



# HHS Public Access

Author manuscript

*West J Nurs Res.* Author manuscript; available in PMC 2017 September 29.

Published in final edited form as:

*West J Nurs Res.* 2013 April ; 35(4): 497–513. doi:10.1177/0193945911416379.

## Nutritional Effects on Sleep

Glenda Lindseth<sup>1</sup>, Paul Lindseth<sup>1</sup>, and Mark Thompson<sup>2</sup>

<sup>1</sup>University of North Dakota, Grand Forks

<sup>2</sup>Naverus Corporation, Kent, WA

### Abstract

The purpose of this study was to examine the nutritional effects on sleep using actigraphy measures. A repeated-measures, counterbalanced, cross-over study design was used to administer treatment diets to 44 adult participants. Participants served as their own control and consumed high-protein, high-fat, high-carbohydrate, and control diets. The study participants wore *Motionlogger* Actigraph sleep watches while consuming weighed food intakes for 4 days over four different treatment periods. Demographic and laboratory data were also analyzed. Actigraph results showed that the wake episodes and sleep latencies were significantly different when comparing the macronutrient intakes of the participants. Post hoc test results showed that high-protein diets were associated with significantly fewer ( $p = .03$ ) wake episodes and high-carbohydrate diets were associated with significantly shorter ( $p < .01$ ) sleep latencies than control diets. Thus, consumption of specific macronutrient intakes may have a significant influence on sleep.

### Keywords

healthy adults; nutrition; sleep

---

More than two thirds (72%) of adults get less than the recommended 8 hr of sleep each night and one fifth (20%) of those adults get less than 6 hr each night (Centers for Disease Control and Prevention, 2011; National Sleep Foundation, 2009). The consequences of a lack of sleep can result in a poor performance of daily activities and reductions in functional capacities (Mahowald, 2007). Furthermore, sleep deprivation is seen as an unmet public health problem (Colten & Altevogt, 2006). Some studies have shown that dietary intakes may significantly affect sleep when macronutrient intakes are manipulated (Husain, Yancy, Carwile, Miller, & Westman, 2004; Szentirmai, Kapás, Sun, Smith, & Krueger, 2010). However, there is a paucity of research on this topic, and study results are mixed and inconclusive.

---

Reprints and permission: [sagepub.com/journalsPermissions.nav](http://sagepub.com/journalsPermissions.nav)

**Corresponding Author:** Glenda Lindseth, University of North Dakota, P.O. Box 9025, Grand Forks, ND 58201, USA. [glenda\\_lindseth@und.nodak.edu](mailto:glenda_lindseth@und.nodak.edu).

#### Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Electroencephalographic sleep changes were studied in eight young healthy male participants using a repeated-measures, within-subjects study design (Phillips et al., 1975). A high-carbohydrate/low-fat diet resulted in significantly less slow-wave (nonrapid eye movement [non-REM]) sleep in comparison with a normal balanced diet or a low-carbohydrate/high-fat diet. Similarly, in an animal-based study, consumption of a protein-rich albumin diet significantly enhanced non-REM sleep (Obál, Kapás, & Krueger, 1998). The most restorative sleep is thought to occur during the deep, REM stage (Bonnet, 1986). Another study of individuals with no previous history of sleep disorders found that those who consumed a high-carbohydrate, low-fat diet spent less time in slow-wave (non-REM) sleep than those who consumed either a control balanced diet or a low-carbohydrate, high-fat diet (Husain et al., 2004).

Using a 7-day sleep diary and a subjective sleep quality index, changes in sleep were measured as part of a double-blind, placebo-controlled study of 49 male and female participants. A protein source food bar of tryptophan with carbohydrate was most effective in significantly reducing awake time during the night (Hudson, Hudson, Hecht, & MacKenzie, 2005). In comparison, dietary carbohydrate consumption significantly affected sleep onset in comparison with dietary intakes from a control diet (Krauchi, Cajochen, Werth, & Wirz-Justice, 2002).

A study of 12 healthy young men showed that when comparing very high (90.4%) carbohydrate meals with high- and low-glycemic indices, consumption of high-glycemic index meals consumed 4 hr before bedtime resulted in shorter sleep latencies (Afaghi, O'Connor, & Chow, 2007). Polysomnographic measures were used to measure the sleep status of the study participants. A significant reduction in the mean sleep-onset latencies of healthy sleepers was observed when a high-glycemic index diet was consumed in comparison with a low-glycemic index meal 4 hr before bedtime. Thus, the investigators felt that the glycemic index of high-carbohydrate foods eaten by study participants may be a factor in sleep latency results. In another study of 14 healthy men by these same scientists (Afaghi, O'Connor, & Chow, 2008), very low-carbohydrate meals were found to significantly decrease sleep latency. Thus, their results contradicted their previous findings showing that high-carbohydrate meals resulted in a significant decrease in sleep latency. The study authors also noted that the higher fat intake levels associated with the low-carbohydrate intakes may have been a factor in the sleepiness of the study participants.

Other research has shown that one of the physiological responses to a meal is the thermic effect of food (TEF): a rise in body temperature after food intake (Driver, Shulman, Baker, & Buffenstein, 1999). TEF occurs because the digestion and absorption of dietary nutrients incur energy expenditure that is released as heat (Tappy, 1996). A lower energy diet is related to a lower TEF (Zammit, Ackerman, Shindledecker, Fauci, & Smith, 1992). Meals inducing postprandial sleepiness may act through the production of a high TEF and subsequent heat loss. As a result, the rate of heat loss may be a good predictor of sleep latency (Krauchi et al., 2002). Driver et al. (1999) found that high-energy food intakes appeared to be related to a long-lasting TEF. Consequently, these factors influenced sleep for up to 2 hr after meal consumption.

Previous studies have also showed improvements in sleep following consumption of a high-protein diet, especially a diet rich in tryptophan (Markus et al., 2005). However, Landström, Knutsson, and Lennernäs (2000) did not discover a significant relationship between diet and the onset of drowsiness. Another study measured sleep duration in 240 adolescents using food recall and wrist-actigraphy devices (Weiss et al., 2010). Adolescents who slept an average of 8 or more hours on weekdays consumed a larger proportion of their calories from high-fat foods than those who slept less. In contrast, a large cross-sectional study from China showed a positive association between decreased sleep duration and increased fat intake (Shi, McEvoy, Luu, & Attia, 2008). Using a national health and nutrition survey, a sample of 2,828 adults in China was studied. Those who slept for less than 7 hr a day consumed a significantly higher ( $p = .005$ ) percentage of their calories from fat than those sleeping more than 7 hr per day. In addition, a study by Rontoyanni, Baic, and Cooper (2007) of 30 healthy Greek women also found a weak, positive relationship between sleeping less and consumption of dietary fat intake. This observational cross-sectional study used a Sleep Habits Questionnaire, a 7-day sleep diary, and two 24-hr dietary recall interviews for measurement.

Finally, foods and beverages containing central nervous system stimulants and depressants, such as caffeine and alcohol, are often discouraged because of their adverse effects on quantity and quality of sleep. Although alcohol is often used to “self-medicate” and to shorten sleep latency, research also indicates that it has been found to affect the quantity of slow-wave sleep and has also affected REM sleep (Danel, Libersa, & Touitou, 2001; Feige et al., 2006). Caffeine, found in beverages and foods such as tea, coffee, or chocolate, is a neurologic stimulant, which has been found to lower the need to sleep and produces sleep disruption when taken just prior to going to bed (Nehlig, Daval, & Debry, 1992).

In summary, although good nutrition intake is prescribed for a variety of health conditions, the literature shows that the potential connection between dietary intake and sleep has not been translated into a sufficient number of clinical studies given the mixed results (Michalsen et al., 2003). Especially noteworthy is the limited number of studies measuring nutrient intakes through the use of weighed food intakes rather than dietary recall methods.

## Purpose

As a result, the purpose of this study was to test the effects of weighed macronutrient food intakes on sleep. The specific aims for this study were as follows: (a) determine actigraph sleep/activity measures of participants receiving a nonmanipulated (control) diet, a high-protein diet, a high-fat diet, or a high-carbohydrate diet; (b) analyze for differences in sleep/activity scores for the groups of participants receiving a nonmanipulated diet, a high-protein diet, a high-fat diet, or a high-carbohydrate diet; and (c) examine relationships among participants' sleep scores and dietary intakes.

Sleep variables were determined by the following measures: sleep efficiency (the amount of time asleep divided by the amount of time spent in bed), sleep latency (the time between going to bed and falling asleep), and wake episodes (the number of awakenings between falling asleep and rising). A macronutrient is referred to an essential nutrient—either

protein, fat, or carbohydrate—that is consumed in sufficient quantities to provide energy for the body (Ambulatory Monitoring, Inc).

## Method

### Design

Using a repeated-measures, counterbalanced, crossover study design, participants served as their own control for each of four dietary treatment sessions that included a high-protein, high-carbohydrate, high-fat, and a control diet. The session order was counterbalanced across dietary groups. A double-blind intervention was used so neither the participant nor the researchers knew when the participant was receiving control or treatment diets.

### Setting and Sample

The sample was comprised of 44 healthy adults recruited through a midwestern university. A power analysis, calculated with Borenstein and Cohen's methodology, determined the required sample size (Borenstein, Rothstein, & Cohen, 2001), based on multiple analysis of variance statistics. The possible range of effects of dietary interventions on sleep was based on previous dietary studies conducted by these investigators with a similar population. The effect size for the power analysis was estimated to be "medium." The power for this sample was set at 0.80, with a confidence level,  $\alpha = .05$ . Therefore, a minimum of 35 participants was estimated for each treatment group to achieve statistical power. Additional participants were entered into the study to allow for attrition. Thus, 44 participants completed the study.

Inclusion criteria for the study were as follows: (a) being between the ages of 18 and 50 years and (b) having an ability to read, understand, and speak English. Exclusion criteria for the study included (a) a self-reported diagnosis of diabetes or pregnancy, (b) reported sleep problems, and (c) taking prescription/over-the-counter medications other than Aspirin or non-Codeine Tylenol. Diabetes and pregnancy were exclusionary because of the special dietary requirements necessary for prenatal or diabetic conditions. Illicit drug use and alcohol use were forbidden during the study. The participants' academic program has a strict policy against illicit drug use as well as a random drug testing program is in effect to deter illicit drug use.

During the first 2 weeks of university classes, participants who met sample selection criteria were invited to participate in this study by the researchers. The purpose and details of the study were explained to potential study participants. Participants' questions were answered by the investigators. Individuals that signed consent forms and met the inclusion criteria were randomly selected for participation in the study by drawing names from a container. Study participants were considered to be free-living participants because they were allowed to attend classes and work in the community, although all meals for the study were to be eaten in the study's dining room. Ethical considerations for participants in the study were reviewed and approved by the University and the U.S. Army Human Research Protection Office.

## Measures

Measurements for this study were accomplished through use of the following study measures: demographic questions, weighed food intakes, sleep actigraph measures, the Pittsburgh Sleep Quality Index (PSQI), for its seven components (Buysee, Reynolds, Monk, Berman, & Kupfer, 1989), and biochemical laboratory tests.

**Demographics**—Participants completed a questionnaire that included place of residence, age, education, marital or social living status, employment status, and ethnic identification.

**Anthropometric measurements**—Each participant was weighed twice per visit using a Cardinal-Detecto balance beam scale, and averages of the two measures were recorded. Weights were measured at the beginning of each study week when the participant received a sleep watch and when they turned in the sleep watch for evaluation at the end of each 4-day treatment session. Height was measured on the first visit using an Accustat wall-mounted height board. Body mass indices (BMIs) were calculated as the weight divided by the height squared based on the weight measured on admission to the study. Weight assessments were recorded to establish whether the participant was considered underweight or overweight at the beginning of the study, and serial weights were completed to monitor weight gains or losses during the four treatment sessions.

**Sleep and activity measures**—The PSQI is a self-report questionnaire. The questionnaire is used to assess sleep quality of participants for the month prior to completing the sleep inventory. The 19-item questionnaire measured important descriptive sleep characteristics. These characteristics were then used to produce seven-component scores (Buysee et al., 1989). The components were summed into a global PSQI score using a Likert-type scale of 0 to 5 (0 = *good quality sleep*, 5 = *poor quality sleep*). Internal consistency of the PSQI score was reported as  $r = .83$  and test-retest reliability (4 weeks) was  $r = .85$ .

Sleep was measured using the *Motionlogger* Actigraph (Ambulatory Monitoring, Inc.), a small portable wristwatch type of device that digitally records integrated measures of gross motor activity. A reliability coefficient of .92 and a validity of .99 were established for the *Motionlogger* model (Tryon, 2005). The actigraph computer interface used ACT Millennium Graphs Software Version 2K3.0 to analyze data. Physical activity was recorded as activity counts on the actigraph. Sleep measures included the following variables: sleep efficiency (the amount of time asleep divided by the amount of time spent in bed), sleep latency (the time between going to bed and falling asleep), and wake episodes (the number of awakenings between falling asleep and rising). The participants wore their actigraph watch continuously throughout each 4-day treatment session. The actigraphs were approved for showering, although participants could remove the watch just to wash their wrist. The actigraph recorded sleep and wake patterns, bed and rise times, sleep efficiency, sleep and wake bouts, mean physical activity scores, nap analysis, sleep latency, wake episodes, and other activity patterns.

**Health assessment data**—A health status assessment modified from Doenges' Health Assessment Checklist was collected and recorded for each participant (Doenges, Geissler, & Moorhouse, 1989). The checklist consisted of nine factors ascertained during a medical history assessment of the participants, including history of chronic systemic disease such as respiratory, cardiac, gastrointestinal, genitourinary, neurological, musculoskeletal, metabolic/endocrine, audiovisual, and integumentary system disorders. Depending on the severity of these disorders, the condition was considered an exclusionary factor for participating in this study to include pregnancy and ingestion of oral contraceptives.

**Biochemical and laboratory measures**—The laboratory data for each participant included serum glucose and serum cholesterol. Serum cholesterol was analyzed to monitor for serum lipids due to the effects of the high-fat dietary treatment meals. Blood glucose testing was completed before the study to assess for potential hypoglycemic episodes or glucose intolerance and, after the 4-day dietary treatments, to assess for possible glucose intolerance due to the high-carbohydrate intakes. Subsequent high-glucose test results were verified by a 1-hr postprandial glucose test. An abnormal postprandial glucose test would result in referral to the university student health services for medical care as well as being excluded from the study. However, no participants needed to be excluded from the study for this reason. To ensure validity, licensed personnel supervised collection of all lab tests.

**Nutrition analysis**—The Food Processor Nutrition Analysis Program, a software program used to analyze nutrient intakes for research or clinical applications and to present the data in a variety of formats, was used to analyze the nutrient data for this study (ESHA Research, 2002). Although the Food Processor Program analyzed for more nutrients, the nutrients selected for assessment and analysis for this study were based on study objectives focusing on macronutrients. Kilocalories and content of protein, carbohydrate, and fat nutrients were the primary dietary variables analyzed for relationships to sleep efficiency, sleep latency, wake episodes, and physical activity. Amounts of caffeine, resting metabolic rates, kilocalories consumed, and anthropometric and laboratory test variables were also analyzed for potential effects on sleep variables (Smith, Kendrick, & Maben, 1992).

## Intervention

**Dietary treatments**—In this design, participants served as their own control and were randomly rotated through each of three dietary treatments: a high-protein diet (56% protein, 22% carbohydrate, and 22% fat), a high-carbohydrate diet (56% carbohydrate, 22% protein, and 22% fat), a high-fat diet (56% fat, 22% carbohydrate, and 22% protein), and a control diet (50% carbohydrate, 35% fat, and 15% protein). The macronutrient percentage for the control diet was based on percentages of macronutrients commonly used in other nutrition intervention studies (Blumenthal et al., 2010; McDowell et al., 1994). The dietary percentage protocols resulted in two nutrients being controlled for dietary treatment comparisons for all of the diets. (For example, in the high-carbohydrate diet, fat remained constant at 22% for both the high-protein and high-carbohydrate diet comparisons. Similarly, the carbohydrate percentages also remained constant at 22% for the high-fat and high-protein diets.) The four separate diet conditions were selected to test macronutrients

that may affect participants' sleep. Participants were fed diets containing daily kilocalorie levels based on the participant's individual indirect calorimetry measurement.

**Intervention procedures**—Only foods prescribed for the study were to be eaten by the study participants. Preparation of foods for the study was completed under the guidance and in consultation with a research dietitian using standardized recipes and exact portion sizes. Meals prepared for the dietary interventions were served by a research team member directly to each participant. The study dietitian confirmed that each meal was prepared properly before administering the meal to the participants. A double-blind intervention plan resulted in neither the participant nor the researchers knowing when the participant was receiving the control diet or the treatment diets. Food consumption was controlled both in terms of what was consumed and how much was consumed. Each participant was given preweighed meals that were required to be eaten in the study dining room. Meal consumption was monitored by the dietitian and the study staff. Food intakes were weighed and recorded before and after eating by the research staff. Weighing food intakes is considered the most accurate method for measuring human food consumption (Gibson, 1990). After the final meal was eaten in the dining room each day, participants signed a food/beverage intake sheet to verify that no foods or beverages from outside of the study were consumed. Space was provided on the sheet to allow participants to record any foods, beverages, or other substances that may have been inadvertently consumed outside of the study within the previous 24-hr period. If a participant was still hungry, they were given additional portions of the foods in the same exact macronutrient percentages required by study protocols.

Beverages were also issued to the participants as part of the study meals. Beverages planned for consumption included (a) beverages, such as water or noncaloric drinks with controlled caffeine, that were not offset by the percentages of fat, protein, or carbohydrates for the meal and (b) beverages that contributed to the fat, protein, and carbohydrate percentages for the meal and snack (this included milk for participants not indicating lactose intolerance). Amounts of caffeine were carefully controlled for in the study because of the potential effects on sleep. Individuals with a history of moderate caffeine consumption were allowed caffeinated drinks with a limit of <200 mg/day. A preliminary food habit questionnaire was given to all selected participants to be sure that they would be willing to consume the prescribed diets/beverages. Anyone who objected to the prescribed foods was advised they should not participate. Participants were given snacks and beverages in insulated lunch bags to take home for consumption between the evening meal and bedtime. Any uneaten foods or beverages along with the respective containers were returned for weighing when the participant came back for the next meal.

### Data Collection

An initial pilot study was conducted 4 months prior to the full study and was used to provide an evaluation and justification for inclusion of planned instrumentation and variables within this study. The psychometric properties of the instruments in this study had been tested by the original authors or researchers as indicated.

A week prior to starting study treatments, a mutually agreed-on time was set for the nurse researcher to meet with the consenting participants. At this meeting, the researcher completed health assessments and anthropometric measurements. Indirect calorimetry values were also obtained. Each study participant also completed a demographic questionnaire. Directions for the dietary treatment sessions, laboratory testing, and wearing the Actiwatch sleep watches were given. Study participants were instructed on the importance of consuming only the food and beverages as prescribed by the study.

To ensure that order of treatment was not a concern for the interpretation of findings, any potential effects were mitigated in two ways: (a) allowing 2 weeks of “washout” time to lapse between treatments and (b) randomly assigning order of treatment to the participants. Participants were randomly assigned to dietary treatment groups according to two sets of 24 possible treatment orders. The order of treatment and control diets was drawn from a container for random assignment without replacement for 44 participants. The planned treatment order for each participant was recorded and treatments were implemented accordingly. Dietary treatment sessions were scheduled for a Monday through Thursday time period with a 2-week washout period between the study sessions. In the literature, 2-week washout periods are commonly used to eliminate carryover effects between dietary interventions (Carson, Burke, & Hark, 2004; Driskell, 2007; Howarth, Petrisko, Furchner-Evanson, Nemoseck, & Kern, 2010). During the weeks of the dietary treatments, participants were given directions for using the actigraph. The importance of strict compliance in participating with the dietary and control group treatments was stressed. Participants were not allowed to consume any foods or beverages that were not provided by the study. Meal times were set according to a desirable and “typical” schedule for each participant. Participants were carefully observed to be certain that their meals were received and eaten at the appropriate times. Food intakes were carefully weighed and recorded (within .05 oz accuracy). The participants completed questionnaires daily to confirm that they had not consumed foods or beverages that were not issued by the study.

The PSQI, a self-rating questionnaire, was administered to participants as they began their dietary treatments. Each participant was given an actigraph on the first day of treatment to wear continuously during the dietary treatment sessions. The actigraph was worn continuously for the four dietary treatment days and returned after the last planned meal was eaten on the fourth treatment day. This provided data on the participants’ sleep experienced during the study treatments.

On the fourth day of receiving a dietary treatment, the participants returned their sleep watches to the investigators. The record of weighed food intakes for each participant was compared with their sleep assessment results that were gathered on the 4th day of each dietary treatment regimen. Anthropometric measures and laboratory tests were also completed by the nurse researcher at this time. Participants received a small compensation of US\$25 for completion of each of the observational interviews. Including baseline measurements, a total stipend of US\$125 was paid as compensation for each participant’s time in the study, inconvenience, and as an expression of appreciation for contributing to this body of knowledge.



## Analysis

Study data were analyzed using the Statistical Package for Social Science (SPSS). The SPSS Explore Procedure was used to screen data, visually examine distributions of group values, and test for normality and homogeneity of variance. Response frequencies were tabulated for measures of dietary intake, demographics, and sleep/activity levels. The data were analyzed by applicable descriptive and inferential statistics; correlations among key variables were also analyzed. Missing data were handled by replacing the missing value with the mean score calculated from the other participants' data for that particular variable. Sleep efficiency, sleep latency, wake episodes, and activity scale scores were calculated for the participants after receiving the non-manipulated (control) diet and the high-carbohydrate, high-protein, and high-fat diets. Significant differences among the sleep/activity scores for participants in the four dietary treatment groups were analyzed by using a RANOVA. Tukey's post hoc tests were calculated when the overall omnibus effect from the RANOVA was statistically significant. The post hoc testing was used to compare specific diet conditions with each other between the significantly different groups. An alpha level of .05 was the criterion for significance.

## Results

### Sample Demographics

Of the 44 participants, the study sample included 39 European American participants, 2 Asian participants, 2 Hispanic participants, and 1 African American participant. Participant ages ranged from 19 to 22 years with a median age of 20.6 years. The median years of education were 14 years, with a range from 13 to 16 years. The median BMI was 24.8 (range = 21.0–28.0). Other variable means and standard deviations are listed in Table 1.

### Sleep and Activity in Dietary Groups

Mean sleep and activity scores were compared among the high-fat, high-protein, high-carbohydrate, and control diet groups. The sleep scores were based on actigraph measures of sleep efficiency, sleep latency, wake episodes, and actigraph activity scores. Table 2 illustrates the intraparticipant analysis using RANOVA to detect differences in a single participant's sleep and activity scores with different dietary intakes. Statistically significant differences were based on the mean sleep and activity scores compared among the different dietary groups. The wake episode scores were significantly different,  $F = 3.6$ ;  $df = 3, 44$ ;  $p = .02$ , when comparing the control group with the fat, protein, and carbohydrate groups. Also, the sleep latency scores were significantly different among control and the other dietary groups,  $F = 5.3$ ;  $df = 3, 44$ ;  $p = .004$ . Differences among the dietary groups were further analyzed using a post hoc test to detect significant differences between specific groups. Post hoc testing indicated that the wake episodes were significantly lower ( $p = .03$ ) for the protein diet as compared with the control diet. Also the sleep latency scores were significantly lower ( $p = .01$ ) for the carbohydrate diet as compared with the control diet.

Other significant correlations were found between sleep efficiency and the following: basal metabolic kilocalories per day ( $r = .26$ ,  $p = .04$ ), random glucose ( $r = -.45$ ,  $p = .001$ ), and total cholesterol ( $r = -.60$ ,  $p < .001$ ).

## Discussion

This study investigated the effects of manipulated macronutrient intakes on sleep and activity variables. Results indicated significant differences in sleep latency and wake episodes when comparing participants' composition of dietary macronutrients. For example, participants who fed on high-carbohydrate diets had significantly shorter ( $p = .004$ ) mean sleep latencies in comparison with those who fed on control diets. However, these results contradict the work from Afaghi et al.'s. (2008) study, indicating that low-carbohydrate meals decreased sleep latency. However, the results are consistent with results of another study that compared the effects of carbohydrate-rich meals with control meals on sleep and body thermoregulation (Krauchi et al., 2002).

Also, the results of our study indicated that consuming a high-protein diet produced significantly fewer ( $p < .03$ ) wake episodes in comparison with consuming a control diet. Some related studies have shown that low-protein diets alone did not significantly affect sleep, although other studies showed that tryptophan depletion with subsequent brain serotonin reductions correlated with more wake periods and greater wake percentages than controls (Voderholzer et al., 1998). Our findings were similar to those studies that showed sleep improvements with high-protein diets, especially diets rich in tryptophan (Markus et al., 2005).

However, our results contrast with the findings of Landström et al. (2000), who did not discover a significant relationship between diet and the onset of drowsiness. The Landström et al. study relied heavily on subjective ratings of the levels of fatigue. In contrast, our study applied actigraphy for more objective measurements of activity and sleep states. Sleep loss has an adverse effect on the body's ability to use glucose. For example, in 1 week of severe sleep deprivation (about 4 hr per night), a healthy, lean, fit individual can be in a prediabetic state (Van Cauter et al., 2007). Consistent with this, our study showed that lower sleep efficiency was associated with high-glucose and high-serum cholesterol levels. In particular, sleep efficiency correlated significantly ( $p < .05$ ) with random glucose, total cholesterol, and resting metabolic kilocalories per day. In a related study, serum lipids and total cholesterol were significantly related ( $p < .001$ ) to the arousal frequency (number of sleep arousals per hour; Ekstedt, Akerstedt, & Soderstrom, 2004).

In addition, our study indicated that higher caloric intake resulted in better sleep efficiency. This result supports the work of Driver et al. (1999) concerning the TEF. Contrary to our study, Zammit et al. (1992) concluded that there were no significant differences in sleep latency noted in participants that consumed high- or low-caloric carbohydrate meals.

In summary, the results of this study show that attention to nutritional intake could be a key to better sleep. For example, consuming a high-protein diet significantly reduced wake episodes compared with the control diet. Also, a high-carbohydrate diet significantly reduced sleep latency as compared with the control diet. In addition, glucose and serum cholesterol levels were negatively correlated, and kilocalorie intake was positively correlated with sleep efficiency. These results provide evidence that specific macronutrient diets may influence a person's sleep quality. However, our results did not support any relationships

between sleep efficiency or mean activity and macronutrient diet composition. The participants spent the same percentage of time sleeping once they fell asleep and maintained the same level of physical activity regardless of the diet they consumed.

Finally, limitations of this study include homogeneity of the study sample, generalizability of the results, and the fact that actigraphic monitoring was used rather than polysomnographic testing. Another shortcoming includes the fact that although actigraph measurements of sleep are accurate for field research (nonlaboratory research results), they do not achieve the accuracy that could be achieved using polysomnographic testing. Therefore, for a more accurate assessment of sleep quality, we recommend that future studies using polysomnography be conducted with laboratory participants while testing macronutrient diets.

## Acknowledgments

This manuscript is written in memory of Dr. Marcia Gragert, who provided invaluable expertise to this work.

### Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was supported by the Peer Reviewed Medical Research Program of the U.S. Army Biomedical Research and Materiel Command (grant number DAMDIT-03-1-0010) and the National Institutes of Health (grant number 1C06RR022088).

## References

- Afaghi A, O'Connor H, Chow C. High-glycemic-index carbohydrate meals shorten sleep onset. *American Journal of Clinical Nutrition*. 2007; 85:426–430. [PubMed: 17284739]
- Afaghi A, O'Connor H, Chow C. Acute effects of the very low carbohydrate diet on sleep indices. *Nutritional Neuroscience*. 2008; 11:146–154. [PubMed: 18681982]
- Blumenthal JA, Babyak MA, Hinderliter A, Watkins LL, Craighead L, Lin PH, Sherwood A. Effects of the DASH diet alone and in combination with exercise and weight loss on blood pressure and cardiovascular biomarkers in men and women with high blood pressure: The ENCORE study. *Archives of Internal Medicine*. 2010; 170:126–135. [PubMed: 20101007]
- Bonnet MH. Performance and sleepiness following moderate sleep disruption and slow wave sleep deprivation. *Physiology & Behavior*. 1986; 37:915–918. [PubMed: 3786485]
- Borenstein, M., Rothstein, H., Cohen, J. Power and precision (Version 2.1) [Computer software]. Englewood, NJ: Biostat; 2001.
- Buysee DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research*. 1989; 28:193–213. [PubMed: 2748771]
- Carson, JAS., Burke, FM., Hark, L. Cardiovascular nutrition: Disease management and prevention. Chicago, IL: American Dietetic Association; 2004.
- Centers for Disease Control and Prevention. Effect of short sleep duration on daily activities—United States, 2005–2008. *Morbidity and Mortality Weekly Report*. 2011; 60:239–242. [PubMed: 21368739]
- Colten, HR., Altevogt, BM., editors. Sleep disorders and sleep deprivation: An unmet public health problem. Washington, DC: National Academies Press; 2006.
- Danel T, Libersa C, Touitou Y. The effect of alcohol consumption on the circadian control of human core body temperature is time dependent. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology*. 2001; 281:52–55.
- Doenges, ME., Geissler, AC., Moorhouse, MF. Nursing care plans: Guidelines for planning patient care. Philadelphia, PA: F. A. Davis; 1989.

- Driskell, JA. Sports nutrition: Fats and proteins. Boca Raton, FL: Taylor & Francis; 2007.
- Driver HS, Shulman I, Baker FC, Buffenstein R. Energy content of the evening meal alters nocturnal body temperature but not sleep. *Physiology & Behavior*. 1999; 68:17–23. [PubMed: 10627057]
- Ekstedt M, Akerstedt T, Soderstrom M. Microarousals during sleep are associated with increased levels of lipids, cortisol, and blood pressure. *Psychosomatic Medicine*. 2004; 66:925–931. [PubMed: 15564359]
- ESHA Research. Food processor [Computer software]. Salem, OR: Author; 2002.
- Feige B, Gann H, Brueck R, Hornyak M, Litsch S, Hohagen F, Riemann D. Effects of alcohol on polysomnographically recorded sleep in healthy subjects. *Alcoholism: Clinical and Experimental Research*. 2006; 30:1527–1537.
- Gibson, R. Principles of nutritional assessment. New York, NY: Oxford; 1990.
- Howarth L, Petrisko Y, Furchner-Evanson A, Nemoseck T, Kern M. Snack selection influences nutrient intake, triglycerides, and bowel habits of adult women: A pilot study. *Journal of the American Dietetic Association*. 2010; 110:1322–1327. [PubMed: 20800123]
- Hudson C, Hudson SP, Hecht T, MacKenzie J. Protein source tryptophan versus pharmaceutical grade tryptophan as an efficacious treatment for chronic insomnia. *Nutritional Neuroscience*. 2005; 8:121–127. [PubMed: 16053244]
- Husain AM, Yancy WS Jr, Carwile ST, Miller PP, Westman EC. Diet therapy for narcolepsy. *Neurology*. 2004; 62:2300–2302. [PubMed: 15210901]
- Krauchi K, Cajochen C, Werth E, Wirz-Justice A. Alteration of internal circadian phase relationships after morning versus evening carbohydrate-rich meals in humans. *Journal of Biological Rhythms*. 2002; 17:364–376. [PubMed: 12164252]
- Landström U, Knutsson A, Lennernäs M. Field studies on the effects of food content on wakefulness. *Nutrition and Health*. 2000; 14:195–204. [PubMed: 11142608]
- Mahowald, MW. Disorders of sleep. In: Goldman, L., Ausiello, D., editors. *Cecil medicine*. 23. Philadelphia, PA: Saunders Elsevier; 2007. p. 2696-2701.
- Markus CR, Jonkman LM, Lammers JH, Deutz NE, Messer MH, Rigtering N. Evening intake of alpha-lactalbumin increases plasma tryptophan availability and improves morning alertness and brain measures of attention. *American Journal of Clinical Nutrition*. 2005; 81:1026–1033. [PubMed: 15883425]
- McDowell MA, Briefel RR, Alaimo K, Bischof AM, Caughman CR, Carroll MD, Johnson CL. Energy and macronutrient intakes of persons ages 2 months and over in the United States: Third national health and nutrition examination survey, Phase 1, 1988–91. *Advance Data*. 1994; 24:1–24.
- Michalsen A, Schlegel F, Rodenbeck A, Ludtke R, Huether G, Teschler H, Dobos GJ. Effects of short-term modified fasting on sleep patterns and daytime vigilance in non-obese participants: Results of a pilot study. *Annals of Nutrition and Metabolism*. 2003; 47:194–200. [PubMed: 12748412]
- National Sleep Foundation. 2009 sleep in America poll. 2009. Retrieved from <http://www.sleepfoundation.org/sites/default/files/2009%20Sleep%20in%20America%20SOF%20EMBARGOED.pdf>
- Nehlig A, Daval JL, Debry G. Caffeine and the central nervous system: Mechanisms of action, biochemical, metabolic and psychostimulant effects. *Brain Research, Brain Research Reviews*. 1992; 17:139–170. [PubMed: 1356551]
- Obál F, Kapás L, Krueger JM. Albumin enhances sleep in the young rat. *Physiology & Behavior*. 1998; 64:261–266. [PubMed: 9748091]
- Phillips F, Crisp AH, McGuinness B, Kalucy EC, Chen CN, Koval J, Lacey JH. Isocaloric diet changes and electroencephalographic sleep. *Lancet*. 1975; 2:723–725. [PubMed: 52766]
- Rontoyanni VG, Baic S, Cooper AR. Association between nocturnal sleep duration, body fatness, and dietary intake in Greek women. *Nutrition*. 2007; 23:773–777. [PubMed: 17884345]
- Shi Z, McEvoy M, Luu J, Attia J. Dietary fat and sleep duration in Chinese men and women. *International Journal of Obesity*. 2008; 32:1835–1840. [PubMed: 18982012]
- Smith AP, Kendrick AM, Maben AL. Effects of breakfast and caffeine on performance and mood in the late morning and after lunch. *Neuropsychobiology*. 1992; 26:198–204. [PubMed: 1299795]

- Szentirmai E, Kapás L, Sun Y, Smith RG, Krueger JM. Restricted feeding induced-sleep, activity, and body temperature changes in normal and preproghrelin-deficient mice. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology*. 2010; 298:467–477.
- Tappy L. Thermic effect of food and sympathetic nervous system activity in humans. *Reproduction Nutrition Development*. 1996; 36:391–397.
- Tryon W. The reliability and validity of two ambulatory monitoring actigraphs. *Behavior Research Methods*. 2005; 37:492–497. [PubMed: 16405145]
- Van Cauter E, Holmback U, Knutson K, Leproult R, Miller A, Nedeltcheva A, Spiegel K. Impact of sleep and sleep loss on neuroendocrine and metabolic function. *Hormone Research*. 2007; 67(Suppl 1):2–9. [PubMed: 17308390]
- Voderholzer U, Hornyak M, Thiel B, Huwig-Poppe C, Keimen A, König A, Hohagen F. Impact of experimentally induced serotonin deficiency by tryptophan depletion on sleep EEG in healthy participants. *Neuropsychopharmacology*. 1998; 18:112–124. [PubMed: 9430135]
- Weiss A, Xu F, Storfer-Isser A, Thomas A, Levers-Landis CE, Redline S. The association of sleep duration with adolescents' fat and carbohydrate consumption. *Sleep*. 2010; 33:1201–1209. [PubMed: 20857867]
- Zammit GK, Ackerman SH, Shindledecker R, Fauci M, Smith GP. Postprandial sleep and thermogenesis in normal men. *Physiology & Behavior*. 1992; 52:251–259. [PubMed: 1523250]

**Table 1**Baseline Means and Standard Deviations for Demographics, Health Status, and Sleep Data ( $N = 44$ ).

<b>Variable</b>	<b><i>M</i></b>	<b><i>SD</i></b>
Demographics of the sample		
Age (years)	20.6	2.0
Education (years)	13.6	0.98
Health status of the participants		
BMI	24.8	3.5
Sleep and activity data		
Sleep index (scores)	91.1	11.0
PSQI (scores)	4.1	1.9
Sleep efficiency (percentages)	92.2	4.3
Wake episodes	14.8	7.0
Sleep latency (minutes)	11.7	11.9
Activity mean (counts)	268.5	177.4

Note: BMI = body mass index; PSQI = Pittsburgh Sleep Quality Index.

**Table 2**

A Repeated-Measures ANOVA of Sleep and Activity Scores According to Dietary Intakes of Participants ( $N=44$ ).

	<b>M</b>	<b>SD</b>	<b>F</b>	<b>p</b>
Physical activity			0.01	<i>ns</i>
High-fat diet	270.3	170.2		
High-protein diet	271.5	194.7		
High-carbohydrate diet	268.2	184.4		
Control diet	274.8	180.8		
Wake episodes			3.6	.02
High-fat diet	14.5	6.9		
High-protein diet <sup>a</sup>	13.5	7.2		
High-carbohydrate diet	14.1	7.3		
Control diet	16.7	6.4		
Sleep efficiency			1.6	<i>ns</i>
High-fat diet	92.2	4.2		
High-protein diet	92.9	4.1		
High-carbohydrate diet	92.5	4.4		
Control diet	91.3	4.8		
Sleep latency			5.3	.004
High-fat diet	11.3	12.7		
High-protein diet	12.8	15.1		
High-carbohydrate diet <sup>b</sup>	9.1	7.6		
Control diet	13.9	11.7		

Note: Post hoc test results are as follows:

<sup>a</sup>Protein diet score was significantly ( $p = .03$ ) lower than the control diet score.

<sup>b</sup>Carbohydrate diet score was significantly ( $p < .01$ ) lower than the control diet score. ( $df = 3$ ),  $p < .05$ .