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Original Article

ORIGINAL ARTICLE

Mediterranean diet pattern and sleep duration and insomnia symptoms in the Multi-Ethnic Study of Atherosclerosis

Cecilia Castro-Diehl[1,*], Alexis C. Wood[2], Susan Redline[3,4,5] Michelle Reid[3], Dayna A. Johnson[3,5], Janice E. Maras[6], David R. Jacobs, Jr[7], Steven Shea[8,9], Allison Crawford[8] and Marie-Pierre St-Onge[8,10]

[1]Section of Preventive Medicine and Epidemiology, Boston University, Boston, MA, [2]USDA/ARS Children's Nutrition Research Center, Baylor College of Medicine, Houston, TX, [3]Department of Medicine, Brigham and Women's Hospital, Boston, MA, [4]Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA, [5]Harvard Medical School, Boston, MA, [6]Department of Health Sciences, Northeastern University, Boston, MA, [7]Division of Epidemiology and Community Health, School of Public Health, University of Minnesota, MN, [8]Department of Medicine, Columbia University Vagelos College of Physicians and Surgeons, New York, NY, [9]Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, NY and [10]Institute of Human Nutrition, Columbia University College of Physicians and Surgeons, New York, NY

*Corresponding author. Cecilia Castro-Diehl, 801 Massachusetts Suite 470, Boston, MA 02118. Email: cecastro@bu.edu.

Abstract

Sleep duration and sleep quality are important predictors of risk for cardiovascular disease (CVD). One potential link between sleep health and CVD is through lifestyle factors such as diet. To clarify the association between diet and sleep, we assessed the associations of sleep duration and insomnia symptoms with current Mediterranean-style diet (aMed) and with historical changes in aMed score. Actigraphy-measured sleep duration and self-reported insomnia symptoms categorized as insomnia with short sleep (<6 hr/night), insomnia without short sleep, no insomnia with short sleep, and no insomnia or short sleep were obtained from 2068 individuals who also had dietary intake data. A 10-point aMed score, derived from a self-report food frequency questionnaire, was collected concurrently with the sleep assessment and 10 years before. Compared with individuals who currently reported a low aMed score, those with a moderate-high aMed score were more likely to sleep 6–7 vs. <6 hr/night ($p < 0.01$) and less likely to report insomnia symptoms occurring with short sleep (vs. no insomnia or short sleep alone; $p < 0.05$). An increase in aMed score over the preceding 10 years was not associated with sleep duration or insomnia symptoms. However, compared with those with decreasing aMed score, individuals with an unchanging score reported fewer insomnia symptoms ($p \leq 0.01$). These results suggest that a Mediterranean-style diet is associated with adequate sleep duration, less insomnia symptoms, and less likely to have insomnia accompanied by short sleep. Further research should identify possible mediators through which diet may promote adequate sleep duration and reduce the risk of insomnia.

Statement of Significance

This paper investigates whether a Mediterranean-style diet is related to sleep duration and sleep quality. We identified that participants who have favorable eating habits sleep longer and report better sleep quality. Achieving a healthy diet could improve sleep, which could further contribute to the effects of a healthy diet on cardiovascular disease. Research is warranted on possible mediators through which diet may promote adequate sleep duration and prevent insomnia.

Key Words: Mediterranean-style diet; sleep duration insomnia

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Introduction

Epidemiological evidence suggests recent reductions in sleep duration [1] and increases in the prevalence of sleep disorders [2]. Sleep duration and insomnia symptoms are associated with numerous health outcomes, including cardiovascular diseases (CVD) [3]. However, whether the associations between sleep and CVD risk reflect a causal relationship or simple cooccurrence is not clear since sleep patterns, including sleep duration and insomnia, are related to other behavioral and lifestyle choices, such as dietary intake, which also influence CVD risk and may confound the association.

Short sleep duration, insomnia symptoms, and poor sleep quality have been associated with preference for fatty foods, skipping breakfast, eating outside the home [4]; lower consumption of vegetables [5]; and higher intakes of confectionery, sugar-sweetened beverages, and energy drinks [6]. Interventional studies have supported the notion that sleep affects dietary intakes and energy balance. For example, sleep restriction to 4 hr/night for five nights increased meal frequency and total caloric intake, compared with usual sleep in healthy, normal weight adults [7]. Others have reported similar [8, 9], or larger effects of sleep restriction on energy intake [10]. However, the relation between sleep parameters and eating patterns may be bidirectional. From an interventional study, using data from participants undergoing four nights of 9 hr sleep opportunity, St-Onge et al. found that higher fiber and lower saturated fat intakes during the day were associated with greater time in slow-wave sleep (stage N3), and that higher sugar intake was associated with a greater number of arousals at night [11]. These data suggested that dietary patterns and nutrient intakes may influence sleep quality.

The association between sleep and diet is important to understand, given the relationship of these factors to health outcomes [12, 13]. A Mediterranean, or Mediterranean-style diet is consistently associated with beneficial health outcomes [14], partly explained by hormonal and molecular mechanism [15]. It has been linked to a reduced risk of hypertension [16] and coronary heart disease [17], as well as reduced colon cancer [18], depression and cognitive impairment [13], and Alzheimer disease [19]. Insomnia, particularly when associated with short sleep duration, is associated with an increase risk in adverse cardiometabolic outcomes [20], hypertension [21], diabetes [22], neurocognitive impairment [23], incident CVD [24], and mortality [25]. To the best of our knowledge, only two other epidemiological studies have evaluated the association between a Mediterranean-style diet pattern and sleep outcomes. One found that higher adherence to a Mediterranean diet pattern was associated with lower variability in sleep duration over 3 years and also lower risk of self-reported poor sleep quality [26]. Another study found that women who adhered to a Mediterranean diet pattern were less likely to have insomnia symptoms than women who did not adhere to the Mediterranean diet [27]. However, these studies lacked objective estimates of sleep duration and were not inclusive of racial and ethnic minorities. No study to date has examined the association between a Mediterranean diet pattern and insomnia symptoms occurring in conjunction with objectively measured sleep duration.

The aim of this study was to examine cross-sectional associations between a Mediterranean-style diet (aMed) at Exam 5 and sleep duration measured by actigraphy, and insomnia symptoms measured by self-report, and to supplement these analyses with associations between sleep measures and changes in the aMed score from Exam 1 to Exam 5. We hypothesized that (1) participants with higher aMed consumption and participants with an increase or unchanged aMed score from Exam 1 to Exam 5 would be more likely to have adequate sleep duration than participants with lower aMed consumption or decrease in score, (2) higher aMed consumption and an increase or unchanged aMed score from Exam 1 to Exam 5 would be associated with lower risk of insomnia symptoms, and (3) participants with higher aMed consumption and participants with an increase or unchanged aMed score from Exam 1 to Exam 5 would be less likely to report insomnia symptoms occurring with short sleep duration than participants with lower aMed consumption or decrease in score.

Methods

Data

Multi-Ethnic Study of Atherosclerosis (MESA) is a longitudinal study of risk factors for subclinical CVD and its progression to clinical disease. From 2000 to 2002, the study recruited adults aged 45–84 years, with no history of clinical CVD at baseline, from six sites across the United States (Los Angeles, CA; St. Paul, MN; Chicago, IL; Forsyth County, NC; Baltimore MD; and New York, NY). During Exam 1 (2000–2002) and Exam 5 (2010–2012), MESA collected detailed measures of dietary intake using a Food Frequency Questionnaire (FFQ), along with medical and medication history, physical activity, and several physiological measures [28]. The Sleep Study was conducted in conjunction with Exam 5 (2010–2013) and collected sleep data from 2261 participants (Supplementary Figure 1).

The MESA and MESA Sleep Study were approved by institutional review boards at each participating institution and written informed consent was obtained from participants.

Sleep study

All MESA participants except those reporting regular use of oral appliances, nocturnal oxygen, or continuous positive airway pressure therapy were eligible to participate. The MESA Sleep protocol included 7 day actigraphy (Actiwatch Spectrum, Philips Respironics, Murrysville, PA) together with a sleep diary. The actigraphy data during 30 s epochs were scored as sleep or wake by Actiware-Sleep v. 5.59 analysis software (Mini Mitter Co., Inc., Bend, OR) and a validated algorithm [29] in which activity counts recorded during the measured epoch were modified by the level of activity in the surrounding 2 min time period (i.e. ±2 min) to yield a final activity count for each epoch. We estimated sleep duration for each day using this algorithm. The intrascorer intraclass correlation coefficient for average sleep duration was 0.91. The sleep diary provided self-reported information on bed and wake times, sleep onset, naps, actigraph removal, and other events.

Objective sleep duration was assessed using actigraphy and was defined as the average duration of sleep between sleep onset (sleep start time) and morning wakening (sleep end time) while in bed after "lights off." The average days of scorable data were 6.9 (standard deviation [SD] = 0.54) for all days, 5.0 (0.42) for weekday data, and 2.0 (0.27) for weekend data. Only participants

with at least 3 days of actigraphic sleep data, including one weekend day, were included in these analyses. An average sleep was calculated from all days recorded. We categorized sleep duration as follows: <6, 6 to <7, 7–8, and >8 hr/night.

Insomnia was assessed by self-report using the Women's Health Insomnia Rating Scale (WHIIRS), a validated five-item questionnaire which asked participants to rate how frequently they experienced the following difficulties over the past 4 weeks using a five-point Likert type scale: (1) "Having trouble falling asleep," (2) "Waking up several times a night," (3) "Waking up earlier than planned," (4) "Having trouble getting back to sleep after waking up too early," and (5) "How was your typical night's sleep." When summed, the final WHIIRS score ranged from 0 to 20. A score of ≥9 may be considered as clinically significant insomnia [30].

Insomnia symptoms in conjunction with objective sleep duration was further classified by grouping participants into those with insomnia symptoms and those without insomnia symptoms, and further separating those groups into those who slept <6 hr/night (short sleep) and those who slept ≥6 hr/night. We then classified the participants as having insomnia symptoms with short sleep duration, insomnia symptoms without short sleep, no insomnia symptoms with short sleep, and no insomnia symptoms or short sleep. Insomnia symptoms with objective short sleep duration have been reported to associate with worse cardiometabolic outcomes than insomnia without short sleep duration [20, 24].

Mediterranean diet

In both MESA Exam 1 and Exam 5, usual dietary intake over the past year was assessed using a modified-Block [31] 120-item self-report FFQ modified to include Chinese food and beverage items [32, 33]. Participants were asked to report their frequency of intake of each food (number of times per day/week/month) and their usual serving sizes (small, medium, and large) from among nine categories. To ascertain conformity to a Mediterranean-style diet, a 10-point a priori Alternate Mediterranean Diet (aMed) score was created. The aMed scoring system was adapted for U.S. populations from a scoring system developed for Greek populations. The score incorporates 10 food components: vegetables (including potatoes), whole grains, nuts, legumes, fruits, ratio of non-saturated to saturated fat, red and processed meat, whole fat dairy, fish, and alcohol [34]. Participants with intakes above the median for traditional foods in the Mediterranean diet received one point, whereas those with intakes below the median received no points. Potentially detrimental foods inversely associated with the Mediterranean diet (red and processed meats and whole fat dairy) were reverse scored. Alcohol intake received one point if consumed in moderate amounts (12–14 g/d) and none otherwise (<12 or >14 g/d). Points were then summed, and the final score ranged from 0 to 10 with a higher score indicating a closer resemblance to the Mediterranean diet. Consumption to an aMed diet was categorized as low (score 0–5) or moderate-high (score 6–10).

Covariates

All models included age at Exam 5 (as a continuous variable), sex, and race/ethnicity (white, black, or Hispanic), as potential confounders. We also controlled for education (less than a high school diploma, completion of high school or some college, or college degree), smoking status (former/current/never), physical activity (measured in metabolic equivalent (MET)- (summed minutes per week of self-reported time spent in walking, dance/sport, and conditioning) [35], total energy intake (kcal/d), body mass index (BMI, weight in kg/height in m^2), hypertension (yes/no from diastolic blood pressure ≥90 mmHg or systolic blood pressure ≥140 mmHg or self-report history of hypertension and self-report history of hypertensive medications), diabetes (2013 ADA Fasting Criteria at clinic Exam or self-report), the apnea/hypopnea arousal index (AHI) based on apnea and hypopnea with ≥3 per cent desaturation from the polysomnogram, depressive symptoms (a score of ≥16 on the 20-item Center for Epidemiological Studies Depression scale) [36], and medications used (sleep medications as well as antipsychotics and antidepressants). In sensitivity analysis, waist circumference was substituted for BMI and chronic obstructive pulmonary disease (COPD) and "ever stop walking due to breathlessness" variables, both self-report, were also included in the last model.

Statistical analysis

The Sleep Study dataset provided valid objective sleep duration data for 2151 participants and information on insomnia symptoms for 2222 participants. After excluding 144 participants from the sleep duration data and 154 from the insomnia data due to missing FFQ or implausible energy intakes (<500 or >5,000 kcal/day) at Exam 5, 2068 remained on whom to test the study hypotheses (Supplementary Figure 2). Individuals excluded from this analysis were more likely to be Hispanic or Chinese, less educated, with lower incomes, nonsmokers, less physically active, and with lower mean aMed scores (4.1 [1.7] vs. 4.6 [2.0]) than included participants. Included and excluded participants did not differ in age, sex, BMI, waist circumference, sleep duration, or insomnia symptoms.

To test hypothesis 1, we included participants who had valid aMed Score and sleep duration at Exam 5 (n = 2007) and participants who had repeated measures of valid aMed (Exam 1 and Exam 5) and sleep duration at Exam 5 (n = 1927) (Supplementary Figure 2). We investigated associations between moderate-higher score and sleep duration and changes in the aMed score from Exam 1 to Exam 5 and sleep duration.

To test hypothesis 2, we included participants who had valid a Med Score and available measures of insomnia symptoms at Exam 5 (n = 2068) and participants who had repeated measures of valid aMed (Exam 1 and Exam 5) and insomnia symptoms at Exam 5 (n = 1988) (Supplementary Figure 2). We investigated associations between moderate-higher score and insomnia symptoms and changes in the aMed score from Exam 1 to Exam 5 and insomnia symptoms.

To test hypothesis 3, we combined the sleep duration and insomnia datasets with the aMed data at Exam 5 (n = 1976) and then with repeated measures of valid aMed (Exam 1 and Exam 5) (n = 1896). We compared participants with moderate-high aMed score to those with low aMed score for three different categories of insomnia symptoms and objective sleep duration (insomnia symptoms with short sleep (<6 hr/night), insomnia symptoms without short sleep, and no insomnia symptoms with short sleep (<6 hr/night)) relative to no insomnia symptoms or short sleep duration alone. We also investigated associations between changes in the aMed score from Exam 1 to Exam 5 with insomnia symptoms in conjunction with short sleep duration.

To assess changes in the aMed score, we subtracted the aMed score at Exam 5 (aMed 5 score) from the aMed score at Exam 1 (aMed 1 score). Changes in aMed were grouped in three categories (Supplementary Table S1): "no change in score from Exam 1 to Exam 5" if difference in scores was zero, "decrease in score" difference in aMed 5 and aMed 1 scores < 0, and "increase in score" difference in aMed 5 and aMed 1 scores > 0.

We used multinomial logistic or simple logistic regression models depending on the main outcome to examine the associations of our main exposure of interest (aMed) with sleep duration, insomnia symptoms, and with insomnia symptoms occurring with sleep duration. We calculated the odds ratios (OR) and the 95% confidence intervals (95% CI) from these models to estimate the associations of these outcomes with the aMed score specifying lower aMed consumption as the referent category.

To test each hypothesis, we developed several statistical models based on the literature [26]. In model 1, we adjusted for age, sex, and race/ethnicity. In model 2, we also adjusted for education level, and in model 3, we further adjusted for behavioral factors: cigarette smoking, intentional exercise, and total energy intake. In model 4, we further adjusted for potential health-status mediators or confounders of the aMed-sleep duration/insomnia symptoms association: hypertension, diabetes, depressive symptoms, AHI, BMI, and medication. In our analyses of insomnia symptoms as the main outcome, we used the same models, but we controlled for sleep duration hours, and when sleep duration was the main outcome, we controlled for insomnia symptoms in the last model (model 4).

We also developed multinomial logistic regression models to examine associations between change of aMed score from Exam 1 to Exam 5 and sleep outcomes. The reference group was the decrease in aMed score category. Covariate main effects were included to adjust for confounding as described above.

Lastly, we performed sensitivity analyses in which we excluded from our analysis those participants who slept on average <3 hr/night (1.2% of the sample), and in which we limited our sample to participants without diabetes because individuals with diabetes may follow a special diet. We explored substituting waist circumference for BMI. We also considered other morbidities that may affect sleep patterns, so we added in the last model COPD and separately a surrogate variable for heart failure.

Results

Descriptives

The study sample had a mean aMed score of 4.5 (1.9) at Exam 1 and 4.7 (2.2) at Exam 5 (Supplementary Table S2). Those participants whose aMed scores did not change from Exam 1 to Exam 5 had a mean score of 4.6 (1.9) at both exams. Those participants whose aMed score increased (41% of the sample) had a mean score of 3.6 (1.8) at Exam 1 and 5.7 (1.7) at Exam 5, whereas those participants whose aMed score decreased (38% of the sample) had a mean score of 5.4 (1.7) at Exam 1 and 3.5 (1.7) at Exam 5. (Supplementary Tables S1 and S3). Table 1 presents descriptive statistics by cross-sectional level of aMed consumption. Higher aMed scorers were more likely to be women, White or Chinese, more educated, more physically active, and less likely to be cigarette smokers or have obesity, than those with a low score. Higher scorers were also more likely to sleep 6–7 hr/night and less likely to report insomnia symptoms.

Associations between aMed diet and sleep duration

Table 2 shows multinomial logistic regression models that examined the association of the aMed score with sleep duration (n = 2007) after progressively adjusting for potential confounders and/or mediators. The table displays the odds ratio (OR) and 95% confidence interval (CI) comparing moderate-high aMed score to low aMed score for three different categories of objectively measured sleep duration (6–7, 7–8, and >8 hr/night) relative to the shortest sleep duration category (<6 hr/night). Participants with a moderate-high aMed score were more likely to sleep 6–7 hr/night (vs. <6 hr/night) than those who had a low aMed score after full adjustment (OR = 1.43, 95% CI 1.08–1.88, $p < 0.01$), but no more likely to sleep 7–8 hr/night, or >8 hr/night on average. In sensitivity analysis, these estimates did not differ when we excluded 377 participants with treated diabetes. Similarly, when we replaced BMI for waist circumference, or controlled for COPD or heart failure, the results did not differ.

Associations between aMed diet and insomnia symptoms

Compared with those with low aMed score, participants with high aMed score were less likely to report insomnia symptoms (OR = 0.81, 95% CI 0.68–0.98, $p < 0.05$ after controlling for demographic and SES variables), but the association was not significant after adjustment for behavioral (model 3) and health (model 4) factors (Table 2, last column).

Associations between aMed diet and insomnia symptoms in conjunction with sleep duration

Table 3 shows multinomial logistic regression models that examined the association of the aMed score with a phenotype defined by insomnia symptoms with objectively measured short sleep duration. The table displays the OR and 95% CI comparing moderate-high aMed score to low aMed score for three different categories of insomnia symptoms and sleep duration (insomnia symptoms with short sleep, insomnia symptoms without short sleep, and no insomnia symptoms with short sleep duration) relative to no insomnia symptoms or short sleep duration alone. Participants with moderate-high aMed score were less likely to have insomnia symptoms with short sleep duration compared with those with low aMed score (OR = 0.65, 95% CI, 0.45–0.93, $p < 0.05$ after full adjustment). Moderate-high aMed score was not associated with any of the other two groups: insomnia symptoms without short sleep duration and no insomnia symptoms with short sleep duration.

Associations between historical changes in aMed diet and sleep duration

Table 4 shows multinomial logistic regression models that examined the association of the three levels of change in the aMed score from Exam 1 to Exam 5 (reference: decrease in score) with sleep duration at Exam 5, after progressively adjusting for potential confounders. Participants with an increase in the aMed score were more likely to sleep 6–7 hr/night (vs. <6 hr/night) than those with decrease in the aMed score (OR = 1.36, 95% CI 1.05–1.76, $p < 0.05$), but the association was not significant after adjustment for behavioral and health factors. An increase

Table 1. Characteristics by level of aMed consumption at Exam 5, 2010–2012

	Total 2007	Low (aMed score < 5) *n* = 961	Moderate and high (aMed score ≥ 5) *n* = 1046	*P*
	%, mean ± standard deviation or median (Q_1, Q_3)			
Age, years	68.6 ± 9.2	68.1 ± 9.2	69.0 ± 9.1	0.03
Sex, %				0.001
Female	53.6	49.8	57.2	
Male	46.3	50.2	42.8	
Race/ethnicity, %				0.001
White	38.8	41.0	36.7	
Chinese	11.2	7.2	14.8	
Black	27.1	26.2	27.8	
Hispanic	23.0	25.6	20.7	
Education, %				0.001
Less than high school diploma	13.8	14.6	13.0	
High school or some college	40.6	45.7	36.0	
College degree	45.6	39.7	51.0	
Income, %				0.05
First quintile (lowest)	14.5	17.7	16.3	
Second quintile	16.7	22.9	21.6	
Third quintile	22.5	23.2	21.5	
Fourth quintile	28.0	18.4	17.1	
Fifth quintile (highest)	18.3	17.8	23.5	
Cigarette smoking, %				0.001
Never	46.2	42.3	49.8	
Former	46.5	48.0	45.1	
Current	7.3	9.7	5.1	
Alcohol consumption*, %	44.5	44.4	44.7	0.89
Intentional physical activity, MET-min/week	1800 (720, 3690)	1470 (585, 3225)	2100 (892, 4053)	0.001
Body mass index, kg/m^2	28.8 ± 5.6	29.7 ± 5.7	27.9 ± 5.3	0.001
Waist circumference, cm	99.6 ± 14.5	102.0 ± 14.6	97.4 ± 14.2	0.001
Total energy (kcal)	1560 (1118, 2169)	1415 (978, 1907)	1703 (1249, 2350)	0.001
Diabetes, %				0.004
No diabetes	60.3	56.6	63.8	
Impaired glucose	20.7	23.1	18.5	
Diabetes	19.0	20.4	17.7	
Hypertension, %	57.5	58.8	56.2	0.24
Seated heart rate, beats/min	64 ± 11	65 ± 11	63 ± 10	0.001
Any hypertension medication, %	54.2	55.2	53.3	0.39
Depression scale, CESD ≥ 16	14.4	15.9	13.1	0.08
Anti-depressive medications†	13.8	15.8	12.1	0.02
Sleep variables				
All apneas and hypopneas index (AHI)‡	24.0 ± 19.2	25.8 ± 20.3	22.4 ± 18.6	0.001
WHI§ insomnia rating scale ≥ 9		38.3	33.7	0.04
Sleep duration (from actigraphy)				<0.05
< 6 hr (*n* = 609)	30.3	31.4	29.4	
6 to ≤ 7 hr (*n* = 640)	31.9	29.1	34.4	
7 to ≤ 8 hr (*n* = 532)	26.5	26.7	26.3	
> 8 hr (*n* = 226)	11.3	12.7	9.9	
Mediterranean-style diet score (aMed)	4.6 ± 2.0	2.9 ± 1.0	6.2 ± 1.1	0.001
aMed components				
Vegetables	1.90 ± 1.4	1.23 ± 1.0	2.51 ± 1.5	0.001
Legumes	0.43 ± 0.6	0.26 ± 0.4	0.58 ± 0.8	0.001
Fruit	3.18 ± 2.2	2.26 ± 1.6	4.00 ± 2.3	0.001
Nuts	0.47 ± 0.6	0.27 ± 0.4	0.65 ± 0.7	0.001
Whole grains	0.94 ± 0.8	0.64 ± 0.6	1.22 ± 0.8	0.001
Fish	0.27 ± 0.3	0.18 ± 0.2	0.35 ± 0.3	0.001
Red and processed meat	0.41 ± 0.4	0.43 ± 0.4	0.39 ± 0.4	0.03
Whole fat dairy	0.49 ± 0.7	0.56 ± 0.7	0.42 ± 0.6	0.001
Ratio of nonsaturated to saturated fat	2.35 ± 0.9	1.91 ± 0.6	2.74 ± 1.0	0.001
Alcohol consumption (%, moderate)	2.64	1.46	3.73	0.002

p-Value t-test or chi-square.

*alcohol consumption was classified as yes/no.

†anti-depressive, anti-psychotic and sleep medication.

‡all apneas +hypopneas with ≥3% desaturation or arousal Index (AHI).

§Women's Health Initiative (WHI) insomnia rating scale.

Table 2. Odds ratios (95% CIs) for the associations of sleep duration (n = 2007) and insomnia symptoms (n = 2068) with a moderate to high aMed diet score at Exam 5

	Sleep duration‡ *(reference:* <6 hr/night)			Insomnia symptoms§ *(reference:* no insomnia)
aMed score†	6 to <7 hr/night$n$ = 640	7–8 hr/night$n$ = 532	>8 hr/night$n$ = 226	Insomnia$n$ = 739
Moderate-high aMed score (≥5)				
Model 1	**1.30 (1.03, 1.63)***	1.05 (0.82, 1.34)	0.83 (0.60, 1.14)	**0.81 (0.67, 0.97)***
Model 2	**1.32 (1.05, 1.66)***	1.05 (0.82, 1.34)	0.84 (0.61, 1.16)	**0.81 (0.68, 0.98)***
Model 3	**1.38 (1.07, 1.78)****	1.05 (0.80, 1.38)	0.97 (0.68, 1.40)	0.82 (0.67, 1.00)
Model 4	**1.43 (1.08, 1.88)****	1.05 (0.78, 1.40)	0.95 (0.64, 1.42)	0.85 (0.68, 1.06)

†Reference= low aMed score (<5).
‡For sleep duration (ref <6 hr, n = 609).
§For insomnia symptoms (ref no insomnia symptoms, n = 1329).
MODEL 1: adjusted for age, gender, race/ethnicity.
MODEL 2: adjusted for covariates in Model 1 as well as education.
MODEL 3: adjusted for covariates in Model 2 as well as cigarette smoking, intentional exercise, and total energy intake (Kcal/day).
MODEL 4: adjusted for covariates in Model 3 as well as, body mass index (Kg/m^2), hypertension, diabetes, depressive symptoms (CESD score ≥ 16), AHI (continuous), insomnia symptoms, antidepressants and anti-psychotic medication.
In the models with insomnia symptoms as main predictor, we also adjusted for sleep duration (hr) in the last model.
*p < 0.05; **p ≤ 0.01. Bold values indicate significance.

Table 3. Odds ratios (95% CIs) for the associations between insomnia symptoms with short sleep duration and a moderate to high aMed score (n = 1976) at Exam 5

aMed score†	Insomnia with short sleep duration‡ n = 228	Insomnia without short sleep duration‡ n = 481	No insomnia with short sleep duration‡ n = 373
Moderate-High aMed score (≥5)			
Model 1	**0.68 (0.50, 0.92)****	0.91 (0.72, 1.14)	1.03 (0.80, 1.32)
Model 2	**0.67 (0.49, 0.90)****	0.92 (0.73, 1.16)	1.04 (0.80, 1.34)
Model 3	**0.66 (0.48, 0.92)****	0.90 (0.70, 1.15)	0.94 (0.71, 1.24)
Model 4	**0.65 (0.45, 0.93)***	0.92 (0.71, 1.21)	0.91 (0.67, 1.23)

†Reference= low aMed score (<5).
‡Reference = No insomnia symptoms or short sleep duration (n = 894).
MODEL 1: adjusted for age, gender, race/ethnicity.
MODEL 2: adjusted for covariates in Model 1 as well as education.
MODEL 3: adjusted for covariates in Model 2 as well as cigarette smoking, intentional exercise, and total energy intake (Kcal/day).
MODEL 4: adjusted for covariates in Model 3 as well as body mass index (Kg/m^2), hypertension, diabetes, depressive symptoms (CESD score ≥ 16), AHI (continuous), antidepressants, and antipsychotic medication.
*p < 0.05; **p ≤ 0.01. Bold values indicate significance.

in the aMed score from Exam 1 to Exam 5 was not associated with 7–8 or >8 hr/night. An unchanged aMed score was not associated with sleep duration.

Associations between historical changes in aMed diet and insomnia symptoms

Participants with unchanged aMed score from Exam 1 to Exam 5 were less likely to have insomnia symptoms than those with decrease in aMed score (OR = 0.61, 95% CI 0.45–0.82, $p \leq 0.01$, after full adjustment). An increase in the aMed score was not associated with insomnia symptoms (Table 4, last column).

Associations between historical changes in aMed diet and insomnia symptoms in conjunction with objectively measured short sleep duration

Table 5 shows multinomial logistic regression models that examined the association of change in the aMed score from Exam 1 to Exam 5 with insomnia symptoms with objectively measured short sleep duration. Participants with unchanged aMed score from Exam 1 to Exam 5 were less likely to have insomnia symptoms with short sleep (OR = 0.57, 95% CI 0.35–0.93, $p < 0.05$) or insomnia symptoms without short sleep (OR = 0.64, 95% CI 0.45–0.92, $p < 0.05$; vs. no insomnia symptoms or short sleep) than those with decrease in the aMed score. Participants with an increase in the aMed score from Exam 1 to Exam 5 were also less likely to have insomnia symptoms with short sleep (vs. no insomnia symptoms or short sleep; OR = 0.70, 95% CI 0.50–0.97, $p < 0.05$), but the association was no longer significant after models were adjusted for lifestyle and health factors.

Discussion

In the only study to date to assess associations between objectively estimated sleep duration, insomnia symptoms, and a Mediterranean-style diet, we report that individuals with a moderate-high aMed score were more likely than

Table 4. Odds ratios (95% CIs) for the associations of sleep duration (n = 1927) and insomnia symptoms (n = 1988) at Exam 5 with changes in aMed score between Exam 1 and Exam 5

	Sleep duration† *(reference:* <6 hr/night)			Insomnia symptoms‡ *(reference:* no insomnia)
Changes in the aMed Score from Exam 1 to Exam 5	6 to <7 hr/night n = 614	7–8 hr/night n = 516	>8 hr/night n = 219	Insomnia n = 711
No change vs. decrease in score				
Model 1	1.05 (0.77, 1.45)	1.11 (0.79, 1.54)	1.08 (0.71, 1.65)	**0.64 (0.49, 0.83)*****
Model 2	1.07 (0.78, 1.47)	1.10 (0.79, 1.54)	1.09 (0.71, 1.68)	**0.64 (0.49, 0.83)*****
Model 3	1.04 (0.74, 1.45)	1.13 (0.80, 1.60)	1.13 (0.72, 1.80)	**0.65 (0.50, 0.85)****
Model 4	0.98 (0.68, 1.40)	1.06 (0.73, 1.55)	0.96 (0.57, 1.61)	**0.61 (0.45, 0.82)****
Increase vs. decrease in score				
Model 1	**1.35 (1.04, 1.75)***	1.30 (0.98, 1.71)	1.04 (0.72, 1.50)	0.90 (0.73, 1.11)
Model 2	**1.36 (1.05, 1.76)***	1.29 (0.98, 1.71)	1.04 (0.72, 1.50)	0.90 (0.73, 1.11)
Model 3	1.30 (0.99, 1.71)	1.26 (0.94, 1.69)	1.18 (0.80, 1.75)	0.90 (0.72, 1.12)
Model 4	1.34 (0.99, 1.80)	1.30 (0.95, 1.79)	1.15 (0.74, 1.77)	0.92 (0.72, 1.17)

†For sleep duration (ref <6 hr, n = 578).
‡For insomnia symptoms (ref no insomnia symptoms, n = 1277).
MODEL 1: adjusted for age, gender, race/ethnicity.
MODEL 2: adjusted for covariates in Model 1 as well as education.
MODEL 3: adjusted for covariates in Model 2 as well as cigarette smoking, intentional exercise and total energy intake (Kcal/day).
MODEL 4: adjusted for covariates in Model 3 as well as body mass index (Kg/m^2), hypertension, diabetes, depressive symptoms (CESD score ≥ 16), AHI (continuous), antidepressants, and antipsychotic medication, plus insomnia symptoms when main outcome was sleep duration and sleep duration when main outcome was insomnia symptoms
*$p < 0.05$; **$p \leq 0.01$; ***$p \leq 0.001$. Bold values indicate significance.

Table 5. Odds ratios (95% CIs) for the associations between insomnia symptoms with short sleep duration at Exam 5 and changes in aMed score between Exam 1 and Exam 5 (n = 1896)

Changes in the aMed score from Exam 1 to Exam 5	Insomnia with short sleep duration† n = 217	Insomnia without short sleep duration† n = 464	No insomnia with short sleep duration† n = 353
No change vs. decrease in score			
Model 1	**0.60 (0.39, 0.91)***	**0.64 (0.46,0.88)****	1.00 (0.72, 1.41)
Model 2	**0.60 (0.39, 0.91)***	**0.64 (0.46,0.88)****	1.00 (0.72, 1.41)
Model 3	**0.61 (0.39, 0.96)***	**0.64 (0.45,0.89)****	0.96 (0.67, 1.37)
Model 4	**0.57 (0.35, 0.93)***	**0.64 (0.45, 0.92)***	1.05 (0.72, 1.53)
Increase vs. decrease in score			
Model 1	**0.70 (0.50, 0.98)***	0.92 (0.71, 1.19)	0.84 (0.63, 1.13)
Model 2	**0.70 (0.50, 0.97)***	0.92 (0.71, 1.19)	0.84 (0.63, 1.13)
Model 3	0.73 (0.51, 1.04)	0.87 (0.67, 1.14)	0.79 (0.58, 1.08)
Model 4	0.71 (0.49, 1.04)	0.92 (0.69, 1.23)	0.77 (0.55, 1.08)

†Reference = No insomnia symptoms or short sleep duration (n = 862).
MODEL 1: adjusted for age, gender, and race/ethnicity.
MODEL 2: adjusted for covariates in Model 1 as well as education.
MODEL 3: adjusted for covariates in Model 2 as well as cigarette smoking, intentional exercise and total energy intake (Kcal/day).
MODEL 4: adjusted for covariates in Model 3 as well as body mass index (Kg/m^2), hypertension, diabetes, depressive symptoms (CESD score ≥ 16), AHI (continuous), antidepressants, and antipsychotic medication.
*$p < 0.05$; **$p < 0.01$. Bold values indicate significance.

those with low aMed score to sleep 6–7 hr/night than <6 hr/night. Individuals whose aMed score increased over the preceding 10 year period were also more likely, although not significantly, than those whose score decreased to sleep 6–7 hr/night than <6 hr/night. Although we did not find an association of the aMed score with a sleep duration of 7–8 or >8 hr/night, 6–7 hr/night of sleep duration vs. <6 hr/night was consistently associated with higher aMed scores in cross-sectional and analyses of change in score. The sleep duration most typically associated with health benefits is the 7–8 hr/night range [37] based on self-reports. Since actigraphy estimates of sleep duration are approximately 1 hr less than self-report [38, 39], our findings showing that higher aMed score associated with 6–7 hr/night of actigraphy-estimate sleep duration is consistent with the favorable health profiles reported to associate 7–8 hr/night by self-report.

We also found that participants with a moderate-high aMed score were less likely than those with a low score to have insomnia symptoms with short sleep duration. This is the first epidemiological study to the best of our knowledge to document that an inverse association between an exposure to higher aMed score and insomnia symptoms occurring with objectively measured short sleep duration. This finding is important because insomnia symptoms in conjunction with short sleep duration carry a high risk of cardiometabolic outcomes. Insomnia accompanied by objective short sleep duration has been associated

with increased risk of hypertension [21] and diabetes [22], and increased risk of morbidity and mortality [21–25].

In our analysis of change of the aMed score, we also found that participants with unchanged aMed score from the preceding 10 year period were less likely to present insomnia symptoms and this association was independent of sleep duration hours. However, we did not find significant association between increased aMed score from Exam 1 to Exam 5 and insomnia symptoms or insomnia symptoms in conjunction with short sleep duration.

Our data support evidence from previous cross-sectional and longitudinal studies that sleep is associated with the type and quantity of food that people eat such that those who have lower quantity and quality of sleep are more likely to consume a less healthful diet, indicated by more snacks and less fruits and vegetables, which may increase the risk of weight gain [10, 40]. Although the relationship between dietary patterns and sleep duration and sleep quality may be bidirectional, intervention studies suggest a casual association between diet and sleep: Higher fat and carbohydrate intakes close to bedtime have been associated with higher sleep latency [41]. More rrecently, in a randomized-crossover study, low fiber and high saturated fat and sugar intake were associated with less deep sleep and more nocturnal arousals during sleep [11].

Most studies that have investigated the associations between diet and sleep have used specific nutrient or type of food, whereas in the Mediterranean diet, the scores represent an overall profile to adherence to several components of a dietary intake and the interaction among the components. Few studies have examined the association of a Mediterranean diet pattern with sleep considering the Mediterranean diet as the exposure. In a cross-sectional study with more than 5000 French participants, a Mediterranean diet pattern was protective of insomnia symptoms only among women [27]. In a recent study with ≥60-year-old participants from Spain, higher Mediterranean Diet Adherence Screener score was associated with a lower risk of large changes (≥2 hr) in self-reported sleep duration and with better self-reported sleep quality over a follow-up of 2.8 years [26]. Our study adds to these findings by using objectively measured sleep duration in a multiethnic U.S. cohort. Objective estimation of sleep overcomes the misclassification of sleep duration that results from use of self-report. For example, in both the CARDIA Sleep Study [38] and the Study of Latinos [42], self-report mean sleep duration was almost an hour greater than the actigraphy mean sleep duration. Furthermore, there is only weak to moderate agreement in classification of short and long sleep, underscoring the need for objective measurements to elucidate relationships between sleep and health-related behaviors.

One mechanism postulated to explain the benefits of the Mediterranean diet is an anti-inflammatory effect [43] together with reduced oxidative stress [44]. In regard to sleep, increased neuroinflammation is postulated to contribute to poor sleep [45]. The Mediterranean diet has a high level of monounsaturated fatty acids which has been linked to a decrease in the risk of CVD [17] and to improved glycemia and insulin resistance, especially among patients with type 2 diabetes [46]. It has also been described that the Mediterranean diet improves glucose and cholesterol homeostasis [47, 48], which may help not only to control weight [48] but also to improve brain function and mood, which are important to sleep. Although the impact of dietary interventions on cognition is unclear, improved glucose and LDL cholesterol were associated with less global cognitive decline, among more than 2100 T2D participants aged 55 years or older [49]. An alternative explanation linking the Mediterranean diet with less short sleep and less insomnia symptoms may relate to the effects of specific dietary components of sleep processes. For example, the Mediterranean diet contains a high content of plants and seeds, which contain melatonin and serotonin at various levels [50, 51]. Serotonin and melatonin are known to interact with sleep–wake brain centers; low serotonin levels may result in sleep disruption and sleep disorders [52], whereas melatonin plays a fundamental role in sleep initiation [53].

Our study has several limitations. First, although we used aMed change over 10 years to address possible reverse causation, we had objective measurements of sleep at only one time. Second, the possibility of measurement error in the assessment of the exposure, aMed cannot be excluded nor can it be excluded from actigraphy. Third, diet and sleep data at Exam 5 were not collected at the same time. The median interval between those measurements was approximately 1 year (diet preceding sleep measurement). However, in a separate study in participants with and without insomnia symptoms, sleep duration was moderately to strongly stable (intrascorer intraclass correlation coefficient ≥ 0.60) based on the average of three consecutive nights separated by an average of 2.6 years [54]. Fourth, correlation of aMed score at both Exams (Exam 1 and Exam 5) was only moderate ($R = 0.46$)—it is not clear the extent to which this reflects error in the diet assessment, or true change in dietary intake. Promisingly, the mean aMed score was similar at both exams (4.5 at baseline vs. 4.7 at Exam 5) as was the percentage of the lowest score of aMed (49% of the sample at baseline vs. 47% at Exam 5). Our sample size also limited us to classify change based on a one unit change. Thus, power was limited in the change analyses. However, among the participants who had an increase in the aMed score from Exam 1 to Exam 5, the percentage of those with an aMed score ≥5 at baseline was 32 per cent, whereas it was 76 per cent at Exam 5. Even as little as a two-point increase in adherence to the Mediterranean diet has been associated with a reduction in risk of CVD and overall mortality in a meta-analysis of cohort studies [14, 55]. Fifth, we assessed insomnia using a validated questionnaire rather than a clinical assessment. Sixth, we did not have enough power to investigate the role of race/ethnicity or other demographic factors in the association of aMed with sleep. Seventh, we could not investigate the effect of aMed in the longer sleepers because in our sample, based on actigraphy data, only 1.9 per cent participants slept ≥9 hr and our data did not suggest a U-shaped pattern. Finally, we cannot rule out a coadoption of healthy diet and sleep behaviors [1] that may explain our findings.

These limitations need to be considered in the light of several strengths. MESA is a large, multiethnic cohort study, which may increase the generalizability of our results to the U.S. population as whole. We also present the first study that examines the association between Mediterranean diet pattern and insomnia symptoms occurring in conjunction with objectively measured sleep duration collected using 7 day actigraphy.

Our results suggest that in the MESA sample, consumption of a Mediterranean-style diet has more favorable sleep patterns. Mechanistic studies are needed to examine how diet may influence sleep, independent of health and mood. Clinical trials are also needed in which adherence to the Mediterranean diet and its relationship with sleep patterns can be assessed more precisely.

Supplementary Material

Supplementary material is available at *SLEEP* online.

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