

Insufficient Sleep is a Contributor to Overweight and Type 2 Diabetes in Adults: Any Contribution from Caffeine?

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Abstract Objective: To test if sleep insufficiency has a direct impact on being overweight and the 10 years-risk of developing type 2 diabetes in non-diabetic adults. Also to test if there is any possible impact of caffeine consumption on the occurrence of type 2 diabetes in the same population. **Research Design and Method:** We conducted a cross-sectional study on 504 non-diabetic participants, aged between 18 and 64 years. This study took place in North Lebanon between December 2016 and June 2017. Study subjects completed a self-administered questionnaire concerning sleep duration and sleep quality using the Athens Insomnia Scale (AIS) and the 10 years risk of developing type 2 diabetes using the American Diabetes Association Questionnaire. In addition, the participants underwent later on a full analysis of their body fatness, body muscles percentages and their resting energy expenditure (REE). **Results:** Sleep duration of more than 6 hours per day is associated with the lowest risk of developing diabetes and overweight ($p=0.03$ and $p=0.004$) respectively. This was observed in women ($p<0.001$ and $p=0.003$ respectively) but not in men. When stratifying our data by gender and according to the Body Mass Index (BMI) cutoff classification, we observed that women with healthy body weight ($BMI<25 \text{ Kg/m}^2$) and those having a slight obesity, and who both sleep more than 6 hours per day, do not present a risk of developing type 2 diabetes ($p=0.03$ and $p=0.089$ respectively). However, as measured by the Athens Insomnia Scale, Insomnia was not associated with nor diabetes type 2 neither obesity ($p=0.859$ and $p=0.871$ respectively). No association was found when splitting this data by gender. Using binary logistic regression, the subjects who sleep more than 6 hours per day have 0.61 times less risk of developing diabetes compared to those who sleep less than 6 hours per day (0.61 and 95% CI [0.39-0.97]). However, when testing the association between Insomnia and the risk of developing type 2 diabetes in 10 years, no association was found (1 and 95% CI [0.66-1.62]). Despite being a stimulator factor, caffeine, in our study, shows that subjects who drink caffeine had 3.6 times lower risk of developing type 2 diabetes compared to non-drinkers (3.6 and 95% CI [1.5-3.6]). This association was so strong in women (0.3 and 95% CI [0.24-0.63]) compared to men. **Conclusion:** Based on our study results, we conclude that sleeping duration of 6 hours per day or more could be a protective factor against developing diabetes and overweight mainly in women. Also, caffeine consumption is a must to prevent the high risk of developing type 2 diabetes in women, but in the right time to avoid sleep insufficiency.

Keywords Insomnia, Sleeping hours, Obesity, Diabetes, Adults, Caffeine

1. Introduction

According to the International Diabetes Federation, 415 million adults have type 2 diabetes in the world and the estimated number of diabetic patients worldwide will rise to 642 million by 2040 [1].

Diabetes was responsible for 342,000 deaths in 2015 in the

Middle Eastern Region [2]. Over half (51.3%) of all deaths from diabetes in the region occurred in people under 60. These early deaths may be the result of a combination of factors: the rapidly changing environments and lifestyles in the region, late diagnoses and health systems that are not equipped to provide optimal management to the increasing numbers of people with diabetes [2]. Countries with high type 2 diabetes prevalence include Saudi Arabia (raw diabetes prevalence of 17.6%) and Kuwait (14.3%). Due to their different population structures, these countries both have an age-adjusted comparative prevalence of 20%. The countries with the largest number of adults with diabetes are

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Egypt (7.8 [3.8-9.0] million), Pakistan (7.0 [5.1-10.0] million) and Iran (4.6 [3.6-6.3] million) [2]. In Lebanon, till today, 464,200 adults is the number of diabetic where 41.2% were undiagnosed. Overall estimation of the prevalence in Lebanon shows that 1 in 8 Lebanese adults have diabetes [3]. Sleeping is essential in many physical, cognitive, and psychological processes and an optimal sleeping condition appears to be a health imperative [4].

A recent meta-analysis of prospective studies have provided facts of U-shaped relationship between sleep duration and a higher incidence of type 2 diabetes, the most frequent consequence of obesity, with both short and long sleep duration associated with greater risk [5-7]. The mechanisms underlying the sleep-diabetes relationship remain unsettled. Prior studies have shown that short sleep is related to the mechanism of glucose intolerance, insulin resistance, and reduced acute insulin response to glucose [8].

Because of the lack of clinical trials and surveys in this field, the objective of this study was to assess, retrospectively, the temporal relationship between sleep adequacy and type 2 diabetes and overweight.

2. Research Design and Methodology

This is a cross-sectional study that took place in Lebanon, mainly in North Lebanon between December 2016 and June 2017. We contacted a total of 504 non-diabetic participants (234 women and 270 men), aged between 18-64 years who filled a self-administered questionnaires that included information on socio-demographic status, education, sleep duration/quality, medical history, family history, lifestyle, risk factors and working conditions. In order to investigate the sleep adequacy and insomnia in these participants, the Athens Insomnia Scale (AIS) was used (see Appendix 1). As well as, in order to investigate the 10 years-risk assessment of type 2 diabetes, the American Diabetes Association Questionnaire (see Appendix 2) was filled with these participants. All participants were not diabetic, nor having any other chronic diseases. Diabetes in this study was defined as taking medications for treating diabetes, or laboratory test values (fasting blood sugar and HbA1c) indicating the medical occurrence of type 2 diabetes after a final reporting from an endocrinologist. Information about sleep duration was assessed by the question: "How long on average, in hours and minutes, do you normally sleep?" It was categorized into ≤ 5 , 5-6; 6-7, and >7 h. The Athens Insomnia Scale (AIS) was used to assess sleep quality, the consistency, reliability, and validity of which has been ascertained as a screening or diagnosis tool for insomnia and it consists of eight items: the first five of which pertain to sleep induction, awakening during the night, final awakening earlier than desired, total sleep duration (not quantity but perceived sufficiency), and overall quality of sleep (sleep satisfaction); the subsequent three items refer to the sense of well-being, functioning, and sleeping during

the day. Participants will be requested to score on a scale of 0-3, with a score of 0 signifying no problem at all, a score of 1 signifying a minor or slight problem, a score of 2 signifying marked or considerable problems, and a score of 3 signifying serious problems or being unable to sleep at all. Answers were based on any sleep difficulties participants may have experienced at least three times a week during the previous month. For the analyses, a higher score expressed a more aggravated sleep quality. The type 2 diabetes risk test was used to indicate whether the participant is at risk of developing type 2 diabetes in the coming 10 years or not and it consists of seven items: age, gender, gestational diabetes, family history of diabetes, blood pressure, physical activity and body mass index. Participants were requested to score on a scale from 0 to 3 points. For the analysis, if the score was 5 or higher, the participant is considered at increased risk for having type 2 diabetes.

A full body analysis of fat and muscles was performed for participants using an impedance body composition analyzer machine that show numerical values of weight, body mass index, percentage of body fat, percentage of visceral fat, body water, muscle mass and resting energy expenditure (REE).

3. Statistical Analyses

All statistical analyses were performed using the SPSS software (Version 22). For descriptive statistics, basic information was expressed as percentage, means, and standard deviation. The chi-square test was used to investigate the relationship between the dependent variable (Insomnia, sleep duration, Type II Diabetes risk) and each independent variable. Furthermore a binary logistic regression was used to calculate the odd ratio values of the dependent variable in the multivariate analysis after adjustment of confounders. A significance level of $\alpha=0.05$ was used.

4. Results

The total number of participants is 504 adults where 54% were men. The mean age of our population was 30 ± 13 years old. 37% of our population was smoker and more than 50% smokes water pipe. All characteristics of the study participants are summarized in Table 1.

As presented in Table 1, men were taller and heavier than women. Using proportion distribution, women were more educated than men in this study ($p<0.001$) (data not shown). More than 70% of men work however more than half of women are unemployed ($p<0.001$) (data not shown). Half of them work more than 36 to 48 hours per week. Concerning physical activity, 45% of men are physically active compared to 18% of women only ($p<0.001$). More than 94% of our study participants were healthy and free off non-communicable diseases (hypertension, CVD, cholesterol, cancer and Asthma). After completing the full

body analysis for body fatness, visceral fat and skeletal muscles, it was observed that the majority of men had overweight however the majority of women had normal body mass index ($p<0.001$). The analysis of body fatness shows that the majority of men had very high percentages of body fat, and the majority of women were having that too ($p=0.095$) despite of that the majority of women had normal BMI. The deep analysis of visceral fat shows that half of men

were having normal levels of visceral fat, 30% were having high levels and 17% were having very high levels of visceral fats (Table 1) compared to women where 92% of them were having normal levels of visceral fats. The analysis of skeletal muscle percentage shows that 74% of men were having low to normal level of skeletal muscles compared to 93% of women ($p<0.001$).

Table 1. Characteristics of study participants

Characteristics *	Overall 504(100%)	Men 270(53.6%)	Women 234(46.4%)	P-value†
Age**	30±12.5	30.8±12	29±13	<0.098
Height**	168±13.9	173.7±16.0	161.4± 6.4	<0.001
Weight**	74.5±17.1	83.4±15.9	64.2±11.9	<0.001
Occupation*				
Yes	270 (53.6)	191 (70.7)	79 (33.8)	
No	234 (46.4)	79 (29.3)	155 (66.2)	<0.001
Working hours*				
<36h	80 (29.6)	36 (18.8)	44 (55.7)	<0.001
36-48h	102 (37.8)	81 (42.5)	21(26.6)	
>48h	88 (32.6)	74 (38.7)	14 (17.7)	
Smoking				
Currently	185 (36.8)	114 (42.2)	71 (30.5)	
Previously	27 (5.5)	22 (8.1)	5 (2.1)	
Never	291 (57.7)	134 (49.6)	157 (67.4)	<0.001
Type				
Cigarette	25 (10.7)	58 (43.9)	25 (32.1)	<0.124
Water pipe	48 (20.5)	62 (47.0)	48 (61.5)	
Both	5 (2.1)	12 (9.1)	5 (6.4)	
Alcohol				
Never	434 (86.1)	226 (83.7)	208 (88.9)	
Rarely	53 (10.5)	31 (11.5)	22 (9.4)	<0.106
Often	17 (3.4)	13 (4.8)	4 (1.7)	
Physical Activity				
No	195 (38.7)	80 (29.6)	115 (49.2)	<0.001
Yes<2.5h/week	145 (28.8)	68 (25.2)	77 (32.9)	
Yes>2.5h/week	164 (32.5)	122 (45.2)	42 (17.9)	
Hypertension				
Yes	13 (2.6)	7 (2.6)	6 (2.6)	0.984
No	491 (97.4)	263 (97.4)	228 (97.4)	
CVD				
Yes	8 (1.6)	4 (1.5)	4 (1.7)	0.838
No	496 (98.4)	266 (98.5)	230 (98.3)	
Cholesterol				
Yes	29 (5.8)	20 (7.4)	9 (3.8)	0.087
No	475 (94.2)	250 (92.6)	225 (96.2)	
Cancer				
Yes	1 (0.2)	0 (0)	1 (0.4)	0.282
No	503 (99.8)	270 (100)	233 (99.6)	
Asthma				
Yes	12 (2.6)	5 (1.9)	7 (3)	0.403
No	492 (97.4)	265 (98.1)	227 (97)	
BMI				
Underweight	12 (2.4)	3 (1.1)	9 (3.8)	<0.001
Normal	211 (41.9)	86 (31.9)	125 (53.5)	
Overweight	179 (35.5)	110 (40.7)	69 (29.5)	
Obese	102 (20.2)	71 (26.3)	31 (13.2)	

Characteristics *	Overall 504(100%)	Men 270(53.6%)	Women 234(46.4%)	P-value†
Body fat				
Low	8 (1.6)	6 (2.2)	2 (0.9)	0.095
Normal	114 (22.6)	56 (20.7)	58 (24.8)	
High	155 (30.8)	75 (27.8)	80 (34.2)	
Very high	227 (45.0)	133 (49.3)	94 (40.2)	
Skeletal muscle				
Low	195 (38.7)	102 (37.8)	93 (39.7)	<0.001
Normal	228 (45.2)	100 (37.0)	128 (54.7)	
High	61 (12.1)	52 (19.3)	9 (3.8)	
Very high	20 (4.0)	16 (5.9)	4 (1.7)	
Visceral fat				
Normal	353 (70.0)	139 (51.5)	214 (91.5)	<0.001
High	102 (20.2)	83 (30.7)	19 (8.1)	
Very high	49 (9.7)	48 (17.8)	1 (0.4)	

*N (%), ** (μ±SD)

†T-test was used to compare age, height and weight; Chi² test was used for all other variables

CVD: Cardiovascular diseases

Table 2. Consumption of caffeine sources and Energy Drinks by the study participants

Consumption of Caffeine and energy drink	Overall 504(100%)	Men 270(53.6%)	Women 234(46.4%)	P value†
Caffeine*				
Yes	421 (83.5)	224 (83)	197 (84.2)	0.4
No	83 (16.5)	46 (17)	37 (15.8)	
Quantity of Caffeine (mg)	201.9±183.6	243±213.5	155.2±127.6	<0.001
Time of Caffeine Drinking*				
>7h before bedtime	221 (52.5)	115 (51.3)	106 (53.8)	0.61
<7h before bedtime	200 (47.5)	109 (48.7)	91 (46.2)	
Coffee 80 ml**	1.54±2.44	2.10±2.93	0.91±1.50	0.004
Espresso 30 ml**	0.21±0.76	0.34±0.82	0.06±0.65	<0.001
Green tea 180 ml**	0.28±0.78	0.25±0.66	0.31±0.83	0.42
Black tea 180 ml**	0.72±1.19	0.84±1.41	0.57±0.85	0.02
Cacao 180 ml**	0.72±1.19	0.05±0.25	0.05±0.24	0.77
Nescafe 240ml**	0.68±0.95	0.49±1.03	0.90±0.80	<0.001
Energy drinks** (one can)	0.08±0.38	0.11±0.47	0.04±0.26	0.03
Soft drink** (one can)	0.40±0.85	0.51±0.83	0.27±0.86	<0.001

*N (%), ** (μ±SD)

† Chi² test was used to compare caffeine consumption and time of drinking; T-test was used to for all other variables

Table 2 describes the consumption of caffeine and energy drinks by the study participants. There was no difference in caffeine consumption between men and women (p=0.4) that's mean both gender consume equally caffeinated beverages. However, men drinks more caffeinated beverages than women (p<0.007) for example: Turkish coffee (p=0.004), Espresso (<0.001), Black tea (p=0.022), Nescafe (p=0.007), energy drinks (p=0.03) and soft drinks (p<0.001).

Table 3 shows the percentages of sleeping hours for the study participants. There was no differences in sleeping hours between genders (p=0.09) (Table 3). In addition, Insomnia was also calculated using the Athens Insomnia questionnaire, shows that 56% of men had normal sleep quality compared to 56% of women (p=0.007). However a non-negligible percentage of men and women had insomnia

(p=0.07).

Concerning Diabetes assessment risk score, study results show that 25% of men and only 12% of women had a high risk of developing type 2 diabetes (p<0.001). In order to investigate the association between sleep quality and the risk of diabetes occurrence in our study population, a binary logistic analysis was conducted to calculate the odd ratio accordingly. It was shown that subjects who sleep more than 6 hours have 0.61 times less risk of developing type 2 diabetes compared to those who sleep less than 6 hours (0.61 and 95% CI [0.39-0.97]). However, when testing the association between Insomnia and the risk of developing type 2 diabetes, no association was found (1 and 95% CI [0.66-1.62]) (Table 4). Despite being a stimulator factor, caffeine, in our study, shows that subjects who drink caffeine

had 3.6 times lower risk of developing diabetes compared to non-drinkers (3.6 and 95% CI [1.5-3.6]). This association was so strong in women (0.3 and 95% CI [0.24-0.63]) compared to men. If we compare study subjects who have low risk of developing type 2 diabetes to those who have a high risk, we observe that the majority of subjects having low risk sleep more than 6 hours per day (around 70%) ($p=0.03$). No significance was observed when testing these association but with Insomnia Athens score ($p=0.85$). Furthermore, concerning the relationship between sleeping insufficiency and overweight, we observed that the majority of participants who have healthy weight (74%) sleep more

than 6 hours ($p=0.004$). No relation was observed when testing the association between insomnia score and overweight ($p=0.56$) (Table 4). The resting energy expenditure (REE) was also calculated for study participants. We observed that subjects who have a high risk of developing diabetes and who sleep less than 6 hours presented a low REE of 1652 Kcal/day compared to those who sleep more than 6 hours of 1842 Kcal/day ($p=0.023$). On the other hand, this association was not found in those who neither don't have a risk of developing diabetes nor when splitting this data by gender (data not shown).

Table 3. Sleeping hours, Athens Insomnia questionnaire results and the 10 years assessment of developing type 2 diabetes in study participants

Sleeping hours Insomnia Diabetes assessment risk *	Overall 504	Men	Women	P-value†
Sleeping hours				0.097
5h or less	51(10.1)	35(13.0)	16(6.8)	
5-6h	114(22.6)	55(20.4)	59(25.2)	
6-7h	212(42.1)	110(40.7)	102(43.6)	
7h or more	127(25.2)	70(25.9)	57(24.4)	
Athens questionnaire items and score				
Q1				0.080
No problem	202 (40.1)	113 (41.9)	89 (38.0)	
Slightly delayed	200 (39.6)	94 (34.8)	106 (45.3)	
Markedly delayed	77 (15.3)	47 (17.4)	30 (12.8)	
Very delayed	25 (5)	16 (5.9)	9 (3.9)	
Q2				0.370
No problem	274 (54.4)	152 (56.3)	122 (52.1)	
Minor problem	167 (33.1)	87 (32.2)	80 (34.2)	
Considerable problem	58 (11.5)	27 (10.0)	31 (13.3)	
Serious problem	5 (1)	4 (1.5)	1 (0.4)	
Q3				0.450
Not earlier	269 (53.4)	148 (54.8)	121 (51.7)	
A little earlier	187 (37.1)	93 (34.4)	94 (40.2)	
Markedly earlier	44 (8.7)	26 (9.6)	18 (7.7)	
Much earlier	4 (0.8)	3 (1.1)	1 (0.4)	
Q4				0.006
Sufficient	231 (45.8)	124 (45.9)	107 (45.7)	
Slightly insufficient	198 (39.3)	93 (34.4)	105 (44.9)	
Markedly insufficient	69 (13.7)	49 (18.2)	20 (8.5)	
Much earlier	6 (1.2)	4 (1.5)	2 (0.9)	
Q5				0.210
Satisfactory	270 (53.6)	141 (52.2)	129 (55.1)	
Slightly unsatisfactory	171 (33.9)	88 (32.6)	83 (35.5)	
Markedly unsatisfactory	47 (9.3)	32 (11.9)	15 (6.4)	
Very unsatisfactory	16 (3.2)	9 (3.3)	7 (3.0)	
Q6				0.210
Normal	257 (51.0)	147 (54.4)	110 (47.0)	
Slightly decreased	161 (31.9)	84 (31.1)	77 (32.9)	
Markedly decreased	58 (11.5)	28 (10.4)	30 (12.8)	
Very decreased	28 (5.6)	11 (4.1)	17 (7.3)	
Q7				0.006
Normal	314 (62.3)	187 (69.3)	127 (54.3)	
Slightly decreased	151 (30.0)	66 (24.4)	85 (36.3)	
Markedly decreased	35 (6.9)	16 (5.9)	19 (8.1)	
Very decreased	4 (0.8)	1 (0.4)	3 (1.3)	

Q8				0.006
None	95(18.8)	66 (24.4)	29 (12.4)	
Mild	292(57.9)	149 (55.2)	143 (61.1)	
Considerable	93(18.5)	43 (15.9)	50 (21.4)	
Intense	24(4.8)	12 (4.4)	12 (5.1)	
Total score classification				0.073
No problem<6	284 (56.3)	152 (56.3)	132 (56.5)	
Insomnia problem>6	220 (43.7)	118 (43.7)	102 (43.6)	
Diabetes Questionnaire items and score				
Family history of diabetes				0.966
No	358 (71)	192 (71.1)	166 (70.9)	
Yes	146 (29)	78 (28.9)	68 (29.1)	
Diabetes risk score classification				0.001
No risk	405 (80.4)	200 (74.1)	205 (87.6)	
Risk	99 (19.6)	70 (25.9)	29 (12.4)	

* N (%); † Chi² test was used to compare variables

Table 4. Association between sleeping quality and type 2 diabetes and overweight

		Risk of Diabetes		P†	BMI		P†
		Low Risk	High Risk		Healthy weight	Overweight	
Sleeping hours*	<6 h/d	123 (30.4%)	41 (41.4%)	0.03	58 (25.9%)	106 (37.9%)	0.004
	>6 h/d	282 (69.6%)	58 (58.6%)		166 (74.1%)	174 (62.1%)	
Insomnia score*	<Score 6	229 (56.5%)	55 (55.6%)	0.85	123 (54.9%)	161 (57.7%)	0.56
	>Score 6	176 (43.5%)	44 (44.4%)		101 (45.1%)	119 (42.5%)	

*N (%)

†P-value was calculated using Chi-square

5. Discussion

Insufficient sleep is considered a risk factor for weight gain and development of type 2 diabetes. We showed in this study that people who sleep more than 6 hours per day were more protected against weight gain and type 2 diabetes than people who sleep less than 6 hours per day. In addition a decrease in the REE was observed when there is a lack of sleep in participants. This decrease in REE develops overweight and type 2 diabetes consequently. Our results came hand in hand with many experimental studies as reported by a summary report of Chaput et al in 2012 [9]. These experimental studies summarized that two consecutive nights of sleep deprivation to 4 h instead of 10 h decreased by 18% the circulating concentrations of the anorexigenic hormone leptin and a 28% increase in the concentrations of the orexigenic hormone ghrelin in conjunction with increased sensations of hunger and appetite [10]. Other studies discussed the effect of sleep deprivation on energy intake summarizing the possible mechanism by the fact that inadequate sleep has been reported in some studies to decrease leptin levels, increase ghrelin level and cortisol levels, alter glucose homeostasis, and activate the

orexin system [11].

In addition, the temporal release of the counter-regulatory hormones growth hormone (GH) and cortisol as well as the release of hormones that play a major role in appetite regulation, such as leptin and ghrelin, is partly dependent on sleep timing, duration and quality. Glucose tolerance and insulin secretion are also markedly modulated by the sleep-wake cycle [12].

However, our results are contradicted by the study published by Markwald et al, who showed that in 16 adults for a 14- to 15-d-long that insufficient sleep increased total daily energy expenditure by ~5% and insufficient sleep led to a weight gain of 0.82 ± 0.47 kg (\pm SD) weight gain despite changes in hunger and satiety hormones ghrelin and leptin, and peptide YY, which signaled excess energy stores [13]. Subsequent study examined appetite regulation after 2 nights of 4 h in bed and after 2 nights of 10 h in bed, in a randomized cross-over design. This study confirmed the decrease in leptin levels seen in the previous study, with a 18% decrease of leptin levels after the short nights relative to the long nights. Furthermore, ghrelin was assayed and showed a 28% increase after the 2 nights of 4 h in bed. Appetite for high carbohydrate nutrients was the most

affected with a 32% increase [14]. Importantly, the subjective report of increased hunger was correlated with the increase in ghrelin to leptin ratio [15]. An important observation was reported by Spiegel et al [16] concerning the effect of sleep deprivation on metabolic and endocrine function that overall leptin levels, evening cortisol levels, and the HOMA response to breakfast varied in a dose-response relationship with sleep duration. Shorter sleep duration was associated with greater disturbances in these hormonal and metabolic variables. Leptin levels were lowest when the subjects were in a state of sleep debt, signaling the brain an unnecessary need for extra caloric intake. Evening cortisol levels were highest when the subjects were in a state of sleep debt. A state of sleep debt therefore appears to delay the normal return to low levels of corticotrophic activity [15]. HOMA levels post-breakfast were the highest in a state of sleep debt indicating a decrease in glucose tolerance and/or a decrease in insulin sensitivity. The 24-hour Growth hormone (GH) profiles in the 8- and 12-hour bedtime conditions were qualitatively similar, with a trend for lower post-sleep peak values in the extended bedtime that is consistent with a reduced homeostatic drive for sleep with the decreased duration of the wake period [15]. In the state of sleep debt, a GH pulse prior to sleep onset was observed, in addition to the normal post-sleep onset GH pulse. The elevation of GH concentrations during waking could have an adverse impact on glucose metabolism [15]. Concerning Caffeine consumption, our study results show that subjects who drink caffeine had 3.6 times lower risk of developing diabetes compared to non-drinkers (3.6 and 95% CI [1.5-3.6]). This result is parallel to a meta-analysis of 28 prospective studies on 1,109,272 study participants and 45,335 cases of type 2 diabetes, published by Ding et al [17] who showed that Coffee consumption was inversely associated with the risk of type 2 diabetes in a dose-response manner. Both caffeinated and decaffeinated coffee was associated with reduced diabetes risk. Another meta-analysis of 42 studies on more than 1,091,000 persons with or without diabetes shows that the association of coffee, decaffeinated coffee and caffeine intake with type 2 diabetes incidence was stronger for women than that for men. Dose-response analysis suggested that incidence of type 2 diabetes decreased by 12% [0.88 (0.86-0.90)] for every 2 cups/day increment in coffee intake, 11% [0.89 (0.82-0.98)] for every 2 cups/day increment in decaffeinated coffee intake and 14% [0.86 (0.82-0.91)] for every 200 mg/day increment in caffeine intake [18]. First, coffee contains compounds that are antioxidants [19] which may be related to the risk of diabetes because the oxidative stress may be a pathogenic mechanism linking insulin resistance with dysfunction of both beta cells and endothelium [20]. Second, phenol chlorogenic acid and its degradation products (quinides) in coffee contribute to inhibition of glucose absorption in the intestine, reducing hepatic glucose output through inhibition of glucose-6-phosphatase [21]. Third, caffeine may also contribute to the inverse association between coffee and diabetes through increasing of basal energy expenditure

stimulating fat oxidation and mobilization of glycogen in muscle, and weight loss [22]. Caffeine has also been related to acute decrease of insulin sensitivity, although prolonged use of caffeine may result in tolerance for such acute effect [23]. Finally, other components in coffee such as magnesium have been associated with a lower risk for type 2 diabetes [24].

Potential limitations of our study include the lack of data that could assist in managing this study prospectively. We considered a study design of cross sectional a non-conclusive study that could only explain possible relation between both variables (sleep quality and type 2 diabetes or overweight) but it cannot confirm. Thus, further prospective studies are needed to confirm this relation.

6. Conclusions

This study suggests that sleep disturbances, including insufficient sleep due to bedtime curtailment and poor sleep quality, may represent novel risk factors for obesity and type 2 diabetes. While laboratory studies have been conducted in adults only, a large number of epidemiologic studies in pediatric populations should be well elaborated to decrease the rate of obesity worldwide.

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