SHORT-TERM ATKINS DIET ALTERS BEHAVIOR AND GLYCEMIC STATUS OF HEALTHY HUMAN VOLUNTEERS

Ahmad Afaghi¹, Helen O’Connor² and Chin Moi Chow²

¹Discipline of Exercise and Sport Science, Faculty of Health Science, The University of Sydney, NSW 1825 Australia; and Qazvin University of Medical Science, School of Medicine, Qazvin Metabolic Disease Research Center (QMDRC), Qazvin, Iran; and ²Discipline of Exercise and Sport Science, Faculty of Health Science, The University of Sydney, Sydney, Australia.

[Received October 22, 2009; Accepted November 4, 2009]

ABSTRACT: The low carbohydrate, high-fat, high-protein Dr Atkins weight-reduction diet is popular worldwide. In addition to evidence of short-term efficacy for weight reduction, there are consistently reported side effects including constipation, halitosis, headache and fatigue. We examined the effect of the Atkins diet compared to a control mixed diet on sleepiness, mood, fatigue and related symptoms. Fourteen, healthy subjects (18-35 y, BMI = 23.2 ± 1.9 kg.m⁻²) were maintained on a control diet followed by the Atkins diet for 48 h. Subjects’ daytime mood, fatigue intensity and sleepiness on the Epworth Sleepiness Scale were assessed before the evening test meals. Symptoms that developed during the Atkins diet were scored using a modified Atkins diet symptom questionnaire. The number of subjects with dream recall was recorded on awakening after each polysomnographic night. Subjects developed ketosis and mild hypoglycemia 48 h after consuming the Atkins diet with significant increase in daytime symptoms of fatigue, sleepiness, depressed mood and impaired cognition. The findings indicate that macronutrients in food can significantly influence behavioral symptoms and dream recall in response to resultant biochemical changes. Thus mild hypoglycemia resulting from the diet may mediate the subjective responses of daytime sleepiness, depressed mood and intense fatigue.

KEYWORDS: Atkins diet; Dream recall; Fatigue; Macronutrients; Mood; Sleepiness

INTRODUCTION

In his introductory speech at the 1982 Conference on Research Strategies for Assessing the Behavioral Effects of Food and Nutrients Dr. Richard Wurtman said “That what one eats can affect the way one feels and behaves is probably obvious to most laymen” (Lieberman and Wurtman, 1982). There is now ample evidence to support the claim that the constituents of food can positively and negatively affect mood, cognition and sleep via metabolites that act on the brain. A number of amino acids are known to influence these responses. For example, L-tryptophan (Trp) induces an earlier sleep onset (Hartmann and Elion, 1977; Hartmann and Spinweber, 1979; Spinweber et al., 1983; Wurtman and Wurtman, 1995). This effect on the improvement in sleep onset behavior has also been confirmed following consumption of a high glycemic index (GI) carbohydrate meal with a low protein content (Afaghi et al., 2007).

Previous studies have shown that different macronutrients, especially high levels of each macronutrient of fat, protein and carbohydrates promote a sedative action and sleepiness (Wells et al., 1995; Wells et al., 1998). High carbohydrate meals were observed to induce a state of calmness and sleepiness. These effects are probably mediated via increased serotonin levels following carbohydrate consumption (Young, 1991; Wurtman and Wurtman, 1995; Zmarzty et al., 1997). Following a lunch meal high in protein or carbohydrates but with a standardized fat content, similar postprandial effects were observed with subjects feeling more feeble, lethargic and mentally slower (Wells and Read, 1996). Similarly, in comparison to the period prior to lunch a high fat lunch also has been shown to produce subjective feelings of sleepiness, boredom, and feebleness (Wells et al., 1997). In addition, subject’s ratings of fatigue were higher three hours after the high fat meal in comparison to after a high-carbohydrate meal (Wells et al., 1997). Lunch containing a high level of fat (50%), but low carbohydrate content induced greater drowsiness, decreased mood, and impaired cognitive performance in comparison to a lunch with medium-fat and carbohydrate levels (Lloyd et al., 1994).
cholecystokinin (CCK) (Wells et al., 1997; Zmarzty et al., 1997). The timing of meal ingestion also has a direct influence on the postprandial mood and alertness. A high-fat meal compared to a high-carbohydrate meal given in the morning, irrespective of energy content, caused greater depression in mood and alertness than when given at lunch time (Wells and Read, 1996).

The Atkins diet, which consists of 60% fat, 30% protein and 10% carbohydrates (Atkins, 1992; Kossoff, 2004), has been shown to produce modest weight loss, but long-term compliance is generally poor (Westerterp et al., 1996; Toubro and Astrup, 1997; Brehm et al., 2003). Numerous studies have shown that restriction of glucose intake elicited carbohydrate craving and suppressed mood (Atkins, 1992; Wurtman and Wurtman, 1995; Wells et al., 1997; Wurtman et al., 2003). Although increased daytime sleepiness has been reported following the Atkins diet (Atkins, 1992), patients with narcolepsy prescribed the Atkins diet demonstrated a modest reduction in subjective daytime sleepiness on the Narcolepsy Symptom Status Questionnaire, but non-significant small changes on the Epworth Sleepiness Scale (Husain et al., 2004).

Adverse effects of the Atkins diet include constipation, halitosis, headache and fatigue (Atkins, 1992). These side effects may be due to macronutrient content or metabolic by-products of the diet. The diet is not prescribed to be hypoenergetic, but evidence indicates that the diet enhances satiety and reduces overall energy intake (Foster et al., 2003). The reduction of carbohydrate energy metabolism leads to fatigue. Glucose levels are decreased in comparison to normal levels and ketosis is evident after a few days on the Atkins diet (Atkins, 1992). Inadequate carbohydrate intake induces fatty acid metabolism to sustain energy levels. Ketones (acetoacetic acid, β-hydroxybutyric acid and acetone) are produced as metabolites of fat oxidation and released into the blood. Via high hepatic production, ketones provide an alternative metabolic fuel for the brain. With increasing levels of ketones passing through the kidneys, they can be detected by the spill over into the urine. An intake of less than 25 g/d carbohydrate leads to ketones passing through the kidneys, they can be detected by the spill over into the urine. Adequate ketone intake induces fatty acid metabolism to sustain energy levels.

MATERIALS AND METHODS

Subjects

Fourteen healthy men (18-35 years, BMI = 20-27 kg.m⁻²) were recruited from 45 screened volunteers. Subjects were recruited based on a medical questionnaire and an interview, from the student population attending Sydney University. Each subject signed a consent form that stated the purpose and nature of the study. Subjects were excluded if they had a self-reported current or past history of significant medical, psychiatric or sleep disorders (nocturnal eating inclusive), used prescribed medication (including sedatives or antidepressants), recreational drugs, or regularly had an alcohol intake of greater than 20 g per day or 100 g per week. Subjects exercising more than three times a week were also excluded. During the 48 h prior to the study subjects abstained from any vigorous exercise. Subjects were asked to go to bed at the same time on each of the study nights. They were also asked to abstain from alcohol for 48 h, and caffeinated beverages for 12 h prior to and during the entire testing period spanning five nights.

Measures

The measures of behavior included subjective rating of mood, fatigue (visual analogue scale, VAS) (Black et al., 2005), sleepiness during the day (Epworth Sleepiness Scale, ESS) (Johns, 1991), postprandial sleepiness (Likert, 1932), the modified Atkins diet related symptoms (Atkins, 1992) and frequency of dream recall. Biochemical measures included urine ketones and blood glucose.

Protocol

This observational study on the behavioral responses to the Atkins diet was part of a study conducted to evaluate objective sleep changes via polysomnography on each test night. The study consisted of five study nights, commencing with a familiarization night, followed by a day and evening meal based on a balanced, mixed macronutrient profile (Control) and then the Atkins diet for 48 h. The night immediately followed by the first evening Atkins test meal is designated ‘Atkins Acute’ phase, and the night following 48 h on the Atkins diet, with an Atkins evening test meal was designated ‘Atkins Ketosis’ phase (Table 1). All evening test meals were eaten 4 h before subject’s usual bedtime.

<table>
<thead>
<tr>
<th>TABLE 1. Study meal plan.</th>
<th>*Control test meal: 1090 kcal; 15.5% protein, 12.5% fat and 72% carbohydrate; #Atkins test meal: 1090 kcal; 38% protein, 61% fat and &lt;1% carbohydrate; Daily energy consumption: 2400 kcal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day</strong></td>
<td><strong>Familiarization</strong></td>
</tr>
<tr>
<td>Breakfast</td>
<td>Mixed meal</td>
</tr>
<tr>
<td>Lunch</td>
<td>Mixed meal</td>
</tr>
<tr>
<td>6-8 h fast on days 2, 3 &amp; 5</td>
<td></td>
</tr>
<tr>
<td>Evening meal</td>
<td>Mixed meal</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleep study</td>
</tr>
</tbody>
</table>
Behavioral data were collected for the control day, the Atkins Acute and Atkins Ketosis days. The measures of ESS, mood and fatigue were taken before each evening test meal (Table 1), as was the modified Atkins diet-related symptom questionnaire. Only sleepiness via Likert scale was measured postprandial. The number of subjects with dream recall was recorded each morning following waking. Urine was collected for ketone analysis before the evening test meal and at bedtime. Finger-prick blood samples for glucose analysis were taken before (zero time) and after each test meal at 15, 30, 60, 90 and 120 min.

**Meals**

Standard isocaloric test meals (approximately 1090 kcal) were provided. The control mixed meal consisted of 16.5% protein, 12.5% fat, 71% carbohydrate (Table 2), and the Atkins meals had a macronutrient distribution of 38% protein, 61% fat, and less than 1% carbohydrate for the Atkins Acute and Atkins Ketosis nights (Table 3).

**TABLE 2. Control mixed meal composition.** Diet composition was analyzed using Food works analysis software, version 3.

<table>
<thead>
<tr>
<th>Food</th>
<th>Wt/g</th>
<th>E/kJ</th>
<th>E/kcal</th>
<th>Protein/g</th>
<th>Fat/g</th>
<th>CHO/g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tomato past</td>
<td>10.0</td>
<td>28.1</td>
<td>6.7</td>
<td>0.3</td>
<td>0.0</td>
<td>1.0</td>
</tr>
<tr>
<td>carrot, 1 (medium)</td>
<td>61.0</td>
<td>78.7</td>
<td>18.8</td>
<td>0.5</td>
<td>0.1</td>
<td>3.23</td>
</tr>
<tr>
<td>Apple red, large</td>
<td>212.0</td>
<td>483.4</td>
<td>115.5</td>
<td>0.6</td>
<td>0.2</td>
<td>26.5</td>
</tr>
<tr>
<td>Rice, white, raw Jasmine</td>
<td>170.0</td>
<td>2510.9</td>
<td>599.8</td>
<td>11.2</td>
<td>0.9</td>
<td>134.5</td>
</tr>
<tr>
<td>Chicken 2 medium drumstick (without skin)</td>
<td>118.0</td>
<td>836.6</td>
<td>199.9</td>
<td>30</td>
<td>8.9</td>
<td>0.0</td>
</tr>
<tr>
<td>Vegetable oil</td>
<td>5.0</td>
<td>188.1</td>
<td>45.0</td>
<td>0</td>
<td>5.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Orange juice (contain 30% juice)</td>
<td>262.0</td>
<td>437.5</td>
<td>104.5</td>
<td>0.0</td>
<td>0.0</td>
<td>26.5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>838.0</td>
<td>4563.3</td>
<td>1090.2</td>
<td>42.6</td>
<td>15.0</td>
<td>191.7</td>
</tr>
</tbody>
</table>

**TABLE 3. Atkins' meal composition.** Diet composition was analyzed using Food works analysis software, version 3.

<table>
<thead>
<tr>
<th>Food</th>
<th>Wt/g</th>
<th>E/kJ</th>
<th>E/kcal</th>
<th>Protein/g</th>
<th>Fat/g</th>
<th>CHO/g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chicken drumstick, roasted with skin (5 medium)</td>
<td>335.0</td>
<td>3102.0</td>
<td>741.0</td>
<td>76.0</td>
<td>48.9</td>
<td>0.0</td>
</tr>
<tr>
<td>Vegetable oil</td>
<td>10.0</td>
<td>376.2</td>
<td>90.0</td>
<td>0.0</td>
<td>10.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Egg, whole, boiled (3 medium)</td>
<td>150.0</td>
<td>948.0</td>
<td>223.0</td>
<td>19.7</td>
<td>15.9</td>
<td>0.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>445.0</td>
<td>4802.4</td>
<td>1054.0</td>
<td>95.7</td>
<td>74.8</td>
<td>0.0</td>
</tr>
</tbody>
</table>

**Ketosis and urine ketone level**

Subjects’ urine was collected on three of the study nights (control, Atkins Acute and Atkins Ketosis) before the evening test meal and at bedtime to monitor urinary ketone levels with Multiple Reagent Strips for Urinalysis (Multistix 10 SG Bayer) using the following semi quantitative scale: none, trace (0.5 mM), low (1.5 mM), moderate (4 mM), high (= 8 mM) (Husain et al., 2004). Ketone levels at bedtime on the evening of the Atkins Ketosis phase also served to monitor subjects’ adherence to the Atkins diet. A minimum concentration of urinary ketones level of 1.5 mM (Husain et al., 2004) had to be reached for subjects to continue in the final sleep study night.

**Blood Glucose**

Finger prick blood samples for glucose analyses using a glucometer (Medisense Optium TM) were collected at baseline before each test meal and at 15, 30, 45, 60, and 120 min after each meal. The area under the curve (Cajochen et al.) for the blood glucose response over the 120 min was calculated using Microsoft Office Excel.

**Behavioral instrument**

**Daytime Sleepiness by Epworth Sleepiness Scale (ESS)**

Subjects were instructed to choose the most appropriate number on a scale of 0-3 for each of the eight situations (see below). Scales included: 0 = would never doze or sleep, 1 = slight chance of dozing or sleeping, 2 = moderate chance of dozing or sleeping, 3 = high chance of dozing or sleeping. The situations were: Sitting and reading, Watching TV, Sitting inactive in a public place, Being a passenger in a motor vehicle for an hour or more, Lying down in the afternoon, Sitting and talking to someone, sitting quietly after lunch (no alcohol), Stopped for a few minutes in traffic while driving. A total score of less than 10 was considered normal, a score of 10 or more was considered sleepy, and a score of 18 or more was very sleepy. The ESS is a reliable and valid method for measuring daytime sleepiness (Johns, 1991).

**Subjective rating of mood, fatigue and postprandial sleepiness (Likert and visual Analogue Scale, VAS)**

Subjects ranked their overall mood for each day using a VAS with a 100 mm horizontal line with “0” being their best possible overall mood and “100” being their worst possible overall mood (Black et al., 2005). Subjects also ranked their daily level of fatigue with “0” being lack of fatigue and “100” being highest intensity of fatigue (Black et al., 2005). Subjective rating of postprandial sleepiness was assessed after the meal at time points 1, 2, 3, and at 4 h (immediately) before bedtime by marking appropriately on a four-point sleepiness Likert scale, from zero ‘not at all sleepy’, +1 ‘slightly sleepy’, +2 ‘sleepy’ to +3 ‘very sleepy’. The validity and reliability of this scale has been demonstrated (Likert, 1932; Hindmarch, 1980).

**Modified Atkins diet-related symptom questionnaire**

The Atkins questionnaire of 49 items that described the symptoms related to ketosis and hypoglycemia was reduced to a 27 item modified questionnaire that included only the most frequently reported symptoms during the ketotic period (Atkins, 1992).
Subjects were instructed to use the questionnaire to document the symptoms felt during the day.

**Dream recall**

The number of subjects who experienced dreams was recorded each morning following an overnight polysomnographic study (sleep data reported elsewhere). Subjects were then asked to rate how they felt about their dreams on a three-point scale (1 = pleasant, 2 = neutral, 3 = unpleasant).

**Statistics**

Data were inspected for normality of distribution prior to use of parametric statistics. Data are reported as mean ± standard deviation (SD). The Likert scale (Likert, 1932) ratings for postprandial sleepiness (at 4 time points and interaction of meal types and these time points), mood and fatigue intensity, and day time sleepiness (ESS) were analyzed by repeated measures ANOVAs. Similar statistical analyses were applied to compare the effect of meal types on blood glucose levels at baseline (zero time) and 6 time points following the test meals, and the area under the curve (Cajochen et al.) of blood glucose response. Non parametric (K-related samples, Cochrane) test was used to compare the effect of meal types on dream recall.

The Sydney University’s Ethics Committee approved the experimental protocol and consent of the subjects was obtained after the purpose of the study and the nature of the procedures were fully explained.

**RESULTS**

**Urinary ketones**

Urinary ketone level showed negative traces before the meal and at bedtime for both the control and Atkins Acute nights (Table 4). This contrasted with an increased ketone level 48 h following commencement of the Atkins diet at Atkins Ketosis where 2 subjects showed a low ketone level of 1.5 mM, 6 showed moderate levels of 4 mM, and 6 had high levels of = 8 mM.

**Blood glucose**

Table 4 shows the baseline blood glucose level before the evening meal and the glucose response curve (Cajochen et al.) for the control, Atkins Acute and Atkins Ketosis phase. Statistical difference was not seen for the baseline blood glucose level measured prior to either the control mixed meal or Atkins first test meal. However, the baseline glucose level for the Atkins Ketosis condition showed a significant fall from the control mixed meal level (P < 0.001).

After the control mixed meal, blood glucose peaked at 45 min followed by a gradual fall back to the baseline level. In contrast, a flat glucose response was observed following the Atkins meals at Atkins Acute and Atkins Ketosis. Significant differences were found for the AUC between the control mixed meal and the Atkins test meals at Acute and Ketosis phase (P < 0.001) (Figure 1).

![FIGURE 1. Blood glucose response following the Control and Atkins test meals (Acute and Ketosis) given 4 h before bedtime. n = 10. A significant fall in baseline glucose level was observed for the Atkins Ketosis night compared with Control and Atkins acute phase (P < 0.001). Significant differences were found between the area under curve (*) for the Control night compared to Atkins test meals (P < 0.001).](image)

![TABLE 4. Biochemical and behavioral measures following a mixed meal compared to the Atkins diet at the Acute and Ketosis phase. *Comparison of meals between Control and Atkins Ketosis; †Comparison of meals (baseline blood glucose) between Atkins Acute and Atkins Ketosis; ‡ ± SD (all such values); ‡Comparison of meals (AUC) between Atkins Acute and Control. n = 14. AUC, area under curve; ESS, Epworth sleepiness scale; VAS, visual analogue scale.](table)

**Epworth sleepiness scale (ESS)**

The subjective rating of daytime sleepiness recorded before the control mixed meal and before the Atkins first test meal (Atkins Acute) was not significantly different as expected.
However, at Atkins Ketosis phase, all participants showed a significant increase in daytime sleepiness score (p < 0.001) (Table 4). Ten subjects had ESS score of 10 or more and 4 subjects had an ESS score of 8 or 9.

**Overall mood (VAS)**

The subjective ratings of overall daytime mood recorded before the control mixed meal and before the Atkins first test meal (Atkins Acute) were not significantly different. However, worsened mood was recorded at Atkins Ketosis phase (p <0.001) (Table 4).

**Fatigue intensity (VAS)**

The rating of “Fatigue” intensity recorded before the control mixed meal and before the Atkins first test meal (Atkins Acute) were not significantly different. A significant increase in rating for this item was recorded at Atkins Ketosis (p <0.001) (Table 4).

**Postprandial Sleepiness (Likert scale)**

The subjective ratings showed progressive increase in sleepiness each hour up to bedtime (P <0.001) following the evening test meals for Control, Atkins Acute and Atkins Ketosis (Figure 2). The interaction of meal type and time after meal ingestion was not significant (P >0.1).

**Modified Atkins diet-related symptom responses**

No symptoms were experienced on the control day or the Atkins Acute day. In contrast, at the Atkins Ketosis phase many symptoms were experienced including ‘fatigue’, ‘craving for sweets’, ‘tired all the time’, and ‘sleepiness during the day’ (Figure 3). Subjective reports of cognitive function included ‘poor motivation’, ‘difficulty with concentration’ and ‘difficulty with decision making’. Negative emotions of boredom, depression and anger were also expressed at the Atkins Ketosis phase.

**Dream recall**

There was a trend in increased number of subjects reporting dreams during Atkins Acute and Ketosis phase compared to control (overall P = 0.09) (Table 2). Unpleasant dreams were reported in 50% of those who dreamt during the Atkins Ketosis phase.

**DISCUSSION**

The present study reported the biochemical and behavioral responses and dream recall frequency in response to a short-term Atkins diet in healthy adult males. The subjects did not experience any unusual response following either the control mixed meal or Atkins first test meal (Atkins Acute). Mild hypoglycemia (3.42 ± 0.22 mM) and ketosis developed 48 h (Atkins Ketosis) after commencement of the Atkins diet. These symptoms coincided with subjective reports of increased daytime sleepiness, increased fatigue intensity and poorer concentration, with an increased number of subjects reporting dreams of an unpleasant nature. These symptoms experienced by our subjects represent a direct response to the macronutrient profile or metabolites associated with the Atkins diet. Thus, factors such as ketosis, hypoglycemia and possible serotonin diminution from reduced carbohydrate ingestion or elevated CCK from high fat and high protein ingestion (Atkins, 1992; Wells et al., 1997; Wurtman et al., 2003) are considered below.

The appearance of the symptoms including increased sleepiness, decreased mood and increased fatigue intensity followed the time course of ketosis development. However, a search of the literature failed to demonstrate a causal relationship between ketosis and these symptoms. These symptoms of the Atkins diet, along with poor cognition, negative mood related to irritability, anger, a lack of motivation and a craving for sweets suggest that hypoglycemia
may be a trigger factor.

During the Atkins Ketosis phase subjects showed a significant increase in daytime sleepiness with an Epworth Sleepiness Scale score of greater than 10. This score suggests an increased sleep propensity unrelated to any pre-existing sleep disorders or sleep deprivation, since our subjects had no history of sleep apnea (as assessed via polysomnography on the familiarization night), and they were regular sleepers (as indicated by their sleep log) respectively. The observed daytime sleepiness in our subjects thus was a result of the Atkins diet, which may be explained by the mild hypoglycemia. A blood glucose concentration of \( \approx 4 \) mM has previously been shown to increase drowsiness and decrease alertness that coincided with an increased level of electroencephalographic (EEG) theta (4-8 Hz) waves (Cox et al., 2000). Hypoglycemia at a level = 3.6 mM also impaired cognitive functioning (Cox et al., 2000).

Although our subjects reported increased daytime sleepiness, the subjective ratings of postprandial sleepiness indicated 'not at all sleepy' or only 'slightly sleepy' for the first three hours after each of the evening meals (Control, Atkins Acute and Ketosis). The post-prandial period (1800 - 2000h) for our subjects appears to fall within the 'forbidden zone' for sleep (Wells et al., 1998) and thus may explain why our subjects did not feel sleepy after the meals regardless of the meal type, mixed or Atkins. It would be expected that meals that are served outside of the 'forbidden zone' should induce sleepiness. Indeed, alertness was suppressed in subjects fed a high-fat, low-carbohydrate meal in the morning or at lunch time (Wells et al., 1995; Wells et al., 1997). The onset of sleepiness coincided with CCK release approximately 2-3 h after the high fat, high protein meal (Wells et al., 1997).

It was not until four hours after the meal, at their usual bedtime, that our subjects indicated that they were ‘sleepy’ (Figure 2). However, this increased sleepiness was not significantly different between the three meals, suggesting that factors other than the meals, such as the circadian timing of sleep, may bear a strong influence on sleep propensity (Van den Heuvel et al., 1998).

One half (%50) of subjects reported feeling of "moodiness" during Atkins Ketosis (Figure 3). Their daytime moodiness may be related to a diminished level of serotonin resulting from a low blood glucose level and high protein content of the diet. Serotonin is involved in mood control (Young, 1991; Wurtman and Wurtman, 1995) and its levels in the brain dependent on its precursor tryptophan (Trp). However, the Atkins diet provided a low level of Trp to brain due to a high protein-high fat and low carbohydrate intake (Wurtman et al., 2003; Halyburton et al., 2007). Low level of serotonin along with hypoglycemia may be responsible for the worsening of mood (Halyburton et al., 2007) and hypoglycemia also accounts for the tiredness and fatigue (McAulay et al., 2001) experienced by our subjects. At Atkins Ketosis, 13 (%98) subjects experienced ‘fatigue’ and 11 (%79) subjects reported being ‘tired all the day’ (Figure 3). Thirteen (%79) subjects reported ‘carbohydrate craving’, 9 (%64) ‘slow start in the morning’, 8 (%57) ‘poor motivation’, and 8 (%57) ‘difficulty with concentration’ (Figure 3). These symptoms, inclusive of sleepiness and hypoglycemia during the short-term Atkins diet may have implications for work performance and driving (Frier, 2000). The highest rates of traffic accident victims has been reported in Saudi Arabia, United Arab Emirates, and in a London hospital during the food avoidance period attributable to reduced alertness (Roky et al., 2004) and low blood glucose concentration of 3.7 ± 0.6 mM compared to normal situation of 5.2 ± 0.4 mM (P <0.001) (Aybak et al., 1996). Subjects who did not consume food for several hours reported an increased irritability (Kaderi et al., 2000; Roky et al., 2004) as experienced by our subjects at Atkins Ketosis. Irritability may exaggerate the incidence of motor vehicle accidents.

It was interesting to note that our subjects recalled more dreams with progression of the Atkins diet. The highest proportion of subjects with dream recall occurred at the Atkins Ketosis phase. The dream recall may be explained by a higher rate of transient EEG arousals that occurred during light sleep stages 1-2 and increased wake time during the sleep period (reported in a separate study) during both Atkins Acute and Atkins Ketosis. The mean arousal index (stages 1-2) per hour for the control night was 11.2 ± 4.2, which was significantly lower than that for the Atkins Acute (14.9 ± 6.0, P = 0.02) and Atkins Ketosis (14.2 ± 6.4, P = 0.02) nights. It is known that dreams are usually fixated into memory by the event of awakening itself (Muzur, 2005). When awoken during the periods of increased spontaneous eyelid movements (i.e. EEG arousals) during stage 2 and REM sleep, the experimental subjects reported imagery dreams (Conduit et al., 2004). Several investigations have confirmed a positive correlation between frequency of dream recall and the frequency of nocturnal awakenings in healthy subjects (Cory et al., 1975; Halliday, 1988; Schredl and Montasser, 1996-1997a; Schredl and Montasser, 1996-1997b).

Dream content most likely reflects waking life stressors (Schredl et al., 1998) and incorporation of stressful elements of wakefulness into dreams (Koulack et al., 1985). Unpleasant dreams experienced by the subjects may be due to physiological stress related to the metabolic processes or low blood glucose of the Atkins diet. In conclusion the observed biochemical and behavioral responses to the Atkins diet on the short term suggest that ingested macronutrients and their by-products can significantly influence mood, fatigue and sleepiness during the daytime. Mild hypoglycemia resulting from the diet may explain these responses.

ACKNOWLEDGMENTS

We are most grateful for the help of Dr. Liz Barnes at Human Research Committee of Sydney University for her helpful guideline of statistical analysis. We also wish to thank the volunteers who participated in this study.

CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests. No author had any financial interest in the organization supported this research.

REFERENCES


