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Dietary Macronutrients and Sleep

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Abstract

This study examined the effects of macronutrient diets on sleep quantity and quality. Using a repeated-measures, randomized crossover study design, 36 young adults served as their own control, and consumed high protein, carbohydrate, fat, and control diets. Treatment orders were counterbalanced across the dietary groups. Following consumption of the study diets, sleep measures were examined for within-subject differences. Fatty acid intakes and serum lipids were further analyzed for differences. Sleep actigraphs indicated wake times and wake minutes (after sleep onset) were significantly different when comparing consumption of macronutrient diets and a control diet. Post hoc testing indicated high carbohydrate intakes were associated with significantly shorter ($p < .001$) wake times. Also, the Global Pittsburgh Sleep Quality Index© post hoc results indicated high fat intake was associated with significantly better ($p < .05$) sleep in comparison with the other diets. These results highlight the effects that dietary manipulations may have on sleep.

Keywords

proteins; carbohydrates; fats; sleep; actigraphs

Over two thirds of adults get less than 8 hr of sleep during a typical night; 20% of those individuals get less than 6 hr of sleep (National Sleep Foundation, 2009). Some investigators believe that dietary modifications might lead to better quality sleep (Grandner, Jackson, Gerstner, & Knutson, 2014; Shi, McEvoy, Luu, & Attia, 2008). Although some studies have suggested that the macronutrient (the body's fuel and major building blocks: carbohydrates, fats, and proteins [Molnar, 2006]) content of food intakes may affect both the quality and quantity of sleep (Crispim et al., 2011; Markus et al., 2005; Nehme et al., 2014), other studies have produced mixed results (Adam & Oswald, 1979; Landström, Knutsson, & Lennernäs, 2000; Santana et al., 2012). Therefore, this study focused on the effects of specific macronutrients on sleep.

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Declaration of Conflicting Interests

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Fat Intake and Sleep

The literature on fat intake and sleep relationships can vary, depending on how sleep is measured. Therefore, study results have been inconsistent. Some studies show sleep quality was improved (Irmisch, Schläfke, Gierow, Herpertz, & Richter, 2007; Santana et al., 2012) when individuals consumed a high fat diet, whereas another study indicated no relationship (Landström et al., 2000). For example, in a study of the impact of fatty acids on sleep in 118 hospitalized male and female participants diagnosed with depression, significant negative correlations resulted among sleep disturbances and fatty acid intakes (Irmisch et al., 2007). Santana et al. (2012) studied 58 obese elderly patients (60–80 years of age) of both genders and found that a preference for high energy, dense foods resulted in shorter sleep duration (Santana et al., 2012).

In other studies, more dietary fat hindered sleep quality and/or sleep onset. Crispim and colleagues (2011) studied the relationship among dietary intakes and sleep patterns of 52 healthy male and female participants. Nocturnal polysomnography measured sleep, and 3-day food diaries were used to measure food intake. Sleep was negatively affected when participants consumed food near or during the nocturnal period (Crispim et al., 2011). The relationships among sleep duration, food intake, and metabolic balance of 2,828 adults were also studied in a 2002 national survey of nutrition and health in China (Shi et al., 2008). The authors found that increased fat and carbohydrate consumption correlated positively with shorter sleep duration.

Landström et al. (2000) studied 12 day-and-night drivers who consumed high and low fat foods, but found no significant differences in wakefulness when comparing the two diets (Landström et al., 2000). In another study, the difficulty of falling asleep or staying asleep was associated with consuming fatty acids (Grandner et al., 2014). Relationships of dietary intakes and sleep symptoms were examined using the 2007–2008 National Health and Nutrition Examination Survey results from 4,552 individuals. Results indicated that a cholesterol-rich diet was associated with non-restorative sleep (Grandner et al., 2014). Different studies have shown that both over- and under-consumption of cholesterol negatively affected sleep quality and duration (Gangwisch et al., 2010; Santana et al., 2012). In a study of individuals with depression, the degree of difficulty in falling asleep was thought to be related to consumption of saturated fatty acids that were precursors of palmitoleic acid (Irmisch et al., 2007).

In a study of 395 healthy children from schools in the United Kingdom, supplementation of polyunsaturated fatty acids (docosahexaenoic acid [DHA]) over a 16-week period led to improved sleep quality in gender- and age-specific groups. However, the authors cautiously concluded that DHA supplementation in comparison with placebo consumption may lead to better sleep according to both subjective parental data and objective data gathered by Actigraph measurements (Montgomery, Burton, Sewell, Spreckelsen, & Richardson, 2014).

Protein and Sleep

The essential amino acid, tryptophan, has a very important and direct role in the functioning of brain neurotransmitters (Fernstrom, 2013). Amino acids are the primary building blocks of protein. Two factors help determine the availability of tryptophan to the brain: (a) plasma tryptophan levels and (b) ratios between large neutral amino acids. Changes in these ratios can affect tryptophan availability. Because tryptophan serves as a precursor to serotonin and melatonin, it has a relationship in enhancing sleep in humans. A lack of tryptophan can result in less serotonin and may lead to sleep disturbances (Fernstrom, 2012). Tryptophan levels depend on dietary consumption, especially of protein, because the body is unable to synthesize tryptophan. Consequently, a high protein meal would contribute less tryptophan to the circulating blood in comparison with the other large neutral amino acids, thus reducing brain serotonin levels and resulting in less sleep.

The consumption of foods high in protein or enhanced with tryptophan has also led to improved sleep quality in some studies (Bravo et al., 2013; Markus et al., 2005). Conversely, other researchers (Adam & Oswald, 1979; Voderholzer et al., 1998) found that sleep latency and consumption of diets high in protein did not have a significant relationship. For example, in a repeated-measures study of 35 healthy subjects with some sleep difficulties, consumption of cereals enhanced with a high dose of tryptophan increased sleep efficiency, sleep time, and improved sleep latency in comparison with a low dose tryptophan cereal or a control cereal (Bravo et al., 2013). Sleep was measured using wrist actimeter devices over a 3-week period with diets changed each week.

In another study, 12 healthy volunteers (*M* age = 34 years) who received a tryptophan-depleted diet significantly increased their rapid eye movement sleep (Voderholzer et al., 1998). The participant's sleep was measured with nightly polysonograms in the randomized, double-blinded study. In a study of participants (ages 38–65 years), tryptophan supplements were consumed before bedtime for two nights with an inert placebo given for two other nights (Adam & Oswald, 1979). However, no association was found between tryptophan consumption and the participants' sleep latency times.

Carbohydrates and Sleep

Some studies have indicated that high carbohydrate diets had a positive impact on sleep latencies (Lindseth, Lindseth, & Thompson, 2013; Nehme et al., 2014), whereas other studies showed no significant relationship or a negative relationship between carbohydrate consumption and sleep onset (Afaghi, O'Connor, & Chow, 2007; Phillips et al., 1975). In Lindseth and colleagues' (2013) study, Actigraphs were used to measure the sleep and activity levels of 40 healthy young adults. The researchers found that high carbohydrate intakes led to shorter sleep latencies than a control diet and higher protein intakes resulted in fewer wake episodes. Nehme and colleagues (2014) studied 24 night security guards (*M* age = 30.8 years) for 3 weeks using Actigraph devices and found that complex carbohydrate diets resulted in significantly longer sleep durations that improved the sleep quality of obese workers when compared with other diets (Nehme et al., 2014).

Phillips and colleagues (1975) studied the possible relationship between dietary intakes and sleep in eight young, male participants. The authors used an electroencephalogram to measure brain wave activity during sleep. A significant correlation was found between high carbohydrate/low fat diets and decreased incidence of slow-wave sleep when compared with either a low carbohydrate/high fat diet or a balanced diet (Phillips et al., 1975). Using polysomnographic measures, Afaghi et al. (2007) found that sleep latencies of 12 healthy males were significantly shorter when consuming high glycemic index foods before bedtime in comparison with low glycemic index meals. Sleep quality was found to be diminished after consumption of high glycemic index beverages in a study involving eight children, ranging in age from 8 to 12 years. After three nights, participants who consumed the beverages 1 hr before bedtime had increased night-time arousals, resulting in a negative impact on the quality of sleep (Jalilolghadr, Afaghi, O'Connor, & Chow, 2011).

Study Purpose

Given the inconsistent results of previous studies on macronutrient intakes and sleep, the purpose of this study was to examine the effects of dietary fat, protein, and carbohydrates on sleep. This study also differed from previous studies by further examining diets high in specific fatty acids and related biochemical lab tests (serotonin, serum lipids and glucose levels) to determine possible associations with sleep quantity and quality.

Method

Study Design

This within-subjects, crossover, designed study aimed to determine differences in sleep when each participant consumed four distinct diets: a high protein diet, a high carbohydrate diet, a high fat diet, and a non-manipulated (control) diet. The orders of dietary treatment were written on slips of paper and were randomly selected for each participant by drawing the paper slip from a container. Each treatment session consisted of 4 days (Monday through Thursday) of monitored dietary intakes with 2-week "wash-out" intervals between each of the four treatment sessions. Further analysis of saturated and polyunsaturated fatty acids and low fat intakes were also calculated for their effects on sleep quality and duration. In addition, the effects of serum lipids and serotonin levels were analyzed for the study participants.

For each treatment session, participants were evaluated for sleep with the *Actiwatch* Spectrum® (Royal Philips; Bend, OR) sleep watch and the Pittsburgh Sleep Quality Index® (PSQI; University of Pittsburgh, Pittsburgh, PA; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Throughout the study, both the participant and the nurse who collected the sleep data were blinded to the types of diets consumed during the study's dietary treatment periods.

Population Description and Sample Selection Plan

Study participants were randomly selected from a population of 300 eligible students at a Midwestern university. They were recruited during their third semester of academic coursework at a student orientation session held during the first 2 weeks of classes for that

semester. Course faculty did not engage in the recruitment process to avoid coercion. A full description of the study and the consent forms were presented to potential participants. Questions from students about the study were answered by the researchers. Students were informed that not everyone who consented to participate may meet eligibility requirements and, therefore, would not be selected for the randomization process. Participants were informed that participation was voluntary. They were assured responses would be confidential. Also, there would be no prejudice should they refuse to participate. Study responses would be anonymous because the results would be reported as aggregate data.

The inclusion criteria for study participation included (a) enrollment in their third term at a Midwestern university; (b) consented to participate; (c) age 18 to 30 years; (d) able to read, understand, and speak English; (e) taking no regular medications; and (f) no diagnosed medical condition. Also, participants agreed to only consume study foods and beverages that were prescribed for the weeks of dietary treatments.

The University's institutional review board and the Human Research Protection Office of the U.S. Army Medical Research and Materiel Command (the study's funding agency) both approved the study (#A-16115) protocols. Participants each received a US$25 honorarium for each dietary treatment session they completed. The stipend was an expression of appreciation for their time and scholarly contribution.

Statistical Power

Based on a plan to calculate repeated-measures ANOVAs, a statistical power analysis was calculated with Borenstein, Rothstein and Cohen's "Power & Precision" software program. Results from previously published studies with similar study populations provided a basis for potential effects (Lindseth et al., 2013; Lupien, Gillin, & Hauger, 1999). Sample size calculations were set to attain a statistical power of 0.80, an alpha of .05, and a "medium" effect size of 0.30. Given these parameters, a minimum of 30 participants would need to be selected for our study sample. Factoring in a conservative (20%) rate of attrition, 36 participants who met the study criteria and consented to be in the study were randomly selected to be in the study. All 36 participants who consented to be in the study also completed the study.

Participant Selection, Orientation, and Assessment Schedule

Study participants were randomly selected by drawing names of eligible participants from a container and recording the names in the order they were drawn. Participants who consented to participate met with the investigators within 1 week after randomized selection. During this meeting, baseline health assessments, anthropometric measurements, indirect calorimetry measures, and demographic data were collected. Each participant met with the study dietitian to determine an eating schedule. In addition, food preferences for each participant were recorded onto a food preference checklist to facilitate study compliance and ensure compatibility with the foods served throughout the study. Directions and explanations were given for completing the dietary treatments, laboratory tests, and sleep/fatigue assessments.

After each dietary intervention week, participants were assessed for anthropometric parameters, health status, fatigue, and sleep/wake levels; also, serum lab samples were taken within 2 hr after the last meal for each dietary intervention week. Participants collected urine samples upon rising in the morning. Data were recorded prospectively by the research team. The study instruments had been psychometrically tested by their original authors. All study protocols were pretested at the research site. These data were used to confirm validity and reliability of the study measures.

Dietary Interventions

The treatment diets comprised different proportions of macronutrients (the high fat diet—65% fat, 10% protein, 25% carbohydrate; the high carbohydrate diet—80% carbohydrate, 10% protein, 10% fat; the high protein diet—45% protein, 40% carbohydrate, 15% fat; and the control diet—15% protein, 50% carbohydrate, 35% fat; see Table 1). The composition of the study diets were based on previous, similar dietary intervention studies (Lindseth et al., 2013; Lindseth, Petros, & Helland, forthcoming) and according to advice from a consulting dietitian with extensive experience in conducting intervention studies.

Based on previous study results indicating that saturated and polyunsaturated fats had some effects on sleep (Grandner et al., 2014; Irmisch et al., 2007), a further analysis of the fat content of our study diets was calculated. This analysis resulted in participants meeting the following three fat consumption categories: (a) saturated fat intake (35% Saturated Fatty Acids/day), (b) polyunsaturated fat intake (1.3 mg Polyunsaturated Fatty Acids/day), and (c) low fat intake (10% Fatty Acids/day). The saturated and polyunsaturated fat and low fat intakes were then analyzed for their effects on sleep quality and duration. In addition, serum lipids were analyzed for the study participants.

The study dietitian measured the energy requirements of each participant with indirect calorimetry to ensure the study meals were prepared to meet each participant's respective energy requirements. To prevent unplanned effects that could result from possible micronutrient deficiencies, the micronutrient content of the study meals (within 5% of variance) was consistent with the U.S. Recommended Daily Allowances (Food and Nutrition Board, 1989). Meals were planned by the research study dietitian and principal investigator.

During the treatment periods, study meals were served in a metabolic dining room within a behavioral research center. This provided a controlled research environment for serving study meals. Eating study meals in the metabolic dining room was a study requirement. The study dietitian provided information and directions for compliance with the dietary treatments. Research team members served the meals and confirmed that participants received the proper foods and consumed them in strict compliance with study protocols. Under the supervision of the study dietitian, all foods and beverages were weighed and recorded before and after each meal to determine exact food intakes.

The meals were prepared so participants could not determine which macronutrient diet they were consuming for that study week. For example, different combinations of gravies or sauces were served with a particular meat or vegetable to disguise a "high fat" meal from a "high protein," "high carbohydrate," or "low fat" meal. Participants were told and reminded

that noncompliance with the study diet, by consuming non-study foods or beverages, could result in elimination from the study. Beverages included water, milk, and assorted fruit juices. Caffeinated foods and beverages were limited to 100 mg per day. This assisted in avoiding the confounding effects of caffeine on sleep. Each day, all participants verified in writing and verbally, that only those foods or beverages issued by the study were consumed in the previous 24 hr. Also, to prevent the consumption of outside food by participants, weighed snacks and bottled water were issued during the evening and between the study meals. These beverages and snacks were also weighed before and after consumption to ensure accurate food intake records for each participant.

Sleep Measurements

Sleep was measured for all participants during a baseline assessment and at the end of each treatment session, as follows:

Actigraph sleep watch—The *Actiwatch Spectrum*®, an Actigraph, is a small, portable wristwatch device (Royal Philips; Bend, OR). It digitally recorded the participants' integrated measures of gross motor activity and, using a corresponding computer interface, analyzed the data. In a previous study, use of Actigraphs to measure sleep showed a .92 reliability coefficient and a .99 validity (Tryon, 2005). To determine reliability, polysomnography scores were compared with the *Actiwatch Spectrum*® data, and a sleep–wake reliability coefficient of $K = .81$ was determined (Danker-Hopfe et al., 2009). Participants wore Actigraph watches continuously throughout each of the treatment sessions. For this study, the *Actiwatch Spectrum*® recorded (a) Sleep Efficiency, the amount of time (%) asleep divided by the amount of time spent in bed after "lights off"; (b) Sleep Latency, the time between going to bed and falling asleep; (c) Wake Time, intervals when the participant was not sleeping that were scored by Actigraph algorithms and set as epoch counts; (d) Wake Minutes (after sleep onset), the minutes of wake after sleep onset of 20 continuous minutes of sleep following "lights off"; and (e) Sleep Time, the actual minutes per day spent sleeping. The wake time minutes directly corresponded to the sleep time minutes that equated to a 1,440-min day. "Lights off" was monitored by light sensors in the Actigraph watches.

PSQI—The PSQI, a validated self-report measure of subjective sleep quality and sleep disturbances, was completed by the study participants each treatment session (Buysse et al., 1989). A Likert-type scale was used for each individual component of the questionnaire. The scores of the 19 items resulted in seven component scores. All individual component scores were then added up to determine a Global Sleep Score. A global score of greater than 5 was indicative of poor sleep quality. The test–retest reliability of the PSQI sleep measurement questionnaire over a 4-week period was $r = .85$ (Buysse et al., 1989).

Demographic and Health Data

Demographic data—We recorded the participants' age, employment status, years of education, marital/social living status, and ethnic identification.

Anthropometric measurements—Each participant was weighed at the beginning and end of each treatment session with a Detecto® (Detecto Scale Company; Webb City, MO) balance beam scale. An Accu-Stat® (Accu-Stat Diagnostics, Inc.; Lake Forest, CA) wall-mounted height board was used to measure height. Weight-to-height ratios were calculated using the Quetelet Index (kg/m^2) to determine body mass indices. In the Second National Health and Nutrition Examination Survey, the anthropometric (weight and height) reliabilities for men and women were above .97 (Marks, Habicht, & Mueller, 1989).

Indirect calorimetry—The BodyGem® Indirect Calorimeter (Microlife USA, Inc.; Clearwater, FL) was used to determine the Resting Metabolic Rate for each participant. This handheld device worked in tandem with the Body-Gem® Analyzer software to measure the energy requirements of each participant using indirect calorimetry to measure each participant's respective energy requirements. A validity ($r = .92$) and reliability ($r = .90$) of this calorimeter was established in a study of 41 healthy, nonsmoking adults by Melanson et al. (2004).

Health Assessment Checklist (modified from Doenges, 1989)—Baseline health assessments of participants included a medical history based on the following checklist of health factors: endocrine, cardiac, respiratory, metabolic, gastrointestinal, urinary, sensory, integumentary, and neurological disorders. The assessment results were recorded by the research nurse and analyzed to identify any conditions or medications that might compromise performance outcomes.

Biochemical/laboratory tests—Biochemical/laboratory tests included measurements for serotonin levels, due to its possible relationship with sleep (Fernstrom, 1991; Markus et al., 2005). Serum glucose was recorded to test for hyper- and hypoglycemic effects of high protein and high carbohydrate diets. Serum lipids (cholesterol, low density lipoproteins, high density lipoproteins, and triglycerides) were measured to monitor the effects of high and low dietary fat intakes. Parikh, Mochari, and Mosca (2009) found the test results analyzed by the Cholestech-LDX© correlated with the Columbia University General Clinical Research Center Core Laboratory analysis of total cholesterol ($r = .91$), low density lipoproteins ($r = .88$), high density lipoproteins ($r = .77$), and triglycerides ($r = .93$). The same licensed personnel performed all blood draws and all laboratory tests to ensure validity of the study's lab samples.

Data Entry and Analysis

On the final day of each treatment session, sleep watches were removed and data were downloaded for analysis. The participant's health status was inventoried and recorded; and blood tests were drawn and analyzed. The SPSS statistical computer program was used for data entry and analysis. A double-entry procedure was implemented to reduce data entry errors. The Food Processor® nutrition analysis system (ESHA Research, Salem, OR) was selected to assign nutrient values to the weighed foods and beverages consumed by each study participant. This software system was used because it can analyze dietary intakes for a comprehensive nutrient inventory.

Frequencies were tabulated for the demographic and anthropometric data. The quality and quantity of the participants' sleep was determined by the *Actiwatch*es® and PSQI questionnaires. These data were evaluated following consumption of macronutrient diets during the treatment sessions. Descriptive and inferential statistics using repeated-measures ANOVA and correlational analysis were calculated. Tukey's post hoc tests were calculated to confirm where the differences occurred between the groups when the repeated-measures ANOVA calculations were significant. The minimum statistical significance for the study was set at $p \leq .05$.

Results

Demographic Characteristics

Of the 36 participants, 31 were European American, one was Native Hawaiian, three were Hispanic, and one was African American. The gender breakdown of the participants was 89% male and 11% female. The proportion of female participants was limited primarily because hormonal oral contraceptives and pregnancy were exclusionary criteria for the study, according to the Human Research Protections Office. The study participants averaged 20.9 years of Age ($SD = 1.9$), had completed an average of 14.1 years of School ($SD = 0.9$), and had a mean body mass index of 24.6 ($SD = 4.1$).

Effects of Macronutrients on the Global PSQI Sleep Measures

Global PSQI sleep scores were significantly different ($F = 5.1$, [df 4, 35], $p = .002$) among the participants when they consumed the high fat, protein, carbohydrate and the control diets. Also, the Tukey's post hoc test indicated that when the participants consumed the high fat diet, they had a significantly lower Global PSQI sleep score ($p < .001$) than when they consumed the high carbohydrate, high protein, and control diets. A lower Global PSQI sleep score indicates better sleep (Table 2).

Effects of Macronutrient Consumption on Individual Sleep Measures

Intra-subject analyses were performed using a repeated-measures ANOVA to detect differences in each participant's sleep scores following consumption of the different macronutrient diets. The participants' sleep efficiency, sleep latency, wake minutes (after sleep onset), wake times (using epoch counts), sleep duration (in minutes), and wake time (in minutes) were measured using sleep Actigraphs. The mean sleep scores for each participant were compared among the different diets. Participants' wake times (epoch counts) were significantly different ($F = 5.9$, [$df = 4, 35$], $p = .001$) when they had eaten the high fat, protein, carbohydrate and control diets. The wake minutes (after sleep onset) also differed significantly ($F = 2.8$, [$df = 4, 35$], $p = .05$) among the different dietary interventions (Table 3).

The Tukey's post hoc test indicated that when the participants consumed the high carbohydrate diet, they had significantly shorter ($p < .001$) wake times than when they consumed the high protein, high fat, and control diets (Table 3).

Effects of Dietary Fatty Acid Intake on Sleep Measures

The Actigraph sleep measures were also evaluated using a repeated-measures ANOVAs to determine differences in the sleep scores of each individual when they consumed different types of fat intakes during the treatment sessions. These evaluations compared the participants' mean sleep scores after they had consumed high saturated fat, high polyunsaturated fat, and low fat intakes. The participants' Actigraph wake times using epoch counts were significantly ($F = 3.1$, [$df = 3, 35$], $p = .05$) different when they had consumed high saturated or polyunsaturated fat and low fat intakes. Their Actigraph sleep duration and wake times (in minutes) were also significantly different ($F = 3.0$, [$df = 3, 35$], $p = .05$) when they had consumed high saturated and polyunsaturated fat and low fat intakes. The Global PSQI sleep scores indicated the quality of sleep was significantly higher ($F = 12.3$, [$df = 3, 35$], $p = .001$) after consuming saturated and polyunsaturated fat intakes in comparison with sleep quality scores after consuming the low fat intakes. The post hoc Tukey's test indicated the low fat intakes were significantly higher ($p \leq .05$) among the participants' PSQI sleep scores when comparing the dietary fat intakes (Table 4). A higher Global PSQI sleep score (> 5.0) indicates poor sleep.

Sleep Scores, Serotonin, Glucose, and Lipid Levels

Serotonin and serum glucose and lipid samples were analyzed from blood samples drawn at the end of each of the macronutrient and control dietary treatments. Sleep Actigraphs were used to quantify the sleep measures.

After a treatment session of consuming a high fat diet, the sleep efficiency scores were significantly associated with high density lipoprotein levels ($r = .35$, $p = .03$) and serum triglyceride levels ($r = -.38$, $p = .02$). Sleep latency times were also significantly associated with increased serum triglyceride levels ($r = .39$, $p = .02$) and total cholesterol levels ($r = .37$, $p = .03$). After a treatment session of consuming a high carbohydrate diet, there was a significant association between serotonin levels and wake times ($r = .94$, $p = .005$). However, after consuming a high protein diet, there were no significant associations among serotonin, serum lipid or glucose levels. Also, the serum glucose levels were not significantly ($p > .05$) associated with sleep levels after consuming the high carbohydrate diet.

Discussion

Our results indicated that Actigraph wake times (measured as epoch counts) were significantly different ($p = .001$) when participants had consumed diets high in fat, protein, and carbohydrates and a control diet. Likewise, wake minutes (after sleep onset) recorded on Actigraphs, were significantly different ($p \leq .05$) when participants had consumed the control diet and diets high in fat, protein, and carbohydrates. Following post hoc testing, significantly ($p < .001$) shorter wake times were recorded on the Actigraphs when participants consumed a high carbohydrate diet in comparison with when they had consumed the high fat, high protein, and control diets. In contrast, the Global PSQI sleep measures indicated the high fat diet resulted in significantly ($p < .001$) better sleep quality than the control diet. Our results are in agreement with prior reports showing carbohydrate diet consumption resulted in shorter wake times and high fat diets resulted in longer wake

times. However, there is conflicting data (Landström et al., 2000; Lindseth et al., 2013; Nehme et al., 2014; Rontoyanni, Baic, & Cooper, 2007; Voderholzer et al., 1998; Weiss et al., 2010).

Our post hoc study results of Actigraph sleep data showed high carbohydrate diets resulted in significantly shorter wake times in comparison with the other treatment or control diets. This differed from another study of 40 healthy young adults that used Actigraphs to measure sleep. In that study, higher protein intakes resulted in fewer wake episodes (Lindseth et al., 2013). Similarly, in Nehme et al.'s (2014) study of night security guards over 3 weeks using Actigraphs, high protein meals induced significantly fewer wake episodes compared with a (baseline) control diet; and the carbohydrate-rich meals induced significantly longer sleep durations. Another Actigraph study using 24-hr food recalls to measure macronutrient intakes of 240 adolescents, resulted in carbohydrate kilocalories leading to shorter sleep durations when average daily increases of fat followed with a concomitant decrease in carbohydrate kilocalories (Weiss et al., 2010). Also, Voderholzer et al. (1998) found a low protein diet led to tryptophan depletion, and subsequently serotonin levels dropped in the brain. This condition led to more waking periods in comparison with a control diet. Landström et al. (2000) reached a somewhat different conclusion, finding that a balance of macronutrients (fat, protein, and carbohydrate) did not affect wakefulness in 12 day-and-night drivers.

Our PSQI sleep data indicated the quality of sleep was significantly better after consuming a high fat diet in comparison with other diets. Also, the Global PSQI sleep scores indicated the quality of sleep was significantly better ($p = .001$) after consuming saturated and polyunsaturated fat intakes in comparison with the low fat intakes. Furthermore, the Tukey's post hoc test indicated the low fat intakes were significantly ($p > .05$) higher, meaning the participants' sleep was poorer when they had consumed low fat intakes. Our results were inconsistent with those of a cross-sectional study of 30 healthy Greek women in which increased saturated fat intakes correlated with shorter sleep duration (Rontoyanni et al., 2007). Sleep-habit questionnaires, 7-day sleep diaries, and two 24-hr dietary recalls were used for measurements in that study.

The beneficial effects of the fats on the PSQI sleep measures in our study may be partially explained as follows: Long-chain polyunsaturated fatty acids, such as arachidonic acid (AA) and DHA, have shown some promise for enhancing sleep initiation and duration. Both AA and DHA are thought to be important precursors in promoting and regulating sleep because higher levels of DHA resulted in increased levels of melatonin in a study by Catalá (2010). DHA may also have a possible role for enzymatic activity needed to transform serotonin into melatonin; thereby, promoting sleep by increasing serotonin and melatonin levels (Peuhkuri, Sihvola, & Korpela, 2012).

Tryptophan-enriched high protein diets have also resulted in sleep improvements in another study (Markus et al., 2005). Still another study examined narcolepsy patients who consumed a low carbohydrate, ketogenic diet for 8 weeks. Those participants experienced reduced daytime sleepiness and fewer narcolepsy symptoms with a low carbohydrate diet compared with a control diet (Husain, Yancy, Carwile, Miller, & Westman, 2004).

Our study results showed that increased sleep latency (time to fall asleep) was significantly associated with increased serum triglyceride and total cholesterol levels. Similar to our study, Adam and Oswald (1979) found no differences in sleep latencies for 12 male and female participants (*M* age = 54 years) after they had consumed high carbohydrate or low carbohydrate meals, compared with a placebo treatment. Conversely, other studies showed that high carbohydrate diets had a positive impact on with shorter sleep latencies (Lindseth et al., 2013; Nehme et al., 2014), while other studies showed no significant relationship or a negative relationship between carbohydrate consumption and sleep onset (Afaghi et al., 2007; Phillips et al., 1975). For example, in a study by Afaghi et al. (2007) of 14 healthy men, decreased sleep latencies and slow-wave sleep resulted after consuming very low carbohydrate meals. The authors suggested that the high fat content of their low carbohydrate meals may have influenced the men's sleep. In support of that notion, our Global PSQI sleep measures indicated that a high fat diet resulted in better PSQI sleep scores than the high protein, carbohydrate, or control diets.

The results in our study indicated a significant positive association between sleep efficiency scores and high density lipoprotein levels and a significant negative association between sleep efficiency scores and serum triglyceride levels in young, healthy adults. Similarly, Lindseth et al. (2013) found that serum lipid levels (total cholesterol) were significantly elevated when sleep efficiency was low. Serum lipids and total cholesterol were also related to the number of sleep arousals per hour in a study by Ekstedt, Åkerstedt, and Söderström (2004). Our study results also showed that increased sleep latency was significantly associated with increased serum triglyceride and total cholesterol levels.

After consuming a high carbohydrate diet, we also found no significant association between serum glucose levels and sleep levels, thus contrasting with a previous study's results showing low sleep efficiency was significantly associated with high glucose levels (Lindseth et al., 2013). Although, the high carbohydrate diet in our study resulted in a significantly strong correlation between serum serotonin levels and wake time. A likely explanation is that serum serotonin fluctuations may have served as a precursor to sleep disturbances (Fernstrom, 2012). Carbohydrate intakes can play a factor in promoting the entry of tryptophan into the brain. In addition, carbohydrates with a high glycemic index have the ability to increase the ratio of tryptophan to large neutral amino acids (Wurtman et al., 2003). This is also affected by the insulin response that carbohydrate intakes have on the selective muscle uptake of large neutral amino acids. Thereby, sleep is promoted by increasing tryptophan and serotonin levels in the brain following the intake of high glycemic index foods (Fernstrom, 1991).

This study had some limitations. First, the results were based on a small sample size. However, we used a repeated-measures study design and recruited a sufficient number of participants to meet statistical power requirements to enhance the reliability of the study. Providing baseline sleep data in addition to the data acquired for the control diet would also add strength to this study. While we used Actigraph sleep watches to record sleep measures; the literature has indicated that polysomnography or electroencephalographic monitoring may provide greater accuracy for measuring sleep (Marino et al., 2013). However, given that the purpose of our study was to investigate free-living study participants in a non-laboratory

setting, we decided the *Actiwatches*® could achieve the accuracy needed for this study. Our study was also based on a short-term (4 days per diet) dietary treatment plan. A longer term study is recommended for the future to generalizability of the results. Taken together, some of our results suggested contradictory conclusions, as found in previous studies. Therefore, further study is recommended.

In summary, our results indicated that the proportions of macronutrients in a diet may affect sleep, particularly the wake time. For example, significantly shorter wake times were associated with the high carbohydrate diet. Also, our PSQI sleep measurements indicated that the high fat diets induced significantly better sleep quality than the control diet. When analyzing the biochemical lab data, the high fat diet showed significant associations between sleep efficiency scores and serum low density lipoprotein and triglyceride levels. Sleep latency times were also significantly associated with serum triglyceride and cholesterol levels. Interestingly, following the high carbohydrate diets, we found a significantly high association between serotonin levels and wake times, which supports Fernstrom's (2012) assertion that serotonin levels can serve as a precursor to sleep disturbances.

Thus, our results suggest that the proportions of specific macronutrients in a diet may influence a person's sleep quality, by either inducing or reducing sleep time. Future studies should test the effects of dietary macronutrients on sleep by monitoring sleep more closely with electroencephalogram or polysomnography devices in a laboratory setting.

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Table 1

Nutrient Content of Study Diets (n = 36).

Study Diets	Fat %	Carbohydrate %	Protein %
Macronutrient diets			
Control diet	35	50	15
Fat diet	65	25	10
Protein diet	15	40	45
Carbohydrate diet	10	80	10

Table 2

Repeated-Measures ANOVA for PSQI Global Sleep Scores During Diets With Different Macronutrient Contents ($n = 36$).

Measure	*M*	*SD*	*F*	*p*
Global PSQI			5.1	.002**
High carbohydrate diet	5.7	1.7		
High protein diet	5.3	2.6		
High fat diet[a]	4.2	1.7		
Control diet	5.2	2.9		

Note. PSQI = Pittsburgh Sleep Quality Index®.

[a]Post hoc results: Fat diet resulted in significantly ($p < .001$) lower Global PSQI scores (better sleep) than the other diets.

** $p \leq .01$.

Table 3

Repeated-Measures ANOVA for Actiwatch Sleep Measures Following Macronutrient Consumption ($n = 36$).

Measure	*M*	*SD*	*F*	*p*
Sleep duration (minutes)			2.4	.07
High carbohydrate diet	389.5	76.4		
High protein diet	380.5	60.2		
High fat diet	398.4	58.9		
Control diet	409.6	64.5		
Wake time (minutes)			2.4	.07
High carbohydrate diet	1,050.5	76.4		
High protein diet	1,059.5	60.2		
High fat diet	1,041.6	58.9		
Control diet	1,030.4	64.5		
Sleep efficiency (%)			1.0	.37
High carbohydrate diet	81.2	11.5		
High protein diet	79.8	10.7		
High fat diet	82.4	8.8		
Control diet	81.3	9.1		
Sleep latency (minutes)			0.49	.69
High carbohydrate diet	40.6	46.0		
High protein diet	36.5	38.1		
High fat diet	43.5	40.0		
Control diet	44.9	36.1		
Wake time (epoch counts)			5.9	.001***
High carbohydrate diet[a]	532.0	134.1		
High protein diet	610.7	110.6		
High fat diet	577.2	108.3		
Control diet	582.9	106.2		
Waking minutes (after sleep onset)			2.8	.05*
High carbohydrate diet	25.2	13.4		
High protein diet	30.8	19.5		
High fat diet	23.0	10.0		
Control diet	29.0	18.7		

[a]Post hoc results: Carbohydrate diet resulted in significantly ($p < .001$) shorter wake times than the other diets.

*$p \leq .05$.

***$p \leq .001$.

Table 4

Repeated-Measures ANOVA for Sleep Measures of Participants With Different Fatty Acid Intakes (n = 36).

Measure	*M*	*SD*	*F*	*p*
Actiwatch				
Wake time (epoch counts)			3.1	.05*
High saturated fatty acid intake	587.8	119.1		
High polyunsaturated fatty acid intake	581.7	114.2		
Low fat intake	542.6	146.6		
Waking minutes (after sleep)			0.31	NS
High saturated fatty acid intake	23.0	10.0		
High polyunsaturated fatty acid intake	23.7	10.3		
Low fat intake	24.8	13.6		
Wake time (minutes)			3.0	.05*
High saturated fatty acid intake	1,041.6	59.0		
High polyunsaturated fatty acid intake	1,038.6	57.7		
Low fat intake	1,059.1	70.8		
Sleep duration (minutes)			3.0	.05*
High saturated fatty acid intake	398.4	59.0		
High polyunsaturated fatty acid intake	401.4	57.7		
Low fat intake	380.9	70.8		
PSQI				
Global PSQI			12.3	.001***
High saturated fatty acid intake	4.2	1.8		
High polyunsaturated fatty acid intake	4.3	1.9		
Low fat intake[a]	5.4	2.8		

Note. PSQI = Pittsburgh Sleep Quality Index®.

[a]Post hoc results: Low fat intakes resulted in significantly (p < .05) higher Global PSQI sleep scores (poorer sleep) than other fatty acid intakes.

*$p \leq .05$.

***$p \leq .001$.