Chapter 5

INFLUENCE OF THE ATKINS’ DIET ON

BEHAVIOURAL RESPONSES
Influence of the Atkins’ diet on behavioural responses

Abstract

Objective: The effect of the Atkins’s diet was examined over 48 h compared to a Control mixed diet on sleepiness, mood, fatigue and related symptoms in healthy subjects.

Design: This study employed a repeated measure design where subjects were given isocaloric diets and matching evening test meals, which were either mixed (15% protein, 25% fat, 60% carbohydrate) or Atkins (38% protein, 61% fat, <1% carbohydrate). Subjects’ overall 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 recalls was recorded on awakening after each polysomnographic night.

Subjects: Fourteen, healthy subjects (18-35 y, BMI 20-27 kgm⁻²) underwent a Control diet followed by the Atkins’ diet for 48 h.

Measurement: Subjects’ overall 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 recalls was recorded on awakening after each polysomnographic night.

Results: Subjects developed mild hypoglycemia and ketosis 48 h following the Atkins’ diet. The daytime symptoms of fatigue, sleepiness and depressed mood were significantly increased following the Atkins’ diet compared to the Control diet. An increased proportion of subjects reporting dreams and cognitive impairments (increasing difficulty with concentration and decision making) and other symptoms (craving for sweets, and tired all the time) was also noted 48 h after the Atkins’ diet.

Conclusion: The findings suggest that mild hypoglycemia resulting from the diet may mediate the subjective responses of daytime sleepiness, depressed mood and intense fatigue. The increased proportion of subjects with dream recalls may be related to increased transient arousals from sleep during which dreams may be incorporated into memory.

Keywords: Atkins’ diet; Fatigue; Sleepiness; Mood; Dream recalls
**Introduction**

“That what one eats can affect the way one feels and behaves is probably obvious to most laymen.” Dr. Richard Wurtman wrote in his Introduction for the 1982 Conference on *Research Strategies for Assessing the Behavioral Effects of Food and Nutrients* (Lieberman & Wurtman 1982).

Today, there is evidence to support the claim that components of food can affect mood, cognition and sleep through biochemical factors acting 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 & Elion 1977; Hartmann & Spinweber 1979; Spinweber et al 1983; Wurtman & Wurtman 1995). This sleep behaviour was also confirmed following consumption of a high glycemic index (GI) carbohydrate meal with a low protein content (Afaghi et al 2007). The high GI effect on sleep onset is most likely mediated by a high insulin response, which promoted uptake of large neutral amino acids (LNAAs) by muscles, giving rise to a high Trp to LNAA ratio (Trp:LNAA), thereby facilitating the entry of Trp into the brain (Fernstron & Wurtman 1971; Layons & Truswell 1988; Berry et al 1991). In the brain, TRP is converted to serotonin, which both induces sleep and improves mood (Cole et al 1980).
Previous studies showed that different macronutrients, especially high-fat and high-carbohydrate content of meals conferred a sedative action and promoted sleepiness (Wells et al 1995; Wells et al 1998). High carbohydrate meals were observed to reduce pain perception, induce a state of calmness, sleepiness and satiety. These effects are likely mediated through serotonin following carbohydrate consumption (Young 1991; Wurtman & Wurtman 1995; Zmarzty et al 1997). High-protein and high-carbohydrate meals with standardized fat content showed similar postprandial effects with subjects feeling more feeble, lethargic and mentally slower after lunch (Wells & Read 1996). Amongst macronutrients, fat similarly showed a potent effect on subjective feelings of sleepiness, boredom, and being feeble after lunch than before eating (Wells et al 1997), and the rating of fatigue was higher 3 h after the meal compared to a high-carbohydrate meal (Wells et al 1997). A lunch meal containing a high proportion of fat (50%), but low content of carbohydrate induced greater drowsiness, decreased mood, and impaired cognitive performance with a muddled mind compared to a medium-fat medium-carbohydrate lunch (Lloyd et al 1994). Many of these responses of satiety, analgesia, the state of relaxation (Zmarzty et al 1997) and sleepiness observed after a high fat, high protein and to a much lesser extent high carbohydrate meal are thought to be mediated via
cholecystokinin (CCK) (Wells et al 1997; Zmarzty et al 1997). However, post-lunch sleepiness and fatigue may be due to circadian rhythm affect (Monk 2005) and the timing of meal ingestion also has a direct influence on postprandial mood and alertness. A high-fat meal compared to a high-carbohydrate meal given in the morning, irrespective of energy content, caused a greater depression in mood and alertness than when given at lunch time (Wells & Read 1996).

The Atkins’ diet, consisting of 60% fat, 30% protein and averaging 10% carbohydrate (Atkins 1992; Kossoff 2004), is associated with modest weight reduction, but with poor long-term compliance (Westerterp et al 1996; Toubro & Astrup 1997; Brehm et al 2003). Numerous studies have shown that restriction of glucose intake elicited carbohydrate craving and suppressed mood (Atkins 1992; Wurtman & Wurtman 1995; Wells et al 1997; Wurtman et al 2003). Although an increased daytime sleepiness has been reported following the Atkins’ diet (Atkins 1992), patients with narcolepsy prescribed the Atkins’ diet demonstrated modest improvements in subjective daytime sleepiness when scored on the Narcolepsy Symptom Status Questionnaire, but nonsignificant small changes when scored on the Epworth Sleepiness Scale (Husain et al 2004).
The adverse effects of the Atkins’ diet include constipation, halitosis, headache and fatigue (Atkins 1992). These side effects may be associated with the macronutrients or metabolic by-products of the diet. Although the diet is not prescribed to be hypoenergetic, evidence shows that the diet enhances satiety and reduces overall energy intake (Foster et al 2003) and that reduced carbohydrate energy metabolism may lead to fatigue. Decreased glucose from normal level and ketosis are evident a few days following commencement of the Atkins diet (Atkins 1992). Inadequate carbohydrate intake turns on fatty acid metabolism to sustain energy supply. Ketones (acetoacetic acid, β-hydroxybutyric acid and acetone) that are produced as metabolites of fat oxidation are then released into the blood. Ketones, through high hepatic production, provide an alternative metabolic fuel for the brain. As an increased level of ketones passes through the kidneys, some spill into the urine. An intake of less than 25 g/d carbohydrate leads to substantial urinary excretion of ketones (Westman et al 2002).

Dr. Atkins (Atkins 1992) reported that ketosis and hypoglycemia were factors responsible for the symptoms of “tired all the time”, “moodiness”, “difficult with concentration”, “poor memory”, “bored”, “headache”, “anger”, “sleepiness during day” and “fatigue”. A systematic
documentation of these behavioral changes in a controlled study has not been undertaken. Furthermore, subjects who were on the Atkins’ diet reported anecdotally an increased dream tendency. Therefore, in a study with a repeated measure design, healthy subjects were investigated in terms of subjective behavioural responses and dream recall frequency after consuming the Atkins diet for 48 h period.

Subjects and methods

Subjects

Fourteen healthy men (18-35 years, BMI 20-27 kgm\(^{-2}\)) 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 of the study and the nature of the experiment. 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 5 nights.

Protocol

This observational study on the behavioural 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 5 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 is designated ‘Atkins Ketosis’ phase (Table 1). Behavioral data were collected for the Control day, the Atkins Acute and Atkins Ketosis days. All evening test meals were eaten 4 h before their usual bedtime.

Behavioral data were collected for the control day, the Atkins Acute and Atkins Ketosis days. The measures of ESS (Johns 1991), mood and
fatigue (Black et al 2005) were taken before each evening test meal (Table 1) as was the modified Atkins’ diet-related symptom questionnaire (Atkins 1992). Only sleepiness via the Likert scale (Likert 1932) was measured postprandially at 1, 2, 3 and 4 h (immediately) prior to bedtime. 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.

**Meal**

Standard isocaloric (2400 kcal) diets and matching evening test meals (approximately 1090 kcal) were provided. The Control mixed meal consisted of 16.5% protein, 12.5% fat, 71% carbohydrate, and the Atkins’ meals had a macronutrient distribution of 38% protein, 61% fat, <1% carbohydrate for the Atkins Acute and Atkins Ketosis nights.

**Ketosis and urinary 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 urine ketone levels with Multiple Reagent Strips for Urinalysis (Multistix 10 SG Bayer) using the following semi quantitative scale: none, trace (0.5 mM), small (1.5 mM), moderate (4 mM), large (≥ 8 mM ) (Husain et al 2004). The ketone level on the evening of the Atkins Ketosis phase served as a check on 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 analysis using a glucometer (Medisense Optium TM) were collected at baseline before each test meal and at 15, 30, 45, 60, and 120 min after the meal.

_Behavioural 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. These situations were referred to subject’s usual way of
life in recent times. Even if subjects had not done some of these things
recently, they were instructed to work out how they would have affected
them. 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 (Visual
Analogue Scale and Likert scale): Subjects ranked their overall daily
mood for that day using a VAS with a 100mm 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 (Hindmarch 1980).

*Modified Atkins’ diet-related symptom questionnaire:* The Atkins’ questionnaire of 49 items that described the symptoms related to ketosis and hypoglycemia were reduced to 27 in the modified questionnaire to include 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 the next morning following an overnight polysomnographic testing (sleep data reported elsewhere). Subjects were then asked 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). Likert scale ratings for postprandial sleepiness, VAS for mood and fatigue intensity, and day time sleepiness (ESS) were analyzed by repeated measures ANOVA and “tests of within-subjects effects”. Non parametric (K-related samples, Cochrane) test was used to compare the effect of meal types on dream recall.

The current protocol has been approved by the Sydney University’s Ethics Committee and consent of the subjects was obtained after the nature of the procedures had been fully explained.

| TABLE 1 |
| Study meal plan |

<table>
<thead>
<tr>
<th>Day</th>
<th>1 Familiarization</th>
<th>2 Control</th>
<th>3 Atkins’ Acute</th>
<th>4 Atkins’ diet</th>
<th>5 Atkins’ Ketosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>Mixed meal</td>
<td>Mixed meal</td>
<td>Mixed meal</td>
<td>Atkins’ diet</td>
<td>Atkins’ diet</td>
</tr>
<tr>
<td>Lunch</td>
<td>Mixed meal</td>
<td>Mixed meal</td>
<td>Mixed meal</td>
<td>Atkins’ diet</td>
<td>Atkins’ diet</td>
</tr>
<tr>
<td>6-8 h fast on days 2, 3 &amp; 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evening meal</td>
<td>Mixed meal</td>
<td>*Control test meal</td>
<td>#Atkins’ test meal</td>
<td>Atkins’ diet</td>
<td>#Atkins’ test meal</td>
</tr>
<tr>
<td></td>
<td>Sleep study</td>
<td>Sleep study</td>
<td>Sleep study</td>
<td>Sleep Study</td>
<td></td>
</tr>
</tbody>
</table>

*Control test meal: 1090 kcal; 15.5% protein, 12.5% fat and 72% carbohydrate
#Atkins’ test meal: 1090 kcal; 38% protein, 61% fat and <1% carbohydrate
Daily energy consumption: 2400 kcal.
Results

Urine ketones

Urine ketone level showed negative traces before the meal and at bedtime for both the Control and Atkins Acute nights (Table 2). 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 level of 4 mM, and 6 had large level of ≥ 8 mM.

<table>
<thead>
<tr>
<th>Test</th>
<th>Control meal</th>
<th>Atkins Acute</th>
<th>Atkins Ketosis</th>
<th>Overall P</th>
<th>P-value&lt;sup&gt;c&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine ketone, mM</td>
<td>Negative</td>
<td>Negative</td>
<td>1.5-16</td>
<td></td>
<td>Ketosis</td>
</tr>
<tr>
<td>VAS Mood (mm)</td>
<td>45.8±4.6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>47.4±4.0</td>
<td>54.1±2.5</td>
<td>P&lt;0.001</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>VAS Fatigue (mm)</td>
<td>2.5±1.7</td>
<td>3.2±1.6</td>
<td>29.9±5.0</td>
<td>P&lt;0.001</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td>*Dream recalls</td>
<td>3</td>
<td>6</td>
<td>8</td>
<td>P=0.09</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>n = 14. VAS, visual analogue scale

<sup>b</sup>X ± SD (all such values).

<sup>c</sup>Comparison of meals between Control and Atkins Acute and Atkins Ketosis

*Number of subjects with dream recalls
Blood glucose

The baseline blood glucose level before the evening meal for the Control, Atkins Acute and Atkins Ketosis phase were 4.28 ± 0.38, 4.28 ± 0.43, and 3.42 ± 0.22 mM respectively. There was no difference in the baseline blood glucose level between the Control and the Atkins Acute night. However, a significant fall in baseline glucose level was observed for the Atkins Ketosis night (range 3.10 - 3.70 mM). Following the Control meal, the blood glucose response showed a peak at 45 min, followed by a gradual fall back to baseline level. In contrast, following the Atkins meals at Atkins Acute and Atkins Ketosis, a flat glucose response was observed.

Epworth sleepiness scale (ESS)

The daytime sleepiness score was not significantly different between the Control day (4.2 ± 2.1) and at Atkins Acute (4.4 ± 1.9). At Atkins Ketosis, all participants showed a significant increase in the subjective rating of daytime sleepiness with an ESS score of 11.1 ± 2.4 (P <0.001) (Figure 1). Ten subjects had ESS score of 10 or more and 4 subjects had an ESS score of 8 or 9.
Overall mood

The subjective ratings of overall daytime mood between the Control condition (45.8 ± 4.6) and Atkins Acute condition (47.4 ± 4.0) were not significantly different. However, worsened mood was recorded at a rating of 54.1±2.5 at Atkins Ketosis condition (P <0.001) (Table 2).

FIGURE 1. Comparison of the sleepiness score using the Epworth sleepiness scale (ESS) between the 3 test meals (Control mixed, Atkins Acute and Atkins Ketosis). Mean ESS score ( - - - ) on Atkins Ketosis was significantly increased compared to Control and Atkins Acute (n = 14, repeated measures ANOVA and "tests of within-subjects effects P < 0.001").
Fatigue intensity

The “Fatigue” intensity at the Atkins Acute phase (3.2 ± 1.6) did not change significantly from the Control day (2.5 ± 1.7). A significant increase in rating for this item was recorded at Atkins Ketosis (29.9 ± 5.0, P <0.001) (Table 2).

Postprandial Sleepiness scale (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).

FIGURE 2. The time course of subjective sleepiness scores over a 4 h period post-test meals to bedtime. n = 14
**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.
FIGURE 3. Comparison of the proportion of subjects experiencing the Atkins diet-related symptoms between Control and Atkins Ketosis for 14 subjects. Symptom items (17 only) are displayed.

**Discussion**

The present study reports the behavioural responses to short-term Atkins’ diet and dream recall frequency in healthy adult males. Subjects did not experience any unusual responses on the Control day or following the first Atkins’ 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 the subjective report of increased daytime sleepiness, increased fatigue intensity, worsened mood and concentration, and increased number of subjects with dream recalls of an unpleasant nature. The symptoms experienced by subjects may implicate origins from a number of factors that act either singly or in combination. The factors of ketosis, hypoglycemia, possible serotonin diminution, and increased cholecystokinin (CCK) resulting from a high fat, high protein and low carbohydrate diet (Atkins 1992; Wells et al 1997; Wurtman et al 2003) are considered below.

The time course of the observed symptom changes at Atkins Ketosis and their coincidence with hypoglycemia and ketosis provides a feasible link for the latter to explain daytime sleepiness, intense fatigue and depressed mood. However a search of the literature, failed to demonstrate the causal relationship between ketosis and these symptoms. In contrast, there is much evidence linking hypoglycemia to these responses. Our observation of hypoglycemia following the Atkins’ diet confirms the previously observed (Atkins 1992).
Subjective reports of daytime sleepiness, fatigue, poor cognition, negative mood related to irritability, anger, a lack of motivation and a craving for sweets during Atkins Ketosis strongly suggest that hypoglycemia was a primary factor responsible for these sensations. This claim is supported by published literature discussed below.

During the Atkins Ketosis phase subjects showed a significant increase in the Epworth Sleepiness Scale >10 (Figure 1). This score indicated an increased sleep propensity which was not influenced by the presence of sleep disorders or previous sleep deprivation as evidenced by their normal sleep schedules recorded in their sleep diary, and full polysomnograph on the familiarization night. The observed daytime sleepiness in our subjects was a result of the Atkins’ diet, which was likely explained by the mild hypoglycemia. A blood glucose concentration of ≤4 mM has previously been observed to lead to increased drowsiness and decreased 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 subjects reported an 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). It was not until the fourth hour post meal, at their usual bedtime, before they indicated that they were ‘sleepy’ (Figure 2). This increased feeling of sleepiness just before bedtime, however, was not significantly different between the three meals suggesting that factors other than the meals may dictate sleep propensity.

All of the meals were served at the same time for each subject between 1800 - 2000 h, that is 4 h before the subjects’ usual bedtime of 2206 - 2346 h. The meal period appears to fall within the ‘forbidden zone’ (sleep propensity is lowest) 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’. On the other hand, the subjects were increasingly sleepy close to their usual bedtime (P < 0.001, Figure 2) suggesting that circadian timing of sleep bears a strong influence on sleepiness (Van den Heuvel et al 1998). It would be expected that meals that are served outside of the ‘forbidden zone’ should induce sleepiness. Subjects fed a high-fat, low-carbohydrate meal in the morning or at lunch time showed suppressed alertness 2-3 h after the meal compared to a low-fat, high-carbohydrate meal (Wells et al 1995; Wells et al 1997). The changes in
postprandial sleepiness were thought to be related to cholecystokinin (CCK) release; the timing of its maximum release coincided with the onset of sleepiness (Wells et al 1997). It has been shown that CCK is released approximately 2-3 h after a high fat, high protein meal, and to a much lesser extent after a high carbohydrate meal, but not significantly affected by a mixed meal (Wells et al 1997).

Fifty percent of subjects reported feeling of “moodiness” during Atkins Ketosis (Figure 3). Subjects’ overall mood ratings taken during the day before the evening meal 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 & Wurtman 1995) and its level in the brain is dependent on its precursor Trp. In turn Trp entry into the brain depends on plasma Trp:LNAA. A high ratio thus facilitates its entry into the brain, whereas a low ratio resulting from a low carbohydrate-high protein meal similar to that of the Atkins’ meal would impede Trp entry into the brain (Wurtman et al 2003).

Tiredness and fatigue are symptoms of hypoglycaemia (McAulay et al 2001). At Atkins Ketosis, 98% of subjects experienced ‘feeling of
fatigue’ and 79% reported ‘tired all the day’ (Figure 3). Indeed, many of the symptoms experienced during Atkins Ketosis are tied to mild hypoglycemia, with 79% of subjects reporting a carbohydrate craving, 64% ‘slow start in the morning’, 57% ‘poor motivation’, and 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 Ramadan fasting period attributable to reduced alertness (Roky et al 2004). The blood glucose concentration during Ramadan has been reported as low as 3.7 ± 0.6 mM, which was significantly lower than compared to before the start of Ramadan 5.2 ± 0.4 mM (P <0.001) (Aybak et al 1996). Those who had fasted reported an increased irritability (Kaderi et al 2000; Roky et al 2004) similar to subjects in the present study who expressed feelings of ‘anger’ during Atkins Ketosis. Irritability may compound the incidence of motor vehicle accidents.

It was interesting to note that the number of subjects with dream recalls increased with progression of the Atkins’ diet. The highest proportion of subjects with dream recalls occurred at the Atkins Ketosis phase. These
dream recalls may be explained by a higher rate of transient EEG arousals that occurred during light sleep stages 1-2 (part of a study conducted to evaluate objective sleep changes) during both Atkins Acute and Atkins Ketosis. The mean arousal (stage 1-2) index 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. Transient arousals from sleep leading to dream fixation has been documented. 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).

It is known that dreams are usually consolidated into memory by the event of awakening itself (Muzur 2005). 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 & Montasser 1996-1997). We observed a non significant trend in increased total wake time (P > 0.1, data reported elsewhere) during Atkins Ketosis night compared to the Control and Atkins Acute nights. It is feasible that the wake time during the sleep period may contribute to the increased number of dreams being recalled.
This is consistent with a higher proportion of subjects recalling dreams during the Atkins Ketosis phase.

Dream content most likely reflects waking life stressors (Schredl et al 1998) and the incorporation of stressful elements of wakefulness into dreams (Koulack et al 1985). In the study self-description of dream emotions which were characterized with themes of “depression”, “negatives” and “unpleasant” may reflect the physiological stress related to the metabolic process and biochemical changes that occurred following the Atkins’ diet, in particular during Atkins Ketosis with mild hypoglycemia or ketosis.

In conclusion we explored behavioral responses to the Atkins’ diet on the short term. It is suggested that that mild hypoglycemia resulting in from the diet may explain the subjective responses of daytime sleepiness, depressed mood and intense fatigue. Following the Atkins diet a greater proportion of the subjects reported dream recalls, particularly during Atkins Ketosis.
References


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