A 12-week low carbohydrate, high fat (LCHF) diet improves metabolic health outcomes over a control diet in a randomised controlled trial with overweight defence force personnel.

<table>
<thead>
<tr>
<th>Journal:</th>
<th>Applied Physiology, Nutrition, and Metabolism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Manuscript ID</td>
<td>apnm-2017-0260.R1</td>
</tr>
<tr>
<td>Manuscript Type:</td>
<td>Article</td>
</tr>
<tr>
<td>Date Submitted by the Author:</td>
<td>02-Jun-2017</td>
</tr>
</tbody>
</table>
| Complete List of Authors: | Zinn, Caryn; Auckland University of Technology, Human Potential Centre  
McPhee, Julia; Auckland University of Technology, Human Potential Centre  
Harris, Nigel; Auckland University of Technology, Human Potential Centre  
Williden, Micalla; Auckland University of Technology, Human Potential Centre  
Prendergast, Kate; Auckland University of Technology, Human Potential Centre  
Schofield, Grant; Auckland University of Technology, Human Potential Centre |
| Is the invited manuscript for consideration in a Special Issue?: | |
| Keyword: | Low-carbohydrate, high-fat, LCHF, metabolic health, weight loss |
Title

A 12-week low carbohydrate, high fat (LCHF) diet improves metabolic health outcomes over a control diet in a randomised controlled trial with overweight defence force personnel.

Authors

Caryn Zinn, Julia McPhee, Nigel Harris, Micalla Williden, Dr Kate Prendergast, Grant Schofield

Corresponding author

Dr Caryn Zinn. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Private Bag 92006, Auckland 1142, New Zealand. PH: +64 9 921 9999 ext. 7842. Fax: +64 9 921 9960. Email: caryn.zinn@aut.ac.nz.

Other authors

Julia McPhee. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Email: julia.mcphee@aut.ac.nz

Dr Nigel Harris. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Email: nigel.harris@aut.ac.nz

Dr Micalla Williden. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Email: mwilliden@gmail.com

Dr Kate Prendergast. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Email: kprendergast.nzl@gmail.com

Professor Grant Schofield. AUT; Human Potential Centre. Faculty of Health & Environmental Sciences. Email: grant.schofield@aut.ac.nz
Abstract

Introduction. Overweight, obesity and poor health is becoming a global concern for defence force personnel. Conventional nutrition guidelines are being questioned for their efficacy in achieving optimal body composition and long-term health. This study compared the effects of a 12-week low-carbohydrate, high-fat diet with a conventional, high-carbohydrate, low-fat diet on weight reduction and metabolic health outcomes in at-risk New Zealand Defence Force personnel.

Materials and methods. In this randomised controlled trial, 41 overweight personnel were assigned to intervention and control groups. Weight, waist circumference, fasting lipids and glycaemic control were assessed at baseline and at 12 weeks. Within-group change scores were analysed using the t-statistic and interpreted using a p<0.05 level of statistical significance. Between-group mean differences and confidence intervals were analysed using effect sizes and magnitude-based inferences.

Results. Twenty-six participants completed the trial (14, intervention; 12, control). Both groups showed statistically significant weight and waist circumference reductions; the intervention group significantly reduced triglycerides and serum glucose, and significantly increased HDL cholesterol. Relative to control, the intervention group showed small, possibly-to-likely beneficial effects for weight, triglycerides, glucose, insulin and HOMA-IR, moderate, likely beneficial effects for HDL cholesterol, triglyceride: HDLc ratio and HbA1c, and a small, likely harmful effect for LDL cholesterol.

Discussion. This dietary approach shows promise for short-term weight loss and improved metabolic health outcomes conditions, compared to mainstream recommendations. It should be offered to defence force personnel at least as a viable alternative means to manage their weight and health.
Keywords

Low-carbohydrate, high-fat; LCHF; metabolic health; weight loss; lipids; defence force
Introduction

The conditions of overweight and obesity, alongside ill-health, continue to rise on a global level (World Health Organization 2016b). Evidence from US defence force personnel indicates that this population group is not spared from weight and health-related issues, with reports of an all-time high in overweight and obesity documented in 2005 (60.5%). (Smith et al. 2012; Tanofsky-Kraff et al. 2013). The increasing health consequences of an overweight military population is cause for concern given the association between obesity and operational effectiveness and long-term health (Robbins et al. 2001).

The current mainstream national nutrition guidelines which are considered to be “best practice” for weight control and optimal health are having little efficacy in achieving a long-term healthy body mass and optimal health in specific populations (Howard et al. 2006; Pirozzo et al. 2002). These recommendations are based on a high carbohydrate, moderate protein, low fat approach, centred on the energy balance concept of “eating less and moving more” (Ministry of Health and Clinical Trials Research Unit 2009). While there might very well be an element of portion control and exercise that contributes to weight loss maintenance and health, the current formula for dietary macronutrient contribution to total energy intake and calorie restriction does not take into account the complex intrinsic milieu of nutrient-hormone interactions (Bruning et al. 2000; Wieser et al. 2013). In particular, it is the post-prandial insulin surge and its consistent elevation following a high carbohydrate load that is problematic, particularly for populations with insulin resistance. Consistently elevated levels of insulin, or hyperinsulinaemia, not only makes for a challenging body fat-burning environment, but also has been shown to play a causal role in chronic diseases such as metabolic syndrome, diabetes and cardiovascular disease (Crofts et al. 2015).
A low-carbohydrate, high-fat (LCHF) dietary approach for weight control is certainly not new; (Banting 1865; Osler 1978) however, more recently, it has become substantially more popular in both research and practice settings. On the whole, randomised controlled trials (RCTs) show promise, and demonstrate that it is at least as effective as a conventional high carbohydrate, low fat approach for reducing body weight and waist circumference, and is more effective for improving triglyceride levels, HDL cholesterol (HDLc) and glycaemic control (Bazzano et al. 2014; Forsythe et al. 2008; Gardner et al. 2007; Nordmann et al. 2006).

In this study, we tested the hypothesis that a 12-week LCHF dietary intervention would be at least as effective as a control diet (high carbohydrate, low fat) in reducing weight and be more effective at improving metabolic health outcomes in at-risk personnel. The desired longer term aims are to improve the long-term health and wellbeing risk profile of at-risk individuals, to help reduce the number of personnel unfit for operational service, and to reverse the growing proportion of overweight and obese individuals within the New Zealand Defence Force (NZDF).

Materials and Methods

This was an RCT allocated with a computer-assisted 1:1 randomisation technique, and was conducted at a Naval Base in Auckland, New Zealand. The trial was registered with the Australian New Zealand Clinical Trials Registry (Registration number: ACTRN12616001579482). The study was approved by the AUT ethics committee (approval number 14/246).
The details of the study were advertised at the local naval base via posters, the intranet, a newsletter, and an email invitation during the months of October and December 2014. Interested individuals were sent an information sheet, consent form, and screening questionnaire via email and were provided with instructions to return signed documents by email or via a drop box at a designated location on the base. A 3-day dietary recall was submitted as part of this package (two week days and two weekend days) which was assessed for carbohydrate quantity. Participants were excluded if they were consuming a diet that contained 250g or less of carbohydrate, and if they had a BMI < 25 (as assessed via screening questionnaire). A sample size was calculated for magnitude based inferences for the primary outcome, weight (Hopkins 2006). Using Cohen’s smallest standardised change for beneficial (-0.2) and harmful (0.2) between-group differences, the minimum sample required was estimated to be 23. We recruited the total available, and eligible, sample (i.e., 41 participants), thereby accounting for a potential attrition rate of 56%.

Protocol
A total of 41 suitable NZDF personnel were randomly assigned to one of two groups: 1) a control group (standard Ministry of Health guidelines for weight loss i.e., a high carbohydrate, low fat diet) or, 2) Intervention - LCHF group. Participants were advised to maintain existing exercise habits. Prior to beginning the 12-week dietary intervention, all participants were required to attend a compulsory workshop specific to their dietary protocol and were directed to specific resources for guidance. The workshops were designed to provide participants with information on the study protocols, outline weekly data collection requirements, and to inform participants of the dietary programme assigned to them. Each week during the study period participants were required to complete a 0-10 satiety scale (0 = very hungry; 10 – much too full), a 0-100% adherence scale (0% = ate none of the foods from
the recommended list; 100% = ate only food from the recommended food list), and a food frequency questionnaire (FFQ), which was a shortened and modified FFQ from the New Zealand Ministry of Health 2008/2009 adult nutrition survey (Ministry of Health and University of Otago 2011). It comprised nine questions, asking participants how many times in the previous week they consumed foods from the following categories: breads; pasta; root vegetables; rice; snacks, confectionary, takeaways, baked goods, and sugary drinks. These measures were used for self-monitoring purposes and as a talking point in participants’ individual meetings at the naval base conducted every four weeks.

**Intervention Components**

*Workshops.* Control group participants attended a workshop defining the low fat, high carbohydrate dietary approach. This way of eating was aligned with the existing food pyramid which endorses the predominant consumption of carbohydrate foods (i.e., whole grain products, fruit and vegetables) alongside the minimisation of fat (i.e., lean meats, low fat dairy products, use of small amounts of added fats for cooking, amongst other low fat food choices). Participants were not prescribed a specific diet with a macronutrient breakdown or calorie requirement. Instead, they were provided with a food guide (i.e., preferred foods and foods to avoid). It was anticipated that this style of eating reflected a macronutrient profile that aligned with the New Zealand Food & Nutrition Guidelines (Ministry of Health 2011) (i.e., Carbohydrate: 45-60% of total energy; Protein 15-25% of total energy; Fat 30-33% of total energy). From a weight loss perspective, participants received dietary advice based on the standard key weight loss principles of portion control and a reduction of energy-dense sources (i.e., foods or beverages high in fat and alcohol).

LCHF study participants attended a workshop designed to inform them on the LCHF dietary approach. This dietary approach focused on a moderate level of carbohydrate
restriction and was based on a whole food approach, discouraging consumption of processed food wherever possible. Again, participants were not prescribed a specific diet with a macronutrient breakdown or calorie requirement. It was anticipated from their food guide that this style of eating reflected a carbohydrate intake approximating less than the lower range of standard Ministry of Health recommendations i.e., <45% of total energy; a higher fat intake, approximating more than 33% of total energy, and a moderate protein intake, approximating 15-25% of total energy. Foods that were encouraged included vegetables (with a focus on non-starchy vegetables), fruit, meat and full fat dairy products, and foods high in fats such as nuts, avocado and healthy oils. Some of these food choices were encouraged to be used in limited quantities (fruit, legumes, starchy vegetables) due to their high carbohydrate load. Foods that were discouraged included foods containing refined sugar, junk food, cereals and grains.

Support Resources. All participants were provided with information and resources to support them through the 12-week trial. The control group was advised to access two websites which promote mainstream dietary guidelines, The Ministry of Health Food & Nutrition Guidelines (Ministry of Health 2011) and The Healthy Food Guide (Healthy Life Media Limited 2016). The intervention group was advised to access a specifically designed website to support participants in food choices and information on adhering to this way of eating. Figure 1 presents the participant flow throughout the duration of the study.

Insert Figure 1 here

Outcome variables
Weight was the primary outcome and was measured using a set of SECA 813 weighing scales. The remaining anthropometric measures, height and waist circumference (a secondary outcome) were measured using a SECA213 portable stadiometer and a Lufkin W606PM tape measure, respectively. Other secondary outcomes were lipid profiles (HDLc, LDL cholesterol (LDLc), triglycerides) and glycaemic control (serum glucose, serum insulin, HbA1c) and were determined by venepuncture technique using laboratory serum assays. HOMA-IR was used to depict insulin resistance and was calculated using the formula: (fasting insulin x fasting glucose) / 22.5, at baseline and at the completion of the intervention. At weeks 4 and 8 participants attended a weigh-in session and a meeting with a Registered Nutritionist to review their progress and receive additional dietary support.

Data analysis
Baseline group differences in categorical variables were described in percents and interpreted with the effect thresholds of <10%, <30%, <50%, and <70% corresponding to a modified Cohen’s scale of trivial, small, moderate, and large effects, respectively (Hopkins et al. 2009). Effect sizes of the group differences for all other variables at baseline were determined by dividing the mean group difference by the standard deviation of the pooled sample, and interpreted with the equivalent effect thresholds of <0.2, <0.6, <1.2, and <2.0.

Final data was analysed on study completers only, using the IBM statistical software package, SPSS (Version 23), and Microsoft Excel (2016) spreadsheets designed to analyse controlled trials using the t-statistic (Hopkins et al. 2009). All variables are presented in their raw values; however, analyses of their change scores have been performed with log transformation. For the variables weight and waist, a covariate of gender was included in the analysis. Means and standard deviations of pre- and post-scores for all outcome measures are presented; within-group change scores were calculated, analysed using the t-statistic and
interpreted using a statistical significance threshold of p<0.05. Between-group mean differences in variables are presented with 90% confidence limits. Effect sizes were determined by dividing the mean group difference by the standard deviation of the pooled sample. Negative and positive effect sizes can be interpreted as being beneficial and harmful, respectively, for the changes in the LCHF group compared with the control group. The only exception is for HDLc, where the direction of beneficial change is reversed. Using inferential statistics, qualitative and quantitative probabilities were used to assess the clinical significance of an effect as follows: a clinically clear beneficial effect was at least possibly beneficial (>25% chance) and almost certainly not harmful (<0.5% risk), and if the odds ratio for benefit to harm was >66; an unclear effect was at least possibly beneficial (>25%) with an unacceptable risk of harm (>0.5%); the effect was otherwise clearly trivial or harmful, depending on which outcome had the greater probability. The quantitative probabilities are not shown, but the qualitative terms were applied to each clear effect with its qualitative magnitude (e.g., likely small benefit). Weekly self-reported adherence and dietary FFQ data were presented as mean and standard deviations for the 12-week duration. For the FFQ data, the nine food categories were combined into a total weekly score for the number of times high carbohydrate-load food items were consumed. Scores were compared between groups, analysed using the t-statistic and interpreted using a statistical significance threshold of p<0.05.

Results
A total of 41 NZDF personnel were recruited into the study (21 in the LCHF group; 20 in the control group). Table 1 presents the baseline participant characteristics and blood marker variables for the two groups.
There were small differences between the group baseline characteristics for gender, ethnicity (NZ European only), HDLc, LDLc, all glycaemic control variables and weight and BMI (for males only); remaining variable differences were trivial. Both groups were considered to be obese according to the World Health Organization reference scale (World Health Organization 2016). Both groups showed blood marker parameters within the normal reference ranges for all markers (Labtests New Zealand 2016). Due to travel and professional commitments throughout the study period, 15 participants withdrew at various time points (i.e., 37% attrition rate, with roughly equal attrition from both groups). Data analysis was conducted on 26 completing participants (14 in the LCHF group; 12 in the control group).

Table 2 presents a summary of the pre- and post-intervention outcomes for each of the anthropometric and blood marker variables, and their respective mean values and standard deviations.

Within-group statistically significant differences are denoted by symbols, where appropriate. Between-group mean differences are presented along with the corresponding effect sizes and their qualitative clinical inference. Overall, both groups showed significant reductions in the anthropometric variables, weight and waist circumference, with a greater reduction noted in the LCHF group for both variables. For the control group, none of the remaining blood markers showed statistical significance in their tracking, while for the LCHF group, a significant reduction in triglycerides, triglyceride: HDLc ratio and glucose, and a significant increase in HDLc was observed. All between-group outcomes showed clear
effects, and ranged from small to moderate in favour of the LCHF group, apart from LDLc, which showed a small, likely harmful outcome.

Figure 2 shows the outcomes as individual responses, which help to illustrate the individual magnitude and direction of change both within and between groups. Individual responses to take note of are as follows: one LCHF participant showed a substantial increase in LDLc, and another showed a substantial decrease in fasting insulin, and subsequently, HOMA; one control group participant showed a large increase in HbA1c and another showed an increase in triglycerides, and subsequently, triglyceride: HDLc ratio.

Over the 12 weeks, the LCHF and control groups’ self-reported adherence (mean ± standard deviation) was 77.0 ± 19.9% and 59.1 ± 20.6%, respectively (p<0.05); FFQ dietary scores were 18.0 ± 7.4 and 22.7 ± 11.7, respectively.

Discussion

The key findings of this study are that relative to standard Ministry of Health dietary guidelines, the LCHF dietary approach was successful in achieving beneficial outcomes in all anthropometric and metabolic health measures apart from that of LDLc. To our knowledge, this is the first study using the LCHF dietary approach, conducted on military personnel; the findings corroborate with that of similar studies undertaken in other settings with overweight or obese individuals (Bazzano et al. 2014; Forsythe et al. 2008; Hu and Bazzano, 2014; Nordmann et al. 2006).

The mechanisms by which superior weight loss was achieved by the LCHF group could be attributed to several factors. Firstly, it is likely that water contributed to some of the weight lost by this group, as a result of the reduced glycogen content and associated water
storage that accompanies carbohydrate-restriction (Olsson and Saltin 1970). Secondly, despite the deliberate lack of calorie prescription for either group, a greater spontaneous calorie reduction likely occurred in the LCHF group than in the control group. Again, this is in line with available evidence suggesting that appetite seems to be better regulated with LCHF eating (Hu et al. 2016; McClernon et al. 2007). Another explanation for why LCHF eating might produce superior outcomes is due to the physiological and hormonal changes observed when undertaking an LCHF diet. Firstly, with carbohydrate-restriction, insulin becomes down-regulated as hepatic glucose output is reduced (Westman et al. 2007). With a down-regulation of insulin, and under energy-deficit conditions, body fat is able to be utilised readily as a fuel source, hence resulting in weight loss. A final possible mechanism for enhanced weight reduction over and above the control group is the possibility that eating LCHF resulted in spontaneous increased calorie expenditure. In a well-controlled, metabolic ward RCT, Ebbeling and colleagues (2012) identified an extra 300 Cal energy expenditure in overweight LCHF subjects compared with those on the low fat diet. The mechanism in this case resulting from altered macronutrient dietary composition was attributed to either metabolic efficiency (i.e., enhanced fat utilisation, and the ability of the body to easily switch between fat and carbohydrate fuel utilisation), an alteration in hormones, an effect of autonomic tone on skeletal muscle, or a possible improvement in leptin sensitivity, thereby causing enhanced skeletal muscle efficiency. In the present study, participants were instructed to continue with their existing exercise regime and advised not to make any changes. While this was the case, as documented in a qualitative manner at the final set of data collection measures, without this being measured quantitatively (and considered a study limitation) this contributory mechanism to weight loss remains speculative.

While we acknowledge that both styles of eating (LCHF and standard Ministry of Health dietary guidelines) can result in weight reduction, what appears to be echoed
throughout the literature is that the LCHF style of eating supersedes standard dietary guidelines in reducing triglycerides and raising HDLc. While several aspects of diet and lifestyle have the ability to impact these lipid markers, these outcomes are consistent with the supportive physiological evidence that dietary fat raises HDLc (Mensink and Katan 1992; Mensink et al. 2003) and that the consumption of dietary carbohydrates raises blood triglyceride levels (Parks and Hellerstein 2000). These outcomes are important because triglyceride: HDLc ratio is becoming increasingly well-recognised and utilised in a clinical setting as a useful predictor of insulin resistance and cardiovascular disease risk factors (McLaughlin et al. 2005). While we cannot say for certain as to whether these metabolic improvements have occurred independent of weight loss, evidence suggests that both lipid and glycaemic control markers have shown improvements in the absence of weight loss (Krauss et al. 2006; Gannon and Nuttal 2006; Mayer et al. 2014). Furthermore, greater improvements in triglyceride and HDLc have been shown in low carbohydrate diets compared with isocaloric low fat diets in the context of equal group weight loss, thereby suggesting an independent impact of macronutrient re-distribution. (Brinkworth et al. 2009).

In our study, we showed a non-significant upward and downward trend in LDLc in the LCHF and control groups, respectively, with eight participants in the LCHF group, compared to four participants in the control group showing an increase in LDLc. These trends, along with the small, likely harmful between-group effect were not unexpected as an increase in LDLc has been identified to accompany LCHF eating in some studies (Mansoor et al. 2016). There is current debate surrounding LDLc and its overall effect on disease risk. Briefly, it is the LDL sub-fractions and their varying effects on health, which is suggested to impart more meaning to the overall prediction of cardiovascular risk than LDLc itself (Ip et al. 2009; Ravnskov et al. 2016; Rizzo and Berneis 2006). Compared to the large, buoyant, LDL particles, it is the small, dense, LDL (sdLDL) particles, that are said to be atherogenic,
as they pass more easily into arterial walls where they undergo oxidation and cause inflammation and damage to the blood vessel wall. While these fractions are not yet routinely measured in research or clinical practice, evidence suggests that a reduced HDLc and a raised triglyceride lipid profile are predictors of the atherogenic LDLc phenotype, with the reverse (i.e., high HDLc and low triglycerides) being true for predicting large, buoyant LDL particles. (Austin et al. 1990; Ip et al. 2009; Krauss and Siri 2004; Toth 2007). In our sample, it could be that in this metabolic context, the benefits from a statistically significant improvement in a cluster of risk factors impacting chronic disease, may outweigh any non-significant impact from a raised LDLc. However, we state this with caution as without direct measurement of the LDLc particles themselves, there is still uncertainty regarding the true meaning of the risk associated with an elevated LDLc. Further research on LDLc is warranted in the context of the LCHF dietary approach, particularly in longer duration studies with larger sample sizes.

The small, likely beneficial between-group effects seen for insulin and for HOMA-IR need to be interpreted with caution. What appears a substantial reduction in these markers in the LCHF group may be accounted for by their large inherent variability, as noted by the large standard deviations and that considerable decreases were experienced by individuals whose baseline fasting insulin levels were above recommended reference levels (<174pmol/l). For example, one individual in the LCHF group experienced a reduction in insulin of 258 pmol/L, and another, 112 pmol/L. Due to the small sample size of completing participants, the presentation of our results as individual responses alongside mean values provides more clarity and meaning to their interpretation.

For the markers, HbA1c, one control group participant went from an HbA1c of 34 to 41.7mmol/l, which is above the normal reference threshold. Despite the moderate, likely beneficial between-group effect shown for HbA1c, all participants apart from the one just
described were within the normal reference range post-intervention (<41mmol/mol) (Labtests New Zealand 2016).

Aside from the limitations already mentioned, the study had two further limitations. Firstly, the study numbers were modest. Despite much interest in this trial, the realities of working in a defence force environment meant commitments, which included unexpected sea travel, took preference to completing the trial or being present at the final data collection point. While travel did not deter individuals from being part of a trial, we understand that all meals are provided in sea travel situations, leaving no control for participants to adhere to their respective study protocol, either the LCHF or the control groups. What is somewhat encouraging is that attrition was equal between groups.

Another limitation was that we did not obtain diet records to assess adherence. The FFQ data showed a statistically significant difference between consumption of high carbohydrate-load food items between the groups over the 12 weeks, however the true meaning of this finding is limited. While this tells us about the number of times participants consumed these foods, we are unable to report on the amount consumed. The self-reported measure of adherence showed a greater percentage score in the LCHF group compared with control. This could have had an impact on findings, however it was a subjective measure, and without more comprehensive diet records, this is merely speculative.

We conclude that the LCHF dietary approach showed promise in being able to reduce weight and improve metabolic health outcomes in this group of NZDF personnel over the short term. Longer term studies are required to assess adherence to both dietary approaches before judgement can be made on which should be considered “best practice” for health. From a practical translational standpoint, we have recommended that the LCHF dietary
approach be considered as an alternative (or at least an adjunct) therapy for the promotion of weight loss, and improved health outcomes in susceptible NZDF personnel.

Conflict of Interest Disclosure: Two of the authors, Dr Caryn Zinn and Professor Grant Schofield have co-authored two books called “What The Fat? - Fat’s in, Sugar’s out”, and “What The Fat – Sports performance”.

Funding Disclosure: This study was funded by a contestable research grant supplied by AUT. The funding body had no input into any aspect of the study.

References


doi:10.1017/S0007114515004699


doi:10.1038/oby.2007.516


Table 1: Baseline participant characteristics and blood marker variables for the two groups

<table>
<thead>
<tr>
<th></th>
<th>Control (n= 20)</th>
<th>LCHF (n=21)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Participant characteristics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>9 (45)</td>
<td>5 (24)</td>
</tr>
<tr>
<td>Male</td>
<td>11 (55)</td>
<td>16 (76)</td>
</tr>
<tr>
<td>Ethnicity, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NZ / Other European</td>
<td>15 (75)</td>
<td>11 (52)</td>
</tr>
<tr>
<td>Maori</td>
<td>3 (15)</td>
<td>8 (38)</td>
</tr>
<tr>
<td>PI</td>
<td>1 (5)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Indian</td>
<td>0 (0)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Euroasian</td>
<td>1 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Age, mean ± SD (y)</td>
<td>39.7 ± 9.6</td>
<td>39.6 ± 7.8</td>
</tr>
<tr>
<td><strong>Body composition and blood marker, mean ± standard deviation (SD)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>85.0 ± 13.4</td>
<td>85.6 ± 14.8</td>
</tr>
<tr>
<td>Male</td>
<td>95.1 ± 10.8</td>
<td>101.3 ± 15.1</td>
</tr>
<tr>
<td>BMI (kg.m(^2))</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>30.8 ± 3.8</td>
<td>31.2 ± 5.5</td>
</tr>
<tr>
<td>Male</td>
<td>30.5 ± 3.0</td>
<td>31.7 ± 4.6</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>92.2 ± 12.6</td>
<td>92.8 ± 7.2</td>
</tr>
<tr>
<td>Male</td>
<td>101.3 ± 8.5</td>
<td>101.1 ± 13.9</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/l)</td>
<td>1.2 ± 0.22</td>
<td>1.1 ± 0.2</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/l)</td>
<td>3.0 ± 0.7</td>
<td>2.8 ± 0.7</td>
</tr>
<tr>
<td>Triglyceride (mmol/l)</td>
<td>1.5 ± 1.1</td>
<td>1.4 ± 0.7</td>
</tr>
<tr>
<td>HbA1c (mmol/mol)</td>
<td>35.2 ± 2.2</td>
<td>36.0 ± 2.1</td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>5.1 ± 0.6</td>
<td>5.2 ± 0.5</td>
</tr>
<tr>
<td>Insulin (pmol/l)</td>
<td>64.4 ± 35.4</td>
<td>81.5 ± 76.9</td>
</tr>
</tbody>
</table>
Table 2: Pre- and post-intervention anthropometric and blood marker outcomes, with respective means and standard deviations.

<table>
<thead>
<tr>
<th></th>
<th>LCHF (n=14)</th>
<th>Control (n=12)</th>
<th>Between-group difference Mean (90% CI)</th>
<th>Between-group difference Cohen’s d (90% CI)</th>
<th>Effect size, qualitative clinical inference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Baseline</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Body Composition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>96.2 ± 13.3</td>
<td>90.7 ± 11.8†</td>
<td>94.8 ± 11.8</td>
<td>92.8 ± 12.2†</td>
<td>-3.6 (-5.8, -1.4)</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>98.0 ± 11.9</td>
<td>93.2 ± 10.3†</td>
<td>100.8 ± 10.5</td>
<td>97.5 ± 10.2†</td>
<td>-1.5 (-4.1, 1.1)</td>
</tr>
<tr>
<td><strong>Lipid profile</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDLc (mmol/l)</td>
<td>2.98 ± 0.70</td>
<td>3.24 ± 1.04†</td>
<td>3.12 ± 0.76</td>
<td>2.93 ± 0.65</td>
<td>0.5 (0.0, 0.9)</td>
</tr>
<tr>
<td>HDLc (mmol/l)</td>
<td>1.09 ± 0.26</td>
<td>1.31 ± 0.40†</td>
<td>1.16 ± 0.25</td>
<td>1.13 ± 0.30</td>
<td>0.2 (0.1, 0.4)</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.38 ± 0.55</td>
<td>1.01 ± 0.38†</td>
<td>1.59 ± 0.69</td>
<td>1.35 ± 0.62</td>
<td>-0.1 (-0.4, 0.2)</td>
</tr>
<tr>
<td>Triglyceride: HDLc ratio</td>
<td>1.38 ± 0.79</td>
<td>0.95 ± 0.64†</td>
<td>1.49 ± 0.82</td>
<td>1.33 ± 0.85</td>
<td>-0.4 (-0.8, 0.1)</td>
</tr>
<tr>
<td><strong>Glycaemic control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose (mmol/l)</td>
<td>5.19 ± 0.58</td>
<td>4.81 ± 0.28†</td>
<td>4.97 ± 0.42</td>
<td>4.86 ± 0.36</td>
<td>-0.3 (-0.6, 0.1)</td>
</tr>
<tr>
<td>Insulin (pmol/l)</td>
<td>83.3 ± 82.3</td>
<td>48.9 ± 20.0</td>
<td>64.8 ± 36.8</td>
<td>69.6 ± 31.0</td>
<td>-39.2 (-78.0, -0.5)</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>3.07 ± 3.64</td>
<td>1.50 ± 0.72</td>
<td>2.06 ± 1.12</td>
<td>2.18 ± 1.04</td>
<td>-1.8 (-3.7, 0.00)</td>
</tr>
<tr>
<td>HbA1c (mmol/mol)</td>
<td>35.7 ± 2.1</td>
<td>35.6 ± 2.1</td>
<td>35.2 ± 2.4</td>
<td>35.5 ± 3.4</td>
<td>-2.2 (-4.2, -0.2)</td>
</tr>
</tbody>
</table>

* SD Standard deviation
† significant within group difference from baseline to week 12
Figure 1. Participant flow through the study (F=females; M=males).

Figure 2. Individual responses for anthropometry and blood marker outcomes.

●  = males; ○ = females
Enrolment
Expression of interest (n=155)
Excluded (n=114)
- Not returned screening questionnaire (n=100)
- Not met inclusion criteria of BMI>25 (n=11)
- Declined to participate once confirmed (n=3)
Randomised (n=41)
Allocated to LCHF group (n=21)
Lost to follow-up (n=7: 1F; 6M)
Discontinued intervention
Analysed (n=14; 4F; 10M)
Excluded from analysis (n=0)
Allocated to Control group (n=20)
Lost to follow-up (n=8: 5F, 3M)
Discontinued intervention
Analysed (n=12; 4F; 8M)
Excluded from analysis (n=0)