Chapter 6

SUMMARY AND GENERAL DISCUSSION
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Despite wide ranging popular advice regarding foods or beverages that either help or hinder sleep and behaviour responses, there is a paucity of research regarding the specific benefits of dietary manipulation on sleep and behaviour responses. This thesis aimed to investigate the effects of manipulating macronutrient intake and the glycemic index (GI) on sleep and behaviour responses.

The first study explored the effect of GI and meal timing on the sleep pattern of healthy volunteers. This study revealed that a high GI meal shortens sleep onset latency (SOL) by 48.6% compared to an identical low GI meal (Afaghi et al 2007). The high GI meal ingested 4 h before the usual bedtime was more effective in reducing the SOL than the same meal taken 1 h before (Afaghi et al 2007). It was proposed that the reduction in SOL was mediated via an increased plasma tryptophan (Trp) to large neutral amino acids (LNAAs) ratio (Trp:LNAA) and the subsequent rise in brain serotonin level. The basis for this proposed mechanism lies in the increased insulin response to a high GI carbohydrate meal, which increases circulating insulin and facilitates
greater muscle uptake of LNAAxs, but not Trp resulting in an increased Trp:LNAA. Although did not measure Trp:LNAA or plasma serotonin did demonstrate a significantly increased area under the curve for glucose following a high GI compared to a low GI meal, so the insulin response to both of these meals would have been different. It is anticipated that analysis of plasma Trp and serotonin would reveal the direct mechanism and may also explain the differences in the greater effectiveness of the 4 h meal vs. 1 h meal given before the usual bedtime, since the time interval between meal ingestion and the metabolic process leading to increased serotonin level may be a critical factor.

A major finding of this study was a reduction in SOL following a high GI meal compared with the low GI meal. Given that good sleepers with limited room for sleep improvement were studied it could be suggested that a high GI meal consumed by a patient with sleep initiation insomnia may prove efficacious in improving the sleep pattern and sleep consolidation, in addition to reducing the SOL. Future research should explore this area in patients with insomnia.

The high GI meal consisted almost entirely of carbohydrate (rice and vegetables) with only a small proportion from protein (8%). This is vastly
different to a normal mixed diet with a recommended protein level of 15-20% usually from animal and vegetable sources. Thus, the high GI meal with a low percentage of protein is not typical and may not be either sufficiently palatable nor appropriate for regular consumption, particularly for certain groups such as the elderly who are a high risk group for insomnia, but also can be nutritional deficiency. However, addition of a higher percentage of protein to the meal may compromise its effect on SOL by lowering Trp:LNAA.

To ensure satiety in our study, large portions were served resulting in a very high glycemic load (GL) of 175 for the high and 81 for the low GI meal. Meals with a GL >20 are considered to be high, and over a day a GL of 120 is rated high. Meals containing a high GL are not suitable for daily consumption or for people who have obesity or diabetes.

The high GI meals may be applicable in people with circadian disorders or episodic insomnia such as travelers with jet-leg and shift-workers with circadian shifts to assist them to induce somnolence at the appropriate time. Additionally, high GI meals may be effective in subjects with delayed sleep phase syndrome. In the blind people who can not synchronize to the day/night cycle or do so at an abnormal time, the high
GI meal may be effective and comparable with melatonin administration (Lewy et al 2006). These applications are yet to be tested. Epidemiological studies in relation to eating pattern (high GI starchy food vs. low GI mixed meal) at night, and sleep quality may reveal new information about eating and sleep behavioral.

In summary, future research should include individuals with insomnia of both gender and with the age range extended to include older individuals for outcomes to be more generalisable. A dose response study of glycemic load is needed to assess the optimal load for clinically significant changes in SOL.

The second study outlined in this thesis investigated the short-term effect of the Atkins’ diet on sleep pattern and behaviour. The study revealed that the Atkins’ diet increased slow wave sleep (SWS), reduced rapid eye movement sleep (REM), increased electroencephalogram (EEG) arousal and increased the proportion of subjects reporting dream recalls. However, 48 h following commencement of the Atkins’ diet with development of hypoglycemia and ketosis, the participants reported increased sleepiness, fatigue, and suppressed mood. These findings are important and clearly suggest that dieters be aware of these symptoms.
Sleep pattern changes may be linked to changes in the macronutrients of the Atkins diet, whereas reduced alertness and increased sleepiness and fatigue are likely a result of hypoglycemia (Cox et al 2000) observed during the Atkins Ketosis phase. Increased proportion of subjects reporting dream recall may be related to increased EEG arousals following which dreams are fixated into memory. The Atkins diet-related symptoms such as “lack of concentration”, “slow in the morning”, “carbohydrate craving” and “being bored” expressed by the subjects may be a result of the low carbohydrate content (Dye et al 2000). A restricted carbohydrate intake together with a high protein content of the Atkins’ diet could be interpreted as decreased availability of serotonin (Wurtman et al 2003) thereby suppressing mood (Wurtman & Wurtman 1995).

It has been reported that obese and overweight subjects showed an abnormality in SWS, REM sleep and sleep duration (Vorona et al 2005; Taheri 2006). Following weight reduction these were normalized (Charuzi et al 1992; Willi et al 1998; Dixon et al 2005; Gangwisch et al 2005; Vorona et al 2005; Taheri 2006; Bjorvatn et al 2007). In addition, low carbohydrate diet improves SWS in normal subjects. These observations together with our findings appear to suggest that sleep
changes have a direct link to dietary changes and weight reduction through the Atkins diet improves sleep quality.

The Atkins’ diet induced behavioral responses including suppressed mood, sleepiness and fatigue. The intensity of sleepiness after ingestion of the Atkins’ diet may increase especially when the meal coincides with the 12-h harmonic rhythm of temperature nadir (e.g. at lunch time) that is associated with diminished alertness (Monk 2005), the post-lunch dip. The observed behavioural responses and symptoms are usually reduced or abolished as a gradual increase of carbohydrate content occurs with progression of the diet (Atkins 1992).

In monitoring the behavioural responses during the short-term Atkins’ diet, a major limitation has been the restricted measurement of symptoms of overall mood, fatigue and sleepiness only in the evening before the meal. Measures of mood, fatigue and sleepiness at different time points during the day would enable a determination of the time point during the day that subjects experience the alternated mood, increased fatigue or sleepiness.
The effects of the Atkins’ diet on sleep and behaviour should be further perused, since there are several publications indicating that the diets similar to the Atkins’ diet can produce significant weight reduction. Therefore, future systematic, long-term effects of the Atkins’ diet on sleep, metabolic changes and behavior response relationships should be investigated.
References


Cory TL, Orniston DW, Simmel E, Dainoff M (1975): Predicting the frequency of dream recall. *J Abnorm Psychol* 84: 261-266.


*Nutrition* 16(10): 1021-1034.

Edinger JD, Wohlgemuth WK, Radtke RA, Marsh GR, Quillian RE (2001):  
Cognitive Behavioral Therapy for Treatment of Chronic Primary  

Ellenbogen MA, Young SN, Dean P, Palmour RM, Benkelfat C (1996): Mood  
response to acute tryptophan depletion in healthy volunteers: sex  

5: 61-65.

Effect of rapid tryptophan depletion on sleep electroencephalogram and  
mood in subjects with partially remitted depression on Bupropion.  
*Neuropsychopharmacology* 27(6): 1016-1026.

(2007): Glucose regulation of insulin gene transcription and pre-mRNA  


Fernstron JD, Wurtman RJ (1971): Brain serotonin content: increase following  


_Psychoneuroendocrinology_ 18(8): 567-78.