Low- and high-carbohydrate weight-loss diets have similar effects on mood but not cognitive performance

Angela K Halyburton, Grant D Brinkworth, Carlene J Wilson, Manny Noakes, Jonathan D Buckley, Jennifer B Keogh, and Peter M Clifton

ABSTRACT

Background: Low-carbohydrate diets are often used to promote weight loss, but their effects on psychological function are largely unknown.

Objective: We compared the effects of a low-carbohydrate, high-fat (LCHF) diet with a conventional high-carbohydrate, low-fat (HCLF) diet on mood and cognitive function.

Design: Ninety-three overweight or obese participants \([\bar{x} \pm \text{SEM}]\): body mass index (in kg/m\(^2\)): 33.6 \(\pm\) 0.4 were randomly assigned to an energy-restricted \((\approx 6–7 \text{ MJ}, 30\% \text{ deficit})\) planned isocaloric LCHF diet or an HCLF diet for 8 wk. Body weight and psychological well-being were measured by using the Profile of Mood States, Beck Depression Inventory, and Spielberger State Anxiety Inventory instruments at baseline and fortnightly. Cognitive functioning (working memory and speed of processing) was assessed at baseline and week 8.

Results: The LCHF diet resulted in significantly greater weight loss than did the HCLF diet \((7.8 \pm 0.4 \text{ and } 6.4 \pm 0.4 \text{ kg}, \text{ respectively}; \text{ } P = 0.04)\). Both groups showed improvements in psychological well-being \((P < 0.01 \text{ for time})\), with the greatest effect occurring during the first 2 wk, but there was no significant difference between groups. There were no significant between-group differences in working memory \((P = 0.68)\), but there was a significant time \(\times\) diet interaction for speed of processing \((P = 0.04)\), so that this measure improved less in the LCHF than in the HCLF diet group.

Conclusions: Both dietary patterns significantly reduced body weight and were associated with improvements in mood. There was some evidence for a smaller improvement in cognitive functioning with the LCHF diet with respect to speed of processing, but further studies are required to determine the replicability of this finding. Am J Clin Nutr 2007;86:580–7.

KEY WORDS Psychological function, weight loss, ketogenic diet, low-carbohydrate diet, obesity

INTRODUCTION

Whereas current guidelines recommend a high-carbohydrate, low-fat (HCLF), energy-restricted diet for obesity treatment \((1, 2)\), there has been considerable public interest in using low-carbohydrate, high-fat (LCHF) ketogenic diets, such as the Atkins diet, which are usually high in protein and fat (particularly, saturated fat), to attain successful weight loss \((3)\). There has however, been concern that an LCHF diet may alter serotonergic expression \((4)\) and adversely affect aspects of psychological function, including mood and cognition \((5, 6)\). Whereas a high-carbohydrate intake can increase serotonin synthesis, fat and protein intakes can reduce serotonin concentrations in the brain \((4, 7, 8)\). But the effect of an LCHF diet on psychological function has been poorly studied, and the effects remain largely unknown. Thus, many persons adopt this dietary plan without regard for potential negative effects.

Observational studies have shown that diets low in carbohydrate and high in fat and protein are associated with higher levels of anxiety and depression \((9, 10)\), but such studies do not allow for the drawing of inferences about causation. Acute feeding studies have shown that participants felt less vigorous and imaginative and more dreamy and sociable after consuming an LCHF meal than after consuming an isonenergetic HCLF meal \((11, 12)\). However, there is a paucity of well-controlled studies examining the chronic effects on mood state of altering the entire composition of a diet—in particular, changing to an LCHF—over a longer period. Two small studies in healthy, obese females showed that mood state was not affected by diet composition after the consumption of a low-energy \((800–950 \text{ kcal/d})\) HCLF or LCHF diet for 4–6 wk \((13, 14)\). However, because of the low energy intakes, the absolute carbohydrate concentrations in both diets were relatively low, which limited the generalizability of the findings. Conversely, compared with an HCLF diet, an LCHF diet increased the levels of tension, depression, anger, and total mood disturbance and decreased vigor in trained female cyclists \((15)\). Butki et al \((6)\) also showed that physically active men and women experienced greater fatigue, more negative affect, and less positive affect in response to exercise training after 3 wk of a carbohydrate-restricted Atkins diet than after 3 wk of an HCLF diet. However, that study did not control for energy intake, and both of these latter trials \((6, 15)\) examined normal-weight participants, whereas the use of weight-loss diets is more prevalent.
in obese populations. Common methodologic limitations among previous studies include small sample sizes, brief treatment durations, and limited experimental control, as well as the use of very low energy intakes in most instances. Consequently, it is difficult to draw any definite conclusions regarding the chronic effects on psychosocial health of an LCHF diet combined with moderate energy restriction, and further studies are warranted.

Other studies also have suggested that dietary intake can influence cognitive function and performance (16), but, again, few studies have investigated the chronic effects of diet composition, in particular an LCHF diet, on cognitive function. A small study in obese women found impairment of cognitive function after the consumption of a very-low-energy (≈600 kcal), low-carbohydrate, ketogenic diet but not after the consumption of an isocaloric nonketogenic diet with higher carbohydrate and lower fat (17). Impaired cognitive performance has also been observed in rats fed a high-fat diet, and greater impairment was observed with greater proportions of saturated fat (18, 19). Epidemiologic studies also have suggested that diets high in complex carbohydrates and low in saturated fat may prevent cognitive decline (20, 21). Together, this evidence suggests that an LCHF diet may adversely influence cognitive function.

Considering the lack of well-controlled intervention studies, the purpose of the present study was to compare the effects on mood and cognitive function of a moderately energy-restricted LCHF diet with those of an isocaloric, conventional HCLF diet in overweight and obese persons.

**SUBJECTS AND METHODS**

**Participants and design**

Overweight or obese men and women [n = 121; body mass index (in kg/m²): 26–43; age range: 24–64 y] with abdominal obesity and ≥1 other metabolic risk factor, as defined by the International Diabetes Foundation (22), were recruited by public advertisement to participate in an 8-wk outpatient clinical trial. Before study commencement, all participants completed a health screening questionnaire, and potential participants were excluded if they had a history of liver, cardiovascular, peripheral vascular, respiratory, or gastrointestinal disease; diabetes; a malignancy; or a psychological disorder.

The protocol and the potential risks and benefits of the study were fully explained to the participants before they provided written informed consent. All experimental procedures were approved by the Human Ethics Committee of the Commonwealth Scientific and Industrial Research Organisation, the University of Adelaide, and the University of South Australia.

**Study design**

In a parallel study design, participants were matched for age, sex, and body mass index and then randomly assigned to consume either an energy-restricted low-carbohydrate ketogenic (LCHF) diet or an isocaloric conventional high-carbohydrate (HCLF) diet for 8 wk. At baseline (week 0) and after the intervention (week 8), participants attended the clinical research unit at the Commonwealth Scientific and Industrial Research Organisation after an overnight fast; at that time, a series of psychological mood assessment questionnaires were completed, and cognitive functioning tests assessing working memory and speed of processing were undertaken. The participants’ height and weight were measured before a venous blood sample was taken for measurement of plasma ketone bodies. Throughout the intervention, participants attended the clinic fortnightly for a weight check and completed the questionnaires to quantify time-course changes in mood. Apart from the prescribed dietary intervention, participants were asked to maintain their usual lifestyle throughout the study.

**Dietary intervention**

The planned macronutrient profiles of the dietary interventions were as follows: the LCHF diet provided 35% of total energy as protein, 61% as fat (20% as saturated fat), and 4% as carbohydrate; the HCLF diet provided 24% of total energy as protein, 30% as fat (<8% as saturated fat), and 46% as carbohydrate. The diets were designed to be isocaloric and to have a moderate energy restriction of ≈30% energy (≈6000 kJ for women and 7000 kJ for men) for 8 wk. Key foods for each diet representative of the diet’s macronutrient profile were supplied on a fortnightly basis during the 8 wk to aid compliance. The foods provided mainly were uncooked but were weighed in advance to facilitate compliance. The dietary plan was structured to include specific daily quantities of foods to ensure the correct macronutrient and energy requirements (Table 1). These foods were listed in a food record that participants completed on a daily basis. Detailed dietary advice and information on meal planning and recipes were provided at baseline and every 2 wk by a qualified dietician. Scales for weighing food were provided. Three consecutive days (1 weekend day and 2 weekdays) from the semiquantitative food record of each 2-wk period were analyzed while the volunteer was present to ensure accuracy. This analysis was conducted with the use of FOODWORKS.
Body height and weight

Body height was measured to the nearest 0.1 cm by using a stadiometer (SECA, Hamburg, Germany) while participants were barefoot and in a free-standing position. Body weight was measured to the nearest 0.05 kg by using calibrated electronic digital scales (AMZ 14; Mercury, Tokyo, Japan) while participants wore light clothing and no footwear.

Psychological questionnaires and cognitive tests

Participants completed the mood assessment in a quiet, temperature-controlled room. Mood was assessed by using 3 validated paper-based questionnaires: Profile of Mood States (POMS; 23), Beck’s Depression Inventory (BDI; 24), and Spielberger State Anxiety Inventory (SAI; 25). The POMS questionnaire is a sensitive measure of mood in normal healthy participants; it measures several subscales: tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewildernment; a higher score represents a higher level. The POMS global score [total mood disturbance score (TMDS)] was determined by subtracting the vigor or activity score from the sum of the 5 negative mood factors. The BDI and SAI are well-validated measures that were used to determine each participant’s state (depression and anxiety, respectively) during the intervention period. For all mood measures, higher scores indicated higher levels of negative mood, and these measures were previously used to assess effects of macronutrient manipulation (9, 15, 26).

Cognitive function was assessed by using the computer-based digit span backwards (DSB) and inspection time (IT) tests (27), which assess working memory and speed of processing, respectively. The DSB test is a subtest of the Wechsler Adult Intelligence Scale-III (28) that requires the participant to recall a string of numbers in an order backwards of that in which they were presented. Scores represent the maximum number of digits correctly entered backwards. The IT test involves a simple discrimination task in which participants are required to identify which 1 of 2 lines of a target stimulus displayed for a very short time is the shorter. A cue is presented for 500 ms, and this appearance is followed first by the presentation of the stimulus and then by the presentation of a pattern mask that completely covers the stimulus area. As the test proceeds, each presentation of the stimulus lasts a progressively shorter time, with the use of a staircase procedure. Scores represent the minimum amount of time the participant needs to view the object to achieve a previously specified level of accuracy. Both tests were conducted with PRESENTATION software (version 9.13; Neurobehavioral Systems Inc, Albany, CA).

Biochemical analysis

Fasting blood samples were collected from a forearm vein into tubes containing either no additive for lipids or sodium fluoride and EDTA for glucose and ketone body measurements. Plasma or serum was isolated by centrifugation at 2500 rpm at 4 °C for 10 min (GS-6R centrifuge; Beckman, Irvine, CA) and then stored at −80 °C until it was analyzed. Biochemical assays were performed at one time at the completion of the study. Serum lipid and glucose concentrations were measured in one run on a Hitachi analyzer by using standard enzymatic kits (both: Roche Diagnostics Co, Indianapolis, IN). A modified Friedwald equation was used to calculate LDL cholesterol (29). Plasma ketone concentrations were analyzed in duplicate on a Hitachi autoanalyzer (Roche Diagnostics Co) with the use of a RANBUT d-3-hydroxybutyrate kit (Randox Laboratories Ltd, Crumlin, United Kingdom).

Statistical analysis

Before hypothesis testing, data were examined for normality. Statistical analyses were performed with the use of SPSS for WINDOWS software (version 14.0; SPSS Inc, Chicago, IL). Between-group differences in baseline characteristics were compared by using independent t tests for continuous variables and the Pearson chi-square test for categorical variables. Student’s independent t tests were used to compare dietary data. The effect of the dietary intervention was assessed by using repeated-measures analysis of variance (ANOVA) with time as the within-subject factor and diet (LCHF versus HCLF diet) as sex as between-subject factors. When there was a significant main effect, post hoc comparisons were performed as appropriate with Bonferroni’s adjustment for multiple comparisons to determine differences between group means. Analysis of covariance was used to adjust for differences in weight loss. Age was also used as a covariate in all analyses. Intention-to-treat analysis with the last observation carried forward was performed for those who did not complete the study for the primary outcome measures of weight, mood, and cognitive function. Correlational analysis was used to determine relations between variables. Statistical significance was set at P < 0.05. All data are presented as means ± SEMs. Twenty subjects in the LCHF group and 19 in the HCLF group were missing data for 1 of the 5 measurement times for the psychological variables; the missing data point was replaced by the mean for the subscale.

RESULTS

Participants

Of the 121 participants who were enrolled, 14 withdrew before commencement of the study and another 12 withdrew during the intervention. Of those who withdrew during the intervention, 5 withdrew because of an inability to comply with the dietary protocol (n = 4 and 1 for the LCHF and HCLF diets, respectively), 6 were lost to follow-up and did not attend the follow-up clinic appointments for assessment (n = 3 for both the LCHF and HCLF diets), and 1 withdrew from the LCHF group because of an illness unrelated to the study. An additional 2 people in the LCHF group who were identified as outliers were also excluded from analysis, 1 because of an ongoing illness in the family that affected the participant’s mood state and 1 in whom extreme scores for the primary outcomes (mood and cognitive function measures) in the present study were reported. The final analysis and the data reported are for the 93 remaining participants (Table 2). There were no significant differences between groups in baseline, age, weight, body mass index, or cardiovascular disease risk factors.

Dietary analysis and compliance

The reported dietary intakes are consistent with the prescribed dietary treatments (Table 3). There was no significant difference
in total energy intake between the 2 diet groups (P = 0.25). Carbohydrate intake was significantly higher and fat and protein intakes were significantly lower in the HCLF than in the LCHF diet group (P < 0.001). There was no difference in plasma concentrations of ketone bodies (d-3-hydroxybutyrate) between the groups at week 0 (0.07 ± 0.01 and 0.06 ± 0.01 mmol/L for the LCHF and HCLF diets, respectively; P = 0.40). There was a significant (P < 0.001) time × diet effect for plasma ketone bodies, such that concentrations had increased more in the LCHF diet group than in the HCLF diet group by week 2 (0.41 ± 0.04 and 0.08 ± 0.02 mmol/L, respectively), and they remained higher throughout the intervention, which indicated adherence to a very low carbohydrate intake in the LCHF diet during the study.

**Body weight**

After the intervention, there was a significant (P = 0.02) time × diet interaction for weight loss: the LCHF diet group had a significantly (P = 0.005) greater weight loss than did the HCLF diet group (8.0 ± 0.3% and 6.6 ± 0.4%, respectively) (Figure 1). No significant effect of sex or age was observed. A comparable response was observed with intention-to-treat analysis: the magnitude of the intervention effect was slightly more modest (weight loss of 7.6 ± 0.3% and 6.3 ± 0.4% in the LCHF and HCLF diets, respectively) but significant (P = 0.01).

**Mood measures**

At week 0, there was no significant difference between the groups in scores on the BDI, the SAI, or the TMDS on the POMS (P ≥ 0.64). All participants were within the normal nonclinical range for each psychometric measure. There was a significant reduction in BDI, SAI, and TMDS on the POMS in both groups during the intervention; most of this effect occurred during the first 2 wk of the study (P < 0.001 for time; Figure 2). However, there was no significant differential effect of diet composition for any of the mood scores (P ≥ 0.49, time × diet interaction). There was also no statistically significant effect of sex on these variables. The 6 subscales of the POMS for tension, depression, anger, vigor, fatigue, and confusion all showed significant improvements during the study (P < 0.001 for time), and these subscales followed the same time course pattern of change as did the TMDS: no significant effect of diet was evident for these variables (P ≥ 0.23). Adjustment for weight loss in all of these analyses had no effect.

The intention-to-treat analysis (ie, that in subjects who did not complete the study) showed patterns that did not differ significantly from those in the completers. There were no correlations between the mood state measures and the nutrient intake variables.

**Cognitive function measures**

At week 0, there was no significant difference between the groups with respect to working memory (DSB test: 4.2 ± 0.2 and 3.8 ± 0.2 for the LCHF and HCLF diets, respectively; P = 0.26) or speed of processing (IT test, P = 0.35; Figure 3). After the intervention, DSB test scores increased in both groups (0.8 ± 0.2

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**TABLE 2**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>LCHF diet (n = 48)</th>
<th>HCLF diet (n = 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>50.6 ± 1.1</td>
<td>49.8 ± 1.3</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>18</td>
<td>19</td>
</tr>
<tr>
<td>Women</td>
<td>30</td>
<td>26</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>93.6 ± 2.1</td>
<td>97.0 ± 2.1</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>33.3 ± 0.6</td>
<td>33.8 ± 0.6</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>133.0 ± 2.0</td>
<td>135.8 ± 1.8</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>73.9 ± 1.7</td>
<td>77.7 ± 1.6</td>
</tr>
<tr>
<td>Triglycerol (mmol/L)</td>
<td>1.64 ± 0.10</td>
<td>1.80 ± 0.15</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>5.40 ± 0.15</td>
<td>5.37 ± 0.11</td>
</tr>
<tr>
<td>HDL</td>
<td>3.27 ± 0.15</td>
<td>3.23 ± 0.12</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>1.39 ± 0.04</td>
<td>1.33 ± 0.05</td>
</tr>
<tr>
<td>Glucose (mmol/L)</td>
<td>5.7 ± 0.09</td>
<td>5.6 ± 0.08</td>
</tr>
</tbody>
</table>

1 LCHF, low-carbohydrate, high-fat; HCLF, high-carbohydrate, low-fat. There were no significant between-group differences (independent-sample t test for continuous variables and chi-square test for categorical variables).

2 SEM. LCHF, low-carbohydrate, high-fat; HCLF, high-carbohydrate, low-fat. Participants completed daily semi-quantitative food record analyses at weeks 2, 4, 6, and 8.

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**TABLE 3**

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>LCHF diet (n = 48)</th>
<th>HCLF diet (n = 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kJ)</td>
<td>6640.6 ± 98.2</td>
<td>6471.8 ± 107.3</td>
</tr>
<tr>
<td>Carbohydrate (% of energy)</td>
<td>5.0 ± 0.1</td>
<td>46.7 ± 0.5</td>
</tr>
<tr>
<td>Protein (% of energy)</td>
<td>35.1 ± 0.3</td>
<td>24.0 ± 0.3</td>
</tr>
<tr>
<td>Fat (% of energy)</td>
<td>58.4 ± 0.4</td>
<td>27.8 ± 0.5</td>
</tr>
<tr>
<td>Saturated fat (% of energy)</td>
<td>20.9 ± 0.3</td>
<td>5.9 ± 0.1</td>
</tr>
<tr>
<td>Monounsaturated fat (% of energy)</td>
<td>25.2 ± 0.3</td>
<td>12.3 ± 0.3</td>
</tr>
<tr>
<td>Polyunsaturated fat (% of energy)</td>
<td>7.9 ± 0.1</td>
<td>6.9 ± 0.1</td>
</tr>
</tbody>
</table>

1 All values are ± SEM. LCHF, low-carbohydrate, high-fat; HCLF, high-carbohydrate, low-fat. Participants completed daily semi-quantitative food records. Dietary data from 3 d (2 weekdays and 1 weekend day) were analyzed at weeks 2, 4, 6, and 8.

2 Significantly different from the LCHF diet, P < 0.001 (independent-sample t test).
and 0.7 ± 0.2, respectively; *P < 0.001 for time effect), and the difference between the groups was not significant (P = 0.67). Speed of processing improved (ie, IT test scores decreased) in both treatment groups during the intervention; however, there was a significant (P = 0.04) effect of diet; that is, the HCLF diet promoted greater improvements than did the LCHF diet (effect size (Eta²) = 0.04; Figure 3). This effect remained significant after control for weight loss. There were no significant effects of sex or age on the treatment effects observed. The intention-to-treat analysis resulted in similar effects for the cognitive function measures. Correlational analyses, both within each diet group separately and across the 2 diet groups, showed no significant associations between the changes in DSB or IT test scores with weight loss or change in plasma ketone body concentrations. In addition, there was no significant correlation between the changes in DSB scores or any of the nutrient intake variables. The change in IT test scores was significantly positively correlated with the percentages of energy from fat (r = 0.23, P = 0.03), saturated fat (r = 0.21, P = 0.04), and monounsaturated fat (r = 0.23, P = 0.03) and negatively correlated with the percentage of energy from carbohydrate (r = −0.21, P = 0.04). There were no other significant correlations.

**DISCUSSION**

The main finding of this study was that, under clinical supervision in an outpatient setting, consumption of a hypoenergetic LCHF diet had effects on mood and working memory similar to those of an isocaloric, conventional HCLF diet. Participants following either diet improved their speed of processing over 8 wk, but whether these improvements in cognition reflected outcomes of weight loss or simply arose from a practice effect is a matter of interpretation. However, failure to observe a correlation between IT test scores and weight loss suggests that a practice effect is more likely. Nonetheless, a significant interaction effect for speed of processing was observed, which indicated that the improvements in the LCHF group were less than those seen in the HCLF control group. This finding suggests that the usual practice effect may have been moderated by the LCHF diet. Further work is required to isolate practice effects from treatment effects.
As in a previous study by our group (30), plasma ketones remained low throughout the present study in participants consuming the HCLF diet but were elevated in those consuming the LCHF diet. This finding and the data obtained from the daily weighed-food checklists made us confident that participants in both groups achieved a high level of adherence to the prescribed diets and consumed qualitatively different diets.

Both dietary programs resulted in substantial reductions in body weight, with the LCHF diet producing greater weight loss than the HCLF diet. This finding confirms the results of the previous study (30) and is in accordance with other clinical trials (31, 32).

The principal objective of this study was to evaluate the psychological effects of an LCHF diet in a large, well-controlled trial over a longer term than was previously used. We found that participants consuming an LCHF diet had no differences in mood measures from participants consuming an isocaloric conventional HCLF diet. In direct agreement, previous smaller weight-loss studies in overweight and obese persons showed no difference between low-energy HCLF or LCHF diets with respect to psychological wellbeing after 4–6 wk (13, 14). In contrast, studies in normal-weight, physically active participants undertaking concomitant high-intensity exercise training showed that consumption of an LCHF diet over a 1–3-wk period adversely affected mood state (6, 15). Glucose is the primary fuel for intense exercise (33), and it is possible that undertaking exercise training with reduced muscle glycogen may have been more difficult and thus could have affected mood. On the whole, participants in our study were sedentary and were asked to maintain their habitual physical activity levels throughout the intervention. Whether any differential effects of the diets on psychological state would be observed in obese patients undertaking a concurrent exercise training program remains unknown, but, because a combination of energy restriction and increased physical activity is advocated for weight management (1, 2), this possibility warrants further investigation.

Another possible explanation for the lack of any effect of diet composition on mood in the present study is that both diets were hypocaloric, and, because participants were overweight and obese, their motivation for participating was weight loss. At the end of week 8, mood in both groups had improved significantly from baseline. This finding is consistent with previous research that showed marked improvements in mood with weight loss via energy restriction in overweight and obese persons (34–36). Therefore, it is likely that the effects of weight loss may have had dominant effects on mood that overshadowed any effect of diet composition. However, Schweiger et al (37) showed in normal-weight young women that mood was significantly better after 6 wk of an energy-restricted diet higher in carbohydrate and lower in fat and protein. Hence, the possible protection of weight loss against any otherwise negative effects of dietary composition may be confined to overweight or obese persons. Furthermore, it should still be recognized that, although the 2 diets produced similar mood effects during active weight loss, whether similar effects occur over the long term during weight maintenance remains to be established.

It is also noteworthy that the treatment environment may have been a factor in mitigating any moderating effects of the diets. In the present study, the improvements in mood indexes did not correlate with the amount of weight lost; most of the effect on mood occurred within the first 2 wk of caloric restriction, before substantial weight loss was realized. Studies have reported that active participation in supervised weight-loss programs, such as clinical studies, can improve mood, probably by enhancing feelings of self-control (38, 39). Participation in a controlled study with the associated intensive support for weight loss may have been responsible for subjects’ positive mood responses. This participation may provide an alternative explanation for the absence of any effect of diet on mood or well-being and for the divergent findings of observational studies showing that diets low in carbohydrate and high in fat and protein are associated with higher levels of anxiety and depression (9, 10). Further studies should evaluate psychological adjustment to LCHF diets in persons who are self-managing their diet in an environment without external support.

Cognitive function measures showed significant improvement in both diet groups, which most likely reflected a practice effect, because changes were not related to the degree of weight loss. Previous studies showed that following a prescribed diet has little effect on cognitive performance in overweight or obese participants (40, 41). However, the IT test score (a measure of the speed of visual information processing) was affected by diet composition. The results showed that participants consuming the LCHF had significantly less improvement in the minimum stimulus time required to make a correct response than did those consuming the HCLF diet, and analysis indicated a moderate effect size. Whether this effect was caused by the HCLF diet’s promotion of greater improvement in processing speed or the LCHF diet’s moderation of practice effects is difficult to determine. Our findings are consistent with those of an earlier study in obese women showing that performance of a complex, cognitively demanding task assessing mental flexibility was significantly worse after the consumption of a very-low-energy, low-carbohydrate, ketogenic diet than after the consumption of an isocaloric, nonketogenic diet with higher carbohydrate and lower fat content (17). Similarly, the treatment of young rats with a ketogenic LCHF diet for 1 mo resulted in severe cognitive impairment (19), and a series of rat studies showed that the chronic ingestion of a high-fat diet, in particular a high-saturated-fat diet, can adversely affect cognitive performance (18). Both cross-sectional and prospective human epidemiologic studies have shown that a high intake of fat, especially saturated fat, is associated with poorer cognitive performance (20, 42). In support of this, the improvement in processing speed in the present study was associated with low fat and low saturated fat intakes and a higher intake of carbohydrate. Collectively, this evidence suggests that the differential change in IT test scores was likely caused by some detrimental effect of the LCHF diet, and, although the specific mechanism cannot be determined, the high intake of fat, the restriction of carbohydrate, or a combination of the 2 was likely responsible. It has been postulated, on the basis of recent evidence from animal studies, that a high dietary fat intake may cause cognitive impairment by disrupting neurogenesis in the brain through an increase in serum corticosterone concentrations (43). Alternatively, insulin resistance and glucose intolerance associated with a high intake of saturated fat have been identified as important factors (18, 44, 45). Further work is required to identify the neurobiological mechanisms underlying cognitive impairment.

The large sample size, the matched energy content of the 2 diets, and the high level of dietary compliance are key strengths of this study, but the generalizability of the findings is limited to...
healthy, overweight and obese, young to middle-aged adults with normal mood state and no cognitive impairment who are undertaking a structured weight-loss program. Whether either of these diets would produce similar outcomes over longer periods and under ad libitum conditions or in populations that are more vulnerable to adverse effects, such as persons with specific psychological difficulties, requires further investigation. Moreover, although the DSB and IT tests evaluate components of working memory and speed of processing, respectively (46), and although they were previously found to show sensitivity to nutritional status and manipulation (47–49), they may not evaluate these cognitive domains in their entirety. Future research could be extended to include further measures of cognition and other.

In conclusion, the results of the present study indicate that short-term consumption of a moderately energy-reduced LCHF diet has an effect on the psychological well-being of overweight and obese persons similar to that of consumption of an isocaloric conventional HCLF diet. However, there was evidence of a somewhat smaller improvement in speed of processing with an LCHF diet. Further studies are required to determine the replicability of this finding and to determine whether similar outcomes are evident over the long term.

We thank the volunteers whose participation made the study possible. We gratefully acknowledge Kathryn Bastiaans, Julia Weaver, Anne McGuffin, and Vanessa Courage for coordinating this trial; Belinda Wyld for assisting in the collection of the mood and cognitive function data; Xenia Cleanthous, Julianne McKeough, and Gemma Williams for assisting in the dietary intervention; Rosemary McArthur for nursing expertise; and Candita Sullivan, Julie Turner, Laura Nehez, and Mark Mano for assisting with the biochemical assays.

The authors’ responsibilities were as follows—AKH (who completed this study as part of the requirement of her honors BSc in physiology): participated in study implementation, performed data analyses, and contributed to the interpretation of results and the writing of the manuscript; GDB: the conception and design of the study, trial coordination, all statistical analyses, data interpretation, and coordination of the writing of the manuscript; CW, JDB, and PMC: contributed to the experimental design, data interpretation, and writing of the manuscript; MN: the design of the dietary protocols and contributions to the experimental design, data interpretation, and writing of the manuscript; JBK: the design of the dietary protocols, completed the dietary analysis, and contributed to the preparation of the manuscript; and all authors: agreement on the final version of the manuscript. None of the authors had a personal or financial conflict of interest.

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