Sleep and lifestyle in young adult monozygotic twin pairs discordant for body mass index

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ABSTRACT

Objectives: The causal nature of the sleep-obesity association is unclear. To control for potential confounding by genes and shared environment, we studied monozygotic twin pairs discordant for body mass index (BMI). First, we investigated sleep in relation to BMI. Second, we examined associations of objective and subjective sleep duration and sleep debt (objective or subjective sleep duration minus subjective sleep need) with eating behaviors and physical activity (PA).
Design: Cross-sectional study.
Setting: Finnish twins in everyday life circumstances.
Participants: Seventy-four healthy young adult monozygotic twin pairs, of whom 36 were BMI-discordant (ΔBMI $\geq$ 3 kg/m^2).
Measurements: Clinical measurements estimated BMI and body composition. Sleep, eating, and PA behaviors were measured by self-report and actigraphy.
Results: Compared to co-twins with lower BMI, co-twins with higher BMI reported shorter sleep (P = .043), more snoring (P = .0093), and greater tiredness (P = .0013) and trended toward eveningness (P = .036). Actigraphy-measured sleep duration correlated highly within BMI-discordant twin pairs (r = 0.63, P = .004). Subjective sleep debt was consistently positively associated with disinhibited eating and binge eating, but not with BMI. Subjective and objective sleep debt had negative correlations with moderate-to-vigorous PA.
Conclusions: Twins with higher BMI showed less favorable sleep characteristics than their co-twins with lower BMI. Subjective sleep debt is a potential target for intervention to reduce eating and PA behaviors that promote weight gain. Experimental studies could elucidate mechanisms underlying tiredness in individuals with higher BMI and investigate causal relationships between sleep debt, BMI, and lifestyle.

Introduction

Sleep and obesity are strongly connected in a bidirectional manner.[1] Obesity is related to sleep disorders—such as insomnia, sleep apnea, and restless leg syndrome—that impact the quality and quantity of sleep.[2] Shorter sleep and daytime sleepiness correlate with lower objective physical activity (PA) in young adults,[3] and shorter sleep might lead to overeating through increased appetite,[4] thus promoting weight gain.

Short sleep (<7 h/night) has been associated with obesity,[5] and it correlates with higher body mass index (BMI), particularly in young adults (18-34 years).[6] However, most studies on sleep and obesity have depended on self-reported sleep. Although subjective reports are prone to response bias, a recent retrospective study on >120,000 individuals who consistently wore a commercially available wrist-

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worn device replicated the subjective findings with objective measurements.[7]

Self-reported sleep duration may be more informative in relation to sleep need, which varies between individuals.[8] A sleep duration below one's need indicates sleep debt, a common occurrence in the modern environment. Insufficient sleep, defined by one study as at least a one-hour sleep debt, occurred in 20.4% of 12,423 Finnish adults, of whom 44.1% experienced this as a long-standing condition (9-year follow-up).[9] Data on whether sleep debt is related to BMI or obesogenic behaviors are lacking, however.

Short sleep duration, poor sleep quality, and habitual sleep debt can be linked to obesity through their effects on eating and PA behaviors. Adults who self-reported short sleep in combination with high levels of emotional[10] or disinhibited[11] eating behaviors exhibited elevated food consumption. Experimentally induced short sleep lowered dietary restraint in women and caused weight gain.[12] Self-reported insufficient sleep for at least 14 of the preceding 30 days was linked with a higher prevalence of physical inactivity and obesity.[13] Contrasting evidence suggested that neither dietary habits nor PA mediated the relationship between sleep and obesity.[14]

In addition, self-reported eveningness chronotype has been prospectively associated with higher BMI.[15,16] Heritability estimates of up to 54% and recent genome-wide association studies (GWAS) suggest a genetic component of chronotypes.[17] However, a recent Mendelian randomization study of ~700,000 individuals found no causal association between chronotype and BMI.[18]

Research on sleep and obesity would benefit from controlling for genetic influences. Twin research has shown a modest heritability of sleep characteristics. A recent meta-analysis found moderate heritability for self-reported sleep duration (38%; 95% CI: 16%-56%) and sleep quality (31%; 95% CI: 20%-41%).[19] Unique environmental factors explain most of the variation in sleep variables. Genetic estimates are now consistent with the pedigree-derived heritability for BMI of 40%.[20] Self-reported short (<7 hours) and long (>9 hours) sleep duration and an evening chronotype appear to intensify the genetic risk of obesity.[21] Furthermore, genes with overlapping functions for sleep, obesity, and metabolism have been identified.[22] These studies suggest that genetic influences partly explain associations between sleep and obesity.

In order to control for genetic confounding, we included monozygotic (MZ) twin pairs. MZ twin pairs share an identical genetic predisposition, are of the same age and sex, and due to their upbringing in the same household, they share various early-life environmental influences (eg, in utero, family, and neighborhood environments). Studies with co-twin controls inherently control for shared variables that studies usually cannot control for by screening for twin pairs with co-twins that differ in a specific exposure or outcome, while other factors remain constant. One co-twin study investigating sleep and BMI[23] selected MZ twin pairs that consisted of one co-twin with short sleep duration (<7 hours) and the other with normal sleep duration (7-8.9 hours). The short sleepers had a 1.0 kg/m² higher BMI (25.7 vs 24.7 kg/m²), while controlling for confounding influences of genotype and shared early environmental factors.

In our study, we examined healthy young adult MZ twin pairs discordant for BMI who had been raised in the same household until adulthood. Each pair consisted of one co-twin with lower and one co-twin with higher BMI. This design controls for genetic and within-family influences on BMI, sleep, and lifestyle. This study aimed to uncover within-pair differences in a comprehensive set of self-reported and actigraphy-measured sleep variables. We hypothesized that a higher BMI is associated with shorter sleep duration, higher sleep debt, worse sleep quality, and an eveningness chronotype. Moreover, we investigated the associations of self-reported and actigraphy-measured sleep duration and sleep debt with objective PA and subjective eating behaviors. Since we previously demonstrated that co-twins with greater BMI exhibited lower PA[24] and higher

susceptibility to overeating behaviors[25], we hypothesized that sleep duration and sleep debt are associated with these lifestyle behaviors.

Participants and methods

Study sample

This cross-sectional study included 74 healthy young adult MZ twin pairs. We divided twin pairs into co-twins with lower and higher BMI, which resulted in groups of 36 BMI-discordant (BMI difference ≥3 kg/m²) and 38 BMI-concordant (BMI difference <3 kg/m²) twin pairs. The BMI-concordant twins functioned as a reference group to compare sleep within pairs similar in BMI.

The participants were recruited from two population-based longitudinal studies of 10 full birth cohorts between 1975-1979 and 1983-1987 (FinnTwin16 and FinnTwin12, n = 5,417 pairs).[26,27] This study analyzed data acquired between 2003 and 2013. Participants were enrolled based on their self-reported weight and height as young adults, with the aim to cover a wide range of BMI values between and within pairs to ensure studies of both BMI-discordant and -concordant twin pairs. The twins attended a clinical examination, which included a medical history, physical examination, and laboratory evaluation. We excluded twins with concomitant diseases, regular medication (except contraceptives), anemia, hypo- or hyperthyroidism, psychiatric disorders, eating disorders, and weight cycling (±5 kg within the past 3 months). The protocol was designed and performed according to the principles of the Helsinki Declaration and was approved by the Ethics Committee of the Helsinki University Central Hospital. All participants provided written informed consent.

Clinical measurements

Weight and height were measured barefoot and in light clothing to calculate BMI. Body composition was measured by dual energy X-ray absorptiometry (DEXA) (Lunar Prodigy, Madison, WI, USA; software version 8.8). Fat percentage of the whole body was calculated as fat mass / (fat mass + lean mass + bone mineral content) * 100. Zygosity of the twin pairs was confirmed through genotyping of multiple genetic markers from genome-wide arrays (Illumina 670 & Illumina Human CoreExome chips).

Actigraphy

The Actiwatch AW7 (CamNtech Ltd, Cambridge, UK) is a wrist-worn monitor that records bodily movements to provide an objective estimation of sleep parameters. The units were mounted on the non-dominant arm and positioned using a standardized protocol. The twins were instructed to wear the Actiwatch at all times during the day and night for seven consecutive days, except in the shower, sauna, or other water activities. Activity data were saved in one-minute epochs and scored according to a sleep log or the marker times with Actiwatch Activity and Sleep Analysis Software 7 (CamNtech Ltd, Cambridge, UK). We used medium sensitivity in the scoring program, the sampling frequency was up to 32 Hz, and the filters were set at 3-11 Hz.

In principle, all available valid data were analyzed. Individual nights with reports of a significant illness or symptom (eg, fever, flu) or a technical failure were excluded. However, the night was included if only a minor illness or symptom was reported (eg, short period of stomachache, headache). A completed Actiwatch registration was available for 50 individuals; 3 of these were excluded from statistical analyses (technical failure, absence of sleep log and second registration, and traveling during observation period). Thus, 47 registrations were available for statistical analysis of Actiwatch data, which comprised 23 complete twin pairs. Excluding nonwear time, the twins wore the Actiwatch continuously for mean 6.7 (SD = 1.5) consecutive days.

The Actiwatch approximated objective sleep duration, sleep efficiency, sleep onset latency, and a fragmentation index (see Actiwatch user manual[28]). The Actiwatch AW7 algorithm detects the time of sleep initiation and waking. The time from sleep initiation to waking is the assumed sleep period. When the Actiwatch measures 40 or more arm movement counts per minute (CPM), the participant is considered awake. The assumed sleep period minus the number of minutes awake forms the objective sleep duration. Sleep onset latency is the duration between bedtime and sleep initiation time. Sleep efficiency reflects the proportion of objective sleep duration during the time spent in bed (from bedtime to get up time). The fragmentation index is an indication of restlessness and combines 2 constructs: (1) the percentage of time spent awake during assumed sleep, added to (2) the number of immobility/sleep phases that lasted a maximum of one minute as a percentage of the total number of immobility/sleep phases.

As in our previous study on 22 of the same MZ twin pairs,[24] the hip-worn ActiGraph GT1M (ActiGraph LLC, Pensacola, FL, USA) provided diurnal PA measurements: sedentary time (0-99 CPM), time spent in light (100-1951 CPM), moderate-to-vigorous PA (MVPA; ≥1952 CPM),[29] and steps/day. These PA variables were included for correlations with subjective and objective measurements of sleep duration and sleep debt.

Questionnaires

Five standard questions,[9,30] known as basic sleep items, assessed sleep duration and sleep need (continuous variables) and sleep quality, morning tiredness, and daytime tiredness (categorical variables; Text S.1). To calculate subjective sleep debt, we subtracted self-reported sleep duration from self-reported sleep need. For objective sleep debt, we subtracted actigraphy-measured sleep duration from self-reported sleep need.

Other self-reported sleep dimensions were measured with the Basic Nordic Sleep Questionnaire (BNSQ).[31] From the BNSQ, we calculated sum scores for insomnia (questions 1 and 3-6, score range 5-25), tiredness (questions 8 and 9, score range 2-10), and snoring (questions 16 and 17, score range 2-10) to indicate the severity of the symptoms in these 3 dimensions (Text S.2), comparable to a recent approach.[32] The BNSQ also provided bedtimes and wake times during weekdays and weekends, from which we derived the time spent in bed and differences between weekends and weekdays. All BNSQ-derived variables were treated as continuous variables. We excluded participants who reported less than 4 hours in bed, incomplete bed and wake time data (only the incomplete days were removed), and times recorded in error (eg, times outside the 24-hour time spectrum).

The morningness-eveningness questionnaire (MEQ) estimated chronotype through a sum score of 19 questions (range 16-86).[33] The cut-points for chronotype categories were 16-30 = definitely evening type, 31-41 = moderately evening type, 42-58 = neither/intermediate type, 59-69 = moderately morning type, 70-86 = definitely morning type.

Eating behaviors previously investigated[25] were included for correlations with self-reported sleep duration and sleep debt. Cognitive restraint of eating, disinhibited eating, and susceptibility to hunger were derived from the Three Factor Eating Questionnaire (TFEQ).[34] Information on emotional eating, external eating, and restrained eating was obtained through the Dutch Eating Behaviour Questionnaire (DEBQ).[35] Binge-eating scores were calculated from the Binge-Eating Scale (BES).[36]

We also assessed smoking status (daily smoker, yes/no) and alcohol consumption in standard drinks (12 g ethanol per drink) per week.

Statistical methods

Statistical analyses were performed in Stata/MP 16.0 (StataCorp LLC, College Station, TX, USA). Because of the non-normal distribution of the majority of variables and the small sample size (particularly for the actigraphy data), we applied non-parametric statistical testing.

First, we tested sex differences with Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables. For all pairs, 1-way random-effects intraclass correlation coefficients (ICC) assessed within-pair resemblance on sleep variables, controlled for between-pair age and sex.

Next, we compared co-twins from BMI-discordant and -concordant pairs. Wilcoxon signed-rank tests compared continuous variables, and symmetry tests compared categorical variables for basic characteristics and sleep in co-twins with lower versus higher BMI. We assessed the ICCs of objective sleep duration (Actiwatch AW7) separately for BMI-discordant and -concordant pairs and tested for ICC size differences with Fisher's z transformation to assess whether resemblance was dependent on the within-pair BMI difference.

Partial correlations indicated the association of subjective and objective sleep duration and debt with self-reported eating behaviors and PA from actigraphy, with age and sex as covariates. Partial correlations were measured both for individual measurements of sleep and behaviors, as well as within-pair differences in these variables. Individual correlations suggest an overall association between sleep and behavior, while the within-pair analyses remove the potential confounding factors in twins (eg, genetics, shared early environment).

Results

Basic sleep characteristics

On average, the participants fell within the overweight BMI category, slept 7 hours objectively and 7.5 subjectively, and reported sleep need of 8.0 hours. They slept fairly well, experienced 1-2 days per week morning and/or daytime tiredness, and had low scores on the insomnia [5-25], tiredness [2-10], and snoring [2-10] scales. Furthermore, individuals reported regular sleep and wake timing, indicative of an intermediate chronotype, confirmed by the MEQ score.

Differences by sex

Men and women differed on clinical, lifestyle, and sleep variables (Table 1) and known differential distribution of anthropometrics. In addition, women drank 6 fewer units of alcohol per week than men. Women slept longer (0.5 hours self-reported, 1.2 hours actigraphy)—mainly owing to 45 minutes earlier bedtimes and 56 minutes longer time in bed—and experienced 52 minutes less objective sleep debt), morning tiredness on 1-4 fewer days, 11 minutes shorter sleep latency, 5.8% higher sleep efficiency, and 7.5 units lower fragmentation index than men.

Characteristics and sleep schedule by BMI discordance

Expectedly, co-twins with greater BMI had higher adiposity measurements than co-twins with lower BMI (Table 2), and these differences were considerably larger in BMI-discordant twin pairs than in BMI-concordant pairs (Mann-Whitney U test produced $P < .001$ for all four weight measurements).

Participants predominantly exhibited regular schedules of nocturnal sleep and diurnal activities (eg, work, school) during weekdays (Table S.1), inferred from the interquartile range of bedtimes from 22:12 to 23:12 and wake times from 06:00 to 07:00, with a delay in timing during weekends: interquartile range of bedtimes from 23:00 to 01:00 ($P < .001$) and wake times from 07:30 to 09:40 ($P < .001$).

Table 1
Characteristics and sleep measurements of individuals and by sex

	Individuals	Men	Women	P value	Δ%
Characteristics	n = 148	n = 74	n = 74		
Age, y	29.2 (3.9)	29.7 (3.6)	28.6 (4.2)	.054	−3.7
Height, cm	172.8 (9.7)	179.4 (6.7)	166.1 (7.3)	<.001	−7.4
Weight, kg	80.3 (17.5)	86.6 (15.2)	74.0 (17.5)	<.001	−14.5
BMI, kg/m^2	26.8 (5.1)	26.8 (3.9)	26.8 (6.0)	.31	0
Fat mass, kg	26.1 (12.2)	23.2 (10.8)	29.0 (12.9)	.008	25.0
Body fat, %	31.6 (10.8)	25.6 (8.7)	37.6 (9.3)	<.001	46.9
Daily smoker (yes/no), freq.	35/77	19/35	16/42	.39	−29.8
Alcohol, drinks/week	6.7 (8.3)	9.6 (10.0)	4.2 (5.4)	.0020	−56.3
Actiwatch AW7	n = 47	n = 21	n = 26		
Objective sleep duration, h	7.1 (0.9)	6.4 (0.7)	7.6 (0.6)	<.001	18.8
Sleep latency, min	16 (15)	22 (19)	11 (9)	.012	−50.0
Sleep efficiency, %	85.2 (5.6)	82.0 (5.2)	87.8 (4.4)	<.001	7.1
Fragmentation index	27.6 (8.6)	31.2 (9.5)	24.7 (6.5)	.036	−20.8
Basic sleep items	n = 144	n = 72	n = 72		
Sleep duration, h	7.5 (0.9)	7.3 (0.8)	7.8 (0.9)	.001	6.8
Sleep need, h	8.0 (0.8)	7.9 (0.8)	8.1 (0.7)	.026	2.5
Subjective sleep debt, min	29.2 (55.5)	36.4 (63.6)	22.1 (45.6)	.23	−39.3
Objective sleep debt, min	58.8 (53.7)	88.3 (48.2)	36.3 (47.0)	<.001	−58.9
Sleep quality[a,b]	2 (1-2)	2 (1-2)	2 (1-2)	.70	0
Morning tiredness[a,c]	3 (2-3)	2 (2-3)	3 (2-4)	.014	50.0
Daytime tiredness[a,c]	3 (2-4)	3 (2-4)	3 (2-4)	.70	0
BNSQ	n = 81	n = 38	n = 43		
Insomnia score	11.8 (3.5)	12.5 (3.6)	11.1 (3.3)	.069	−11.2
Tiredness score	5.2 (2.3)	5.4 (2.4)	5.1 (2.2)	.52	−5.6
Snoring score	4.7 (2.2)	5.2 (2.2)	4.3 (2.2)	.074	−17.3
Bedtime weekday[d]	22:52 (63)	23:17 (58)	22:32 (61)	.001	−3.3
Bedtime weekend[d]	23:55 (77)	06:35 (85)	06:44 (71)	.62	2.3
Wake time weekday[d]	06:40 (97)	00:10 (80)	23:41 (109)	.15	−2.0
Wake time weekend[d]	08:41 (103)	08:43 (104)	08:40 (103)	.81	−0.5
Time spent in bed weekday[d]	07:49 (78)	07:17 (71)	08:13 (70)	.002	12.6
Time spent in bed weekend[d]	08:46 (99)	08:32 (71)	08:59 (53)	.10	5.2
Bedtime delay weekend[d]	01:05 (75)	01:00 (65)	01:11 (88)	.58	18.0
Wake time delay weekend[d]	02:05 (63)	02:20 (91)	01:53 (105)	.12	−18.9
MEQ	n = 76	n = 34	n = 42		
Score	52.9 (9.0)	51.1 (8.6)	54.3 (9.1)	.19	6.3

Δ%, difference in percentages [(women-men) / men × 100]; BMI, body mass index; n, number of individuals; freq., frequency; BNSQ, Basic Nordic Sleep Questionnaire; MEQ, morningness-eveningness questionnaire.

Values are mean (SD), P values from chi-square test for categorical variables and Mann-Whitney U test for continuous variables.
  [a] Median and interquartile range for categorical variables.
  [b] 1 = well, 2 = fairly well, 3 = fairly poorly, 4 = poorly.
  [c] 1 = (almost) daily, 2 = 3-5 d/w, 3 = 1-2 d/w, 4 = <1/week, 5 = <1/month.
  [d] Hours:minutes (min).

Sleep duration, sleep quality, and chronotype by BMI discordance

In BMI-discordant twin pairs, co-twins with higher BMI reported, on average, 18 min shorter sleep, more tiredness, and higher snoring scores than co-twins with lower BMI (Table 2). Compared to co-twins with lower BMI, 50% of co-twins with higher BMI slept less (18% 0.5 hours, 18% 1.0 hours, and 15% ≥ 1.5 hours), 24% slept the same amount, and 26% slept more (21% 0.5 hours and 5% ≥ 1.0 hours).

In all twin pairs, ICCs of subjective sleep quantity and quality (all basic sleep items and BNSQ variables) were modest (ICC = 0.21-0.39; Table S.2), and differences in bedtimes and wake times were minor (Table S.1). Within-pair associations for objective sleep duration were fairly strong, ICC = 0.62 (Table S.2, Fig. 1), with no apparent difference (P = .97) in ICCs between BMI-discordant (ICC = 0.63 [0.20, 0.86], and BMI-concordant pairs (ICC = 0.64 [−0.002, 0.91]), indicating that within-pair BMI differences do not affect similarity in objective sleep duration.

In BMI-discordant pairs, co-twins with higher BMI typically had MEQ scores closer to the evening chronotype than co-twins with lower BMI (Fig. 2, Table 2). Differences were small: 62.5% of discordant pairs belonged to the same chronotype. Differences in the sleep items and chronotype were minimal within BMI-concordant pairs.

Sleep duration and sleep debt in relation to lifestyle behaviors

A longer subjective sleep duration was correlated with a shorter subjective sleep debt in individual (r = −0.62, P < .001) and pairwise (r = −0.54, P < .001) analyses. Associations of sleep duration and sleep debt with subjective eating and objective PA parameters are shown in Fig. 3. Sleep duration was negatively correlated with binge-eating in individuals (r = −0.22, P = .019): shorter sleep was associated with higher binge-eating score.

Longer sleep debt was consistently correlated with stronger susceptibility to disinhibited eating in individuals (r = 0.33, P = .0027) and within-pair differences in these behaviors (r = 0.35, P = .031), meaning the correlation holds regardless of genetic and shared environmental influences. Sleep debt was consistently positively correlated with binge-eating score in individuals (r = 0.34, P < .001) and within-pair differences (r = 0.35, P = .013). Longer sleep debt was linked with shorter time spent in MVPA in intrapair correlations (r = −0.53, P = .035).

Moderate correlations were observed between objective sleep duration and sleep debt individually (r = −0.33, P = .031) and pairwise (r = −0.44, P = .065). Longer objective sleep duration was correlated with shorter sedentary time in individuals (r = −0.37, P = .014).

Table 2
Characteristics and sleep measurements of co-twins with lower versus higher BMI from BMI-discordant and BMI-concordant twin pairs

	BMI-discordant twin pairs				BMI-concordant twin pairs			
	Lower BMI	Higher BMI	P value	Δ%	Lower BMI	Higher BMI	P value	Δ%
Characteristics	n = 36				n = 38			
Age, y	29.3 (4.6)	29.3 (4.6)	-	-	29.0 (3.3)	29.0 (3.3)	-	-
Female, %	61	61	-	-	39	39	-	-
Height, cm	171.9 (10.4)	172.3 (10.0)	.31	0.2	173.2 (9.4)	173.5 (9.3)	.43	0.2
Weight, kg	75.8 (16.8)	93.3 (19,2)	<.001	23.1	74.4 (2.2)	78.1 (2.3)	<.001	5.0
BMI, kg/m^2	25.5 (4.8)	31.3 (5.4)	<.001	22.7	24.7 (13.6)	25.8 (14.0)	<.001	4.5
Fat mass, kg	24.5 (11.7)	37.6 (12.2)	<.001	53.5	20.1 (8.2)	22.8 (8.7)	<.001	13.4
Body fat, %	31.5 (10.7)	40.2 (8.9)	<.001	27.6	26.4 (9.3)	28.6 (9.1)	<.001	8.3
Daily smoker (yes/no), freq.	9/20	9/20	1.00	0	8/19	9/18	.71	18.8
Alcohol, drinks/week	6.3 (9.8)	7.0 (7.9)	.20	11.1	5.6 (6.4)	7.8 (7.8)	.028	39.3
Actiwatch AW7	n = 15				n = 8			
Objective sleep duration, h	7.3 (0.8)	7.3 (0.9)	.59	0	6.8 (1.1)	6.9 (0.8)	.95	1.5
Sleep latency, min	12 (8)	20 (22)	.33	66.7	19 (14)	15 (10)	.64	−21.1
Sleep efficiency, %	85.2 (5.3)	85.5 (6.0)	.83	0.4	82.7 (5.1)	86.6 (6.2)	.078	4.7
Fragmentation index	27.8 (9.9)	26.6 (6.8)	.93	−4.3	32.4 (7.9)	24.9 (9.3)	.11	−23.1
Basic sleep items	n = 34				n = 35			
Sleep duration, h	7.6 (0.9)	7.3 (1.0)	.043	−3.9	7.6 (0.7)	7.8 (0.8)	.22	2.6
Sleep need, h	8.1 (0.6)	7.8 (0.9)	.088	−3.7	8.1 (0.9)	8.1 (0.8)	.67	0
Subjective sleep debt, min	26.5 (43.5)	30.9 (60.2)	.53	16.6	34.4 (44.4)	18.5 (66.5)	.23	−46.2
Objective sleep debt, min[a]	58.7 (49.3)	49.8 (17.9)	.54	−15.2	62.5 (40.8)	53.5 (37.5)	.56	−14.4
Sleep quality[b,c]	2 (1-2)	2 (2-3)	.18	0	2 (1-2)	1 (1-2)	.051	−50.0
Morning tiredness[b,d]	3 (2-4)	3 (2-3)	.48	0	3 (2-3)	3 (2-3)	.65	0
Daytime tiredness[b,d]	3 (2-4)	3 (2-4)	.56	0	3.5 (3-4)	3 (2-4)	.24	−14.3
BNSQ	n = 27				n = 12			
Insomnia score [5-25]	11.8 (3.4)	11.9 (3.5)	.51	0.8	12.2 (3.4)	10.4 (3.3)	.26	−14.8
Tiredness score [2-10]	4.9 (2.2)	6.1 (2.2)	.009	24.5	4.2 (2.3)	4.8 (1.9)	.42	14.3
Snoring score [2-10]	3.9 (3.4)	5.4 (2.3)	.001	38.5	4.2 (1.5)	5.3 (2.4)	.087	26.2
MEQ	n = 24				n = 10			
Score	53.3 (8.6)	49.8 (9.8)	.036	−6.6	56.4 (5.5)	55.2 (8.7)	.24	−2.1

BMI, body mass index; *n*, number of pairs; Δ%, difference in percentages [(higher BMI co-twin−lower BMI co-twin)/lower BMI co-twin × 100]; freq., frequency; BNSQ, Basic Nordic Sleep Questionnaire; MEQ, morningness-eveningness questionnaire.
Values are mean (SD), *P* values from Wilcoxon signed-rank test for continuous variables and symmetry test for categorical variables.

[a] *n* = 14 for BMI-discordant, *n* = 6 for BMI-concordant
[b] Median and interquartile range for categorical variables
[c] 1 = well, 2 = fairly well, 3 = fairly poorly, 4 = poorly
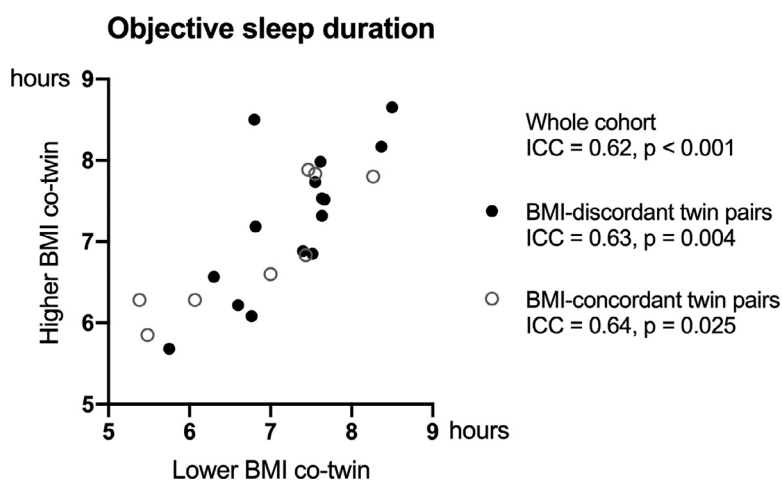[d] 1 = (almost) daily, 2 = 3-5 d/w, 3 = 1-2 d/w, 4 = <1/week, 5 = <1/month.

Fig. 1. Scatterplot and intraclass correlation coefficient (ICC) of actiwatch-measured sleep duration in hours in all co-twins with lower versus higher body mass index (BMI) with a separate display of twin pairs discordant (black circles) and concordant (white circles) for BMI.

Longer objective sleep debt was correlated with longer sedentary time (r = 0.64, P = .077) and shorter time spent in MVPA (r = −0.57, P = .021) in pairwise analyses.

Discussion

We hypothesized that a higher BMI in BMI-discordant twin pairs would be associated with shorter sleep duration, longer sleep debt, worse sleep quality, and an eveningness chronotype. In support of this, in BMI-discordant pairs, twins with higher BMI reported shorter sleep, more snoring, more tiredness, and scores toward eveningness compared to their co-twins with lower BMI. However, differences in objective sleep measurements were minor. Furthermore, all twin pairs had modest within-pair associations among subjective sleep quality, quantity, and chronotype, and strong correlations in objectively measured sleep duration. We also hypothesized that sleep
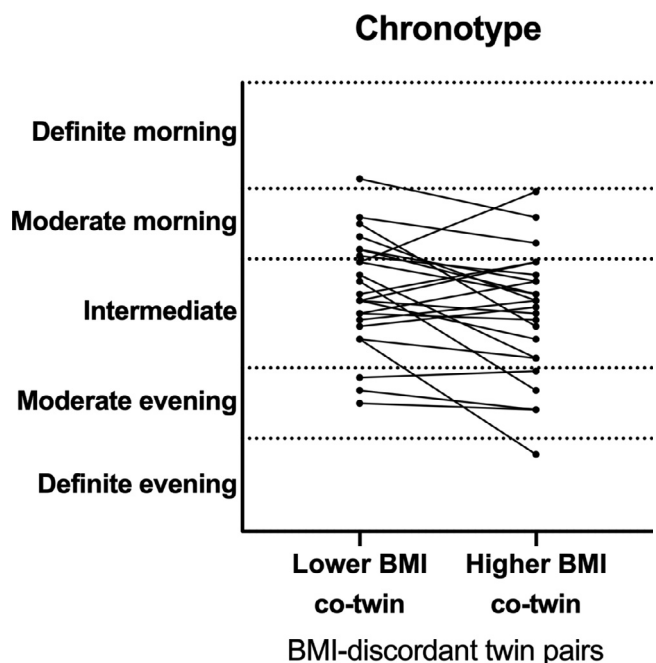
Chronotype

Fig. 2. Continuous scores on morningness-eveningness questionnaire plotted within their respective chronotype categories in co-twins with lower versus higher body mass index (BMI) from twin pairs discordant for BMI.

debt, in addition to sleep duration, would be associated with obesity and related lifestyle behaviors. Evidence was lacking for an association between subjective or objective sleep debt or duration and obesity, but, compared to shorter sleep duration, greater subjective sleep debt was associated with unhealthier behaviors. All findings were independent of the otherwise confounding influences of age, sex, genotype, and shared early environment.

When subjectively reported, twins with greater BMI acquired ~18 minutes shorter sleep than their co-twins with lower BMI (7.3 hours vs 7.6 hours). Although this difference seemed small, 50% of the twins with higher BMI reported at least 30 minutes shorter sleep than their co-twins with lower BMI (50% evenly distributed between 0.5 hours, 1.0 hours, and ≥1.5 hours shorter sleep). The majority of the remaining 50% consisted of equal sleep durations (24%) and 30 minutes longer sleep than co-twins with lower BMI (21%). This trend follows the commonly observed association of self-reported[6] and objectively measured[7] shorter sleep with higher BMI. It also complements a study on twin pairs discordant for subjective sleep duration, which found that those sleeping less than 7 hours had higher BMI (1.0 kg/m²) than co-twins sleeping 7-8.9 hours.[23] However, sleep duration and BMI were not correlated in the full sample. The BMI-concordant pairs might have diluted the strength and statistical support for the correlations, as they lack a difference in BMI and sleep duration.

BMI-discordant co-twins attained an identical average of 7.3 hours of objective sleep, with strong resemblance over a range of 5.4-8.7 hours. Our findings agreed with extant studies that observed a lack of association between sleep duration and BMI, measured both subjectively and objectively.[37] Our findings suggest the strong influence of a genetic component on actigraphy-measured sleep duration, possibly combined with acquired habitual sleep behaviors during early life. An alternative explanation for the similar objective sleep duration comes from recent evidence from a Mendelian randomization study, which found that a higher waist-to-hip ratio (corrected for BMI), as well as a higher BMI, were causally associated with shorter objective sleep duration.[38] Thus, co-twins with greater BMI might be in too early a stage of acquired overweight to experience its negative consequences on objective sleep duration.

The shorter perceived sleep duration in co-twins with higher BMI may explain their greater tiredness. Obesity has been prospectively linked to incident and persistent excessive daytime sleepiness, while weight loss resulted in its remission, even when controlling for baseline subjective sleep duration.[39] Furthermore, newly discovered
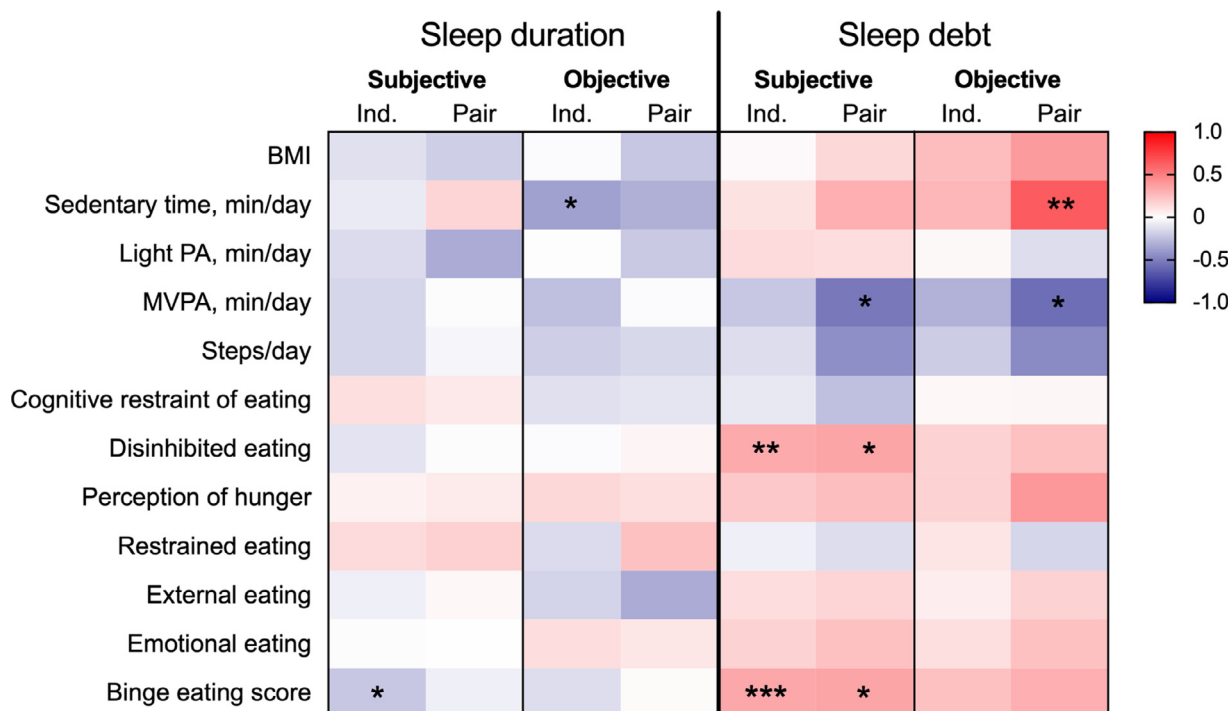

Fig. 3. Heatmap of partial correlations of subjective and objective sleep duration and sleep debt with body mass index (BMI), physical activity (PA), and eating behaviors, corrected for age and sex. "Ind." denotes correlations of individuals and "pair" indicates correlations of within-pair differences in sleeping and outcome variables. MVPA, moderate-to-vigorous physical activity. The number of individuals and pairs can be found in Table S.3. *P < .05, **P < .01, ***P < .001.

genetic variants for daytime sleepiness overlapped with obesity variants.[22] In this sense, in MZ twins with an identical genetic predisposition, BMI and tiredness might increase simultaneously. Co-twins with greater BMI additionally experienced higher severity and frequency of snoring. BMI is strongly associated with sleep apnea,[40] so the elevated snoring levels may indicate symptoms of sleep apnea,[41] which can also explain tiredness.

The co-twins with higher BMI trended closer to the evening chronotype than their co-twins with lower BMI. One study investigated long-term (mean >7 years) weight-loss (mean >33 kg) maintainers compared with treatment-seeking individuals with overweight or obesity.[42] The weight maintainers identified more often as morning types (34% vs 25%) and less often as evening types (20% vs 31%). Hence, morningness may benefit weight maintenance in general, or weight may influence chronotype. Furthermore, compared to morningness, an evening chronotype has seemed to reinforce the genetic risk of obesity.[21] The evening chronotype has also been associated with weight gain in college freshmen over an 8-week study period[15] and with higher BMI before bariatric surgery and 4 years afterward through stronger weight regain.[16] However, a recent and very large Mendelian randomization study found no evidence for a causal relationship between chronotype and BMI.[18] Perhaps, instead, a mismatch between one's chronotype and food intake associates with BMI,[43] but we were unable to quantify this. Furthermore, environmental factors, such as school and working hours, may impact whether individuals can live according to their chronotype and desired sleep duration.

Knowledge about behavioral influences on the sleep-BMI association is limited. One study found that individuals ate more and gained more weight over six years when they reported both short sleep (≤ 6 h) and high disinhibited eating levels.[11] Here, rather than self-reported sleep duration, a larger subjective sleep debt was associated with higher disinhibited eating and binge-eating scores, independent of genetic and shared environmental influences. Short sleep affects food-related hedonic features of the brain that may override the homeostatic drive for food intake,[44] contributing to overeating behaviors. In a similar study of BMI-discordant twins, we demonstrated associations between overeating behaviors and BMI.[25] However, we were unable to replicate connections between sleep (or BMI[25]) with emotional[10] and restrained eating.[12]

Longer subjective and objective sleep debts were linked, in pairwise analyses, with shorter objective time spent in MVPA. In a partly overlapping sample of twins, less time spent in MVPA was associated with a higher BMI.[24] Similarly, a higher frequency of subjective insufficient sleep (days/month) was linked to physical inactivity.[13]

In our study, longer objective sleep duration and shorter objective sleep debt were both associated with less sedentary time. Increasing the amount and intensity of PA may be relevant for more restorative sleep or lowered sleep debt, and vice versa. Nevertheless, sleep debt appeared unrelated to BMI but was associated with various behaviors that potentially stimulate weight gain. If the co-twins maintain the sleep debt in the long term, this might contribute to obesity development. Assessing subjective sleep debt through two simple questions could help identify individuals at risk for unhealthy behaviors, informing timely intervention.

This study has strengths and limitations. The cross-sectional study design precluded causal inferences, but the twin design excluded influences of age, sex, genotype, and shared early environment from associations. The sample size was fairly small because of the unique rare BMI-discordant co-twin control design. We used both subjective and objective sleep measurements, each with benefits and drawbacks. Self-reported sleep is a subjective estimate and perception of sleep, whereas actigraphy translates arm movements into sleep variables. Furthermore, self-reported sleep inquired about habitual sleep, while accelerometry measured 1 week of sleep. Compared to polysomnography (the gold standard), actigraphy lacks the ability to determine wakefulness while lying still or to distinguish restless sleep from wakefulness.[45] Questionnaires are subject to response bias, and they tend to overestimate actigraphy-measured sleep duration.[46] Overall, it is challenging to report habitual sleep quantity and quality, especially when night-to-night variations are large or sleep quality is poor. Self-reported sleep duration might exceed the actigraphy-measured duration due to an individual's mix-up between bedtime and sleep initiation time, as well as wake time and get up time. Therefore, self-reports of sleep duration indicate a subjectively estimated sleep time (and possibly a comprehensive sleeping experience).[46] Actigraphy and self-reported sleep are thus not directly comparable, and therefore we would not attribute the differences in findings to sample size or measurement accuracy per se. Regardless, questionnaires and actigraphy provide complementary information, are useful for the assessment of individuals under natural conditions, and cause minimal sleep disturbance. The eating behavior questionnaires are vulnerable to response bias. The ActiGraph GT1M provides reasonably accurate objective estimates of PA.[47] It is designed to detect most forms of human movement, but it filters high-frequency signals and may therefore not correctly record the intensity of highly strenuous activities. The generalizability of the ICCs (as estimates of max. heritability) is dependent on a lack of association with BMI (our sampling variable). The ICCs in this study resembled those from previous research,[48] suggesting little bias in the overall estimate.

Polysomnography could elucidate the underlying sleep mechanisms that accelerometry fails to detect, possibly explaining, for example, the lack of perceived restorative sleep in twins with higher BMI compared with lower BMI. The relationship between chronotype and BMI should be assessed in relation to the timing of other lifestyle behaviors (eg, food intake, PA, sleep). Prospective studies with larger sample sizes should investigate (1) determinants of sleep debt, (2) the causal relationship between sleep debt and BMI and the contribution of unhealthy behavioral patterns, and (3) individual variation in the consequences of sleep debt (observational and experimental).

Conclusions

In BMI-discordant twin pairs, co-twins with higher BMI reported shorter sleep, felt less rested, exhibited more snoring, and trended more toward eveningness than their co-twins with lower BMI. However, differences in objective sleep measurements were negligible. All co-twins, irrespective of BMI-discordance, revealed a fairly strong resemblance in objectively measured sleep duration and a modest resemblance in subjective sleep quantity, quality, and chronotype. Longer subjective sleep debt appeared unrelated to BMI, but it was consistently correlated with stronger susceptibility to disinhibited and binge eating. Additionally, longer subjective and objective sleep debt had intrapair correlations with shorter MVPA. These correlations were absent for sleep duration. Thus, sleep debt may contribute to weight gain with the co-occurrence of unhealthy lifestyle behaviors, which would suggest that interventions should target sleep debt instead of sleep duration. All findings were independent from otherwise confounding influences of age, sex, genotype, and early environmental factors.

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Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.sleh.2021.04.002.

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