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The effect of prior walking on coronary heart disease risk markers in South Asian and European men

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Running Head
Walking and CHD risk markers in South Asian and European men

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ABSTRACT

**Purpose:** Heart disease risk is elevated in South Asians possibly due to impaired postprandial metabolism. Running has been shown to induce greater reductions in postprandial lipaemia in South Asian than European men but the effect of walking in South Asians is unknown.

**Methods:** Fifteen South Asian and 14 White European men aged 19-30 years completed two, 2-d trials in a randomised crossover design. On day 1, participants rested (control) or walked for 60 min at approximately 50% maximum oxygen uptake (exercise). On day 2, participants rested and consumed two high fat meals over a 9h period during which 14 venous blood samples were collected. **Results:** South Asians exhibited higher postprandial triacylglycerol (geometric mean (95% confidence interval) 2.29(1.82 to 2.89) vs. 1.54(1.21 to 1.96) mmol·L\(^{-1}\)·hr\(^{-1}\)), glucose (5.49(5.21 to 5.79) vs. 5.05(4.78 to 5.33) mmol·L\(^{-1}\)·hr\(^{-1}\)), insulin (32.9(25.7 to 42.1) vs. 18.3(14.2 to 23.7) µU·mL\(^{-1}\)·hr\(^{-1}\)) and interleukin-6 (2.44(1.61 to 3.67) vs. 1.04(0.68 to 1.59) pg·mL\(^{-1}\)·hr\(^{-1}\)) than Europeans (all ES ≥ 0.72, P≤0.03). Between-group differences in triacylglycerol, glucose and insulin were not significant after controlling for age and percentage body fat. Walking reduced postprandial triacylglycerol (1.79(1.52 to 2.12) vs. 1.97(1.67 to 2.33) mmol·L\(^{-1}\)·hr\(^{-1}\)) and insulin (21.0(17.0 to 26.0) vs. 28.7(23.2 to 35.4) µU·mL\(^{-1}\)·hr\(^{-1}\)) (all ES ≥ 0.23. P≤0.01), but group differences were not significant.

**Conclusions:** Healthy South Asians exhibited impaired postprandial metabolism compared with White Europeans, but these differences were diminished after controlling for potential confounders. The small-moderate reduction in postprandial triacylglycerol and insulin after brisk walking was not different between the ethnicities.

**Key Words:** cardiovascular disease, exercise, inflammation, physical activity, postprandial lipaemia
### Abbreviations:

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
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<tbody>
<tr>
<td>AUC</td>
<td>Area under the curve</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary heart disease</td>
</tr>
<tr>
<td>CRP</td>
<td>C-reactive protein</td>
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<tr>
<td>ES</td>
<td>Effect size</td>
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<tr>
<td>HDL-C</td>
<td>High-density lipoprotein cholesterol</td>
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<td>IL-6</td>
<td>Interleukin-6</td>
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<tr>
<td>RER</td>
<td>Respiratory exchange ratio</td>
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<tr>
<td>RPE</td>
<td>Ratings of perceived exertion</td>
</tr>
<tr>
<td>TAG</td>
<td>Triacylglycerol</td>
</tr>
<tr>
<td>TC</td>
<td>Total cholesterol</td>
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<tr>
<td>UK</td>
<td>United Kingdom</td>
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<tr>
<td>V̇O₂ max</td>
<td>Maximum oxygen uptake</td>
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INTRODUCTION

South Asians represent the largest ethnic minority (4.1%) in the United Kingdom (UK) and are comprised of individuals with ancestral origins in India, Pakistan, Sri Lanka, Nepal, and Bangladesh (Blackledge et al. 2003). Numerous studies have shown that South Asian descendants have an increased risk of coronary heart disease (CHD), both in South Asia and after migration to Western nations (Ghaffar et al. 2004; Wild et al. 2007).

Physical inactivity is a well-established risk factor for CHD and has been estimated to explain > 20% of the excess CHD mortality experienced by UK South Asians after adjustment for potential confounders including socioeconomic status, smoking, diabetes and existing cardiovascular disease (Williams et al. 2011a). Although observational evidence suggests a cardio-protective role of physical activity on CHD and CHD risk markers in South Asians (Rastogi et al. 2004; Mohan et al. 2005), to the authors’ knowledge, only one study has investigated the acute effects of exercise in this population. In this regard, previous work from our laboratory demonstrated that 60 min of running exercise induced a greater reduction in postprandial triacylglycerol (TAG) concentrations in South Asian compared with White European men (Arjunan et al. 2013). Additionally, running exercise decreased other CHD risk factors, including fasting total cholesterol (TC) and postprandial interleukin-6 (IL-6) concentrations, to a similar extent in both ethnicities (Arjunan et al. 2013).

Considering the high levels of physical inactivity within the South Asian population (Yates et al. 2010; Williams et al. 2011b), physical activity interventions are likely to focus on lower intensity exercise. In this regard, walking exercise is achievable for the majority of the population, has little risk of injury and is generally regarded as the main option for increasing physical activity in sedentary populations (Morris and Hardman 1997). Furthermore, walking exercise is associated with a reduced risk of cardiovascular disease and has been shown to
acutely reduce postprandial TAG concentrations in Western populations (Wannamethee and Sharper 2001; Miyashita et al. 2008). The acute effect of low to moderate intensity exercise such as walking on CHD risk markers in South Asians is currently unknown and requires investigation. This topic is particularly prominent considering the recent development of physical activity guidelines specific to South Asian populations (Misra et al. 2012).

Thus, the aim of this study was to compare the effects of a single bout of brisk walking on CHD risk markers in South Asian compared with White European men. The primary outcome variable in this study was postprandial TAG concentration, as an established CHD risk factor (Bansal et al. 2007; Nordestgaard et al. 2007) that is frequently reported to be lowered by exercise (Maraki and Sidossis 2013). In addition, several other disease risk markers were examined in this study including TC, high-density lipoprotein cholesterol (HDL-C), glucose, insulin, C-reactive protein (CRP) and IL-6. It was hypothesised that South Asian men would exhibit an impaired postprandial metabolic profile compared with White European men, but a single bout of brisk walking would be equally, if not more, effective for reducing postprandial TAG concentrations in South Asian than White European men.

METHODS

Participants

With the approval of Loughborough University’s Ethics Advisory Committee, 15 South Asian and 14 White European men were recruited and they gave their written informed consent to participate in this study. This sample size was deemed sufficient to detect a 10% difference in TAG area under the curve (the primary outcome variable) based on previous data from our laboratory (Miyashita et al. 2008). This calculation was performed using G*power with an alpha value of 5% and a power of 90% (Faul et al. 2007). All participants had a body mass index < 30 kg·m⁻². Information from a general health questionnaire revealed
participants were non-smokers, not taking medication, had no personal history of cardiovascular or metabolic disease, were not currently dieting and were aged 19 to 30 years. A physical activity questionnaire revealed participants were habitually recreationally active but no difference in habitual activity levels was seen between the South Asian and European men. To verify ethnicity each South Asian participant completed a form providing details of their place and country of birth, their mother tongue language, their religion and race, the country of their parents’ birth and their family history of migration. This revealed that seven of the participants were Indian nationals, five were UK Indians, one was a UK Pakistani and two were Sri Lankan nationals. All of the European participants were white with white parents, and all were British citizens.

**Preliminary Measures**

Prior to the main trials participants visited the laboratory to undergo screening, familiarization, anthropometric measurements and a maximal walking exercise test to determine maximum oxygen uptake (\( \dot{V}O_2 \max \)). Height and body weight were measured and body mass index was subsequently calculated. Body fat percentage was estimated via skinfold measurements (Durnin and Womersley 1974) and waist circumference determined as the narrowest part of the torso between the xiphoid process and the iliac crest. Blood pressure was measured using a digital monitor (Omron M5-1, Matsusaka Co., Japan).

After familiarisation with the testing equipment, walking \( \dot{V}O_2 \max \) was determined using an incremental uphill walking protocol at a constant speed until participants reached volitional exhaustion. The initial treadmill gradient was set at 3.5% and the gradient was increased by 2.5% every 3 minutes (Taylor et al. 1955). Walking \( \dot{V}O_2 \max \) was determined from an expired gas sample collected during the final minute of the test when participants signalled that they could only continue for an additional 1 min. Heart rate (Polar T31; Polar Electro,
Kempele, Finland) and ratings of perceived exertion (RPE) (Borg 1973) were monitored throughout the test. The calculated \( \dot{V}O_2 \) max was used to determine the relative intensity of exercise during the main trials.

**Main trials**

Participants completed two, 2-day trials (exercise and control) in a random order separated by an interval of at least one week. Trials were undertaken in block random order (block size of two) using software available at http://www.randomization.com/. On day 1 of the trials, participants arrived at the laboratory between 8.00 am and 9.00 am and rested (reading, working at a computer, watching television, listening to music or playing video games) throughout the day until 5.00 pm. Participants consumed a standardized lunch at approximately 12.00 pm containing white bread, chocolate spread, butter, whole fat milk, chocolate muffin, apple, pear and water. Trials were identical except that 60 min brisk walking was performed at 3.30 pm during the exercise trial. Participants were encouraged to walk as fast as was comfortably possible at a sustainable pace. The objective was to simulate a natural setting/daily activity, hence intensity was not fixed. One minute samples of expired gas were collected at 15 minute intervals during the walk to monitor the exercise intensity. If necessary, adjustments were made to the treadmill speed to ensure participants were walking briskly. Heart rate and RPE were also monitored during the walk. Participants rested throughout day 1 of the control trial and expired gas was collected into Douglas bags for 5 min every 15 min at equivalent time points to the exercise trial i.e. between 3.30 pm and 4.30 pm equivalent to minutes 10-15, 25-30, 40-45 and 55-60 during the walk. Net energy expenditure (gross energy expenditure of exercise minus energy expenditure at rest) was subsequently calculated using the equations of Frayn (1983).
On day 2 of the main trials participants arrived at the laboratory between 7.45 am and 8.00 am having fasted overnight (no food or drink except plain water) for 10 hours. On arrival to the laboratory, participants rested in a semi-supine position for 5 minutes before a cannula (BD Venflon, Becton-Dickinson, Helsingborg, Sweden) was inserted into an antecubital vein and a fasting blood sample was collected. Participants then consumed a prescribed test meal (see below). A clock was started at the moment participants commenced the meal and this was identified as 0 h. The trial continued for nine hours during which a total of 14 (including the fasting sample), 9 mL venous blood samples were collected. At 4 h, a second test meal, identical to the first, was consumed by the participants. Participants rested throughout day 2 of both the control and exercise trials and hence day 2 of these trials was identical.

**Control of diet and exercise**

The day before each main trial and on the first day of each main trial, participants recorded their food intake using a weighed food diary. Participants replicated this food intake for the subsequent main trial. Participants abstained from coffee, tea and alcohol on the day prior to and during the main trials. Participants were also asked to refrain from strenuous physical activity during the day preceding the main trials and on day 1 and day 2 of the main trials.

**Test meals**

The test meals consisted of white bread, butter, cheese, mayonnaise, crisps, chocolate milk shake powder and whole milk. The amount of food consumed was adjusted for each participant based on their body weight and was kept constant throughout the trials. The macronutrient content of the test meal was 57% fat, 32% carbohydrate and 11% protein and each meal provided 60 kJ (14.3 kcal) energy per kg body mass. Participants consumed each meal within 15 minutes and water was available *ad libitum.*
**Blood sampling**

Venous blood samples were collected for the measurement of TC, HDL-C, TAG, glucose, insulin, CRP and IL-6. Samples were collected in the fasted state and at hourly intervals thereafter for 9 h. Additional samples were collected 15 and 30 min after each meal, i.e. at 0.25, 0.5, 4.25 and 4.5 h. Concentrations of TC, HDL-C and CRP were measured only from fasting samples. IL-6 concentrations were measured from samples collected at 0, 3, 6 and 9 h. Insulin was measured at 0, 0.5, 2, 4, 4.5, 6 and 9 h. Glucose and TAG were measured from all samples. Participants rested in a semi-supine position during blood sampling. Venous blood samples were drawn into pre-cooled 9 mL EDTA monovette tubes (Starstedt, Leicester, United Kingdom) and immediately centrifuged at 1165 x g for 10 minutes at 4ºC (Labofuge 400R, Thermo Scientific, Langenselbold, Germany). The plasma supernatant was dispensed into eppendorf tubes and stored at -20ºC for later analysis. Measurements of haemoglobin and haematocrit were taken at 0 and 9 h to estimate the acute change in plasma volume across the trial (Dill and Costill 1974).

**Biochemical analysis**

Plasma TC, HDL-C, TAG and glucose concentrations were determined spectrophotometrically using commercially available kits and a bench top analyser (Penta 400, HORIBA ABX Diagnostics, Montpellier, France). Enzyme linked immunosorbent assays were used to determine plasma concentrations of IL-6 (high sensitivity kit; R&D Systems, Abingdon, UK), insulin (Mercodia, Uppsala, Sweden) and CRP (high sensitivity kit, IBL International, Hamburg, Germany). To eliminate inter assay variation, samples from each participant were analysed in the same run. The within batch coefficient of variation for each assay was as follows: 0.7% for TC, 0.7% for HDL-C, 0.7% for TAG, 0.9% for glucose, 4.3% for IL-6, 4.5% for insulin, and 4.3% for CRP.
**Statistical analyses**

Data was analysed using Predictive Analytics Software version 18.0 for Windows (SPSS, Inc., Somers, NY, USA). Physical characteristics and exercise responses are presented as mean (SD) and were compared between South Asians and Europeans using Student’s independent t-tests. Time-averaged area under the curve (AUC) values were calculated using the trapezoidal method. Concentrations of plasma metabolites were not normally distributed and were natural log transformed prior to analyses. These data are presented as geometric mean (95% confidence interval). Linear mixed models, both unadjusted and adjusted for age and percentage body fat, were employed to examine fasting and AUC differences for plasma constituents with fixed (condition and group) and random (participants) factors. Absolute standardised effect sizes (ES) were calculated to supplement important findings by dividing the difference between the mean values (exercise versus control or South Asian versus European) with the pooled standard deviation. An ES of 0.2 was considered the minimum important difference for all outcome measures, 0.5 moderate and 0.8 large (Cohen 1988). Statistical significance for this study was accepted as P < 0.05. Graphical representations of results are presented as mean (SEM) to avoid distortion of the graphs.

**RESULTS**

**Participant characteristics**

The physical characteristics of the participants are displayed in Table 1. There were no significant differences between South Asian and White European participants for weight, resting systolic blood pressure and resting diastolic blood pressure. Age, body mass index, percentage body fat and waist circumference were significantly higher in South Asian compared with White European participants, while height and VO₂ max were significantly lower in South Asian compared with White European participants (all P < 0.05).
Responses to treadmill brisk walking

The European participants walked faster than the South Asian participants but experienced a lower heart rate (walking speed: 7.1 (0.3) vs. 6.4 (0.5) km·h⁻¹; heart rate: 126 (11) vs. 145 (16) beats·min⁻¹ for European and South Asian participants, respectively; both P ≤ 0.001). There were no significant differences between groups for relative exercise intensity, energy expenditure, respiratory exchange ratio (RER) or RPE (exercise intensity: 48.3 (6.8) vs. 52.7 (8.0) % \( \dot{V}O_2 \) max; energy expenditure: 1715 (343) vs. 1555 (359) kJ; RER: 0.95 (0.05) vs. 0.95 (0.05); RPE: 11 (2) vs. 11 (2) for European and South Asian participants, respectively; all P ≥ 0.13).

Fasting plasma metabolite concentrations

Fasting plasma metabolite concentrations are detailed in Table 2. Linear mixed models revealed lower fasting concentrations in the exercise compared with the control trial for TAG (ES = 0.31, P = 0.004) and insulin (ES = 0.48, P = 0.045). No other differences were seen in fasting metabolite concentrations between trials (P ≥ 0.11). Main effects of group revealed higher fasting glucose (ES = 0.72, P = 0.04), insulin (ES = 1.11, P = 0.002), IL-6 (ES = 0.96, P = 0.004) and CRP (ES = 1.36, P < 0.001) concentrations and a higher TC/HDL-C ratio (ES = 1.27, P = 0.002) in the South Asian participants compared with White Europeans, as well as a lower HDL-C concentration in South Asians (ES = 1.61, P < 0.001). Fasting plasma TAG and TC concentrations were similar in South Asians and White Europeans (P ≥ 0.11). After adjustment for age and percentage body fat, between-group differences remained significant and/or meaningful for glucose (ES = 1.00, P = 0.04), CRP (ES = 0.72, P = 0.07), HDL-C (ES = 1.14, P = 0.04) and TC/HDL-C ratio (ES = 0.94, P = 0.09), but were no longer significant for insulin and IL-6 concentrations (P ≥ 0.36). The magnitude of change in fasting
metabolite concentrations following exercise was similar in South Asians and White Europeans (P ≥ 0.07).

**Postprandial plasma metabolite concentrations**

Area under the curve values are presented in Table 3 (and displayed graphically in Figures 1 and 2). A main effect of trial revealed a lower AUC concentration for TAG (ES = 0.23, P = 0.01) and insulin (ES = 0.58, P = 0.01) in the exercise compared with the control trial; AUC concentrations for glucose and IL-6 were not different between trials (P ≥ 0.63). Linear mixed models revealed higher AUC concentrations in South Asians compared with White Europeans for TAG (ES = 0.97, P = 0.02), glucose (ES = 0.72, P = 0.03), insulin (ES = 1.09, P = 0.002) and IL-6 (ES = 0.86, P = 0.01). Between-group differences were no longer significant after adjustment for age and percentage body fat for postprandial TAG, glucose and insulin concentrations (P ≥ 0.19), but a tendency for a higher IL-6 AUC concentration in South Asians remained (ES = 0.74, P = 0.08). The magnitude of change in postprandial metabolite concentrations following exercise was similar in both groups (P ≥ 0.47).

Figure 3 displays the difference in the AUC concentrations for TAG (panel A), glucose (panel B) and insulin (panel C) between the exercise and the control trial for each individual participant. Negative values indicate a lowering of the postprandial metabolite concentration on the exercise trial compared with the control trial. This figure demonstrates that the postprandial TAG concentration was lower on the exercise trial in nine out of 15 (60%) South Asian participants and ten out of 14 (71%) European participants. The postprandial glucose concentration was lower on the exercise trial in nine out of 15 (60%) South Asian men and eight out of 14 (57%) European men. The postprandial insulin concentration was lower on the exercise trial in 11 out of 15 (73%) South Asian men and ten out of 14 (71%) European men.
DISCUSSION

The primary finding of this study is that South Asian men exhibited elevated postprandial plasma TAG, glucose, insulin, and IL-6 concentrations in comparison with White European men after consuming high-fat meals. However, the stark between-group differences in postprandial metabolism were diminished or eliminated completely after controlling for differences in age and percentage body fat. Additionally, an acute bout of brisk walking reduced postprandial plasma TAG and insulin concentrations.

To the author’s knowledge, only two previous studies have compared postprandial TAG responses in South Asian and White European men (Cruz et al. 2001; Arjunan et al. 2013). In accordance with previous research from our laboratory (Arjunan et al. 2013), the present study demonstrated elevated postprandial TAG concentrations in South Asian compared with White European men. While previous research has demonstrated elevated fasting TAG concentrations in South Asians compared with white populations (Anand et al. 2000; Tziomalos et al. 2008), confirmation of elevated postprandial concentrations is significant considering the superior association between postprandial TAG and CHD risk (Bansal et al. 2007; Nordestgaard et al. 2007). In support of previous research, elevated postprandial glucose and insulin responses were also observed in South Asian participants, suggesting a degree of insulin resistance (Cruz et al. 2001; Arjunan et al. 2013). Insulin resistance is thought to be a major contributor to CHD risk in South Asians, even in those with normal glucose tolerance (Sandeep et al. 2011). Furthermore, it seems plausible that the elevated plasma insulin concentrations in the present study may be linked with the observed elevations in postprandial TAG. In this regard, hyperinsulinaemia, resulting from insulin resistance, is thought to decrease muscle lipoprotein lipase activity (Pollare et al. 1991), therefore impairing one of the major mechanisms for the removal of TAG from the blood. In addition, insulin resistance may exaggerate postprandial lipaemia by inhibiting the normal suppressive
action of insulin on hepatic very low-density lipoprotein production (Malmström et al. 1997). Although the underlying mechanisms require further investigation, the findings of the present study substantiate preliminary evidence that currently healthy South Asians exhibit impaired postprandial metabolic responses compared with White European men (Cruz et al. 2001; Arjunan et al. 2013).

One contrast with the findings of the present study is that Cruz and colleagues (2001) did not observe an elevated lipaemic response to high fat meals in South Asian participants. A possible explanation for this discrepancy is that percentage body fat did not differ between ethnic groups in the study conducted by Cruz and colleagues (2001), but body fat percentage was higher in the South Asians in this study and the between-group difference in postprandial lipaemia was no longer significant after controlling for age and percentage body fat in the present study. It has been reported previously that centrally obese but otherwise healthy men exhibit greater postprandial perturbations in TAG, glucose and insulin concentrations compared with age-matched healthy weight men (Gill et al. 2004), and indices of adiposity (e.g. BMI, percentage body fat) are positively correlated with the postprandial TAG response (Couillard et al. 1998). Although a higher body fat in South Asians is representative of Western society (Banerji et al. 1999; Misra and Shrivastava 2013), this may have enhanced any differences in TAG between ethnicities. Nevertheless, previous findings from our laboratory with better-matched groups found greater postprandial TAG concentrations in South Asians even after adjusting for body fat differences (Arjunan et al. 2013). Clearly further work is required to determine the role that adiposity and ethnicity play in modifying postprandial metabolism in South Asians. A second possible explanation for the higher postprandial TAG response in the present study is the provision of two test meals, which increased the scope to detect meaningful differences between ethnicities and better reflects normal dietary practice in Western society. Whatever the explanation for the disparity
between these studies, the contrast in postprandial TAG concentrations between South Asians and White Europeans is worthy of further investigation.

An important finding from the present study is that a single session of brisk walking significantly reduced postprandial TAG and insulin concentrations. This adds to the debate about the effectiveness of walking exercise in reducing postprandial lipaemia. In this regard, although several studies have demonstrated reductions in postprandial TAG after walking exercise of 30 – 120 min in duration (Gill et al. 2001, 2003, 2004; Miyashita et al. 2008, 2013; Burns et al. 2009; Hurren et al. 2011; Kim et al. 2014), this is not a universal finding (Gabriel et al. 2012; Heden et al. 2013). In accordance with previous findings, walking exercise did not influence the glucose response to high fat feeding (Miyashita et al. 2008; Gabriel et al. 2012; Heden et al. 2013); however, the exercise-induced reduction in the postprandial insulin concentration appears to contrast these studies (Miyashita et al. 2008; Gabriel et al. 2012; Heden et al. 2013). Nevertheless, the current study highlights the potential for metabolic health benefits following a single session of brisk walking exercise in South Asian and White European men.

A consensus of research has demonstrated that the energy cost of exercise is predictive of the magnitude of reduction in postprandial TAG concentrations the next day (Gill and Hardman 2003). However, meaningful reductions in postprandial lipaemia are still evident after modest doses of moderate-intensity exercise (~1 MJ) (Miyashita et al. 2008). In addition to observations regarding the energy cost of exercise, recent research has also found the intensity of exercise to be predictive of decreases in postprandial TAG concentrations (Gabriel et al. 2012). Furthermore, decreases in insulin AUC during an oral glucose tolerance test have also been reported to be a function of exercise intensity (Kang et al. 1996; Seals et al. 1984). Considering the elevated CHD risk profile of South Asians (Cruz et al. 2001),
further research is required to elucidate the effectiveness of walking exercise to reduce CHD risk in this population.

In the present study, brisk walking reduced the postprandial TAG concentration to a similar extent in South Asian and White European men (8% vs. 10% respectively), contrasting previous findings from our laboratory reporting a greater exercise-induced reduction in the South Asian men after 60 min running at 70% \( \dot{V}O_2 \) max (22% vs. 10% respectively; Arjunan et al. 2013). The reason for this discrepancy is unclear but it is possible that South Asians may require a greater exercise intensity and/or energy expenditure than 60 min of brisk walking to maximise the acute reductions in TAG. However, this is only the second study to investigate the metabolic response to exercise in South Asian participants and future research is warranted before recommendations are made. Furthermore, considering the high levels of physical inactivity in South Asians (Yates et al. 2010; Williams et al. 2011b), walking exercise may represent the most suitable exercise mode for many individuals beginning exercise programs before advancing to higher intensity exercises such as running.

Concentrations of IL-6 and CRP were also assessed in the present study as independent indicators of chronic low grade inflammation and predictors of future CHD (Pradhan et al. 2001; Anand et al. 2004; Langenberg et al. 2006; Danesh et al. 2008). In support of previous research, South Asian participants exhibited elevated fasting concentrations of CRP (Chambers et al. 2001; Anand et al. 2004). Furthermore, fasting and postprandial IL-6 concentrations were also elevated in South Asian compared with White European participants, although these between-group differences were diminished after controlling for age and percentage body fat. It has previously been suggested that an increased disposition to chronic low grade inflammation may explain the elevated CHD risk experienced by South Asians (Tziomalos et al. 2008). However, current evidence also suggests that pro-
inflammatory IL-6 is secreted from adipose tissue and subsequently stimulates CRP release (Bastard et al. 1999). It is therefore plausible that the differences in inflammation between ethnicities in the present study may have been influenced by higher adiposity in the South Asian participants. Further research is required to differentiate the effects of adiposity and ethnicity on inflammatory profiles in South Asians. Furthermore, although running exercise has been shown to reduce postprandial IL-6 concentrations in both White Europeans and South Asians (Arjunan et al. 2013), walking exercise did not have any effect on this inflammatory marker in the present study.

Although this study provides novel data regarding the effects of exercise on fasting and postprandial metabolic responses, this study is limited by the small sample size and the majority of South Asian participants originated from India. Additionally, the recruitment of participants using convenience sampling precluded the exact matching of possible physical and physiological determinants of the metabolic outcome measures between the two groups. Subsequently, the findings from the present study require confirmation with a larger sample appropriately matched for physical and physiological characteristics (e.g. age, percentage body fat) and in other South Asian groups (e.g. Bangladeshis, Pakistanis and Nepalese). The effect of exercise on postprandial responses in females also requires investigation.

In conclusion, the present study supports preliminary findings that fasting and postprandial CHD risk markers are elevated in South Asian compared with White European men, but these differences were diminished after controlling for confounding factors. In addition, a single session of brisk walking reduced postprandial TAG and insulin concentrations to a similar extent in South Asian and White European men. Further research into the metabolic responses to exercise in South Asians is warranted.
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ETHICAL STANDARDS

This investigation was conducted according to UK ethical standards for scientific research involving human participants.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.
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Fig. 1 Mean (SEM) postprandial plasma triacylglycerol (top panel), glucose (middle panel), and insulin (bottom panel) concentrations measured on day 2 of the exercise and control trials for South Asian \((n = 15)\) and European \((n = 14)\) men. South Asian control trial (●); South Asian exercise trial (○); European control trial (▼); European exercise trial (▽)

Fig. 2 Mean (SEM) postprandial plasma concentrations of interleukin-6 measured on day 2 of the exercise and control trials for South Asian \((n = 15)\) and European \((n = 14)\) men. South Asian control trial (●); South Asian exercise trial (○); European control trial (▼); European exercise trial (▽)

Fig. 3 Individual changes (exercise minus control) in the total area under the plasma metabolite concentration versus time curve in response to the 60 minute brisk treadmill walk for South Asian \(\square; n = 15\) and White European \(\square; n = 14\) men: A) triacylglycerol (TAG); B) glucose; C) insulin. Negative values indicate lower concentrations on the exercise trial. Participant data are organised according to the size of the intervention-induced change in the total area under the curve response; thus, the order of the individual participants is not identical in A, B and C
Table 1. Participant characteristics.

<table>
<thead>
<tr>
<th>Variable</th>
<th>South Asians (n = 15)</th>
<th>Europeans (n = 14)</th>
<th>Europeans vs. South Asians 95% CI (^a)</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24 (3)</td>
<td>22 (1)</td>
<td>0.4 to 3.6(^*)</td>
<td>0.96</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170.5 (8.4)</td>
<td>180.2 (3.9)</td>
<td>-14.7 to -4.7(^*)</td>
<td>1.48</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>74.1 (11.5)</td>
<td>73.6 (8.3)</td>
<td>-7.2 to 8.2</td>
<td>0.05</td>
</tr>
<tr>
<td>Body mass index (kg·m(^{-2}))</td>
<td>25.4 (3.3)</td>
<td>22.7 (2.2)</td>
<td>0.6 to 5.0(^*)</td>
<td>0.99</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>21.7 (6.0)</td>
<td>12.5 (4.6)</td>
<td>5.2 to 13.3(^*)</td>
<td>1.74</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>81.6 (9.2)</td>
<td>75.4 (5.3)</td>
<td>0.5 to 11.9(^*)</td>
<td>0.82</td>
</tr>
<tr>
<td>Resting SBP (mm Hg)</td>
<td>129 (10)</td>
<td>136 (14)</td>
<td>-16 to 2</td>
<td>0.62</td>
</tr>
<tr>
<td>Resting DBP (mm Hg)</td>
<td>76 (7)</td>
<td>77 (6)</td>
<td>-6 to 5</td>
<td>0.07</td>
</tr>
<tr>
<td>(\dot{V}O_2) max (mL·kg(^{-1})·min(^{-1}))</td>
<td>40.7 (7.2)</td>
<td>49.2 (8.0)</td>
<td>-14.3 to -2.8(^*)</td>
<td>1.12</td>
</tr>
</tbody>
</table>

All values are mean (SD). Means were compared using Student’s independent \(t\)-tests. \(^a\)95% confidence interval of the mean absolute difference between the groups.

\(^*\) Significant difference between South Asians and Europeans (P < 0.05)

SBP, systolic blood pressure; DBP, diastolic blood pressure; \(\dot{V}O_2\) max, maximal oxygen uptake.
Table 2. Fasting plasma concentrations on day two of the main trials.

<table>
<thead>
<tr>
<th>Variable</th>
<th>South Asians (n = 15)</th>
<th>Europeans (n = 14)</th>
<th>Control vs. Exercise</th>
<th>Control vs. South Asians</th>
<th>Model 1&lt;sup&gt;a&lt;/sup&gt; Europeans vs. South Asians</th>
<th>Model 2&lt;sup&gt;b&lt;/sup&gt; Europeans vs. South Asians</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Exercise</td>
<td>Control</td>
<td>Exercise</td>
<td>95% CI&lt;sup&gt;c&lt;/sup&gt;</td>
<td>95% CI&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>TC (mmol·L&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>3.98(3.66 to 4.31)</td>
<td>3.94(3.63 to 4.27)</td>
<td>3.79(3.48 to 4.13)</td>
<td>3.80(3.49 to 4.14)</td>
<td>-4 to 3%</td>
<td>-7 to 17%</td>
</tr>
<tr>
<td>HDL-C (mmol·L&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>0.91(0.83 to 1.01)</td>
<td>0.94(0.85 to 1.03)</td>
<td>1.21(1.09 to 1.34)</td>
<td>1.21(1.09 to 1.34)</td>
<td>-2 to 5%</td>
<td>-33 to -12%**</td>
</tr>
<tr>
<td>TC/HDL-C ratio</td>
<td>4.35(3.81 to 4.97)</td>
<td>4.21(3.68 to 4.81)</td>
<td>3.13(2.73 to 3.60)</td>
<td>3.14(2.73 to 3.60)</td>
<td>-4 to 1%</td>
<td>13 to 65%**</td>
</tr>
<tr>
<td>TAG (mmol·L&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>1.26(1.00 to 1.60)</td>
<td>1.00(0.79 to 1.27)</td>
<td>0.89(0.70 to 1.14)</td>
<td>0.84(0.66 to 1.07)</td>
<td>-21 to -5%*</td>
<td>-6 to 80%</td>
</tr>
<tr>
<td>Glucose (mmol·L&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>5.59(5.36 to 5.83)</td>
<td>5.40(5.18 to 5.63)</td>
<td>5.19(4.97 to 5.42)</td>
<td>5.18(4.96 to 5.41)</td>
<td>-4 to 1%</td>
<td>0 to 12%**</td>
</tr>
<tr>
<td>Insulin (µU.mL&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>6.6(5.1 to 8.6)</td>
<td>5.2(4.0 to 6.7)</td>
<td>3.9(2.9 to 5.1)</td>
<td>3.2(2.4 to 4.1)</td>
<td>-36 to -1%*</td>
<td>22 to 131%**</td>
</tr>
<tr>
<td>IL-6 (pg·mL&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>0.80(0.50 to 1.28)</td>
<td>1.11(0.70 to 1.78)</td>
<td>0.34(0.21 to 0.55)</td>
<td>0.45(0.28 to 0.74)</td>
<td>-7 to 100%</td>
<td>36 to 329%**</td>
</tr>
<tr>
<td>CRP (µg.mL&lt;sup&gt;-1&lt;/sup&gt;)</td>
<td>0.45(0.17 to 1.16)</td>
<td>0.54(0.21 to 1.40)</td>
<td>0.04(0.02 to 0.11)</td>
<td>0.05(0.02 to 0.13)</td>
<td>-46 to 152%</td>
<td>244 to 3450%**</td>
</tr>
</tbody>
</table>

All values are geometric mean (95% confidence interval). Statistical analyses are based on log transformed data. Comparisons were made using linear mixed models. <sup>a</sup> Model 1: unadjusted. <sup>b</sup> Model 2: adjusted for age and percentage body fat. <sup>c</sup> 95% confidence interval for the ratio of geometric means.

* Significant difference between exercise and control trials (P < 0.05)

** Significant difference between South Asians and Europeans (P < 0.05)
TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; TAG, triacylglycerol; IL-6, interleukin-6; CRP, C-reactive protein.