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Journal

Chronobiology International, 39(8)

ISSN

0742-0528

Authors

Maghsoudipour, Maryam
Allison, Matthew A
Patel, Sanjay R
et al.

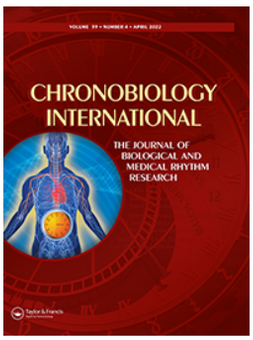
Publication Date

2022-08-03

DOI

10.1080/07420528.2022.2069030

Peer reviewed



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Chronobiology International

The Journal of Biological and Medical Rhythm Research

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/icbi20

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To cite this article: Maryam Maghsoudipour, Matthew A. Allison, Sanjay R. Patel, Gregory A. Talavera, Martha Daviglus, Phyllis C. Zee, Kathryn J. Reid, Nour Makarem & Atul Malhotra (2022): Associations of chronotype and sleep patterns with metabolic syndrome in the Hispanic community health study/study of Latinos, Chronobiology International, DOI: 10.1080/07420528.2022.2069030

To link to this article: https://doi.org/10.1080/07420528.2022.2069030

CHRONOBIOLOGY INTERNATIONAL
https://doi.org/10.1080/07420528.2022.2069030

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ORIGINAL ARTICLE

Associations of chronotype and sleep patterns with metabolic syndrome in the Hispanic community health study/study of Latinos

Maryam Maghsoudipour[a], Matthew A. Allison[b], Sanjay R. Patel[c], Gregory A. Talavera[d], Martha Daviglus[e], Phyllis C. Zee[f], Kathryn J. Reid[f], Nour Makarem[g], and Atul Malhotra[a]

[a]Department of Medicine, University of California San Diego, La Jolla, California, USA; [b]Department of Family Medicine, University of California San Diego, La Jolla, California, USA; [c]Department of Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, USA; [d]Department of Psychology, San Diego State University, San Diego, California, USA; [e]Institute for Minority Health Research, University of Illinois at Chicago, College of Medicine, Chicago, Illinois, USA; [f]Center for Circadian and Sleep Medicine, Department of Neurology, Feinberg School of Medicine, Northwestern University, Chicago, Illinois, USA; [g]Department of Epidemiology, Mailman School of Public Health, Columbia University Irving Medical Center, New York, USA

ABSTRACT

Sleep duration, sleep efficiency, and sleep timing have been shown to have potential effects on metabolic functions relevant to circadian rhythms. It is not clear if the impact of sleep patterns on metabolic risk factors is through sociocultural and environmental factors or circadian misalignment. We investigated the associations of sleep patterns, chronotype, and social jet lag with metabolic syndrome among non-shift worker Hispanic/Latino adults. We used cross-sectional data from the Sueño Ancillary Study of The Hispanic Community Health Study/Study of Latinos (HCHS/SOL). Data from a subsample of 2189 participants aged 18–64 years were used in the analysis. Mean nightly sleep duration, mean sleep onset time, mean sleep offset time, mean sleep midpoint time, sleep efficiency, sleep variability (standard deviation (SD) of sleep duration, and SD of sleep midpoint), and time spent above light exposure threshold (1000 lux) in a day were assessed by wrist actigraphy (Acti-watch Spectrum). Chronotype was determined by the reduced Morningness-Eveningness Questionnaire. Medical conditions including dyslipidemia, hypertension, and diabetes mellitus were determined from a fasting blood specimen and physical exam at the baseline visit. To determine whether sleep patterns, light levels, chronotype, and social jetlag are associated with metabolic syndrome, multivariable logistic regression models were fitted, including variables with $P < .15$ in the univariate analysis. The results of the multivariable analysis demonstrated that in participants older than 40 years, intermediate chronotype (vs early) was significantly associated with a higher risk of metabolic syndrome (Odds ratio (95%CI): 1.33 (1.04,1.7)), while later chronotype (vs. early) in participants younger than 40 years was significantly associated with a lower risk of metabolic syndrome (Odds ratio (95%CI): 0.37 (0.14, 0.96)). Also, higher sleep efficiency was significantly associated with decreased odds of metabolic syndrome (Odds ratio (95%CI): 0.98 (0.96, 0.99)). Nightly sleep duration was not significantly different between two groups of participants with and without metabolic syndrome in multivariable analyses. There was no significant association between social jet lag and metabolic syndrome in multivariable analysis ($p = .286$). Moreover, there was no significant association between chronotype and social jet lag in multivariable analysis. The association between metabolic syndrome and chronotype is age-dependent. While early chronotype is associated with metabolic syndrome in younger individuals, it tended to be associated with lower odds for metabolic syndrome in older individuals.

ARTICLE HISTORY
Received 21 December 2021
Revised 5 April 2022
Accepted 16 April 2022

KEYWORDS
Sleep patterns; chronotype; circadian misalignment; metabolic syndrome

Introduction

Chronotype is defined as a person's preferred timing of sleep and waking behavior, ranging from early or "morning" type to late or "evening" type and the intermediate type falling between them (Wittmann et al. 2006; Ellis et al. 2009). Human preferences in the timing of sleep and wake are, at least partly, based on genetics. However, the genetics behind chronotype are complex, since they are also modulated by age, gender, work schedule, personality, sun time, and light exposure, among other factors (Martinez-Nicolas et al. 2019; Roenneberg et al. 2003). Moreover, the timing of sleep is driven by both endogenous circadian rhythms that regulate sleep propensity, as well as by sociocultural factors that influence behavior (Knutson et al. 2017).

CONTACT Maryam Maghsoudipour mamaghsoudipour@health.ucsd.edu Sleep Research Laboratory, University of California, San Diego, 9452 Medical Center Drive La Jolla, CA 92037

Signals from the exogenous environment (light) and endogenous metabolism are integrated in the CNS (Central Nervous System), and the output in turn, imparts rhythmicity on sleep and a variety of metabolic outputs, such as thermogenesis, feeding behavior, hormone secretion, and locomotor activity. Hence, the disruption of circadian rhythm results in metabolic dysregulation (Huang et al. 2011). Light mediates the effects via SCN (Supra-Chiasmatic Nucleus) and also directly affects other brain structures, which regulate some physiological functions like sleep, glucocorticoid levels, and heart rate (Rumanova et al. 2020).

Previous studies have shown that alterations in sleep efficiency, sleep duration, and sleep timing have an impact on cardiometabolic profile or metabolic disease risk independent of other obesity-related behaviors (Cespedes Feliciano et al. 2018; Merikanto et al. 2013). Some have shown that there was not an association between sleep duration and cardiometabolic disorders (Ramos et al. 2018). Other studies have examined whether later sleep timing or late chronotype is related to metabolic abnormalities independent of sleep duration and lifestyle, with the results being mixed (Hashemipour et al. 2020; Knutson et al. 2017; Yu et al. 2015).

Fritz et al. evaluated the associations between sleep regularity and metabolic health in The Hispanic Community Health Study/Study of Latinos (HCHS/SOL) and showed lower Sleep Regularity Index (SRI) was associated with higher odds of diabetes and the SRI effect was more prominent in older (aged ≥ 45 years) adults (Fritz et al. 2021). Knutson et al. also evaluated the participants from HCHS/SOL and demonstrated that in younger participants, later sleep timing was associated with lower cardiometabolic risk factors. In their study, sleep timing was determined from self-reported bedtimes and wake times and not by actiwatches (Knutson et al. 2017).

In addition, "social jetlag," a form of circadian misalignment due to a mismatch between social rhythms and the circadian clock, has been shown to be associated with insulin resistance, and higher metabolic risk factors. (Wong et al. 2015). Social jetlag is a measure of the difference in sleep timing between workdays and weekends. Unlike travel-induced jetlag, social jetlag occurs throughout working life (Parsons et al. 2015). Some researchers have speculated that individuals with later chronotype experience a circadian misalignment due to incongruity between social rhythms and the circadian clock (Wittmann et al. 2006). Notably, some investigations measured chronotype and social jetlag simultaneously and the associations between metabolic parameters and chronotype were not significant after adjusting for social jetlag (Parsons et al. 2015), while other investigators demonstrated that later chronotype was associated with higher HbA1c levels in patients with prediabetes, independent of social jetlag (Anothaisintawee et al. 2017). Hence, there is no consensus whether the chronotype effects on metabolic risk factors are related to social jetlag or just related to the sleep patterns independently. In other words, it is not clear if the impact of sleep patterns on metabolic risk factors is through sociocultural and environmental factors or circadian misalignment.

Moreover, chronic metabolic diseases, such as obesity and diabetes, constitute a significant burden on Americans, and Hispanics/Latinos are disproportionately affected compared to non-Hispanic Whites (Ogden et al. 2006). While a few investigators evaluated the associations between sleep patterns and metabolic syndrome in non-Hispanic White populations, such studies on Hispanic/Latino populations are sparse. Considering that metabolic syndrome is more prevalent in these communities, we decided to examine whether chronotype, social jetlag, and sleep patterns are associated with metabolic syndrome in Hispanic/Latino populations independent of lifestyle factors.

Methods

Population

The Hispanic Community Health Study/Study of Latinos (HCHS/SOL) is a multicenter community-based cohort study examining the prevalence of and risk factors for chronic disease among 16,415 Hispanic/Latino adults from four urban areas (Bronx NY, Miami FL, Chicago IL, San Diego CA), between the ages of 18 and 74 years (Lavange et al. 2010; Sorlie et al. 2010). All participants underwent baseline assessments from 2008 through 2011. The study was approved by relevant institutional review boards. All participants provided informed consents and completed the questionnaires in either English or Spanish. Survey questionnaires were completed to assess ethnic background, employment status, education, and sleep related information (Bastien et al. 2001; Johns 1991; Lind et al. 2003).

Sueño ancillary study

From December 2010 to December 2013, the Sueño ancillary study to the HCHS-SOL recruited a subsample of 2252 HCHS/SOL participants aged 18–64 years and free of both narcolepsy and severe obstructive sleep apnea (Apnea Hypopnea Index (AHI) <50 events/hr and no positive airway pressure treatment for sleep apnea).

Participants in the Sueño ancillary study were asked to wear an Actiwatch Spectrum device (Philips Respironics, Murrysville, PA) for 7 days on their non-dominant wrist.

The participants were informed that there was a light sensor on the Actiwatch, and they had to keep it uncovered as much as possible. Also, they were instructed to press the buttons on either side of the Actiwatch face for 3 seconds to mark when they were going to sleep or wake up. Participants were also asked to complete a daily sleep log for seven days of study (Patel et al. 2015a, 2015b).

Actigraphic sleep measurements including sleep duration, sleep timing, sleep efficiency, and sleep variability were calculated from actigraphy. The Acti-watch Spectrum collected light and activity data in 30-sec epochs and data were transmitted to the reading center at Brigham and Women's Hospital to be scored based on previously validated algorithms (Kushida et al. 2001; Marino et al. 2013; Patel et al. 2015b). Days with more than 4 hours of missing data, were considered invalid. Studies with five or more than five days of valid data have been included in data analyses.

In our study, all Sueño participants with complete actigraphy measures were included, while participants employed as shift workers, or unusual work schedules, were excluded. Shift work was identified as any regular work at night, rotating, split, or irregular shift.

Exposures of interest

Actigraphic variables of interest were mean nightly sleep duration, mean napping duration, mean 24-h sleep duration, mean sleep latency, mean sleep maintenance efficiency, mean sleep fragmentation index, mean sleep onset time, mean sleep offset time, and mean sleep midpoint time, as well as the standard deviation (SD) of sleep duration, and SD of sleep midpoint. Mean nightly sleep duration was calculated as the total amount of time (in minutes) scored as sleep within each main rest interval averaged over the number of valid days of recording. Mean sleep onset time is the clock time (HH:MM) of the first epoch scored as sleep in each main rest interval averaged across all main rest intervals containing sleep. Mean sleep offset time is the clock time (HH:MM) of the last epoch scored as sleep in each main rest interval averaged across all main rest intervals containing sleep. Sleep midpoint was calculated as the midpoint between sleep onset and sleep offset. Mean sleep maintenance efficiency was calculated as the proportion of time from sleep onset to sleep offset in each main rest interval that was scored as sleep averaged across all valid days of recording expressed as a percentage. All sleep measures were defined as the mean averaged across all valid days in the recording.

Chronotype was determined by reduced version of Morningness-Eveningness Questionnaire (Adan and Almirall 1991; Horne and Ostberg 1976). This questionnaire is an accepted and validated measure of chronotype (Taillard et al. 2004). *Light exposure* (based on acti-watch data) in 30 second intervals was defined as time above 1000 lux threshold (TAT) in a day (white light), and above 1000 $\mu W/cm^2$ irradiance threshold (green, blue, and red light) (Reid et al. 2014). *Social Jet lag* was calculated as the difference in minutes between the midpoints of actigraphy-derived sleep intervals on workdays vs weekends (Parsons et al. 2015; Wong et al. 2015).

Outcomes

Metabolic syndrome was defined if the participant had three or more of the following: 1) A waistline of 40 inches or more for men and 35 inches or more for women; 2) Hypertension defined as a systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or use of antihypertensive medication (Chobanian et al. 2003); 3) A triglyceride level above 150 mg/dl (1.7 mmol/L); 4) HDL Cholesterol less than 40 mg/dL (1.04 mmol/L) in men or less than 50 mg/dL (1.3 mmol/L) in women; and 5) Elevated fasting blood sugar – 100 mg/dL (5.6 mmol/L) or higher (American Diabetes 2010).

Covariates

Based on previous studies, and our univariable analyses, certain covariates were selected for inclusion in the analysis (Dudley et al. 2017; Patel et al. 2015b). Education was categorized as less than high school versus at least high school. Annual household income was dichotomized as: <$30,000 and ≥$30,000. Alcohol consumption was defined as the total number of drinks per week. Caffeine consumption was defined as the number of cups of caffeinated beverages consumed per day. Tobacco smoke was classified as "Tobacco smoke" or "No Tobacco smoke." Also, smoking was assessed based on the number of cigarettes smoked per day. Marital status was classified as single, married, and widowed.

Data analysis

Independent sample t-test and ANOVA were used to compare the continuous variables between participants with and without metabolic syndrome and among different chronotypes, while the Chi square test was used to compare the categorical variables. Associations of sleep patterns, social jetlag, metabolic syndrome, and age were initially explored using line graphs and histograms.

Univariable logistic regression was used for assessing the associations between metabolic syndrome and covariates. Odds ratios with 95% CIs were computed. To determine whether sleep patterns and chronotypes were associated with metabolic syndrome, multivariable logistic regression models were fitted, including covariates with p < .15 in the univariate analysis. Also, multivariable linear regression models were fitted to evaluate the associations between sleep patterns and chronotypes with components of metabolic syndrome (fasting blood sugar, triglyceride level, HDL cholesterol, and waist). Models were adjusted for age, gender, Hispanic/Latino background, household income, education, marital status, AHI, and employment. The sleep parameters as a function of age were plotted and explored. As the effect of age on the outcome of sleep patterns and parameters varied in participants older and younger than 40 years, further sensitivity analysis was performed in these age strata.

Further models were fitted to determine the influence of social jetlag on metabolic syndrome or chronotype while adjusting for confounders. Finally, the interaction of social jetlag with chronotype was included in the model to explore if the association of chronotype and metabolic syndrome differed by social jet lag status.

Statistical analyses were performed using statistical software Stata 14.2 (Stata Corp LLC, College Station, TX). P values less than .05 were considered statistically significant.

Results

Of the 2189 participants in the Sueño ancillary study meeting eligibility criteria, 2166 (98%) had ≥5 days of valid actigraphy data. After exclusion of shift workers (n = 297), 1,869 participants with a mean age (SD) of 46.9 (11.6) years were included in the final analysis; 1225 (65.5%) were women and 629 (33.6%) had metabolic syndrome, 981 (52.4%) had early chronotype, 711 (38.0%) had intermediate chronotype, and 177 (9.5%) had late chronotype.

Demographics of participants by metabolic syndrome status are shown in Table 1. Participants with metabolic syndrome were significantly older (p <.001), had attained lower education levels (p < .001), and had lower household income (p < .001). Marital status was significantly different between participants with and without metabolic syndrome (p < .001). Current employment was associated with lower likelihood of being in the metabolic syndrome group. Gender, Hispanic/Latino background, caffeine consumption and alcohol consumption were not significantly different between metabolic and non-metabolic syndrome participants (p > .05), (Table 1).

Table 2 shows sleep patterns between metabolic and non-metabolic syndrome participants. Nightly sleep durations (Average in bed duration from main sleep over all days in minutes) were not significantly different between two groups, (p > .05). The average sleep maintenance efficiency (%) was significantly lower in metabolic syndrome group (all days and weekends, p = .09 and .006, respectively). Time of light exposure above threshold (1000 lux) in 24 hours a day was not significantly different between the two metabolic syndrome groups.

Characteristics of participants with different chronotypes are presented in Table 3. Early-type participants were significantly older (p < .001) while late-type participants consumed significantly more alcoholic drinks per week (p = .007), and more cups of caffeine beverages per day (p < .001). Marital status rate was significantly different between chronotype groups. While the proportion of single participants to married was lower in the early-type group, this proportion was higher in the late type (p < .001). White and green light exposure times above thresholds (min) were significantly different between different chronotypes and early chronotypes had increased time of exposure above threshold compared to late chronotypes. Nightly average sleep duration (min) between different chronotypes was not significantly different (p > .05), but the standard deviation of total sleep duration and standard deviation of total sleep maintenance efficiency were significantly higher in late chronotypes (p < .001 and p = .019, respectively) (Table 3).

Table 4 presents results of univariable logistic regression for associations between sleep patterns and metabolic syndrome. The sleep maintenance efficiency (per 10% increase) odds ratio for all days was 0.85 (0.7, 1.03), (p = .094), while being 0.79 (0.67, 0.94), (p = .007) for weekends. The association of mean nightly sleep duration and metabolic syndrome was not significant (for all days and weekends). The associations of sleep timings (sleep onset time, sleep midpoint time, and sleep offset time) with metabolic syndrome were significant and every hour of later sleep timing was associated with lower likelihood of metabolic syndrome (OR < 1). However, the associations between mean nightly sleep duration, and sleep timings with metabolic syndrome were not significant after adjustment for confounders. Higher sleep efficiency was significantly associated with decreased odds of metabolic syndrome (Odds ratio (95%CI): 0.98 (0.96, 0.99), p = .04). The association of AHI >15/h with metabolic syndrome after adjustment for cofounders was significant (Odds ratio (95%CI): 2.76 (1.94, 3.92), p < .001).

The association of chronotype with metabolic syndrome after adjustment for confounders in participants younger than 40 years and older than 40 years are shown in Figures 1 and 2, respectively. Intermediate chronotype

Table 1. Demographic characteristics of participants with metabolic syndrome and without metabolic syndrome.

Variable	Metabolic N = 629	Non-metabolic N = 1,240	*P*-VALUE
Age (year)	51.3 ± 9.5	45.1 ± 11.9	<.001*
Gender (Female: Male)	430: 199 (68.3%: 31.7%)	795: 445 (64.1%: 35.9%)	.068**
Body Mass Index (Kg/m^2)	32.9 ± 5.9	28.6 ± 5.7	<.001*
Education (Less than high school: At least high school)	246:383 (39.1%:60.9%)	377:860 (30.4%:69.6%)	<.001**
Household Income (<30 K: ≥30 K)	443:145 (75.3%:24.7%)	773:371 (67.6%:32.4%)	.001**
Marital Status (Single: Married: Widow)	142:364:121 (22.7%:58.0%:19.3)	385:624:228 (31.1%:50.4%:18.43%)	<.001**
Caffeine Consumption (cups/day)	2.3 ± 1.9	2.3 ± 2.2	.590*
Alcohol Consumption /week (Total drinks/WEEK)	4.3 ± 7.7	4.6 ± 8.4	.690*
Apnea hypopnea index ≥15.0 events/hrs.	100: 521 (16.1%: 83.9%)	62: 1160(5.0%:95.0%)	<.001**
Smoking now (Cigarettes/day)	11.4 ± 8.7	10.4 ± 8.1	.345*
Tobacco smoke (No: Yes)	228:21(91.6%: 8.4%)	552:77 (87.7%: 12.2%)	.106**
Current Employment (No: Yes)	358: 271 (56.9%: 43.1%)	549: 691 (44.3%: 55.7%)	<.001 **

* Student T test
** Chi-square test

Table 2. Descriptive characteristics of sleep patterns in participants with metabolic syndrome and without metabolic syndrome.

Variable	Metabolic	Non-metabolic	*P*-VALUE
Nightly Avg sleep duration (min)			
All days (min)	464.1 ± 69.2	457.9 ± 65.6	.059*
Weekdays (min)	460.2 ± 74.3	453.1 ± 70.3	.044*
Weekends (min)	473.9 ± 89.1	470.9 ± 94.6	.518*
Standard Deviation of total sleep duration	77.06 ± 37.9	81.30 ± 41.9	.033*
Sleep latency (min)			
All days (min)	11 ± 15.4	11.1 ± 16	.849*
Weekdays (min)	11 ± 14.5	11.2 ± 14.1	.718*
Weekends (min)	10.8 ± 19.4	10.8 ± 26.6	.962*
Sleep maintenance efficiency (%)			
All days	88.4 ± 4.9	88.8 ± 5	.094*
Weekdays	88.5 ± 5	88.7 ± 5.3	.281*
Weekends	88.2 ± 5.9	89 ± 5.4	.006*
Standard Deviation of total sleep maintenance efficiency	3.7 ± 2.5	3.6 ± 3	.556*
Avg Sleep onset time			
All days	23:46:11 ±1:21:08	23:57:06 ± 1:27:23	.017*
Weekdays	23:37:26 ± 1:22:03	23:43:59 ± 1:27:54	.067*
Weekends	0:10:12 ± 1:36:38	0:25:30 ± 1:48:08	.003*
Standard Deviation of total Sleep onset time	57.8 ± 33.8	62.9 ± 38.9	.035*
Avg Sleep offset time			
All days	7:31:29 ± 1:23:59	7:35:51 ± 1:27:04	.316*
Weekdays	7:18:22 ± 1:28:49	7:18:22 ± 1:31:46	.860*
Weekends	8:04:15 ± 1:38:07	8:17:21 ± 1:45:47	.008*
Standard Deviation of total Sleep offset time	60.9 ± 33.1	65.5 ± 38.7	.038*
Avg Sleep midpoint time			
All days	3:39:55 ± 1:15:01	3:46:28 ± 1:20:55	.067*
Weekdays	3:26:49 ± 1:17:05	3:31:11 ± 1:22:46	.283*
Weekends	4:08:19 ± 1:26:35	4:21:26 ± 1:36:11	.002*
Standard Deviation of total Sleep midpoint time	48.7 ± 26.5	53.4 ± 31.7	.013*
Chronotype (1:2:3)	340: 238: 51 (54.05%: 37.84%:8.11)	641: 473: 126 (51.69%: 38.15: 10.16)	.071**

*Student t test.
**chi-square test.

(Odds ratio (95%CI): 0.48 (0.26, 0.87)) and late chronotype (Odds ratio (95%CI): 0.37 (0.14, 0.96)), were associated with lower metabolic syndrome in participants younger than 40 years (Figure 1). Conversely, in participants older than 40 years, intermediate chronotype (Odds ratio (95%CI): 1.33 (1.04, 1.7)) and late chronotype (Odds ratio (95%CI): 1.11 (0.72, 1.72)) were associated with increased risk of metabolic syndrome (Figure 2). With older age, the association of later chronotype with metabolic syndrome was increased (interaction of chronotype with age p-value = .004), (Intermediate/early: $\beta = 0.32 \pm 0.10$, $p = .003$; Late/early: $\beta = 0.04 \pm 0.17$, p-value = .028). The association of chronotype and metabolic syndrome did not vary at different levels of social jet lag (p-value = .773).

The associations between social jet lag and metabolic syndrome after adjustment for age were not significant in all participants, participants younger than 40 years

Table 3. Characteristics of the participants with different chronotypes.

	Early type	Normal	Late type	*P*-value
Age(year)	49.8 ± 10	44.8 ± 12.1	42.1 ± 13.4	<.001*
Gender (F/M)	629:352(64.12:35.88)	483:228(67.93:32.07)	113:64(63.84:36.16)	.257**
BMI (Kg/m^2)	29.9 ± 5.8	30.2 ± 6.2	30.6 ± 7.2	.257*
Education				
(Less than high school: At least high school)	340:639(34.73:65.27)	228:482(32.11:67.89)	55:122(31.07:68.93)	.419**
House-hold income				
(<30k: ≥30 K)	629:295(68.07:31.93)	470:178(72.53:27.47)	117:43(73.13:26.88)	.114**
Apnea hypopnea index ≥15.0 events/hrs. (1:0)	90:880(9.28:90.72)	56:641(8.03:91.97)	16:160(9.09:90.91)	.669**
Alcohol Consumption /week (TOTALDRINKS/WEEK)	4.6 ± 8.8	4.2 ± 7.1	5.8 ± 8.9	.267**
Alcohol Use Drinking Level (ALCOHOL USE LEVEL)				.021**
(No current use)	550	385	100	
(Low level use)	392	297	61	
(High level use)	38	29	16	
Caffeine Consumption (cups/day)	2.2 ± 1.9	2.3 ± 2.1	2.9 ± 3.1	<.001*
Smoking now (Cigarettes/day)	11.2 ± 8.4	10.5 ± 8.6	10.3 ± 7	.749*
Tobacco smoke (No: Yes)	453:60	279:32	48:6	.824**
Marital Status				
(Single:Married:Widow)	208:582:188 (21.27:59.51:19.32)	241:340:128 (33.99:47.95:18.05)	78:66:33 (44.07:37.29:18.64)	<.001**
Avg White light time above threshold (TAT) 1000 lux (min)				
All days (min)	94.1 ± 83.3	85.2 ± 76.8	67.7 ± 61.2	<.001*
Weekdays (min)	79.1 ± 73.9	71.6 ± 67.1	58.4 ± 54.6	<.001*
Weekends (min)	62.2 ± 63.3	56.8 ± 62.7	41.6 ± 49	<.001*
Avg Green light time above 1000 μW/cm^2 irradiance threshold (min)				
All days (min)	27 ± 50.9	22.2 ± 28.1	17.2 ± 20.1	.010*
Weekdays (min)	21.9 ± 43.6	18.4 ± 24	15.1 ± 18.8	.020*
Weekends (min)	18 ± 39.4	15.4 ± 24.9	10 ± 14.6	.007*
Avg Blue light time above 1000 μW/cm^2 irradiance threshold(min)				
All days (min)	11 ± 45.4	9.1 ± 12.9	6.7 ± 7.9	.211*
Weekdays (min)	9.2 ± 37.1	7.5 ± 11	6.1 ± 7.5	.254*
Weekends (min)	7.5 ± 32.8	6.4 ± 11.7	3.5 ± 4.8	.137*
Avg Red light time above 1000 μW/cm^2 irradiance threshold (min)				
All days (min)	14.6 ± 46.6	12 ± 16.3	9.3 ± 11	.104*
Weekdays (min)	12.1 ± 38.3	9.9 ± 14	8.3 ± 10.5	.145*
Weekends (min)	9.9 ± 34	8.3 ± 14.3	5 ± 6.8	.057*
Nightly Avg sleep duration (min)				
All days (min)	460.2 ± 63.5	459.7 ± 68.1	459.7 ± 79.7	.988*
Weekdays (min)	455.9 ± 67.6	454.8 ± 73.2	456.3 ± 86.9	.938*
Weekends (min)	471.1 ± 90.4	473.4 ± 93.7	470.2 ± 102.3	.852*
Standard Deviation of total sleep duration	74.4 ± 39.5	83.9 ± 40	94.1 ± 44.3	<.001*
sleep latency (min)				
All days (min)	10.9 ± 19.2	11.1 ± 10.4	12 ± 12.9	.677*
Weekdays (min)	10.7 ± 16.2	11.4 ± 11.5	12.4 ± 13	.313*
Weekends (min)	11.2 ± 30.7	10.2 ± 13.5	11.1 ± 17.8	.713*
Sleep maintenance efficiency (%)				
All days	88.8 ± 4.9	88.6 ± 5	88 ± 5	.141*
Weekdays	88.8 ± 5.1	88.5 ± 5.3	88 ± 5.1	.104*
Weekends	88.8 ± 5.5	88.8 ± 5.5	88.1 ± 5.8	.292*
Standard Deviation of total sleep maintenance efficiency	3.5 ± 2.8	3.8 ± 2.8	3.9 ± 2.9	.019*
Avg Sleep efficiency (%)				
All days	86.1 ± 6.4	85.8 ± 5.9	85.1 ± 5.9	.127*
Weekdays	86.1 ± 6.4	85.6 ± 6.3	84.9 ± 6.1	.044*
Weekends	86 ± 7.4	86.1 ± 6.7	85.4 ± 6.9	.525*
Avg Sleep onset time				
All days	3:13:42 ± 1:03:15	4:01:46 ± 1:16:15	5:09:29 ± 1:22:43	<.001*
Weekdays	3:02:47 ± 1:05:31	3:48:40 ± 1:17:10	4:58:34 ± 1:30:01	<.001*
Weekends	3:46:28±1:18:05	4:41:05±1:34:24	55:40:04 ± 1:27:24	<.001*
Standard Deviation of total Sleep onset time	47.4 ± 28.1	54.1 ± 29.4	62.2 ± 34.4	<.001*
Avg Sleep offset time				
All days	23:24:20 ± 1:10:51 ± 1:10:50	0:12:23 ± 1:22:49 ± 1:22:49	1:20:06 ± 1:30:02	<.001*
Weekdays	23:13:24 ± 1:11:53	0:01:28 ± 1:22:20	1:09:11 ± 1:34:54	<.001*
Weekends	23:50:33 ± 1:31:16	0:42:58 ± 1:46:32	1:46:20 ± 1:37:09	<.001*
Standard Deviation of total Sleep offset time	56.9 ± 35.6	63.8 ± 35.7	68.4 ± 43.1	<.001*
Avg Sleep midpoint time				
All days	7:05:16 ± 1:10:20	7:53:20 ± 1:24:14	9:01:03 ± 1:33:30	<.001*
Weekdays	6:49:59 ± 1:15:11	7:35:51±1:28:26	8:45:45± 1:44:43	<.001*
Weekends	7:42:24± 1:28:43	8:37:01± 1:44:02	9:36:00 ± 1:44:57	<.001*
Standard Deviation of total Sleep midpoint time	58.1 ± 34.3	66.9 ± 36.9	80.1 ± 41.1	<.001*

*One way ANOVA.
**chi-square test.

Table 4. Associations of sleep patterns and metabolic syndrome.

Variable	Odds ratio (95% CI)	*P*-value*
Mean nightly sleep duration (per 10 min increase)		
All days	1.01 (1, 1.03)	.059
Weekdays	1.01 (1, 1.03)	.044
Weekends	1 (0.99, 1.01)	.518
SD of total sleep duration	0.97 (0.95, 1)	.033
sleep maintenance efficiency (per 10% increase)		
All days	0.85(0.7, 1.03)	.094
Weekdays	0.9(0.75, 1.09)	.281
Weekends	0.79 (0.67, 0.94)	.007
SD of Sleep efficiency	1.11(0.79, 1.55)	.556
Sleep latency (per 10 min increase)		
All days	0.99(0.93, 1.06)	.849
Weekdays	0.99(0.92, 1.06)	.719
Weekends	1 (0.96, 1.04)	.963
Sleep onset time (per hour increase)		
All days	0.92 (0.86, 0.99)	.017
Weekdays	0.94 (0.88, 1)	.067
Weekends	0.92 (0.87, 0.97)	.003
SD of total Sleep onset time	0.84 (0.71, 0.99)	.035
Sleep midpoint time (per hour increase)		
All days	0.93 (0.87, 1)	.067
Weekdays	0.96 (0.89, 1.03)	.283
Weekends	0.9 (0.85, 0.96)	.002
SD of total Sleep midpoint time	0.77 (0.63, 0.95)	.013
sleep offset time (per hour increase)		
All days	0.97 (0.9, 1.03)	.316
Weekdays	0.99 (0.93, 1.06)	.860
Weekends	0.93 (0.88, 0.98)	.009
SD of total Sleep offset time	0.84(0.72, 0.99)	.038

*Logistic regression.

and older than 40 years (p = .097, .463, .187, respectively). The results were also not different after stratifying for employment status ($p > .05$).

There was no association between social jet lag and chronotype after adjustment for age and other confounders, in all participants, participants younger than 40 years and older than 40 years (p-values = .201, .270, .643, respectively). The associations of social jet lag and chronotypes stratified by employment are shown in the Supplemental Table.

The associations between metabolic syndrome and components of metabolic syndrome with sleep efficiency and chronotype are shown in Tables 5 and 6.

Discussion

In the present study, and among a subsample of Hispanic/Latino adults participating in the Sueño ancillary study, we found that higher sleep efficiency was significantly associated with a decreased odds of the metabolic syndrome. Also, the later chronotype in participants younger than 40 years was significantly associated with a lower odds of metabolic syndrome, while in participants older than 40 years, early chronotype was significantly associated with lower odds of metabolic syndrome. We did not find any association between sleep timing and metabolic syndrome.

In our study, lower sleep efficiency was significantly associated with a significantly higher odds of metabolic syndrome. Similarly, in a study evaluating the relationship between poor sleep and metabolic syndrome in obese adolescents, decreased sleep efficiency was associated with increased waist circumference and higher triglyceride levels (Simon et al. 2020). Also, Cespedes et al. showed that higher sleep efficiency was associated with a more favorable cardiometabolic profile in adolescents (Cespedes Feliciano et al. 2018).

In our study, AHI >15/h had 2.76-fold higher odds of metabolic syndrome after adjusting for confounders. A recent systematic review and meta-analysis of adolescents with obesity found that obstructive sleep apnea is independently associated with metabolic impairment (Patinkin et al. 2017). Similar to our findings, in the cohort study of the SWAN sleep study, sleep efficiency (OR = 2.1) and AHI (OR = 1.9) were significantly associated with the metabolic syndrome and the relationships did not differ by race (Hall et al. 2012).

In our analysis, sleep timings were earlier in the metabolic syndrome group, as age was strongly associated with metabolic syndrome and older people in the present study had earlier sleep timings. No significant link was found between sleep timings and metabolic syndrome after adjusting for confounders. In a previous analytical study on Hispanic/Latino participants that recruited a total of 16,145 adults, and sleep timings were determined by questionnaires and not by objective methods. Later sleep timing was significantly associated with higher insulin resistance and higher fasting glucose in those with type 2 diabetes, but they explained that the relationship between sleep timing and some metabolic measures differed between younger (<36 years) and older individuals (36–70 years). Among those <36 years, later sleep timing was associated with lower BMI, lower fasting glucose, and lower HbA1c, but the opposite association was observed among older participants (Knutson et al. 2017). In the present analysis, sleep timing was objectively assessed by acti-watches. Thus, our sleep data are less prone to measurement error, but our sample size was smaller. Other investigators also found a link between higher BMI and later sleep timing, caloric consumption after 8:00 PM, and fast-food meals. In multivariate models, sleep timing was independently associated with calories consumed after 8:00 PM but did not predict BMI after controlling for sleep duration (Baron et al. 2011).

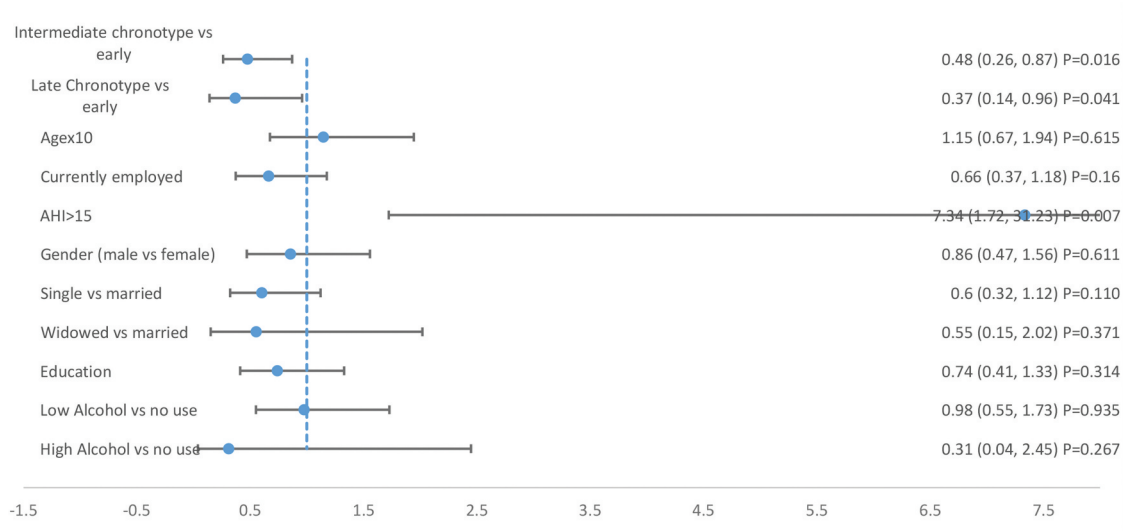

Figure 1. Association of chronotype with metabolic syndrome after adjustment for confounders in participants younger than 40 years.

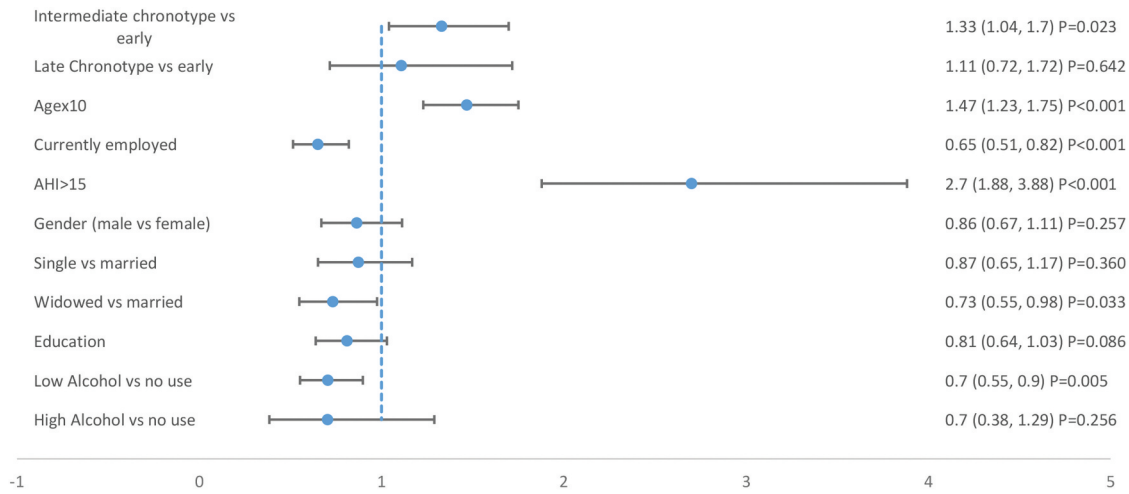

Figure 2. Association of chronotype with metabolic syndrome after adjustment for confounders in participants older than 40 years.

In our participants, the association of different chronotypes with metabolic syndrome was significantly different between younger people and older ones. That is, and in participants younger than 40 years, late chronotypes, compared to early chronotype, had a 0.37-fold odds ratio for metabolic syndrome. Conversely, and in older individuals, intermediate chronotype vs. early had a 1.33-fold odds ratio for metabolic syndrome. In another study, evening types had a 2.5-fold odds ratio for type 2 diabetes and a 1.3-fold odds ratio for arterial hypertension as compared with morning types, after adjustment for sleep duration and sleep sufficiency. (Merikanto et al. 2013) However, their study was performed on the Finnish population from January to March and the effects of long nights on the participants' metabolism and sleeping preferences might be different from our study. Another study more consistent with ours suggested that poor sleep among early type young adults might be more strongly associated with elevated blood pressure and obesity compared to participants with an intermediate chronotype (McMahon et al. 2019). Another study assessed potential associations between individual chronotype and cardiometabolic outcomes in young adults of two independent populations from Europe (Spain) and America (Mexico). Among the Mexican population, evening chronotypes showed higher risk of metabolic syndrome, but it was not significant among the equivalent Spanish chronotypes. Although, evening chronotypes showed increased levels of triglycerides in

Table 5. The associations between metabolic syndrome and components of metabolic syndrome with sleep efficiency and chronotype.

Model	Metabolic Syndrome		Hypertension*		Elevated fasting blood sugar †	
Models	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)
Model 1	0.98 (0.96, 0.99)[a]	1.2 (0.97, 1.49)/ 1.08 (0.74, 1.56)	0.96 (0.94, 0.98)[b]	1.26 (1.01, 1.6)[a]/ 1.36 (0.9, 2.06)	0.98 (0.96, 1)	1.18 (0.94, 1.47)/ 1.3 (0.89, 1.91)
Model 2	0.98 (0.96, 0.99)[a]	1.17 (0.94, 1.47)/ 0.97 (0.66, 1.43)	0.97 (0.95, 0.99)[a]	1.13 (0.89, 1.45)/ 1.08 (0.7, 1.66)	0.99 (0.97, 1.01)	1.11 (0.88, 1.41)/ 1.18 (0.8, 1.75)

Data are presented as Odd Ratio (95% CI) using multivariable logistic regression. Model 1: Adjusted for age and gender. Model 2: Adjusted for Model 1 covariates plus apnea-hypopnea index (AHI>15 vs AHI <15), Marital status, education, currently employment status, and Alcohol use level.

[a]P < .05.

[b]P < .01.

*Increased blood pressure (systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg)

†Fasting blood sugar level above 100mg/dl

Model	High triglyceride level*		Reduced HDL cholesterol†		Large waist‡	
Models	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late /Intermediate Chronotype (vs Early)
Model 1	0.99 (0.97, 1.01)	0.99 (0.8, 1.24)/ 0.95 (0.65, 1.38)	1 (0.98, 1.01)	1.1 (0.9, 1.35)/ 1.06 (0.76, 1.48)	0.98 (0.96, 1)	1.19 (0.96, 1.48)/ 1.24 (0.87, 1.78)
Model 2	0.99 (0.97, 1.01)	1 (0.8, 1.25)/ 0.96 (0.65, 1.42)	0.99 (0.97, 1.01)	1.14 (0.93, 1.41)/ 1.13 (0.8, 1.59)	0.99 (0.97, 1.01)	1.18 (0.94, 1.47)/ 1.13 (0.78, 1.64)

Data are presented as Odd Ratio (95% CI) using multivariable logistic regression. Model 1: Adjusted for age and gender. Model 2: Adjusted for Model 1 covariates plus apnea-hypopnea index (AHI>15 vs AHI <15), Marital status, education, and Alcohol use level, and currently employment status.

[a]P < .05.

[b]P < .01.

*Triglyceride level above 150 mg/dl

†HDL cholesterol less than 50 mg/dL in women or less than 40 mg/dL in men

‡A waistline that measures at least 35 inches (89 centimeters) for women and 40 inches (102 centimeters) for men

both populations, VLDL-c in Spaniards and total cholesterol and LDL-c in Mexicans(Aguilar-Galarza et al., 2021).

Investigators have found that circadian misalignment decreases leptin, increases glucose despite increased insulin, and increases mean arterial pressure. They are acute responses to circadian misalignment as occurs with jet lag (Scheer et al. 2009), or shift work (Hansen et al. 2016). In this study, we did not find any association between social jet lag and metabolic syndrome and not any between chronotype and social jet lag. Thus, social jet lag or circadian misalignments might not be explanations for a disparity in chronotype between younger and older patients who suffer from metabolic syndrome.

In our study on the Sueño ancillary participants of HCHS/SOL study, nightly sleep durations were not significantly different between the two groups of participants with and without metabolic syndrome. In a study on HCHS/SOL short sleep duration and insomnia were associated with diabetes, but short sleepers were defined by self-reported data and not acti-watches (Cespedes et al. 2016). Another previous study on HCHS/SOL reported short sleep duration is associated with obesity but not with diabetes, hypertension, and heart disease (Patel et al. 2015a). Other investigators revealed that short sleepers (≤6 hr) had a lower intake of potassium, fiber, and calcium (Mossavar-Rahmani et al. 2015). Based on a study used data from the continuous NHANES (n = 15,199), short sleepers began eating earlier and ended their eating later in the day, but daily energy intake was not different between shorter and longer sleep duration categories (Kant and Graubard 2014). None of these studies showed any difference between metabolic disorders among different sleep durations (Kant and Graubard 2014; Mossavar-Rahmani et al. 2015; Patel et al. 2015a). Similarly, in the Multi-Ethnic Study of Atherosclerosis, Bakker et al. showed that sleep duration was not significantly associated with abnormal fasting glucose after considering the influence of OSA (Bakker et al. 2015). In a systematic review and meta-analysis, the researchers included 13 studies involving 300,202 participants in which short sleep and long sleep significantly increased the risk of metabolic syndrome, 15% and 19%, respectively. From 13 studies, 11 studies were performed on Asian populations, one study (Quebec family study) from United States (193 participants), and one study (The EpiHealth cohort study) was from Sweden (19691 participants)(Che et al. 2021).

We demonstrated that average times (in minutes) of white and green light above threshold were significantly higher in early chronotype participants compared to late chronotypes. Earlier chronotypes might be exposed to brighter days compared to the later chronotypes. More studies are required to understand whether the differences in light exposure between different chronotypes, are the cause or the consequence of individuals' chronotypes.

Although an elevated nighttime light exposure was associated with metabolic syndrome in some studies (Benedito-Silva et al. 2020), this association was not observed in the present study as the nighttime light exposure data was not calculated in our assessments. Moreover, we did not find any difference in exposure to light (per 24 hours) between participants with and without metabolic syndrome. The negative consequences of exposure to light at inappropriate times were reported to be associated with metabolic diseases, especially in long-term shift-workers who have exposure to bright light at night (Shan et al. 2018; Vetter et al. 2018). Other studies have shown that alterations in the circadian rhythm due to nighttime light exposure, were associated with the change in eating behaviors and weight gain (Fonken et al. 2013; Obayashi et al. 2013). Nonetheless, more extensive research is needed to understand the effects of bright light exposure at night on cardiovascular health and metabolism in humans.

The present study had some strengths like objectively assessed sleep measures and outcomes, and population-based study of diverse Hispanic/Latino adults. Some

Table 6. The associations between components of metabolic syndrome (as continuous variables) with sleep efficiency and chronotype.

Model	Fasting blood sugar		Triglyceride level		HDL cholesterol		Waist	
Models	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late/Intermediate Chronotype (vs Early)	Sleep Efficiency	Late /Intermediate Chronotype (vs Early)
Model 1	−0.29 (−0.6, 0.03)	0.85 (−2.48, 4.17)/ 1.63 (−3.86, 7.13)	0.32 (−0.61, 1.25)	−6.8 (−16.65, 3.06)/ -4.73 (−21.01, 11.55)	−0.02 (−0.14, 0.1)	−0.42 (−1.66, 0.81)/ -0.77 (−2.82, 1.27)	−0.18 (−0.31, −0.06)[b]	0.82 (−0.53, 2.17) 1.82 (−0.4, 4.05)
Model 2	−0.22 (−0.55, 0.11)	0.09 (−3.29, 3.47)/ -0.02 (−5.63, 5.59)	0.28 (−0.7, 1.25)	−5.8 (−15.84, 4.24)/ -3.58 (−20.22, 13.06)	0.01 (−0.11, 0.13)	−0.59 (−1.83, 0.65) -1.2 (−3.25, 0.86)	−0.1 (−0.23, 0.03)	0.52 (−0.82, 1.87) 1.08 (−1.15, 3.31)

Data are presented as Beta coefficient (95% CI) from multivariable linear regression model. Model 1: Adjusted for age and gender. Model 2: Adjusted for Model 1 covariates plus apnea-hypopnea index (AHI >15 vs AHI <15), Marital status, education, currently employment status, and Alcohol use level.

[a]$P < .05$.

[b]$P < .01$.

limitations should be noted in our study. First our conclusions are limited to the population studied and further work is needed to study whether the results generalize to other populations. Second, this investigation was a cross-sectional study. The relationship between chronotype and metabolic syndrome could be bidirectional and in addition to the effect of chronotype on metabolic syndrome, the metabolic syndrome could also affect the evening or morning preferences. Longitudinal studies are needed to decipher relationships between chronotype, sleep patterns, and metabolic syndrome over time.

Conclusion

This study demonstrated that higher sleep efficiency is a protective factor for metabolic syndrome. Moreover, in younger individuals later chronotype was associated with a lower risk of metabolic syndrome. In younger individuals with later chronotypes, later sleeping times might align more closely with their endogenous rhythms. To understand if the discrepancy between endogenous circadian clocks and social clocks can result in disruption of intrinsic rhythms, or if different lifestyle risk factors among individuals with different chronotypes might predispose to higher risks of metabolic syndrome, more research studies are needed.

Acknowledgements

The authors acknowledge the investigators, staff, and participants of HCHS/SOL and SOLNAS for their dedication and commitment to the success of this study. Investigator website: http://www.cscc.unc.edu/hchs/

Disclosure statement

No potential conflict of interest was reported by the author(s).

Funding

Dr. Makarem is supported by a NHLBI Grant # R00-HL148511 and American Heart Association Grant #855050. Dr. Malhotra reports income from the NIH. He has income related to medical education from Corvus, Livanova, Equillium, and Jazz. ResMed provided a philanthropic donation to ucsd.

ORCID

Sanjay R. Patel http://orcid.org/0000-0002-9142-5172

Data availability statement

The data that support the findings of this study are available from Hispanic Community Health Study/Study of Latinos (HCHS/SOL). Restrictions apply to the availability of these data, which were used under license for this study. Data are available with the permission of Hispanic Community Health Study/Study of Latinos (HCHS/SOL) http://www.cscc.unc.edu/hchs/

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