A Very Low Carbohydrate, Low Saturated Fat Diet for Type 2 Diabetes Management: A Randomized Trial

DOI: 10.2337/dc14-0845

OBJECTIVE
To comprehensively compare the effects of a very low carbohydrate, high unsaturated/low saturated fat diet (LC) to a high-unrefined carbohydrate, low fat diet (HC) on glycemic control and cardiovascular disease (CVD) risk factors in type 2 diabetes (T2DM).

RESEARCH DESIGN AND METHODS
Obese adults (n = 115, BMI 34.4 ± 4.2 kg/m², age 58 ± 7 years) with T2DM were randomized to a hypocaloric LC diet (14% carbohydrate [<50 g/day], 28% protein, and 58% fat [<10% saturated fat]) or an energy-matched HC diet (53% carbohydrate, 17% protein, and 30% fat [<10% saturated fat]) combined with structured exercise for 24 weeks. The outcomes measured were as follows: glycosylated hemoglobin (HbA1c), glycemic variability (GV; assessed by 48-h continuous glucose monitoring), antiglycemic medication changes (antiglycemic medication effects score [MES]), and blood lipids and pressure.

RESULTS
A total of 93 participants completed 24 weeks. Both groups achieved similar completion rates (LC 79%, HC 82%) and weight loss (LC 212.0 ± 6.3 kg, HC 211.5 ± 5.5 kg; P > 0.50). Blood pressure (9.8/7.3 ± 11.6/6.8 mmHg), fasting blood glucose (1.4 ± 2.3 mmol/L), and LDL cholesterol (0.3 ± 0.6 mmol/L) decreased, with no diet effect (P > 0.10). LC achieved greater reductions in triglycerides (0.5 ± 0.5 vs. 0.1 ± 0.5 mmol/L), MES (0.5 ± 0.5 vs. 0.2 ± 0.5), and GV indices; P ≤ 0.03. LC induced greater HbA1c reductions (2.6 ± 1.0% [−28.4 ± 10.9 mmol/mol] vs. 1.9 ± 1.2% [−20.8 ± 13.1 mmol/mol]; P = 0.002) and HDL cholesterol (HDL-C) increases (0.2 ± 0.3 vs. 0.05 ± 0.2 mmol/L; P = 0.007) in participants with the respective baseline values HbA1c >7.8% (62 mmol/mol) and HDL-C <1.29 mmol/L.

CONCLUSIONS
Both diets achieved substantial improvements for several clinical glycemic control and CVD risk markers. These improvements and reductions in GV and antiglycemic medication requirements were greatest with the LC compared with HC. This suggests an LC diet with low saturated fat may be an effective dietary approach for T2DM management if effects are sustained beyond 24 weeks.

1Preventative Health National Research Flagship, Commonwealth Scientific and Industrial Research Organisation (CSIRO), Animal, Food and Health Sciences, Adelaide, Australia
2Discipline of Medicine, University of Adelaide, Adelaide, Australia
3Agency for Science, Technology and Research (A*STAR), Singapore
4Nutritional Physiology Research Centre, Sansom Institute for Health Research, University of South Australia, Adelaide, Australia
5Division of General Internal Medicine, Department of Medicine, Duke University Medical Center, Durham, NC
6Center for Health Services Research in Primary Care, Veterans Affairs Medical Center, Durham, NC

Corresponding author: Grant D. Brinkworth, grant.brinkworth@csiro.au.
Received 4 April 2014 and accepted 2 July 2014.
This article contains Supplementary Data online at http://care.diabetesjournals.org/lookup/suppl/doi:10.2337/dc14-0845/-/DC1.
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An energy-reduced, high carbohydrate, low protein, low fat (HC) diet is the traditional dietary approach for type 2 diabetes (T2DM) management (1). However, evidence shows dietary carbohydrate elicits greater postprandial glucose (PPG) responses compared with fat or protein, which independently suppresses this response (2–4). This has increased interest and the use of very low carbohydrate diets (LC; 20–70 g carbohydrates/day) that are also high in protein and fat for diabetes management (5).

Previous studies in T2DM show, compared with an HC diet, an LC diet achieves at least comparable reductions in body weight, blood pressure, and in insulin concentrations (6–8), with greater improvements in glycemic control (6,8–10). However, these studies are limited by poor dietary compliance and the absence and/or control of physical activity, an integral component of lifestyle modification for weight and diabetes management (11). Energy intake and weight loss differences between comparison diets secondary to their ad libitum designs, particularly for the LC diet, may also confound the metabolic outcomes reported. Prior studies also limit glycemic control assessment to glycosylated hemoglobin (HbA1c) and fasting glucose (6–8,10). However, glycemic variability (GV amplitude, frequency, and duration of diurnal glucose fluctuations) and PPG excursions are also considered independent risk factors for diabetes complications, including cardiovascular disease (CVD) risk (12,13), yet no study has systematically evaluated the effects of LC diets on these outcomes. These limitations preclude clear conclusions, highlighting the necessity for well-controlled studies that comprehensively examine effects of LC diets on glycemic control in T2DM.

Previous studies also show that compared with an HC diet, whereas an LC diet favorably lowers triglycerides (TGs) and elevates HDL cholesterol (HDL-C), greater increases in LDL cholesterol (LDL-C), a primary therapeutic target and CVD risk marker (14), are observed (6,8,15–17). LC diets used in previous studies, in addition to increasing total fat intake, concomitantly increased saturated fat intake, which elevates LDL-C (18). Furthermore, a prospective cohort study suggests a vegetable-based LC diet is associated with lower all-cause and CVD mortality risk (19). These data suggest the health effects of LC diets may be influenced by fat quality, and an LC diet with high unsaturated and low saturated fat content may promote greater improvements in glycemic control in T2DM without detrimental effects on LDL-C. However, this hypothesis and the combined effects of these dietary components have not been tested in a well-controlled intervention trial. This study compared the effects of a hypercaloric LC, high unsaturated/low saturated fat diet to an energy-matched HC diet, as part of a holistic lifestyle modification program on glycemic control, including GV and CVD risk factors in T2DM.

**RESEARCH DESIGN AND METHODS**

**Study Population** Overweight/obese adults (n = 115, BMI 26–45 kg/m², age 35–68 years) with T2DM (previously diagnosed with HbA1c ≥ 7.0% [53 mmol/mol] and/or taking antiglycemic medication), recruited via public advertisement, participated in this single-center, randomized, controlled study, conducted between May 2012 and February 2013 at the Commonwealth Scientific Industrial Research Organization (CSIRO) Clinical Research Unit in Adelaide, Australia (Fig. 1). Exclusion criteria were type 1 diabetes; proteinuria (urinary albumin-to-creatinine ratio ≥ 30 mg/mmol); impaired renal function (eGFR < 60 mL/min); abnormal liver function (alanine aminotransferase [ALT], aspartate aminotransferase [AST], or γ-glutamyl transferase [GGT] ≥ 2.5 times the normal upper limit) assessed at screening; any significant endocrinopathy (other than stable treated thyroid disease); history of malignancy (other than nonmelanoma); liver, respiratory, gastrointestinal, or cardiovascular disease; pregnancy or lactation; clinical depression; history of or current eating disorder; or smoking. Participants provided written, informed consent to the study protocol approved by the CSIRO Human Ethics Committee.

**Study Design and Intervention** In a parallel design, participants were block matched for age, sex, BMI, HbA1c, and antiglycemic medication using random varying block sizes before random computer-generated assignment to either an LC or HC diet in a 1:1 ratio. Randomization procedures (sequence generation and allocation concealment) were performed by research associates independent of outcome assessments and intervention delivery. Planned macronutrient profiles of the diet interventions were as follows: LC diet, 14% of total energy as carbohydrate (objective to restrict intake to < 50 g/day), 28% protein, and 58% total fat (35% monounsaturated fat and 13% polyunsaturated fat); HC diet, 53% carbohydrate with emphasis on low glycemic index foods, 17% protein, and < 30% total fat (15% monounsaturated fat and 9% polyunsaturated fat). Saturated fat was limited to < 10% in both diets. Planned nutrient composition of the HC diet comparison group was based on conventional recommendations of current guidelines (1). Diet plans were individualized and matched for energy levels with moderate restriction (500–1,000 kcal/day) (20). Diets were structured to include specific foods (Table 1), listed in a quantitative food record that participants completed daily. To facilitate compliance, participants met individually with a dietitian biweekly for 12 weeks and monthly thereafter. Dietitians provided dietary advice and instruction on the eating plan and reporting requirements. Participants were supplied key foods (− 30% total energy) representative of their allocated diet profile for 12 weeks and key foods or AU $50 food voucher on alternate months thereafter.

Under supervision of exercise professionals, participants undertook, free of charge, 60-min structured exercise classes on 3 nonconsecutive days per week, incorporating moderate intensity aerobic/resistance exercises, consistent with diabetes management guidelines (11). Attendance records were kept and participants were encouraged to make up any missed sessions. Apart from the planned exercise program, participants were instructed to maintain habitual physical activity levels.

**Outcomes** Primary outcome was HbA1c (IMVS, Adelaide, Australia). Secondary outcomes included GV, antiglycemic medication changes, and blood lipids and pressure. Outcomes were assessed at weeks 0 and 24. Although diet assignment was discernible by participants and interventionists, blinding was maintained for outcome assessment and data analysis.
Anthropometric Measurements and Blood Pressure

Height was measured using a stadiometer. Body mass was measured using calibrated electronic scales (Mercury AMZ1, Tokyo, Japan) and waist circumference by tape measure positioned 3 cm above the iliac crest. Body composition was determined by whole-body DEXA (Lunar Prodigy; General Electric Corporation, Madison, WI) to assess total fat (FM) and fat-free mass (FFM). Seated blood pressure was measured by automated sphygmomanometry (SureSigns VS3; Philips, Andover, MA).

Figure 1—Participant flow.
Very Low Carbohydrate Diet for T2DM Management

Diabetes Care

Blood glucose values during successive 24-h periods (MODD, difference between paired daily blood glucose differences) and mean of glucose readings between successive 24-h periods (SD interday); and mean of glucose range, MAGE, CONGA, and MODD were computed by automated algorithm (25). Percentage of total time spent in the hypoglycemic (<3.9 mmol/L), euglycemic (3.9–10 mmol/L), or hyperglycemic range (>10.0 mmol/L), defined by American Diabetes Association glycemic control targets (27), was calculated.

**Medication Changes**

Medications at baseline and changes throughout the study were documented. Medication effects score (MES) (10) based on potency and dosage of antiglycemic agents and insulin usage was used to quantify antiglycemic medication levels. Higher MES corresponds to higher antiglycemic medication usage.

**Dietary Intake and Adherence**

Dietary intake and adherence was assessed from 7 consecutive days (including 2 weekend days) of daily weighed food records for every 14-day period. These data were analyzed using Foodworks Professional Edition Version 7 (Xyris Software 2012, Highgate Hill, Australia) to calculate the average nutrient intake over the entire 24 weeks. Urine samples (24 h) were collected to assess urea-to-creatinine ratio (IMVS), as an objective marker of protein intake (28). Plasma β-hydroxybutyrate levels were assessed monthly as a marker of reduced carbohydrate intake (RANBUT D-3 Hydroxybutyrate kit; Randox, Antrim, U.K.).

**Physical Activity**

Physical activity levels were assessed with 7 consecutive days of triaxial accelerometer (GT3×+model; ActiGraph, Pensacola, FL), using previously defined validity cutoffs (29).

**Statistical Analysis**

Data were examined for normality; non-normally distributed variables (HbA1c, glucose range, MAGE, CONGA-1, CRP, HOMA2-%B, and β-hydroxybutyrate) were logarithmically transformed. Baseline characteristics, dietary data, and exercise session attendance between groups were assessed by independent Student t tests and χ² tests for continuous and categorical variables, respectively. This study used a randomized groups, pretest-posttest design, and data were analyzed using ANCOVA to test between-group differences at posttest assessments (week 24), with baseline and sex as covariates. ANCOVA confers greater statistical power, correcting for regression to the mean (30). Comparisons of regression slopes (test of the interaction between the pretest data and the grouping variable) were conducted to determine whether the ANCOVA assumption of homogeneity of regression slopes was met. For variables that did not meet this assumption (HDL, HbA1c, AUC total min, and mean and maximum glucose), the Johnson-Neyman (J-N) procedure (31) was appropriately used to identify regions of significance along the observed range of the pretest measure that indicated where group (diet) differences on the posttest measures occurred (i.e., where the diet groups differed). For these variables, group means above and below

### Table 1—Food profile of diet interventions

<table>
<thead>
<tr>
<th>LC diet, 1,429 kcal</th>
<th>HC diet, 1,429 kcal</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 30 g high fiber, low Gl cereal*</td>
<td>• 40 g high fiber, low Gl cereal*</td>
</tr>
<tr>
<td>• 1 crispbread (e.g., Ryvita)*</td>
<td>• 5 crispbread (e.g., Ryvita)*</td>
</tr>
<tr>
<td>• 250 g lean chicken, pork, fish, red meat (3-4 times/week)</td>
<td>• 1/2 cup cooked pasta/rice/potato*</td>
</tr>
<tr>
<td>• 40 g almonds and 20 g pecans*</td>
<td>• 2 slices wholegrain bread (70 g)</td>
</tr>
<tr>
<td>• 3 cups low starchy vegetables (exclude potato/sweet potato/corn)</td>
<td>• 80 g lean chicken, pork, red meat (4 times/week)*</td>
</tr>
<tr>
<td>• 200 mL skim (&lt;1% fat) milk</td>
<td>• 80 g fish (2 times/week)*</td>
</tr>
<tr>
<td>• 100 g diet yogurt</td>
<td>• 80 g legumes (1 time/week)*</td>
</tr>
<tr>
<td>• 20 g (1 slice) regular cheese</td>
<td>• 3 cups vegetables</td>
</tr>
<tr>
<td>• 30 g (6 tsp) margarine/oil of monounsaturated variety (e.g., canola oil/margarine)</td>
<td>• 250 mL reduced (1–2%) fat milk</td>
</tr>
<tr>
<td>• 200 mL diet fat milk</td>
<td>• 150 g reduced fat yogurt</td>
</tr>
<tr>
<td>• 2 slices wholegrain bread (70 g)</td>
<td>• 20 g (1 slice) regular cheese</td>
</tr>
<tr>
<td>• 250 mL reduced (1–2%) fat milk</td>
<td>• 25 g (5 tsp) margarine/oil of monounsaturated variety (e.g., canola oil/margarine)</td>
</tr>
</tbody>
</table>

Gl, glycemic index. *Key foods supplied, representing ~30% of total energy intake.
the identified critical points on the pre-
test measures are presented. Percentage
of total time spent in the hypo-, hyper-, or
euglycemic range was analyzed by β-regression using mean and precision
parameterization, which is efficient for
characterizing percentages (SAS software,
version 9.2; SAS Institute Inc., Cary, NC)
(32). Repeated-measures ANOVA with
diet and sex set as between-subject factors
and time as a within-subject factor was used
to assess changes in β-hydroxybutyrate
between groups. No sex effects were
observed for any outcome. The trial
was designed to have 80% power to
detect a 0.7% (7.7 mmol/mol) absolute
difference in HbA1c (primary outcome)
between the diets that has been previ-
ously reported (6,8,10) and considered
clinically significant (33). Data are pre-
sented as means ± SD, unless otherwise
stated. Statistical tests were two tailed
with statistical significance at P < 0.05
and performed using SPSS 20.0 for Win-
dows (SPSS Inc., Chicago, IL) unless other-
wise stated.

RESULTS
Participants
A total of 115 participants commenced
the study. Baseline characteristics were
similar between groups (mean ± SD; LC
and HC): age 58 ± 7 and 58 ± 7 years,
weight 101.7 ± 14.4 and 101.6 ± 15.8
kg, and BMI 34.2 ± 4.5 and 35.1 ± 4.1
kg/m²; sex distribution (males/females)
37/21 and 29/28; HbA1c 7.3 ± 1.1 and
7.4 ± 1.1% (56 ± 12 and 57 ± 12
mmol/mol) (Supplementary Table 1).
A total of 16 participants withdrew prior
to commencement and diet assignment
disclosure (Fig. 1). A total of 93 (81%)
retention) participants completed the
study and were included in the primary
analysis (Table 2). Attrition rates were
comparable between diets (P = 0.50),
with no difference in baseline character-
istics between participants who com-
pleted/withdrew (P ≥ 0.25).

Diet and Physical Activity Compliance
Reported dietary intakes were consis-
tent with diet prescriptions (Supple-
mental Table 2). Energy intake did not
differ between groups (LC 1,563 ±
225 kcal, HC 1,587 ± 171 kcal; P = 0.56).
Relative to the HC diet group, the LC diet
group consumed less carbohydrate
(LC 56.7 ± 8.0 vs. HC 204.9 ± 22.8 g;
14 ± 2 vs. 50 ± 2% total energy) and
dietary fiber (24.7 ± 3.5 vs. 31.1 ± 3.2 g),
more protein (102.8 ± 14.7 vs. 73.6 ± 8.3 g; 27 ± 1 vs. 19 ± 1% total energy),
total fat (96.5 ± 16.5 vs. 44.3 ± 7.4 g;
54 ± 3 vs. 25 ± 3% total energy), satu-
rated fat (10.0 ± 0.9 vs. 7.5 ± 1.1% total
energy), monounsaturated fat (30.4 ±
1.8 vs. 11.5 ± 1.3% total energy), poly-
unsaturated fat (12.2 ± 1.1 vs. 4.1 ±
0.6% total energy), and cholesterol
(243 ± 42 vs. 138 ± 25 mg); P < 0.001
for all. Plasma β-hydroxybutyrate con-
centrations showed a time by diet inter-
action (P < 0.001); levels increased
threefold more on the LC compared
with the HC diet after the initial 4 weeks
and remained higher throughout the
study, indicating a relatively lower car-
bohydrate intake. There was a significant
diet effect for urinary urea-to-creatinine
excretion ratio (P < 0.001), which de-
creased with the HC diet (−2.2 ± 6.2)
and increased with the LC diet (4.2 ±
8.7), indicating a higher protein intake
in the LC diet group.

Exercise session attendance was sim-
ilar between groups (LC 76.7
± 51 and 78.5
± 41; P = 0.51) (Table 2).
Body Weight, Composition, and CVD
Risk Markers
At week 24, body weight, BMI, waist
circumference, FM, FFM, FM-to-FFM,
blood pressure, insulin, HOMA2-IR,
HOMA2-%B, total cholesterol, LDL-C,
and CRP were similar between groups
(P > 0.10) (Table 2). Diet composition
significantly affected TG (P = 0.001) with
fivefold greater reductions with the LC
diet. For HDL-C, due to the heteroge-

enity of regression slopes, indicating the
diet effects depended on baseline levels
for this parameter (significant group ×
baseline interaction), the J-N method
was used to explore the intervention ef-
tect to identify the range on the baseline
measure where differences between
groups were statistically significant.
This revealed that for the range of avail-
able baseline values, greater increases
in HDL-C occurred with the LC diet (P =
0.007) for participants with a baseline
HDL-C < 1.3 mmol/L, with no difference
between groups for participants with
baseline HDL-C ≥1.3 mmol/L.

Glycemic Control and Variability
Due to the significant interaction of
group and baseline HbA1c (P = 0.02), in-
dicating the diet effects depended on
initial HbA1c levels, the J-N method
was used to explore the intervention ef-
fect on HbA1c and identify the range of
the baseline measure where differences
between groups were statistically signifi-
cant (Fig. 2). The result showed the LC diet
reduced HbA1c to a greater extent among
participants with baseline HbA1c > 7.8%
(62 mmol/mol), with no diet effect in
participants with baseline HbA1c ≤ 7.8%.
Percentage weight loss was not different
between the groups for participants with
baseline HbA1c > 7.8% (LC −11.9 ± 5.6%,
HC −11.2 ± 5.4%; P = 0.77).

No significant diet effect on fasting
blood glucose, minimum blood glucose,
and glucose SDinterdays occurred (P ≥
0.06). Compared with HC, the LC diet
gave greater reductions in blood glucose
range, SDintraday, MAGE, CONGA-1,
CONGA-4, and MODD (P ≤ 0.049). Due
to heterogeneity of regression slopes
indicated by the significant interaction of
group and baseline mean blood glu-
cose, maximum blood glucose, and
blood glucose AUCtotal per min (P ≤
0.04), the J-N method was used to ex-
amine the intervention effect in these
parameters. This showed the LC diet
produced greater reductions (P ≤
0.04) among participants with a baseline
mean glucose > 8.6 mmol/L, maximum
blood glucose > 13.2 mmol/L, and
AUCtotal per min > 18.0 mmol/L, for these
parameters respectively (Table 2).

β regression analyses demonstrated
that participants on the LC diet were
85% more likely and 56% less likely to
spend higher proportions of time in the
euglycemic and hyperglycemic ranges,
respectively, compared with their HC
diet counterparts (P ≤ 0.03). The LC
diet group was also 16% less compared
with HC diet group to spend more time
in the hypoglycemic range, but the re-
sidual plots suggested model mis-
fitted (P = 0.42).

Medication Changes
At baseline, medication usage and the
antiglycemic MES were similar in both
groups (P ≥ 0.29 for all) (Supplementary
Table 1). After 24 weeks, the LC diet
group experienced twofold greater reduc-
tions in the antiglycemic MES, with more
participants experiencing a reduction
### Table 2—Body weight and composition, glycemic control and cardiovascular risk markers after 24 weeks on a LC diet or an energy matched HC diet

<table>
<thead>
<tr>
<th></th>
<th>LC diet (n = 46)</th>
<th>Change</th>
<th>HC diet (n = 47)</th>
<th>Change</th>
<th>P value†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Body weight and composition</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>88.1 (13.7)</td>
<td>−12.0 (6.3)</td>
<td>89.9 (14.9)</td>
<td>−11.5 (5.5)</td>
<td>0.57</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>30.0 (4.4)</td>
<td>−4.0 (2.0)</td>
<td>30.9 (4.2)</td>
<td>−4.0 (1.8)</td>
<td>0.74</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>100.5 (10.9)</td>
<td>−10.6 (7.1)</td>
<td>103.2 (11.9)</td>
<td>−9.1 (6.4)</td>
<td>0.25</td>
</tr>
<tr>
<td>Total FFM (kg)</td>
<td>58.8 (10.0)</td>
<td>−1.7 (2.0)</td>
<td>57.7 (10.6)</td>
<td>−1.9 (1.7)</td>
<td>0.66</td>
</tr>
<tr>
<td>Total FM (kg)</td>
<td>29.1 (11.8)</td>
<td>−10.2 (5.7)</td>
<td>32.2 (11.3)</td>
<td>−9.6 (5.2)</td>
<td>0.64</td>
</tr>
<tr>
<td>FM-to-FFM ratio (kg/kg)</td>
<td>0.5 (0.2)</td>
<td>−0.2 (0.1)</td>
<td>0.6 (0.2)</td>
<td>−0.1 (0.1)</td>
<td>0.76</td>
</tr>
<tr>
<td><strong>Glycemic control</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting glucose (mmol/L)</td>
<td>6.8 (1.5)</td>
<td>−1.1 (2.2)</td>
<td>6.7 (1.6)</td>
<td>−1.6 (2.5)</td>
<td>0.67</td>
</tr>
<tr>
<td>Mean glucose (mmol/L)§</td>
<td>6.9 (1.2)</td>
<td>−3.4 (2.2)</td>
<td>7.6 (1.8)</td>
<td>−2.5 (1.6)</td>
<td>0.01‡‡</td>
</tr>
<tr>
<td>Baseline &gt;8.6</td>
<td>9.1 (3.7)</td>
<td>−1.4 (2.3)</td>
<td>9.3 (1.8)</td>
<td>−2.1 (2.1)</td>
<td></td>
</tr>
<tr>
<td>Glucose range (mmol/L)§</td>
<td>5.5 (2.0)</td>
<td>−3.6 (3.1)</td>
<td>7.1 (3.5)</td>
<td>−2.5 (3.8)</td>
<td>0.049</td>
</tr>
<tr>
<td>SDinterday (mmol/L)§</td>
<td>1.1 (0.5)</td>
<td>−0.9 (0.7)</td>
<td>1.5 (0.7)</td>
<td>−0.6 (0.8)</td>
<td>0.004</td>
</tr>
<tr>
<td>MAGE (mmol/L)</td>
<td>2.9 (1.4)</td>
<td>−2.3 (0.0)</td>
<td>3.9 (2.1)</td>
<td>−1.4 (2.3)</td>
<td>0.03</td>
</tr>
<tr>
<td>CONGA-1 (mmol/L)§</td>
<td>1.0 (0.4)</td>
<td>−0.6 (0.5)</td>
<td>1.4 (0.6)</td>
<td>−0.3 (0.6)</td>
<td>0.002</td>
</tr>
<tr>
<td>CONGA-4 (mmol/L)§</td>
<td>1.6 (0.8)</td>
<td>−1.4 (1.1)</td>
<td>2.1 (1.1)</td>
<td>−0.8 (1.2)</td>
<td>0.005</td>
</tr>
<tr>
<td>MODD (mmol/L)</td>
<td>1.1 (0.5)</td>
<td>−0.8 (0.7)</td>
<td>1.5 (0.7)</td>
<td>−0.5 (0.9)</td>
<td>0.002</td>
</tr>
<tr>
<td>AUC_{total per min} (mmol/L)§</td>
<td>13.3 (2.7)</td>
<td>−7.9 (5.0)</td>
<td>15.4 (4.0)</td>
<td>−5.4 (3.7)</td>
<td>0.005‡‡</td>
</tr>
<tr>
<td><strong>CVD risk markers</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>120.1 (11.4)</td>
<td>−11.0 (10.6)</td>
<td>122.9 (14.2)</td>
<td>−8.7 (12.5)</td>
<td>0.26</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>72.4 (6.3)</td>
<td>−8.2 (5.6)</td>
<td>74.3 (7.5)</td>
<td>−6.4 (7.8)</td>
<td>0.10</td>
</tr>
<tr>
<td>Insulin (mU/L)§</td>
<td>8.7 (4.7)</td>
<td>−7.7 (6.2)</td>
<td>9.5 (4.7)</td>
<td>−6.5 (5.7)</td>
<td>0.22</td>
</tr>
<tr>
<td>HOMA2-IR</td>
<td>1.2 (0.6)</td>
<td>−1.1 (0.9)</td>
<td>1.3 (0.7)</td>
<td>−1.0 (0.8)</td>
<td>0.23</td>
</tr>
<tr>
<td>HOMA2-90B§</td>
<td>62.3 (30.8)</td>
<td>−8.8 (19.9)</td>
<td>64.2 (25.1)</td>
<td>−4.7 (22.9)</td>
<td>0.12</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.0 (0.9)</td>
<td>−0.3 (0.70)</td>
<td>4.0 (0.9)</td>
<td>−0.3 (0.9)</td>
<td>0.89</td>
</tr>
<tr>
<td>LDL-C (mmol/L)</td>
<td>2.1 (0.8)</td>
<td>−0.3 (0.5)</td>
<td>2.1 (0.8)</td>
<td>−0.3 (0.7)</td>
<td>0.81</td>
</tr>
<tr>
<td>HDL-C (mmol/L)</td>
<td>1.3 (0.2)</td>
<td>0.2 (0.3)</td>
<td>1.1 (0.2)</td>
<td>0.05 (0.2)</td>
<td>0.007‡‡</td>
</tr>
<tr>
<td>Baseline &lt;1.3</td>
<td>1.5 (0.2)</td>
<td>0.3 (0.2)</td>
<td>1.6 (0.2)</td>
<td>−0.06 (0.2)</td>
<td></td>
</tr>
<tr>
<td>CRP (mg/L)#</td>
<td>1.1 (0.5)</td>
<td>−0.5 (0.5)</td>
<td>1.3 (0.5)</td>
<td>−0.1 (0.5)</td>
<td>0.001</td>
</tr>
<tr>
<td>Mean activity count (counts/min)</td>
<td>232.7 (88.5)</td>
<td>44.3 (57.9)</td>
<td>232.5 (78.2)</td>
<td>51.7 (45.3)</td>
<td>0.51</td>
</tr>
<tr>
<td>MEPVA (min/day)</td>
<td>58.0 (25.9)</td>
<td>11.8 (17.0)</td>
<td>55.7 (21.6)</td>
<td>12.6 (13.0)</td>
<td>0.83</td>
</tr>
<tr>
<td>MEPVA (% of total wear time)</td>
<td>4.3 (1.9)</td>
<td>0.8 (1.2)</td>
<td>4.1 (1.6)</td>
<td>0.9 (1.0)</td>
<td>0.81</td>
</tr>
</tbody>
</table>

Data are means (SD), unless otherwise stated. DBP, diastolic blood pressure; MEPVA, moderate to vigorous intensity physical activity; SBP, systolic blood pressure. To convert mmol/L to mg/dL, multiply by 18 (for glucose), 38.7 (for cholesterol), and 88.6 (for TGs). †Total analyzed n = 93 (LC 46 and HC 47) for all data unless otherwise stated. ‡P value refers to between-group differences over time (diet effect) by ANCOVA and J-N procedure where appropriate. §Total analyzed n = 92 (LC 45 and HC 47) for body composition data; DEXA scan was not performed at baseline for one participant in LC diet group. §§Total analyzed n = 91 (LC 46 and HC 45) for CGM data; CGM device did not collect valid data for two participants in the HC diet group at 24 weeks due to poor system connectivity. ¶Total analyzed n = 83 (LC 42 and HC 41) that meet requirement of 48-h valid CGM data collection to calculate comparisons between 2 successive days. #Total analyzed n = 84 (LC 43 and HC 41) for CRP data; nine participants with CRP >10 mg/L were excluded from these analyses. **Total analyzed n = 91 (LC 45 and HC 46); two participants with accelerometer data that did not meet the validity criteria were excluded. ***Significant group × baseline interaction, with significant group effect for baseline mean glucose >8.6 mmol/L (LC 18 and HC 22). §§Significant group × baseline interaction, with significant group effect for baseline maximum glucose >13.2 mmol/L (LC 26 and HC 28). §§§Significant group × baseline interaction, with significant group effect for baseline HDL-C <1.3 mmol/L (LC 33 and HC 28).
pros in glycemic control, blood glucose the LC diet induced greater improve-
ment of >20% compared with HC diet group (P < 0.005) (Table 2). Six participants reduced (LC 4 and HC 2) and five increased (LC 3 and HC 2) lipid-lowering medication. Eleven participants reduced (LC 10 and HC 1) and six increased (LC 3 and HC 3) antihypertensive medication.

Adverse Events
Eleven participants (LC 5 and HC 6) reported musculoskeletal ailments with exercise training that allowed program continuation following recovery. Two LC diet participants reported gastrointestinal disorders (constipation and diverticulitis); one HC diet participant reported esophageal ulcers with Helicobacter pylori infection; one LC diet participant was diagnosed with prostate cancer; three HC participants had elective surgical procedures performed; four participants (LC 3 and HC 1) experienced non-study-related workplace injuries; one HC diet participant had a motor vehicle accident.

CONCLUSIONS
This study demonstrates that both energy-reduced LC and HC diets with low saturated fat content produce substantial improvements in glycemic control and several cardiometabolic risk markers in obese adults with T2DM. However, the LC diet induced greater improvements in glycemic control, blood glucose profiles, and reductions in diabetes medication requirements compared with the HC diet. The LC diet also promoted a more favorable CVD risk profile by elevating HDL-C and reducing TG levels, with comparable reductions in LDL-C compared with the HC diet. These effects were most evident in participants with greater metabolic derangements, suggesting that an LC diet with high unsaturated/low saturated fat content can improve primary clinical diabetes management targets beyond conventional lifestyle management strategies and weight loss.

One study strength was the energy-matched prescription of diets that achieved comparable weight loss between groups, which removed this potential confounder and enabled metabolic differences between groups to be attributed to differences in the macronutrient profiles. Both groups achieved substantial reductions in HbA1c, although importantly, a further greater reduction of 0.7% (7.7 mmol/mol) (absolute) occurred with the LC diet. This effect size is consistent with previous very low carbohydrate ad libitum studies (6,8,10) and is comparable to those associated with antiglycemic agents (34). A 1% (10.9 mmol/mol) HbA1c reduction is estimated to reduce the risk of diabetes-related death by 21%, myocardial infarction by 14%, and microvascular complications by 37% (33). Therefore, the additional 0.7%ug HbA1c reduction achieved by the LC diet could translate to significant further reductions in diabetes complications risk.

In contrast to previous studies (6,8,10), a diet by baseline score interaction was present for HbA1c indicating that diet effects were dependent on initial levels and that the greater HbA1c reductions with the LC diet were only evident in participants with a baseline HbA1c >7.8% (62 mmol/mol). This difference between studies could be attributed to differences in the statistical approaches used. The current study used ANCOVA combined with the J-N procedure, enabling effects of the covariate (baseline values) on the posttest outcomes to be revealed and regions of significance for any diet (group) differences to be determined. It is therefore possible that the relatively lower mean baseline HbA1c levels of participants in the present compared with previous studies (7.3 vs. 7.4–8.8%; 56 vs. 57–73 mmol/mol) (6,8,10) may have facilitated this response. This suggests greater HbA1c-lowering effects of an LC diet are most evident in those with higher baseline levels. However, given the relatively small subgroup of participants, this result should be interpreted with some caution.

Importantly, the LC diet group also experienced twofold greater reductions in antiglycemic MES, an effect that occurred across the entire study sample. It is therefore possible that the greater reductions in diabetes medication usage with the LC diet tempered the magnitude of HbA1c reductions observed in
of the intervention delivered with high levels of professional support and subsidized food provisions that facilitated high compliance were strengths of this study to deliver its purpose of establishing the efficacy of the diets evaluated. Moreover, inclusion of a closely monitored and professionally supervised physical activity program may have also contributed significantly to the successful weight and cardiometabolic improvements observed in both groups. It is possible this delivery approach may potentially limit success for widescale community adoption. Future initiatives need to integrate these lifestyle program components within cost-effective community-based delivery models. Whether the observed effects are sustained beyond 24 weeks also requires further investigation.

This study shows that both LC and HC diets incorporated as part of a lifestyle modification weight loss program achieve significant improvements in glycemic control and cardiovascular risk markers in overweight and obese adults with T2DM. However, the greatest improvements were achieved following the LC diet. This suggests an LC diet with high unsaturated and low saturated fat may confer advantageous therapeutic potential for T2DM management. Further research is required to establish the longer-term effects.

Acknowledgments. The authors thank the volunteers for their participation. The authors thank the work of the Clinical Research Team at the CSIRO, Animal, Food and Health Sciences (Ann McGuffin, Julia Weaver, and Vanessa Courage for coordinating the trial; Pennie Taylor, Janna Lutze, Paul Foster, Gemma Williams, Hannah Gilbert, and Fiona Barr for assisting in designing and implementing the dietary intervention; Lindy Lawson and Theresa McKinnon for nursing expertise; Vanessa Russell, Cathryn Pape, Candita Dang, Andre Nikolic, and Sylvia Usher for performing the biochemical assays; Dr. Thomas Wycherley for conducting the DEXA scans; Julie Syrette for assisting with the data management; and Kylie Lange for assisting with the statistical analyses) and Luke Johnston and Annie Mondello (Boot Camp Plus, Adelaide, Australia) for conducting the exercise sessions.
Funding. This study was supported by National Health and Medical Research Council project grant 103415. J.T. was supported by a postgraduate research scholarship from the Agency for Science, Technology and Research (ASTAR).

Duality of Interest. No potential conflicts of interest relevant to this article were reported. No sponsor or funding source had a role in the design or conduct of the study; collection, management, analysis, or interpretation of the data; or preparation, review, or approval of the manuscript.

Author Contributions. J.T. conceived and designed the study, analyzed and interpreted data, and drafted the manuscript. N.D.L.-M., C.H.T., M.N., and J.D.B. conceived and designed the study, analyzed and interpreted data, critically revised the manuscript for intellectual content, obtained funding, and supervised the study. G.A.W. and W.S.Y. conceived and designed the study, analyzed and interpreted data, and critically revised the manuscript for intellectual content. G.D.B. conceived and designed the study, analyzed and interpreted data, drafted the manuscript, obtained funding, and supervised the study. All authors read and approved the final manuscript. G.D.B. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Prior Presentation. Some outcome data were presented at the International Diabetes Federation World Diabetes Congress 2013, Melbourne, Australia, 2–6 December 2013.

References


### Supplementary Table 1. Baseline characteristics of study participants

<table>
<thead>
<tr>
<th></th>
<th>LC Diet (n=58)</th>
<th>HC Diet (n=57)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Demographics</strong></td>
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</tr>
<tr>
<td>Age (y)</td>
<td>58 (7)</td>
<td>58 (7)</td>
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<tr>
<td>Sex [n (%)]</td>
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<tr>
<td>Females</td>
<td>21 (36)</td>
<td>28 (49)</td>
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<tr>
<td>Males</td>
<td>37 (64)</td>
<td>29 (51)</td>
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<tr>
<td><strong>Body weight and Composition</strong></td>
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<tr>
<td>Body weight (Kg)</td>
<td>101.7 (14.4)</td>
<td>101.6 (15.8)</td>
</tr>
<tr>
<td>BMI (kg/ m$^2$)</td>
<td>34.2 (4.5)</td>
<td>35.1 (4.1)</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>112.4 (10.6)</td>
<td>112.5 (10.6)</td>
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<tr>
<td>Total FFM (kg)$^\dagger$</td>
<td>62.0 (10.5)</td>
<td>60.1 (11.3)</td>
</tr>
<tr>
<td>Total FM (kg)$^\ddagger$</td>
<td>39.8 (10.5)</td>
<td>41.5 (9.9)</td>
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<tr>
<td>FM:FFM ratio (kg/ kg)$^\ddagger$</td>
<td>0.7 (0.2)</td>
<td>0.7 (0.2)</td>
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<td><strong>Glycaemic control</strong></td>
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<tr>
<td>HbA1c (%)</td>
<td>7.3 (1.1)</td>
<td>7.4 (1.1)</td>
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<tr>
<td>Fasting Glucose (mmol/ L)</td>
<td>7.8 (2.1)</td>
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<tr>
<td>Mean Glucose (mmol/ L)$^\ddagger$</td>
<td>8.4 (2.1)</td>
<td>8.7 (1.7)</td>
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<tr>
<td>Minimum Glucose (mmol/ L)$^\dagger$</td>
<td>4.8 (1.5)</td>
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<tr>
<td>Maximum Glucose (mmol/ L)$^\dagger$</td>
<td>14.0 (3.6)</td>
<td>14.3 (3.2)</td>
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<tr>
<td>Glucose Range (mmol/ L)$^\dagger$</td>
<td>9.1 (3.5)</td>
<td>9.5 (2.9)</td>
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<tr>
<td>SD$_{intraday}$ (mmol/ L)$^\ddagger$</td>
<td>2.0 (0.8)</td>
<td>2.1 (0.7)</td>
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<tr>
<td>SD$_{interday}$ (mmol/ L)$^\ddagger$</td>
<td>0.5 (0.4)</td>
<td>0.5 (0.4)</td>
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<tr>
<td>MAGE (mmol/ L)$^\dagger$</td>
<td>5.2 (2.1)</td>
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<td>CONGA-1 (mmol/ L)$^\dagger$</td>
<td>1.7 (0.6)</td>
<td>1.7 (0.5)</td>
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<td>CONGA-4 (mmol/ L)$^\dagger$</td>
<td>3.0 (1.3)</td>
<td>2.9 (1.0)</td>
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<tr>
<td>MODD (mmol/ L)$^\ddagger$</td>
<td>1.8 (0.8)</td>
<td>2.1 (0.9)</td>
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<tr>
<td>AUC$_{Total per min}$ (mmol/ L)$^\dagger$</td>
<td>16.2 (4.9)</td>
<td>17.0 (3.9)</td>
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<tr>
<td><strong>CVD risk markers</strong></td>
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<tr>
<td>SBP (mmHg)</td>
<td>130.4 (13.1)</td>
<td>132.6 (13.2)</td>
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<tr>
<td>DBP (mmHg)</td>
<td>80.0 (8.9)</td>
<td>80.8 (10.1)</td>
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<tr>
<td>Insulin (mU/L)$^\dagger$</td>
<td>16.3 (8.3)</td>
<td>15.9 (7.6)</td>
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<tr>
<td>HOMA2-IR$^\dagger$</td>
<td>2.3 (1.1)</td>
<td>2.2 (1.0)</td>
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<td>HOMA2-%B$^\dagger$</td>
<td>75.5 (38.7)</td>
<td>67.7 (33.4)</td>
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<tr>
<td>Total Cholesterol (mmol/ L)</td>
<td>4.5 (1.0)</td>
<td>4.3 (1.0)</td>
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<tr>
<td>LDL-C (mmol/ L)</td>
<td>2.5 (0.9)</td>
<td>2.4 (0.9)</td>
</tr>
<tr>
<td>HDL-C (mmol/ L)</td>
<td>1.2 (0.2)</td>
<td>1.3 (0.3)</td>
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<tr>
<td>TG (mmol/ L)</td>
<td>1.6 (0.7)</td>
<td>1.4 (0.6)</td>
</tr>
<tr>
<td>CRP (mg/ L)$^\dagger$</td>
<td>2.8 (2.3)</td>
<td>2.7 (2.2)</td>
</tr>
<tr>
<td><strong>Medications</strong></td>
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<td><strong>Diabetes Medications</strong></td>
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<tr>
<td>Antiglycemic MES</td>
<td>1.3 (1.0)</td>
<td>1.1 (1.1)</td>
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<tr>
<td>Insulin [n (%)]</td>
<td>6 (10)</td>
<td>6 (11)</td>
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<tr>
<td>Metformin [n (%)]</td>
<td>46 (79)</td>
<td>41 (72)</td>
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</table>

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### SUPPLEMENTARY DATA

<table>
<thead>
<tr>
<th>Category</th>
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<tr>
<td>Sulfonylureas [n (%)]</td>
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<tr>
<td>Thiazolidinediones [n (%)]</td>
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<tr>
<td>GLP-1 agonists [n (%)]</td>
<td>1 (2)</td>
</tr>
<tr>
<td>DPP-4 inhibitors [n (%)]</td>
<td>1 (2)</td>
</tr>
<tr>
<td><strong>Lipid lowering medications</strong> [n (%)]</td>
<td>35 (60)</td>
</tr>
<tr>
<td><strong>Antihypertensive medications</strong> [n (%)]</td>
<td>41 (71)</td>
</tr>
<tr>
<td><strong>Physical activity</strong></td>
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<tr>
<td>Mean activity count (counts/min)</td>
<td>188.9 (65.9)</td>
</tr>
<tr>
<td>MVPA (min/day)</td>
<td>46.4 (19.2)</td>
</tr>
<tr>
<td>MVPA (% of total wear time)</td>
<td>3.5 (1.4)</td>
</tr>
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</table>

Abbreviations: LC diet, Very low carbohydrate, high unsaturated/ low saturated fat diet; HC diet, High carbohydrate, low fat diet; BMI, Body Mass Index; FM, Fat mass; FFM, Fat Free Mass; LDL-C, Low density lipoprotein cholesterol; HDL-C, High density lipoprotein cholesterol; TG, Triglycerides; HOMA2-IR, Homeostasis model of assessment index 2- insulin resistance; HOMA2-%B, Homeostasis model of assessment index 2- β cell function; CRP, C-reactive protein; MAGE, Mean amplitude of glycaemic excursions; CONGA- 1, Continuous overall net glycemic action of observations 1 hour apart; CONGA- 4, Continuous overall net glycemic action of observations 4 hours apart; MODD, Mean of daily blood glucose differences; AUC Total per min, Total area under the curve standardised by valid wear time; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; MES, Medication Effect Score; DPP-4 inhibitors, Dipeptidyl-peptidase-4 inhibitors; GLP-1 agonists, Glucagon-like peptide-1 agonists; MVPA, Moderate to vigorous intensity physical activity.

Data are means (SD), unless otherwise stated.

To convert mmol/ L to mg/ dL, multiply by 18 (for glucose), 38.7 (for cholesterol), and 88.6 (for triglycerides).

* Total analysed n=115 (LC:58, HC:57) for all data unless otherwise stated. All baseline characteristics were not significantly different between diet groups (p>0.05) by independent samples t- test (continuous variables) or χ² test (categorical variables).

† Computed from continuous glucose monitoring (CGM) data

‡ Total analysed n=109 (LC:54, HC:55) that met requirement of 48-hours valid CGM data collection to calculate comparisons between 2 successive days.

‖ Total analysed n=103 (LC:52, HC:51) for insulin and HOMA2 data; 12 participants on insulin medication at baseline were excluded from analyses.

§ Total analysed n=105 (LC:54, HC:51) for CRP data; 10 participants with CRP >10 mg/L at baseline were excluded from these analyses.

# Computed from accelerometry data.
**Supplementary Table 2.** Macronutrient composition of diets.

<table>
<thead>
<tr>
<th></th>
<th>LC (n=46)</th>
<th>HC (n=47)</th>
<th>P Value *</th>
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<tbody>
<tr>
<td>Total Energy (Kcal)</td>
<td>1563 (225)</td>
<td>1587 (171)</td>
<td>0.56</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>56.7 (8.0)</td>
<td>204.9 (22.8)</td>
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</tr>
<tr>
<td>Carbohydrate (% energy)</td>
<td>13.9 (1.6)</td>
<td>50.1 (2.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>102.8 (14.7)</td>
<td>73.6 (8.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Protein (% energy)</td>
<td>26.7 (1.3)</td>
<td>18.8 (0.9)</td>
<td>&lt;0.001</td>
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<tr>
<td>Total Fat (g)</td>
<td>96.5 (16.5)</td>
<td>44.3 (7.4)</td>
<td>&lt;0.001</td>
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<tr>
<td>Total Fat (% energy)</td>
<td>54.1 (2.6)</td>
<td>24.5 (2.5)</td>
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<tr>
<td>Saturated Fat (g)</td>
<td>17.7 (3.1)</td>
<td>13.6 (2.9)</td>
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<tr>
<td>Saturated Fat (% energy)</td>
<td>10.0 (0.9)</td>
<td>7.5 (1.1)</td>
<td>&lt;0.001</td>
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<tr>
<td>Monounsaturated Fat (% energy)</td>
<td>30.4 (1.8)</td>
<td>11.5 (1.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Polyunsaturated Fat (% energy)</td>
<td>12.2 (1.1)</td>
<td>4.1 (0.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total Cholesterol (mg)</td>
<td>243 (42)</td>
<td>138 (25)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Dietary Fibre (g)</td>
<td>24.7 (3.5)</td>
<td>31.1 (3.2)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are means (SD)

LC diet - Very low carbohydrate, high unsaturated/ low saturated fat diet, HC diet - High carbohydrate, low fat diet

* P value refers to between group differences by independent t-tests