we performed a pilot study to assess the effects of a DASH-diet intervention on FMD of

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the brachial artery, as a measure of endothelial function. We also assessed subcutaneous ATBF together with its responsiveness to nutrient intake. Although fasting ATBF is regulated by nitric oxide, the ATBF response to nutrients is largely independent of endothelial function,[21] and so we argued that this would provide an independent measure of vascular function. In addition, we measured heart rate variability (HRV) and its components as a measure of sympathetic and parasympathetic control of the cardiovascular system, as increased sympathetic activity is regarded as a hallmark of hypertension.[14]

## Materials and methods

### Subjects
The study was approved by the Oxfordshire Research Ethics Committee and all volunteers gave informed consent. Twenty-seven subjects were recruited from the Oxford BioBank[22] and from local advertisements and posters. Inclusion criteria were age 25–60 years, BMI 20–40 kg m$^{-2}$ and no diabetes or disorders of lipid metabolism requiring treatment. The first subject was recruited in January 2007. All volunteers completed a 3-day food record before a screening visit. At the screening visit, they were interviewed by a dietitian. An electrocardiograph (ECG) was recorded for familiarisation with the procedure for HRV and to check for ectopic beats. A blood sample was taken for measurement of glucose, triglyceride and cholesterol.

Exclusion criteria at the screening visit were judgement by the dietitian that the subject was not capable of making the necessary dietary changes, $>3\%$ ectopic beats on ECG, abnormal glucose ($>6.1$ mmol l$^{-1}$) or lipids (total cholesterol $>6.7$ mmol l$^{-1}$, triglycerides $>2.5$ mmol l$^{-1}$) and any metabolic disease. Subjects were allocated to diet and control groups, bearing in mind that the intention was to observe BP changes, so those whose diet was not easily amenable to change to the DASH pattern were allocated to the control group. This was not designed to be a randomised trial.

After completion of the first study day, subjects allocated to the DASH group met with the dietitian for a detailed consultation of the required dietary changes to adhere to a DASH intermediate sodium-type diet for 30 days.[9] To encourage weight maintenance throughout the study, energy requirements for each subject were predicted using the equation of Schofield,[23] and food lists devised to fit UK food preferences and individual needs. The emphasis of the DASH-type diet was to increase intakes of fruit, vegetables, wholegrain cereals and low-fat dairy products when decreasing intakes of salt and saturated fat. Subjects were provided with a booklet containing the number of daily servings from each food group that was required for weight maintenance. They were also given

suggestions of how to increase fruit and vegetable and reduce salt intakes, lists of low-salt convenience foods and ideas for eating out. To aid compliance, the dietitian was in regular contact with the subjects in the DASH group throughout the study period, and at a mid-point visit (around day 15) subjects met the dietitian to discuss any difficulties in adhering to the diet and to check their weight. Subjects were provided with no-salt sunflower spread and olive oil as spreading and cooking fats. Subjects self-selected and prepared all foods. In addition, subjects were asked to maintain their regular lifestyles, physical activity levels and meal frequencies throughout the study.

Subjects in the control group were asked to maintain their habitual diet and regular lifestyle.

All foods and beverages consumed were weighed or estimated and recorded into food diaries on five representative non-consecutive days (4 week day/1 weekend day) over a 2-week period before coming in for the first study day and again during the last 14 days of the study period. The nutrient composition of the diets was calculated using the food composition data from the Microdiet 2 program (Downlee Systems Ltd, Derbyshire, UK).

More details of the dietary aspects of the study have been presented separately in an assessment of the ease of application of such a dietary pattern to a UK population (Harnden et al., unpublished).

### Clinical measurements
Metabolic study days were carried out on the day before the dietary intervention and on the day after the 30-day intervention. Participants arrived at the Clinical Research Unit having fasted overnight. Subcutaneous abdominal ATBF and HRV measurements were conducted in the fasted state, together with an assessment of FMD of the brachial artery. After collection of fasting blood samples, a glucose load (75 g glucose in water) was given, and measurements of ATBF and HRV continued during the next 2 h, along with blood samples. BP was measured with an automated system (Omron M5-I Intellisense, White Medical, Rugby, UK) 5 min before each blood sample. Cuff size was adjusted according to upper-arm circumference.

ATBF was measured after the injection of 2 MBq $^{133}$Xe into the subcutaneous abdominal adipose tissue, as described earlier.[19] Continuous recordings of residual counts were made, and then analysed in 10 min segments. Fasting ATBF was taken as the average of the values obtained before glucose administration. Peak ATBF after the glucose was assessed as described earlier.[19] The ATBF response to glucose was characterised as peak minus fasting ATBF.

For HRV recording, subjects were semi-supine in a quiet room. Respiration was controlled using an electronic metronome at a rate comfortable for the

subject at 9–11 breaths per minute. A chest strain gauge was worn to assess respiratory compliance. A 3-lead continuous ECG signal was recorded using a Powerlab/8Sp, MLS310 HRV Module (AD Instruments Pty Ltd, Castle Hill, NSW, Australia). The R–R intervals for the last consecutive 5 min for each 10 min recording were manually selected and analysed (minimum of 250 cycles). Time and frequency-domain analyses of HRV and Poincaré plot were performed. All R–R intervals were edited by visual inspection to exclude ectopic beats or artefacts. Two fasting baseline measurements of HRV were recorded for 10 min after the same procedure. Recordings were also made after consumption of glucose at 20, 70 and 130 min.

The various parameters of HRV tended to be highly intercorrelated, and for illustration, the key parameters of total power, low frequency/high frequency ratio and standard deviation of R–R intervals (SDnn) are presented in Results. There was no consistent pattern of change in any of these parameters after glucose ingestion, so average values over the whole experimental day are presented. One subject in the DASH-diet group coughed repeatedly during the HRV measurements and his readings have been excluded.

FMD was recorded using a high-resolution ultrasound machine (ATL HDI 5000, Advanced Technology Laboratories UK Ltd, Letchworth, UK) and a 14-MHz linear array transducer. The limit of axial resolution was 0.1 mm. Subjects rested in the supine position for 10 min before FMD was recorded. Two BP measurements were recorded during this time. Longitudinal scans of the right brachial artery were obtained approximately 2 cm above the antecubital fossa. Three consecutive frames were stored to assess artery diameter at rest. ECG was recorded continuously. A cuff placed on the right forearm was inflated to 50 mm Hg above the systolic BP (maximum inflation 200 mm Hg) for 5 min. One minute after cuff deflation, three consecutive frames were stored to assess peak diameter change during reactive hyperaemia. The brachial artery diameter was measured manually three times per frame, between the media–adventitia interface on the anterior wall and intima–lumen interface on the posterior wall at the peak of the R-wave cycle at rest and post-hyperaemia. FMD was calculated as the percentage change in arterial diameter at peak hyperaemia. Reliable images were obtained on both visits in 10 of the DASH-diet group and 10 controls.

*Laboratory methods*
Plasma concentrations of glucose, insulin and non-esterified fatty acids (NEFA), fasting and during the glucose tolerance test, were measured using enzymatic methods on an Instrumentation Laboratory ILab 600 analyser (Instrumentation Laboratory UK, Warrington, UK). Fasting plasma concentrations of C-reactive protein (CRP), apolipoprotein B (apoB),

triacylglycerol, cholesterol and HDL cholesterol were also measured enzymatically on the ILab analyser.

*Statistical analysis*
The study was powered to assess change in ATBF rather than BP. In this instance, the primary end point (change in ATBF in response to diet) was assessed within subjects. A power calculation using data on repeated ATBF measurements[18] showed that to detect a 30% change within subjects at a significance level of 0.05 with a power of 0.8 on a two-tailed test, we would need nine subjects. The power of the study was also assessed retrospectively as described below and in Results.

All data presented in Results are for paired comparisons: if values from one subject were not available at one visit, that subject has been excluded from the results for that particular parameter for both visits. Reproducibility of the various physiological measurements was assessed from the correlation between values on visit 1 and those on visit 2. These correlation coefficients are presented in Results. They were used retrospectively to assess the power of the study to detect changes in the variables measured, as described by Bland[24]. To test for effects of the dietary intervention, responses of those variables measured before and after glucose administration (plasma concentrations of glucose, insulin, NEFA, ATBF, BP) were analysed by repeated-measures ANOVA with time and dietary status as within-subjects factors. For variables measured at a single time (fasting blood metabolite concentrations), or for which summary measures were used, responses to the dietary intervention were analysed by paired *t*-test. In addition, to test more stringently for differences in response between DASH diet and control groups, the changes from visit 1 to visit 2 were compared between the interventions by unpaired *t*-test.

# Results

*Baseline values and dietary compliance*
Baseline characteristics of the two groups are shown in Table 1. They were closely matched for age, BMI and sex. No volunteers dropped out; although as described below, metabolic and vascular data from one subject with a high CRP concentration were judged unreliable and were not included.

Energy intakes remained reasonably constant over the study in both the DASH and control groups. Subjects in the DASH group consumed significantly more protein ($P<0.001$), carbohydrate ($P<0.001$) and non-starch polysaccharides ($P<0.01$) when on the DASH diet. As intakes of carbohydrate and protein increased, total fat intake was substantially ($P<0.001$) decreased by 9% kJ (22 g per day) in the DASH group, with approximately half of this

**Table 1** Baseline characteristics of the participants

|  | DASH diet group | | Control group | |
|---|---|---|---|---|
|  | n | Male/female | n | Male/female |
|  | 14 | 8/6 | 13 | 8/5 |
|  | Median | Range | Median | Range |
| Age | 46 | 39–58 | 45 | 27–54 |
| BMI ($kg\,m^{-2}$) | 28.7 | 23.7–37.3 | 29.1 | 27.2–35.7 |
| Waist circumference (cm) | 96 | 80–114 | 97 | 76–117 |
| Hip circumference (cm) | 107 | 102–121 | 107 | 97–121 |

No differences between groups were significant.

**Table 2** Fasting biochemical variables and their responses to the dietary intervention

|  | Dash diet (n = 14) | | Control (n = 13) | | Significance of difference in changes[a] | Correlation[b] |
|---|---|---|---|---|---|---|
|  | Visit 1 | Visit 2[c] | Visit 1 | Visit 2[c] |  |  |
| Body weight (kg) | 86.3 ± 3.7 (14) | 85.4 ± 3.7* | 84.8 ± 3.5 | 85.1 ± 3.6 | 0.034 | r = 0.99 P = 0.000 |
| Triacylglycerol ($mmol\,l^{-1}$) | 1.33 ± 0.21 (13) | 1.33 ± 0.20 | 1.07 ± 0.13 (13) | 1.02 ± 0.13 | 0.421 | r = 0.87 P = 0.000 |
| Total cholesterol ($mmol\,l^{-1}$) | 4.86 ± 0.23 (13) | 4.33 ± 0.21** | 4.42 ± 0.18 (13) | 4.33 ± 0.20 | 0.024 | r = 0.77 P = 0.000 |
| HDL-C ($mmol\,l^{-1}$) | 1.08 ± 0.07 (13) | 0.94 ± 0.06* | 1.04 ± 0.09 (13) | 1.02 ± 0.09 | 0.082 | r = 0.83 P = 0.000 |
| LDL-C ($mmol\,l^{-1}$) | 3.17 ± 0.19 (13) | 2.78 ± 0.17** | 2.89 ± 0.12 (13) | 2.84 ± 0.13 | 0.037 | r = 0.70 P = 0.000 |
| Total cholesterol/HDL-C ratio | 4.66 ± 0.25 (13) | 4.75 ± 0.29 | 4.50 ± 0.30 (13) | 4.58 ± 0.38 | 0.967 | r = 0.80 P = 0.000 |
| ApoB ($g\,l^{-1}$) | 0.90 ± 0.05 (13) | 0.82 ± 0.04*** | 0.79 ± 0.04 (13) | 0.75 ± 0.05 | 0.271 | r = 0.87 P = 0.000 |
| Glucose ($mmol\,l^{-1}$) | 4.93 ± 0.10 (13) | 5.13 ± 0.15 | 5.10 ± 0.11 (13) | 5.10 ± 0.10 | 0.142 | r = 0.70 P = 0.000 |
| Insulin ($mU\,l^{-1}$) | 15.2 ± 3.1 (13) | 11.7 ± 1.1 | 10.3 ± 0.9 (13) | 9.2 ± 0.7 | 0.619 | r = 0.83 P = 0.000 |
| NEFA ($\mu mol\,l^{-1}$) | 525 ± 36 (13) | 496 ± 34 | 550 ± 64 (13) | 583 ± 58 | 0.271 | r = 0.70 P = 0.000 |
| CRP ($mg\,l^{-1}$) | 2.00 ± 0.41 (13) | 1.26 ± 0.27** | 1.63 ± 0.42 (13) | 2.57 ± 1.32 | 0.110 | r = 0.68 P = 0.000 |

Abbreviations: -C, -cholesterol; CRP, C-reactive protein.
Values are mean ± s.e.m.
[a]Change on DASH diet versus change on control by unpaired *t*-test.
[b]Correlation between values on visit 1 and those on visit 2.
[c]Asterisks denote significant difference from visit 1 by paired *t*-test: *$P<0.05$, **$P<0.01$, ***$P\leqslant0.001$.

resulting from a decrease in saturated fat intake. Subjects in the DASH group were encouraged to decrease salt intakes, and this resulted in a notable ($P<0.001$) reduction in sodium intakes of 861 mg per day (or 2.2 g per day salt). Reported nutrient intakes did not change in the control group (data not shown).

*Responses to the diet*
*Metabolic variables.* Body weight fell slightly, but significantly on the DASH diet; the mean change was <1 kg (Table 2). There was no significant change in the control group.

One subject in the DASH-diet group on the second visit had a plasma CRP concentration >10 mg $l^{-1}$ (baseline value 1.1 mg $l^{-1}$). He was the only subject in the DASH-diet group whose BP and heart rate

also increased on the second visit. We concluded that he had an acute illness and excluded his biochemical and vascular data.

Fasting concentrations of total- and LDL-cholesterol and apoB fell significantly on the DASH diet (Table 2). HDL-cholesterol concentrations also fell significantly (Table 2), but the total/HDL-cholesterol ratio was not affected by the diet (Table 2). CRP concentrations also fell (Table 2). There were no changes in these variables with the control intervention. In contrast, neither DASH diet nor control intervention affected fasting triacylglycerol, NEFA, glucose or insulin concentrations (Table 2).

Plasma glucose and insulin responses to oral glucose ingestion (not shown) did not change with either DASH diet or control intervention. Plasma NEFA responses to glucose ingestion were also unchanged (data not shown).
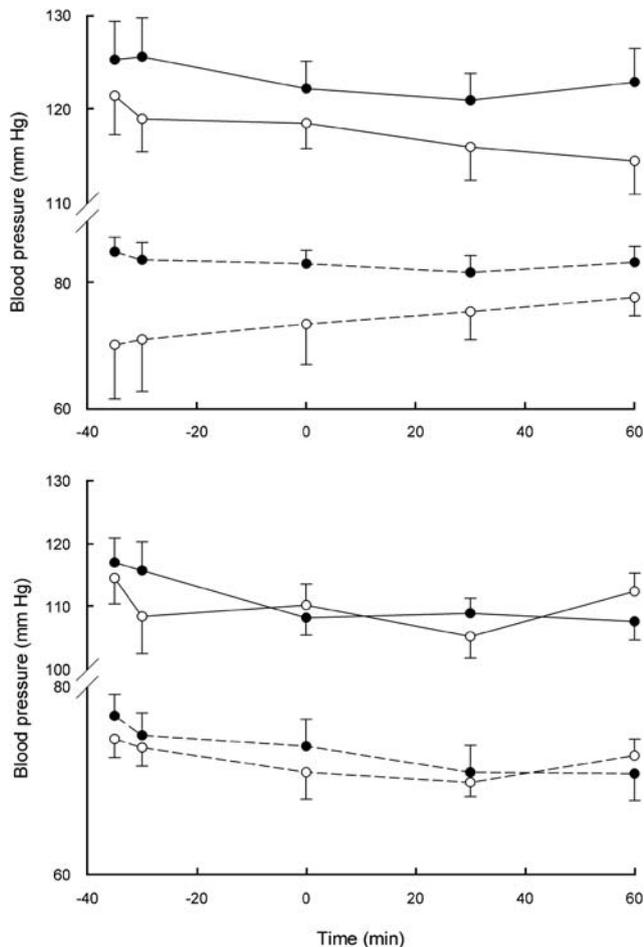
**Figure 1** BPs and their responses to the dietary intervention. Top panel, DASH-diet intervention; lower panel, controls. Solid points, measurements at visit 1; open points, measurements at visit 2. Solid lines, systolic; dashed lines, diastolic. Individual measurement points in the fasting state before the administration of glucose at time 60 min are shown. Repeated measures ANOVA across the OGTT period: DASH- diet group, main effect of diet: systolic, $P < 0.001$; diastolic, $P = 0.005$; control group, changes not significant.

*Vascular variables.* BPs (Figure 1) fell significantly on the DASH diet. Mean fasting heart rate also fell (Table 3). There were no significant changes with the control intervention.

Subcutaneous abdominal ATBF (Figure 2) increased after oral glucose ingestion in both groups. However, there were no effects of either DASH diet or control intervention, either on fasting values or on responses to glucose (summary statistics are in Table 3).

The results for FMD and selected measurements of HRV are given in Table 3. None of these variables was changed systematically by DASH diet, and some of the HRV parameters changed significantly also in the control group. Given the small numbers for FMD, the data were also analysed by plotting visit 2 against visit 1 values (Figure 3). The points lay on either side of the line of identity in both groups.

## Discussion

We found the expected effects of a 30-day DASH-diet intervention. Systolic and diastolic BPs fell, along with LDL-cholesterol concentrations and apoB. We are not aware that the lowering of apoB concentrations with the DASH diet has been reported earlier, but it implies that there was a decrease in the total number of LDL particles, not just in their size. HDL-cholesterol concentrations also fell, presumably because of the decrease in dietary-fat intake, but the total:HDL-cholesterol ratio was not affected. CRP concentrations fell, as has been found earlier.[25] All these parameters remained unaffected in the control group. Nevertheless, we found no effects on measures of peripheral vascular function, although we had expected to. Neither FMD nor the responsiveness of ATBF to glucose intake were altered by DASH diet. Heart rate fell.

We found no effect on plasma glucose and insulin concentrations and their inter-relationships, implying a lack of effect of our DASH-diet intervention on insulin sensitivity. This was surprising because earlier studies have claimed improvement in insulin sensitivity[12] and in aspects of the metabolic syndrome.[26] However, the improvements in insulin sensitivity and metabolic syndrome were seen in the context of weight-losing studies, whereas we strived for weight maintenance. We and others have earlier shown that impaired ATBF responsiveness is associated with insulin resistance and metabolic syndrome features,[19,20,27] so the failure of our intervention to affect insulin sensitivity may also explain why there was no effect on ATBF. In contrast, impairment of endothelial function, as shown by FMD, has been shown in several studies to be distinct from the metabolic syndrome,[28–30] although in patients with peripheral arterial disease, impaired FMD and metabolic syndrome were closely related.[31] Thus, it is more surprising that we found no alteration in FMD with the DASH-diet intervention, especially as impaired FMD and elevated BP have been shown to be related in several studies.[32,33] Furthermore, dietary change to a Mediterranean-style diet has been shown to improve FMD,[34] at least in hypercholesterolaemic men.

The observation that BP and LDL-cholesterol could be altered without effect on other variables generally related to insulin resistance and the metabolic syndrome is consistent with other evidence that these are distinct. A recent analysis of obese women showed that BP is distinct from other core metabolic syndrome features, when analysed by principal components analysis.[35] The distinction between BP and other metabolic syndrome components was also shown when changes over a 5-year period of weight loss were analysed.[36] LDL cholesterol is not considered as part of the metabolic syndrome and behaves differently from metabolic syndrome components in various situations, for

**Table 3** Vascular function measurements and their responses to the dietary intervention

| | Dash diet | | Control | | Significance of difference in changes[a] | Correlation[b] |
|---|---|---|---|---|---|---|
| | Visit 1 | Visit 2[c] | Visit 1 | Visit 2[c] | | |
| Heart rate (fasting) (min$^{-1}$) | 65.7 ± 2.9 (13) | 62.9 ± 2.7* | 58.9 ± 1.4 (13) | 57.4 ± 1.6 | 0.373 | r = 0.91 P = 0.000 |
| FMD (%) | 5.48 ± 1.59 (10) | 6.82 ± 2.01 | 4.11 ± 1.20 (10) | 5.31 ± 1.26 | 0.923 | r = 0.78 P = 0.000 |
| Total power (HRV) × 10$^{-3}$ | 2.3 ± 0.8 (12) | 3.2 ± 1.2 | 3.9 ± 0.9 (13) | 6.5 ± 1.7* | 0.157 | r = 0.88 P = 0.000 |
| LF/HF | 6.9 ± 1.0 (12) | 6.3 ± 1.1 | 5.0 ± 1.3 (13) | 4.3 ± 1.2 | 0.834 | r = 0.81 P = 0.000 |
| SDnn | 41.9 ± 6.7 (12) | 48.9 ± 7.5* | 56.8 ± 7.0 (13) | 71.0 ± 9.5** | 0.179 | r = 0.92 P = 0.000 |
| ATBF fasting (ml 100 g$^{-1}$ min$^{-1}$) | 2.42 ± 0.17 (13) | 2.38 ± 0.28 | 2.38 ± 0.40 (13) | 2.67 ± 0.47 | 0.685 | r = 0.74 P = 0.000 |
| ATBF response (ml 100 g$^{-1}$ min$^{-1}$) | 1.22 ± 0.43 (13) | 1.02 ± 0.35 | 1.27 ± 0.42 (13) | 1.07 ± 0.53 | 0.998 | r = 0.34 P = 0.09 |

Abbreviations: ATBF, adipose tissue blood flow; FMD, flow-mediated vasodilatation; HF/LF, low frequency/high frequency ratio (analysis of heart rate); HRV, heart rate variability; SDnn, standard deviation of R–R intervals.
Values are mean ± s.e.m. (n). Data for paired comparisons only are shown, so n for second visit is same as for first visit.
[a]Change on DASH diet versus change on control by unpaired t-test.
[b]Correlation between values on visit 1 and those on visit 2.
[c]Asterisks denote significant difference from visit 1 by paired t-test: *P<0.05, **P<0.01.
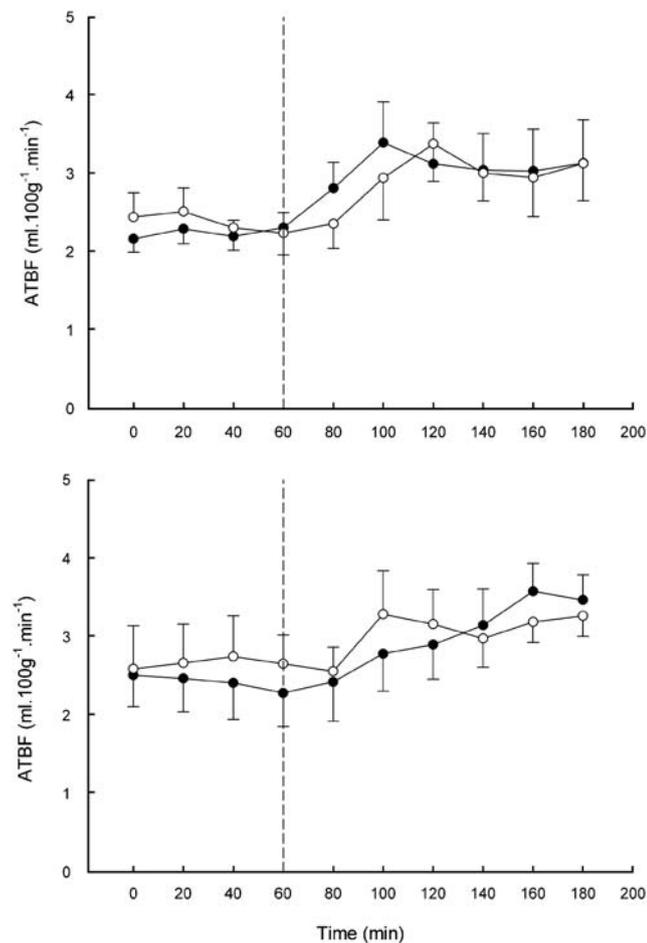


**Figure 2** Subcutaneous abdominal ATBF and its responses to the dietary intervention. Top panel, DASH-diet intervention; lower panel, controls. Solid points, measurements at visit 1; open points, measurements at visit 2. Vertical dashed lines show the time of glucose ingestion. Summary statistics are given in Table 3.
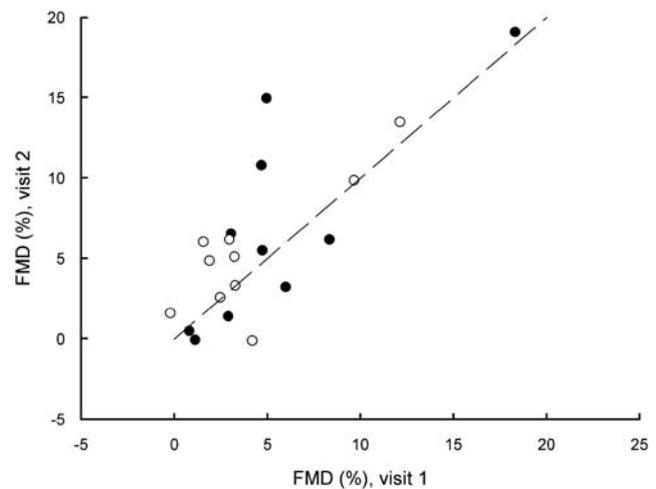


**Figure 3** Flow-mediated vasodilation of the brachial artery at the two visits. Solid points, participants in the DASH-diet group; open points, control subjects. The dashed line shows the line of identity.

instance during weight loss, which improves all metabolic syndrome features, but without effect on LDL-cholesterol.[37]

Our study was small and only relatively large changes in ATBF responsiveness and in FMD would have been detected, although there was no tendency to systematic alteration in the values we measured. We cannot rule out that a larger study would have detected effects, and indeed our data could be used as a pilot for a larger study in the future. A retrospective calculation of statistical power (see Statistical Analysis) showed that the study was powered to detect (power of 0.80 and α 0.05) a change of 2% in systolic BP, of 23% in FMD and of 8% in fasting ATBF.

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In conclusion, in this pilot study, we documented the expected effects of a DASH-diet intervention. However, we found no measurable effects of the diet on aspects of peripheral vascular function, namely FMD and ATBF responsiveness. These findings suggest that the effect of the DASH diet on BP is mediated through autonomic and cardiac mechanisms rather than through alteration of peripheral vascular resistance, at least in the absence of major changes in body weight. A larger study would be required to confirm the results of this pilot study.

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*What is known about the topic?*
- The DASH diet is widely promoted for the prevention and treatment of hypertension.
- The DASH diet effectively lowers blood pressure in normo- and hypertensive individuals, but the mechanisms remain unclear.
- Consuming a DASH diet in conjunction with reducing sodium intake lowers blood pressure to a greater extent.

*What this study adds?*
- The DASH diet does not alter peripheral vascular function.
- A DASH-style diet significantly lowers plasma apolipoprotein B concentrations.

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## Conflict of interest

The authors declare no conflict of interest.

## Acknowledgements

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