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The Association between Sleep Duration and Sleep Timing and Insulin Resistance among Adolescents in Mexico City

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Abstract

Purpose: Emerging evidence suggests that short sleep duration and delayed sleep timing may be independently related to insulin resistance (IR), although findings are mixed. Our aim was to investigate associations between sleep duration and timing with insulin resistance among adolescents.

Methods: The analytic sample included 384 Mexican adolescents from a birth cohort study who took part in a follow-up study beginning in 2015. Insulin and glucose were measured in fasting serum, and HOMA-IR was calculated (marker of insulin resistance; (insulin × glucose)/ 22.5)). Sleep duration and midpoint were measured by actigraphy for 7 days after the visit and for analysis were separated by weekdays and weekends. In full and sex-stratified regression analysis, sleep duration and midpoint were exposures, and the log HOMA-IR was the outcome, adjusting for age.

Results: The mean age of the sample was 13.8±1.93 with 51% female. Shorter sleep duration on weekdays was associated with higher log HOMA-IR (β=−0.049, 95% Confidence Interval (CI) −0.097 to −0.0009). Later midpoints on weekdays and the weekend were independently associated with higher log HOMA-IR (β= 0.0408, 95% CI −0.0049 to 0.087, and β=0.0486, 95% CI 0.0042 to 0.093, respectively). Girls showed stronger associations than boys for both sleep duration and timing with HOMA-IR.

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Conclusions: Sleep duration and sleep timing were independently associated with insulin resistance, and associations were more pronounced among girls.

Keywords

insulin resistance; HOMA-IR; sleep; adolescents

Introduction

Insufficient sleep has become a global public health problem, impacting children and adults alike. Adolescents are among the most vulnerable, with over half classified as sleep deficient in a multi-country meta-analysis[1], based on the recommended 8–10 hours of sleep [2]. While sleep deficiency has multiple adverse outcomes, higher adiposity is one of the most consistent. Further evidence on the impact of sleep on other cardiometabolic outcomes is accumulating. In particular, a small number of studies have linked short sleep duration with insulin resistance among adolescents. Indeed, an association between sleep duration and insulin resistance is biologically plausible, since short sleep duration has been related to risk factors for insulin resistance including poor dietary quality, sedentary behavior, and excess adiposity[3]. However, the nature of the association between sleep duration and insulin resistance is unclear. While some studies have reported inverse or U-shaped associations between sleep duration and insulin biomarkers among adolescents [4–6], others have shown no associations [7,8]. Inconsistences could have to do with the way sleep was assessed, as some studies use objective markers of sleep while others use self-report.

Most investigations on sleep and insulin resistance have focused on sleep duration, despite emerging evidence on the role of delayed sleep timing as an independent risk factor for insulin resistance [9,10]. Delayed sleep timing could represent a misalignment between underlying biological circadian rhythms and behavioral sleep patterns, which could interfere with optimal functioning of glucose metabolism. Delayed sleep timing has similarly been related to multiple behaviors (e.g. poor diet[11], television viewing[12]) that may put adolescents at risk of developing insulin resistance.

Whether sleep duration or timing in relation to insulin resistance differs by sex remains unclear. Among 250 healthy German adolescents, girls had a stronger negative association between sleep duration and insulin resistance compared with boys [13]. However, an observational study among 245 healthy American adolescents [4] and a randomized crossover study among 21 male adolescents [14] found the opposite results: boys had higher insulin resistance with short sleep duration than girls. Only one cross-sectional study has examined sex-specific associations between sleep timing and cardiometabolic outcomes [15], reporting no associations with insulin resistance. The need to examine sex-specific associations has a biological basis, as adolescent males and females have differences in sleep (e.g. female adolescents tend to sleep earlier and longer, as well as have higher prevalence of insomnia[16]) and insulin resistance (e.g. it has been observed that males become more insulin resistant than females over the ages of 11 to 19 despite gaining less adiposity[17]).

We aimed to evaluate the independent relationships of sleep duration and timing with insulin resistance in a cohort of Mexican adolescents. Analyses were run unstratified and sex-stratified, in light of previous sex-specific findings.

Methods

Study Population

This secondary analysis used data from the Early Life Exposure in Mexico to Environmental Toxicants (ELEMENT) cohort study [18]. From 1997 to 2004, eligible mothers and their children were recruited from clinics of the Mexican Social Security Institute that served low-to-moderate income population in Mexico City. The original study excluded women with addiction to illegal drugs, alcohol consumption during pregnancy, and history of infertility, diabetes, or psychosis; and those with a high-risk or non-singleton pregnancy. In this follow up study, 550 participants from the original birth cohorts who were likely to be going through puberty were recruited, starting in 2015. During the 2015–2017 follow-up visit, research staff collected fasting blood samples in addition to information on dietary and lifestyle habits. As a marker of typical peri-pubertal sleep patterns, sleep assessment via actigraphy and sleep diary was conducted in the week immediately following the visit. Of the 550 participants, 528 had sleep information and 400 had glucose and insulin measurements. The final sample size included 384 adolescents with both sleep and insulin resistance biomarkers. Compared to the full study population with sleep information (n=528), the analytic sample was slightly older and had later sleep timing, but had similar sociodemographic characteristics. The Institutional Review Boards at the Mexico National Institute of Public Health and the University of Michigan approved the research protocols, and informed consent was obtained for all participants.

Sleep

The sleep data (including sleep duration, and sleep timing) were collected via an actigraph watch (Actigraph GT3X, ActiGraph, Inc, Pensacola, FL) worn on the adolescent's non-dominant wrist continuously for 7 days following the clinic visit. Sleep duration on each night was estimated with a fused-LASSO based algorithm (more details described here [19]), and was averaged into weekday sleep duration and weekend sleep duration. Sleep duration was also categorized into sufficient sleep versus insufficient sleep, based on the American Academy of Sleep Medicine recommendations (9–12 hours for adolescents 6–12 years, and 8–10 hours for those aged 13–18 years)[2]. To evaluate sleep timing on weekdays and weekends, we calculated average sleep midpoint, or the median of the bedtime and wake time (reported in decimal time). For both sleep duration and sleep midpoint, data were separated into weekdays versus weekends, a standard approach for adolescent studies that use actigraphy [9,20]. Weekdays represent more habitual sleep patterns while weekends are much more variable.

Laboratory assessment

Venous whole blood was taken during the clinic visit after an overnight fast (at least 8 hours), and immediately separated and stored at −80°C. Serum glucose and insulin were measured by an automated chemiluminescence immunoassay (Immulite 1000; Siemens

Medical Solutions) at the Institute of Perinatalogy in Mexico City. HOMA-IR (Homeostatic Model Assessment of Insulin Resistance) was calculated from the product of the fasting serum insulin and fasting serum glucose divided by 405 [21]. Due to right skewness of HOMA-IR, we log-transformed it for analysis. We also dichotomized it at the 90th percentile of the sample (< or ≥ 90th percentile) for logistic regression analysis.

Covariates

Age, sex, maternal education, physical activity, pubertal status, and smoking behavior were included as covariates. Maternal education was collected at the baseline visit during recruitment. Education attainment variable was classified into 4 categories: < 8 years, 9–11 years, 12 years, and >12 years. The collection of the other confounders occurred during the 2015 visit. Physical activity was measured by a frequency questionnaire validated in the Mexican adolescent population. [22] This questionnaire queries the amount of time spent in various activities (e.g. playing soccer, basketball, running) over a typical week. We considered moderate-to-vigorous physical activity (MVPA) as a continuous variable, expressed as hours/week. Pubertal status was assessed by trained physicians with the use of Tanner staging and testicular volume estimation, for boys, and menarche timing, for girls [23]. Pubertal status was categorized dichotomously as later versus earlier pubertal stage. Girls who had experienced menarche or boys with testicular volume>15 mL were considered in later pubertal stages. Smoking behavior was self-reported with a single question from the Mexican National Addictions Survey[24]: “Have you ever tried smoking?” and was categorized dichotomously. It was meant as a marker of risky behavior rather than habitual smoking.

Statistical Analysis

In bivariate analysis of the entire study population, we first estimated means and standard deviations of sleep measures (sleep duration and sleep midpoint in weekdays and weekends) according to the demographic and health characteristics. We also estimated the means and standard deviation (or median and IQR for non-normally distributed variables) of glucose, insulin and HOMA-IR according to categories of the demographic and health characteristics.

The associations between sleep duration and sleep timing (separately for weekend and weekday) with insulin resistance were examined with linear and logistic regression models. As a first step, each sleep exposure (i.e. separate models for sleep duration and sleep timing) was a continuous predictor and continuous log HOMA-IR was the dependent variable. In a second step, we adjusted for age and sex as potential confounders. In the sleep midpoint models only, we also tried adding MVPA hours/week as a potential confounder. Addition of other covariates did not alter estimates and thus were not included in final adjusted models. Finally, we included both sleep measures - timing and duration - in the regression model, along with the sex and age. Weekday sleep measures were analyzed separately from weekend sleep measures in all models.

To examine sex-specific associations, we ran the analyses as described above stratified by boys and girls. In unstratified models, we conducted a formal test for the interaction between sleep measures and sex in relation to log HOMA-IR in linear regression models with a Wald

test of the product term. Given the low power to detect statistically significant interactions, we regard a P for interaction of <0.10 as meaningful.

A few post-hoc analyses were run. First, in all models, informal mediation analyses were conducted by adjusting for BMI for age Z scores. Second, within girls, we evaluated whether menarche modified the association between sleep duration and midpoint with insulin resistance. Third, we examined social jetlag, the difference between weekend and weekday sleep midpoint, as an additional exposure. All analyses were run in SAS version 9.4.

RESULTS

The study population included 384 adolescents, of which 51% were girls. The mean ± SD age of the sample was 13.8±1.9 years, and the mean ± SD BMI-for-age Z score was 0.57 ± 1.25, with 38% of the sample classified as overweight/obese. The mean ± SD sleep duration on weekdays and weekends was 8.5 ±1.2 hours and 9.1 ±1.2 hours, respectively. The mean ± SD midpoint of the sleep period on weekdays and weekends was 3.74 ±1.32 (3:45 AM) and 4.68 ±1.22 (4:41 AM). According to age-specific AASM sleep recommendations, 48% of the sample had insufficient sleep duration on weekdays; and 27% had insufficient sleep duration on weekends. Age was inversely associated with sleep duration and positively associated with sleep midpoint (Table 1). Girls tended to sleep longer (about 15 min) than boys on weekends (Table 1). Higher physical activity was associated with earlier sleep midpoint.

The mean ± SD insulin level was 19 ± 12 μU/mL, and the mean glucose was 77±7 mg/dL. The median (IQR) log HOMA-IR was 1.1 (0.8, 1.5). Age was inversely associated with log HOMA-IR, and girls had higher log HOMA-IR level than boys (Table 2). No other covariates were associated with log HOMA-IR.

In linear regression analysis, shorter sleep duration on weekdays was associated with higher log HOMA-IR after adjusting for age, sex, and sleep midpoint on weekdays (β=−0.049, 95% Confidence Interval (CI) −0.097 to −0.0009, P=0.05) (Table 3). Later midpoints on weekdays and the weekend were associated with higher log HOMA-IR after adjusting for age, sex, and sleep duration (β= 0.0408, 95% CI −0.0049 to 0.087, P=0.08 and β=0.0486, 95% CI 0.0042 to 0.093, P=0.03, respectively). In dichotomous analysis, when the 90th percentile of HOMA-IR was used as a cut-point, there was a positive association in the weekday midpoint model; every one hour later midpoint in weekdays was associated with 1.3 times higher odds of high HOMA-IR (95% CI 0.97 to 1.74, P=0.08, results not shown). Additional adjustment for physical activity did not alter findings.

In the sex-stratified models, the associations between insulin resistance, sleep duration and midpoint were evident among girls but not boys (Table 4). To highlight, every one-hour longer sleep duration in girls was related to −0.091 log HOMA-IR (95% CI −0.15 to −0.029, P=0.004). Each hour later midpoint among girls was associated with 0.091 higher HOMA-IR (95% CI 0.027 to 0.16, P=0.006). It is worth noting that the formal tests for interaction with sex were not statistically significant for sleep duration but were potentially meaningful (P<0.10) for sleep midpoint.

Additional analyses revealed the association between sleep midpoint on weekdays and weekends and insulin resistance was stronger among girls without menarche (N=38) compared with the girls who already had menarche (N=156). For example, later weekday midpoint was associated with 0.25 higher log HOMA-IR (95% CI 0.08 to 0.41, P=0.004) among pre-menarcheal girls, while those who had already had menarche only had 0.05 higher log HOMA-IR (95% CI −0.02 to 0.12, P= 0.14). Informal mediation analyses revealed that after adjusting for BMI for age Z scores, the association between sleep duration and HOMA-IR was attenuated to the point of null. In contrast, the association with sleep midpoint was slightly attenuated but remained statistically significant. There were no associations between social jetlag and HOMA-IR, neither in unstratified nor sex-stratified analysis.

DISCUSSION

In this sample of Mexican adolescents, we found that shorter sleep duration and later sleep midpoint on weekdays were associated with higher insulin resistance (HOMA-IR), associations that were observed among girls. To illustrate, a one-hour later sleep midpoint among girls was related to a 30% higher odds of being in the upper decile of HOMA-IR values. The clinical relevance of these findings is somewhat unclear due to inconsistencies in the definition of a high HOMA-IR. However, even slight elevations during adolescence may signal a propensity for developing insulin resistance later on in adulthood [25]. There were no associations between sleep duration or timing and insulin resistance among boys.

Associations of sleep duration and insulin resistance have been previously reported. A study among 250 German adolescents linked self-reported sleep duration with log HOMA-IR in girls but not boys [13]. In contrast, a US study with 245 healthy adolescents reported a stronger relationship with HOMA-IR among boys than girls [4]. These incongruent results may be attributed to heterogeneity in pubertal stage across studies. For example, adolescents in the US sample were more advanced in their pubertal stage than the Mexican adolescents, and our findings suggested stronger associations between sleep duration and insulin resistance among pre-menarcheal girls. These findings may seem counter to expectation, since there is a period of transient insulin resistance among girls around the time of menarche. However, what our findings suggest is that other factors (e.g. hormonal) besides sleep are responsible for the insulin resistance observed around the time of menarche.

Other reports on the association of sleep duration and insulin resistance did not conduct sex-stratified analysis, and thus are difficult to directly compare with the present study. Nonetheless, the inverse associations between sleep duration on weekdays and insulin resistance are consistent with a US study of 31 obese adolescents, who were primarily female (mean age 16.0 ± 1.4 years). This study associated shorter actigraphy-assessed sleep duration on both weekdays and weekends with worse HOMA-IR [9]. Another US study of 387 adolescents (with mean age of 15.7 ± 2.1; 51% girls) found that both shorter and longer sleep duration on weekdays were related to higher HOMA-IR [6]. In contrast, our data do not suggest a U-shaped association.

Weekday and weekend sleep midpoints were positively associated with insulin resistance independent of sleep duration in girls, but not in boys. A few prior studies have examined the relationships between sleep timing and insulin resistance, with mixed findings [9,15,20]. One US cohort that examined sleep timing and evening preference in relation to cardiometabolic risk factors found no association with insulin resistance in either sex [15]. Similarly, another US study of 125 children aged 8–17 years reported no association between bedtime and HOMA-IR [20]. However, a study among 31 US obese adolescents found positive associations between sleep timing on weekdays and insulin resistance [9]. Further, this study found that a greater discrepancy between melatonin onset (the hormonal cue for sleep initiation) and bedtime had a negative impact on insulin resistance. Intriguingly, each of the prior studies used actigraphy to assess sleep, although the US study of 31 obese adolescents was the only study to utilize an oral glucose test for the assessment of insulin resistance.

The fact that sleep duration and sleep timing were independently associated with insulin resistance suggests that there are separate mechanisms. The pathways involving sleep timing likely involve circadian misalignment. To illustrate, delayed sleep timing is linked to altered meal timing [26], which in turn can interfere with circadian rhythmicity of glucose metabolism [27]. Although related, pathways involving sleep duration may have additional mechanisms beyond circadian rhythms. Indeed, short sleep duration is associated with weight gain over time [28], as well as higher intake of refined carbohydrates[20], such as sugary beverages [29]. Further, sleep duration is positively correlated with physical activity [30]. Both weight gain and physical activity are independent risk factors for insulin resistance, highlighting them as potential mediators in the observed relationship [31]. Indeed, we found that after adjusting for BMI, the association between sleep duration and HOMA-IR was attenuated to the point of null, providing evidence that BMI was a likely mediator.

Objective sleep and insulin resistance assessments represent the major strengths of this study. The analytic approach including sex stratification and potential effect modification by menarche status are additional strengths. However, this study has several limitations. First, the cross-sectional design does not rule out the possibility of reverse causation; i.e. that insulin resistance may cause insufficient sleep duration and late sleep-timing. Second, potential residual confounders including family history of diabetes and the presence of obstructive sleep apnea were not measured. Third, while HOMA-IR is not the gold standard to diagnose insulin resistance, alternative assessment techniques are invasive and infeasible in large cohort studies. Interestingly, HOMA-IR has been found to much better correlated with liver insulin resistance than peripheral insulin resistance[32]; thus, our findings may point especially to the involvement of liver function. Fourth, the original ELEMENT study was not designed with the present study questions in mind and therefore we may have been underpowered to detect associations, especially interactions. Fifth, we did not collect information on self-assessed dimensions of sleep such as sleep satisfaction or daytime sleepiness; thus, we could not evaluate associations between these characteristics of sleep and HOMA-IR.

Shorter sleep duration and delayed bedtime were associated with higher HOMA-IR among adolescents. These findings suggest that sleep duration and sleep timing may independently impact insulin resistance in adolescents, particularly among girls. Within the chronically sleep-deprived population of adolescents, improvement of sleep health could positively influence metabolic function.

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Conflict of Interest:

The authors have indicated they have no potential conflicts of interest to disclose. The study sponsor had no role in the study design, the collection, analysis, or interpretation of data, the writing of the report, or the decision to submit the manuscript for publication. Author PC wrote the first draft of the manuscript. No honorarium, grant, or other form of payment was given to anyone to produce the manuscript.

Abbreviations:

IR	insulin resistance
BMI	body mass index
CI	confidence interval
AASM	American Academy of Sleep Medicine
ELEMENT	Early Life Exposure in Mexico to ENvironmental Toxicants
SD	standard deviation

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Implications and Contributions

Within Mexican adolescents, we found that actigraphy-assessed short sleep duration and delayed sleep timing were each associated with higher insulin resistance among girls only. Although prior literature often focuses on duration, these findings suggest that sleep duration and timing have independent effects on insulin resistance, and in a sex-specific manner.

Table 1.

Mean ± SD (or %) of sleep duration, those not meeting sleep duration recommendations, and midpoint, according to categories of covariates in a sample of 384 Mexican youth

	N	Mean of sleep duration (hour) ± SD		Did not meet sleep recommendations[1], %		Mean of Midpoint (decimal hour) ± SD	
		Weekday	Weekend	Weekday	Weekend	Weekdays	weekends
Age, Quartiles							
Q1, 9.9 to 12.15y	96	8.53 ± 0.97	9.35 ± 1.16	67.71	36.46	3.29 ± 1.10	4.35 ± 0.99
Q2, 12.15 to 13.54y	96	8.53 ± 1.20	9.22 ± 1.14	55.21	29.17	3.68 ± 1.42	4.49 ± 1.15
Q3, 13.54 to 15.62y	96	8.40 ± 1.20	8.95 ± 1.14	34.38	17.89	3.63 ± 1.20	4.66 ± 1.21
Q4, 15.70 to 17.96y	96	8.40 ± 1.39	8.80 ± 1.34	35.42	25.00	4.36 ± 1.32	5.24 ± 1.35
P Value		0.33	0.0005	<0.0001	0.03	<0.0001	<0.0001
Sex							
Male	188	8.40 ± 1.10	8.94 ± 1.20	50.53	32.09	3.77 ± 1.33	4.62 ± 1.27
Female	196	8.52 ± 1.28	9.21 ± 1.22	45.92	22.45	3.71 ± 1.31	4.74 ± 1.18
P Value		0.34	0.032	0.36	0.03	0.67	0.34
Maternal Education							
8 y or less	42	8.39 ± 1.11	9.06 ± 1.21	50	28.57	3.79 ± 1.21	4.79 ± 1.40
9 to 11 y	152	8.49 ± 1.19	9.10 ± 1.32	46.71	30.26	3.74 ± 1.38	4.68 ± 1.15
12 y	134	8.52 ± 1.22	9.11 ± 1.19	44.78	21.80	3.73 ± 1.30	4.65 ± 1.27
> 12 y	55	8.32 ± 1.26	8.93 ± 0.98	58.18	30.91	3.72 ± 1.31	4.69 ± 1.21
P value		0.76	0.59	0.39	0.37	0.82	0.68
Menarche status							
No	38	8.71 ± 1.03	9.43 ± 0.94	47.37	26.32	3.33 ± 1.14	4.49 ± 0.88
Yes	156	8.47 ± 1.34	9.14 ± 1.27	45.51	21.79	3.81 ± 1.33	4.81 ± 1.24
P Value		0.31	0.18	0.84	0.55	0.04	0.13
Testicular Volume (>15mm)							
No	40	8.30 ± 0.83	9.01 ± 1.16	65.00	47.5	3.27 ± 0.91	4.09 ± 0.89
Yes	142	8.45 ± 1.16	8.93 ± 1.23	45.77	26.95	3.87 ± 1.39	4.72 ± 1.31
P Value		0.44	0.74	0.03	0.01	0.01	0.005
MVPA (hours/wk), Quartiles							
Q1, 0 to 5.75	89	8.39 ± 1.27	9.16 ± 1.23	53.93	28.09	3.79 ± 1.28	4.83 ± 1.33

	N	Mean of sleep duration (hour) ± SD		Did not meet sleep recommendations[1], %		Mean of Midpoint (decimal hour) ± SD	
		Weekday	Weekend	Weekday	Weekend	Weekdays	weekends
Q2, 6 to 10	107	8.54 ± 1.24	9.09 ± 1.11	44.86	28.3	3.76 ± 1.36	4.83 ± 1.22
Q3, 10.25 to 14.25	93	8.39 ± 1.22	8.94 ± 1.19	50.54	25.81	3.65 ± 1.40	4.56 ± 1.19
Q4, 14.5 to 29	95	8.52 ± 1.05	9.11 ± 1.34	44.21	26.32	3.76 ± 1.23	4.51 ± 1.15
P Value		0.73	0.62	0.48	0.97	0.78	0.03
Ever smoked cigarettes							
No	317	8.46 ± 1.16	9.17 ± 1.16	50.16	26.27	3.59 ± 1.26	4.52 ± 1.13
Yes	64	8.45 ± 1.36	8.69 ± 1.36	39.06	28.13	4.39 ± 1.37	5.42 ± 1.39
P Value		0.94	0.004	0.11	0.76	<0.0001	<0.0001

[1]According to the American Academy of Sleep Medicine

Table 2.

Median (IQR) or Mean ± SD values of HOMA-IR, insulin, and glucose, according to categories of covariates in a sample of 384 Mexican youth

	Sample size	Median HOMA-IR (IQR)	Median Insulin (IQR)	Mean Glucose ±SD
Age (years), Quartiles				
Q1, 9.9 to <12.2	96	3.23 (2.18, 4.84)	16.55 (11.65, 24.30)	78.32 ± 6.89
Q2, 12.2 to <13.6	96	3.31 (2.17, 5.06)	16.25 (11.20, 24.45)	79.20 ± 6.89
Q3, 13.6 to <15.7	96	3.12 (2.33, 4.33)	16.45 (12.30, 22.50)	77.18 ± 7.51
Q4, 15.7 to 18	96	2.78 (2.06, 4.22)	14.80 (11.10, 21.65)	76.32 ± 7.55
P Value		0.14	0.28	0.02
Sex				
Male	188	2.83 (2.03, 4.26)	14.55 (10.80, 21.10)	78.84 ± 7.30
Female	196	3.31 (2.41, 5.13)	17.45 (13.30, 24.55)	76.71 ± 7.11
P Value		0.01	0.002	0.004
Maternal Education, years				
8 or less	42	3.03 (2.16, 4.32)	15.95 (12.20, 21.40)	76.93 ± 7.83
9 to 11	152	2.99 (2.22, 4.43)	15.35 (12.00, 22.75)	78.24 ± 7.31
12	134	3.09 (2.15, 4.74)	16.45 (11.60, 23.90)	77.47 ± 7.02
≥12	55	3.22 (2.14, 4.50)	16.30 (11.10, 22.90)	77.75 ± 7.50
P value		0.42	0.40	0.97
Menarche status				
No	38	3.26 (2.29, 5.21)	16.55 (12.10, 24.80)	77.37 ± 6.74
Yes	156	3.41 (2.43, 5.13)	18.15 (13.60, 24.55)	76.65 ± 7.17
P Value		0.43	0.34	0.58
Testicular Volume (>15mm)				
No	40	2.58 (2.10, 4.06)	13.35 (10.40, 21.00)	77.30 ± 6.43
Yes	142	2.83 (2.03, 4.31)	14.60 (10.80, 21.20)	79.37 ± 7.42
P Value		0.44	0.59	0.11
MVPA, Quartiles (hours/wk)				
Q1, 0 to 5.8	89	3.32 (2.42, 4.57)	16.30 (13.30, 23.50)	77.81 ± 6.98
Q2, 6 to 10	107	3.43 (2.37, 5.21)	17.60 (13.00, 27.30)	77.64 ± 6.82
Q3, 10.3 to 14.3	93	2.72 (1.98, 4.36)	14.40 (11.10, 21.10)	77.44 ± 7.62

	Sample size	Median HOMA-IR (IQR)	Median Insulin (IQR)	Mean Glucose ±SD
Q4, 14.5 to 29	95	2.96 (2.06, 4.06)	14.90 (11.00, 22.50)	78.14 ± 7.76
P Value		0.04	0.04	0.81
Ever smoked cigarettes				
No	317	3.13 2.39	16.30 (11.70, 23.00)	77.86 ± 7.00
Yes	64	2.79 2.20	14.65 (11.85, 21.25)	77.22 ± 8.65
P Value		0.34	0.39	0.52

Table 3.

Association between sleep duration and midpoint and log of HOMA-IR in a sample of 384 Mexican youth

	Crude Model		Adjusted Model[1]		Adjusted Model 2[2] (Midpoint only)	
	Beta (95% CI)	**P value**	**Beta (95% CI)**	**P Value**	**Beta (95% CI)**	**P Value**
Duration, per hour						
Weekdays	−0.026 (−0.070, 0.017)	0.23	−0.049 (−0.097, −0.0009)	0.05		
Weekends	0.015 (−0.028, 0.058)	0.50	0.0088 (−0.035, 0.052)	0.69		
Recommendation[3]						
Weekdays	0.088 (−0.016, 0.192)	0.10	0.0847 (−0.022, 0.192)	0.12		
Weekends	−0.042 (−0.160, 0.075)	0.48	−0.0342 (−0.152, 0.084)	0.57		
Midpoint, per hour						
Weekdays	0.0105 (−0.029, 0.050)	0.60	0.0408 (−0.0049, 0.087)	0.08	0.0400 (−0.0057, 0.086)	0.09
Weekends	0.039 (−0.0034, 0.081)	0.07	0.0486 (0.0042, 0.093)	0.03	0.044 (−0.0007, 0.088)	0.05

1. In sleep duration models, age, sex and midpoint are adjusted (separately for weekdays and weekends). In the sleep midpoint model, age, sex and sleep duration are adjusted (separately for weekdays and weekends).

2. In the sleep midpoint model, we adjusted for MVPA hour/week, sex, age, and sleep duration.

3. This recommendation is according to the AASM recommendation

Table 4.

Sex-stratified associations between sleep exposures (sleep duration, midpoints, and sleep recommendation) and log of HOMA-IR

		Crude Model		Adjusted Model[1]	
		Beta (95% CI)	**P value**	**Beta (95% CI)**	**P Value**
Duration, per hour					
Weekdays	Male	0.0008 (−0.068, 0.069)	0.98	0.006 (−0.069, 0.081)	0.88
	Female	−0.051 (−0.106, 0.005)	0.08	−0.091 (−0.153, −0.029)	0.004
P for Interaction[2]			0.25		0.24
Weekends	Male	−0.0085 (−0.072, 0.055)	0.79	−0.0109 (−0.074, 0.052)	0.73
	Female	0.0250 (−0.034, 0.084)	0.41	0.0326 (−0.028, 0.094)	0.29
P for Interaction[2]			0.44		0.49
Recommendation					
Weekdays	Male	0.0701 (−0.079, 0.221)	0.36	0.037 (−0.1221, 0.196)	0.64
	Female	0.117 (−0.0270, 0.260)	0.11	0.119 (−0.027, 0.266)	0.11
P for Interaction[2]			0.66		0.63
Weekends	Male	0.073 (−0.088, 0.234)	0.37	0.055 (−0.108, 0.218)	0.50
	Female	−0.145 (−0.316, 0.026)	0.10	−0.146 (−0.318, 0.026)	0.09
P for Interaction[2]			0.07		0.07
Midpoint, per hour					
Weekdays	Male	−0.019 (−0.076, 0.037)	0.50	−0.0092 (−0.075, 0.056)	0.78
	Female	0.042 (−0.0127, 0.097)	0.13	0.091 (0.0267, 0.155)	0.006
P for Interaction[2]			0.12		0.098
Weekends	Male	0.0065 (−0.053, 0.066)	0.83	0.0216 (−0.041, 0.084)	0.50
	Female	0.070 (0.0094, 0.130)	0.02	0.080 (0.0167, 0.143)	0.01
P for Interaction[2]			0.14		0.13

1. In the sleep duration adjusted model, age, and midpoint are adjusted. In the sleep midpoint model, age, and sleep duration are adjusted.

2. P values for interaction come from models that include the main effects of sex and sleep exposure in addition to a sex*sleep measure interaction term.