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Sleep Duration and Obesity in Adults: What Are the Connections?

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Abstract Collectively, cross-sectional and longitudinal studies on self-reported sleep duration and obesity do not show a clear pattern of association with some showing a negative linear relationship, some showing a U-shaped relationship, and some showing no relationship. Associations between sleep duration and obesity seem stronger in younger adults. Cross-sectional studies using objectively measured sleep duration (actigraphy or polysomnography (PSG)) also show this mixed pattern whereas all longitudinal studies to date using actigraphy or PSG have failed to show a relationship with obesity/weight gain. It is still too early and a too easy solution to suggest that changing the sleep duration will cure the obesity epidemic. Given novel results on emotional stress and poor sleep as mediating factors in the relationship between sleep duration and obesity, detection and management of these should become the target of future clinical efforts as well as future research.

Keywords Sleep · Obesity · Adults · Review

Introduction

Sleep duration in the general population has gathered a large interest over the last decade as there have been reports of substantial sleep duration decrease [1, 2] and parallel to this decrease, the prevalence of obesity has increased, with several studies finding associations between the two conditions [3–6]. In addition, sleep complaints are common [7] and an increasing interest in the relationship between sleep and obesity and especially short sleep as a possible cause for obesity has evolved.

Several pathways in which sleep duration can affect development of obesity have been suggested. Short sleep duration has been associated with increased hunger [8, 9] as well as irregular eating habits, with snacking between meals [10] and certain obesity-related behaviors, such as lower physical activity and lower fruit and vegetable consumption [11]. In addition, disrupted eating patterns such as having a dominance of snacks over meals as well as higher intake of fat and sweets and lower intake of fruits and vegetables are associated with both short and long sleep duration [12, 13]. Sleep curtailment has also been shown to undermine dietary efforts to reduce obesity [14]. Furthermore, sleep restriction has also been shown to reduce the plasma leptin concentration [15], the adipocyte-derived hormone that promotes satiety and suppresses appetite [16], and increase ghrelin [17], the stomach-derived appetite-stimulating polypeptide that, together with leptin, regulates dietary intake [18]. Short sleepers (<5 h) have been shown to have lower leptin levels and higher ghrelin levels compared with persons sleeping 8 h [17] and it has been argued that these changes may be mediators of an increased appetite and promotion of obesity.

In the light of the interest in associations between sleep duration and obesity in adults, a review on epidemiological studies on the topic is of interest.

Aim

We aimed to review cross-sectional, longitudinal, and interventional studies on sleep duration and obesity in adults. We

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included studies that reported on associations between sleep duration/sleep time and obesity/weight gain, and we included studies both on reported and measured sleep duration.

Relationship Between Sleep Duration and Obesity

Cross-Sectional Studies

Table 1 shows an overview of the cross-sectional studies on sleep duration and obesity. Most studies have assessed sleep duration through questionnaires (i.e., self-reported sleep duration). Collectively, the studies on self-reported sleep duration and obesity do not show a clear pattern of association with some showing a negative linear relationship (i.e., short sleep duration associated with obesity) [6, 19–31], some showing a U-shaped relationship (i.e., both short and long sleep duration associated with obesity) [17, 26, 32–39], some showing no relationship [40–42], and some showing different results for men and women [43–47] or for different age groups within the study where a relationship is seen in younger adults but not in older [4, 5, 48, 49] (Table 1).

Nonetheless, eight of the cross-sectional studies of sleep duration have objectively measured sleep [31, 50–56] using either actigraphy [50, 52, 53, 55, 56] or one night polysomnography (PSG) [31, 51, 54]. Three of the studies show a negative linear relationship [50, 54, 56], one shows a U-shaped relationship (however, non-significant after adjusting also for sleep fragmentation) [55], and four studies show no association [31, 51–53] between sleep duration and measures of obesity (Table 1).

Longitudinal Studies

Table 2 shows an overview of the prospective studies on sleep duration and obesity. As in the cross-sectional studies, longitudinal studies using self-reported sleep duration and obesity do not show a clear pattern of association with some showing a negative linear relationship [4, 5, 57–60], some showing a U-shaped relationship [3, 33, 61], and some showing no relationship [20, 27, 62, 63]. Also, different results by age [64] or gender [65, 66] have been reported. One recently published study shows increased sleep duration in older women to be associated with increased weight [67] (Table 2).

To date, there are three longitudinal studies where sleep duration has been assessed objectively by either actigraphy [50, 68] or PSG [69••]. None of these studies has shown a relationship with obesity/weight gain.

Interventional Studies

The best way to assess the relation between sleep duration and obesity and whether (in particular) short sleep duration causes obesity or weight gain is to assess if a changing sleep duration changes weight/body status. There has been a number of interventional in-lab randomized, controlled trials (RCTs) looking at the effect of changes in sleep duration on different measures of metabolism [70] but only one of them has had weight change as a primary outcome [71]. Collectively, the experimental RCTs suggest that restricting sleep increases food intake and energy expenditure. However, Capers et al. state in their meta-analysis that the experimental studies do not provide a strong basis for support of a causal relationship between sleep duration and obesity and that future controlled trials that assess impact of increased sleep duration on body weight are warranted [70]. To our knowledge, only one study has set out to study the effect of increased sleep duration on real-life short sleepers, using a RCT setting. The Sleep Extension Study [72•] is a RCT of sleep extension in a group of chronically sleep-deprived (less than 6.5 h per night) obese subjects. The study hypothesis was that sleep extension would cause weight loss and give metabolic and endocrine improvements in the study participants. However, Cizza et al. report that between screening and randomization (i.e., prior to any intervention), substantial improvements in sleep and also biochemical parameters were seen. They conclude that because of these improvements, the "true" study baseline has changed, potentially affecting the outcome [72•].

Discussion

Studies on the relationship between sleep duration and obesity in adults have given mixed answers, and when a relationship has been found, also the direction has varied. Cross-sectional studies have found negative linear, U-shaped, or no association. Not even the cross-sectional studies where an objective measure of sleep duration has been obtained seem to give a clear picture of the relationship. Longitudinal studies using self-reported measures of sleep duration show as in the cross-sectional studies, negative linear, U-shaped, or no association. One longitudinal study in older women has shown increase in sleep duration to be associated with increased weight. However, all of the three longitudinal studies published to date, where objective measures of sleep duration have been obtained, show no association between sleep duration and subsequent obesity, thereby indicating that short sleep duration per se does not cause obesity. In addition, even though experimentally shortened sleep duration seems to correlate with weight-promoting factors and increased weight, the only RCT planned to study if increased sleep duration could reduce obesity in a group of obese real-life short sleepers has not been able to do this as both groups improved in the time between inclusion and randomization [72•]. This was probable due to the "Hawthorne effect" i.e., some people perform better when they are participants in an experiment.

Table 1 Cross-sectional studies on sleep duration and measures of obesity in adults

Study population (author, year)	Sample size	Gender	Country	Ages (years)	Measure of sleep duration	Measure of obesity	Relationship between sleep and obesity
Health and Nutrition Survey in Valencia, Spain (Vioque, 2000) [28]	M: 918 F: 854	M & F	Spain	≥15	Self-reported	Self-reported (BMI)	All: negative linear
Cancer Prevention Study II (Kripke, 2002) [46]	M: 480,841 F: 636,095	M & F	USA	30–102	Self-reported	Self-reported (BMI)	M: slight negative linear F: U-shape (nadir 7/8 h)
Working Scottish men and women (Heslop, 2002) [22]	6797	M & F	Scotland	18–65	Self-reported	Objectively measured (BMI)	Negative linear
Japan Collaborative Cohort Study on Cancer—JACC (Tamakoshi, 2004) [41]	104,010	M & F	Japan	40–79	Self-reported	Self-reported	No association
Wisconsin Sleep Cohort (Taheri, 2004) [17]	1024	M & F	USA	30–60	Self-reported (diary)	Objectively measured (BMI)	U-shape (nadir 7–8 h)
Zurich Psychiatric risk factor cohort (Hasler, 2004) [5]	367	M & F	Switzerland	40	Self-reported	Self-reported (BMI)	No association at age 40 negative linear when participants were younger
Sleep Heart Health Study (Gottlieb, 2005) [40]	1486	M & F	USA	Approx. 70 ± 8.3 SD	Self-reported	Objectively measured (BMI, WC)	No association
National Health and Nutrition Examination Survey 1 (NHANES I) (Gangwisch, 2005) [4]	3682	M & F	USA	32–49	Self-reported	Objectively measured (BMI)	Negative linear NB—no association in those aged 50–86
Detroit Michigan Phone Survey (Singh, 2005) [37]	3158	M & F	USA	18–64	Self-reported	Self-reported	U-shape (nadir 8–9 h)
Patients from primary care practices in Virginia, USA (Vorona, 2005) [29]	924	M & F	USA	18–91	Self-reported	Self-reported	Negative linear
Gothenburg Diabetes Study (Björkelund, 2005) [20]	1462	F	Sweden	38–60	Self-reported	Objectively measured (BMI, WHR)	Negative linear
Coronary Artery Risk Development in Young Adults (CARDIA) (Lauderdale, 2006) [53]	669	M & F	USA	38–50	Actigraphy	Objectively measured (BMI)	No association
Massachusetts Male Ageing (Yaggi, 2006) [42]	1139	M	USA	40–70	Self-reported	Objectively measured (WC)	No association
Keokuk County Rural Health Cohort (Kohatsu, 2006) [6]	990	M & F	USA	48 ± 13 SD	Self-reported	Objectively measured (BMI)	Negative linear
Study in truck drivers in Sao Paolo, Brazil (Moreno, 2006) [24]	4878	M	Brazil	40 ± 10 SD	Self-reported	Self-reported (BMI)	Negative linear
Hordaland Health Study (Bjorvatn, 2007) [32]	8860	M & F	Norway	40–45	Self-reported	Objectively measured (BMI)	U-shape (nadir 7–8 h)
Health 2000 Health Examination Survey (Fogelholm, 2007) [44]	M: 3377 F: 4264	M & F	Finland	M: 52.3 ± 14.8 SD F: 56.4 ± 17.2 SD	Self-reported	Self-reported (waist and BMI)	M: negative linear F: no association
Better health for better Hong Kong study (Ko, 2007) [45]	M: 2353 F: 2440	M & F	Hong Kong	17–83	Self-reported	Objectively measured (BMI)	M: negative linear F: no association
Quebec Family Study (Chaput, 2007) [15]	740	M & F	Canada	21–64	Self-reported	Objectively measured (BMI)	U-shape (nadir 8 h)
Healthy free-living women in Athens, Greece (Rontoyanni, 2007) [26]	30	F	Greece	30–60	Self-reported	Measured BMI, WHR, and %body fat	Negative linear (body fatness and BMI) No association with WHR
Noninstitutionalized men and women over 60y from the Spanish population (Lopez-Garcia, 2008) [36]	M: 1739 F: 2269	M & F	Spain	M: 71.0 ± 8.0 SD	Self-reported	Measured BMI and WC	All: increased OR for overweight and obesity for both short sleep and long sleep duration. No analyses stratified by sex

Table 1 (continued)

Study population (author, year)	Sample size	Gender	Country	Ages (years)	Measure of sleep duration	Measure of obesity	Relationship between sleep and obesity
				F: 72.1 ± 7.6 SD			
Study of Health in Pomerania (Wolff, 2008) [39]	2383	M & F	Germany	20–79	Self-reported	Objectively measured (BMI)	Slight U-shape (nadir 7 h)
Whitehall II Study (Stranges, 2008) [27]	5021	M & F	England	Approx. 55 ± 5.6 SD	Self-reported	Self-reported (BMI, WC)	Negative linear
Men from Osteoporotic Fractures in Men Study (MrOS) and women from the Study of Osteoporotic Fractures (SOF) (Patel, 2008) [56]	M: 3055 F: 3052	M & F	USA	M: 67–96 F: 70–99	Wrist actigraphy	Measured BMI, WC, and %body fat	M + F: negative linear (BMI, WC) Whole group: U-shape (% body fat)
Rotterdam Study (van den Berg, 2008) [55]	983	M & F	Germany	68.4 ± 6.9 SD Range 57–97	Actigraphy	Objectively measured (BMI)	U-shaped (nadir 7–8 h) NB—no association after adj. for sleep fragmentation
Penn State Cohort (Vgontzas, 2008) [31]	M: 561 F: 739	M & F	USA	M: 50.8 ± 12.6 SD F: 54.9 ± 13.6 SD	Self-reported + 1 night PSG	Objectively measured (BMI)	Reported short sleepers had greater obesity. No assoc. between measured sleep duration and obesity
Korean National Health and Nutrition Examination Survey (KNHANES) (Park, 2009) [25]	8717	M & F	Korea	20–65	Self-reported	Self-reported (waist and BMI)	Negative linear
Population-based study in American women (Anic, 2010) [19]	5549	F	USA	20–75	Self-reported (current + lifetime)	Self-reported (BMI)	Current short sleepers were more likely to be obese regardless of their usual sleep duration earlier in life
Sleep and Health in women (SHE) (Theorell-Haglöw, 2010) [54]	400	F	Sweden	22–72	1 night PSG	Measured (WC and sagittal diameter)	Negative linear. Stronger association in younger
45 and Up Study (Magee, 2010) [48]	45,325	M & F	Australia	55–95	Self-reported	Self-reported (BMI)	U-shape in 55- to 64-year olds No association in those aged 65 years and above
Japanese men who underwent health evaluations (Hsieh, 2011) [23]	8157	M	Japan	Approx. 51.4 ± 9.4 SD	Self-reported	Self-reported (BMI)	Negative linear
Sleep and Health in women (SHE) (Theorell-Haglöw, 2012) [49]	6461	F	Sweden	≥20	Self-reported	Self-reported (WC)	U-shape (nadir 6–<9 h) NB: only in younger after adj.
Korean Genome and Epidemiology Study (Kim, 2013) [30]	838	M & F	Korea	40–69	Self-reported	Measured (VFA)	Negative linear
Tromsø Study (Johnsen, 2013) [34]	6413	M & F	Norway	30–65	Self-reported	Self-reported (BMI, waist)	U-shape (nadir 8–9 h)
The Study of Women's Health Across the Nation (SWAN) Sleep Study (Appelhans, 2013) [50]	310	F	USA	49.7 ± 2.0SD	Actigraphy + sleep diary	Objectively measured (BMI)	Negative linear
Study on global AGEing and adult health (SAGE) (Gildner, 2014) [21]	28,980	M & F	USA	>50	Self-reported	Self-reported (BMI and WC)	Negative linear
Patients in an urban hospital affiliated family medicine center (Logue, 2014) [35]	225	M & F	USA	≥18	Self-reported	Self-reported (BMI)	U-shape
National March Cohort (Westerlund, 2014) [38]	M: 14,407 F: 25,790	M & F	Sweden	≥18	Self-reported	Self-reported (BMI)	Slight U-shape NB: only in highest percentiles of BMI
Population of Chinese adults (Sun, 2015) [47]	2981	M & F	China	15–65	Self-reported	Self-reported (BMI)	M: negative linear F: no association
Adult Danish population (Bonke, 2015) [43]	5022	M & F	Denmark	18–64	Self-reported	Self-reported (BMI)	M: short sleep assoc. with obesity

Table 1 (continued)

Study population (author, year)	Sample size	Gender	Country	Ages (years)	Measure of sleep duration	Measure of obesity	Relationship between sleep and obesity
							F: long sleep assoc. with obesity
The HypnoLaus Study (Haba-Rubio, 2015) [51]	2162	M & F	Switzerland	58.4 ± 11.1 SD	1 night PSG	Objectively measured (BMI)	No association
Community-dwelling older adults (Kim, 2015) [52]	189	M & F	Japan	≥80	Actigraphy	Objectively measured (BMI)	No association

Table 2 Longitudinal studies on sleep duration and measures of obesity in adults

Study population (author, year)	Follow-up duration (years)	Sample size	Gender	Country	Ages (years)	Measure of sleep duration	Measure of obesity	Relationship between sleep and obesity
Zurich Psychiatric Risk Factor Cohort (Hasler, 2004) [5]	13	367	M & F	Switzerland	27–40	Self-reported	Subjectively measured (BMI)	Negative linear
Gothenburg Diabetes Study (Björkelund, 2005) [20]	32	1462	F	Sweden	38–60	Self-reported	Objectively measured (weight gain)	No association
National Health and Nutrition Examination Survey (NHANES-I) (Gangwisch, 2005) [4]	5	3355	M & F	USA	32–49 (at baseline)	Self-reported	Self-reported (BMI)	Weak negative linear
	10	3208	M & F	USA	32–49 (at baseline)	Self-reported	Self-reported (BMI)	Weak negative linear NB—no association in those aged 50–86
Nurses' Health Study (Patel, 2006) [3]	16	68,183	F	USA	39–65	Self-reported	Self-reported (BMI, weight gain)	Weak U-shape (stronger for short sleep; nadir 7 h)
The Quebec Family Study (Chaput, 2008) [33]	6	276	M & F	Canada	21–64	Self-reported	Objectively measured (BMI)	U-shape (nadir 7–8 h)
Whitehall II Study (Stranges, 2008) [27]	6	4378	M & F	England	Approx. 55 ± 5.6 SD	Self-reported	Self-reported (BMI, WC)	No association
Coronary Artery risk Development in Young Adults (CARDIA) (Lauderdale, 2009) [68]	5	669	M & F	USA	45.2 ± 3.6 SD	Actigraphy	Objectively measured (weight)	No association
Working men and women in Japan (Watanabe, 2010) [66]	1	31,206	M	Japan	40.5 ± 9.8 SD	Self-reported	Objectively measured (BMI)	Weak U-shape (stronger in short sleep, nadir 7–8 h)
		3646	F	Japan	37.8 ± 9.3 SD	Self-reported	Objectively measured (BMI)	No association
Male workers in Japan (Nishiura, 2010) [59]	4	2632	M	Japan	40–59	Self-reported	Objectively measured (BMI)	Negative linear
Working men and in Japan (Itani, 2011) [58]	7	21,639	M	Japan	All: >18	Self-reported	Objectively measured (BMI)	Negative linear
		2109	F	Japan		Self-reported	Objectively measured (BMI)	Negative linear
Healthy men and women in Japan (Kobayashi, 2012) [61]	3	21,469	M & F	Japan	>20	Self-reported	Objectively measured (BMI)	Weak U-shape (stronger in short sleep; nadir 7 h)
SUM Mediterranean Cohort (Sayón-Orea, 2013) [65]	6.5	10,532	M & F	Spain	All: 39 ± 12 SD	Self-reported	Self-reported (BMI)	Weak U-shape (stronger in short sleep; nadir 7–8 h)
		4689	M	Spain		Self-reported	Self-reported (BMI)	Negative linear
		5843	F	Spain		Self-reported	Self-reported (BMI)	No association
Japanese adult population (Nagai, 2013) [62]	12	13,629	M & F	Japan	40–79	Self-reported	Self-reported (BMI, weight gain)	No association
NIH-AARP Diet and Health Study (Xiao, 2013) [60]	7.5	35,319	M	USA	All: 51–72	Self-reported	Self-reported (BMI, weight)	Negative linear
	7.5	32,025	F	USA		Self-reported	Self-reported (BMI, weight)	Negative linear
The Study of Women's Health Across the Nation (SWAN) Sleep Study (Appelhans, 2013) [50]	4.6 ± 1.0SD	310	F	USA	49.7 ± 2.0 SD	Actigraphy + sleep diary	Objectively measured (BMI)	No association
Penn State Cohort (Vgontzas, 2014) [69••]	7.5	815	M & F	USA	48.9	1 night PSG	Objectively measured (BMI)	No association
Sleep and Health in women (SHE) (Theorell-Haglöw, 2014) [64]	10	4903	F	Sweden	43.9 ± 15.2 SD (at baseline)	Self-reported	Self-reported	U-shape for habitual sleep duration NB—no association for women ≥40 years
Pizarra Cohort Study (Gutiérrez-Repiso, 2014) [57]	6	968	M & F	Spain		Self-reported		

Table 2 (continued)

Study population (author, year)	Follow-up duration (years)	Sample size	Gender	Country	Ages (years)	Measure of sleep duration	Measure of obesity	Relationship between sleep and obesity
					18–65 (at baseline)		Objectively measured (BMI)	Habitual short sleepers had the highest incidence of obesity
	11	673	M & F	Spain	18–65 (at baseline)	Self-reported	Objectively measured (BMI)	Habitual short sleepers had the highest incidence of obesity
Male workers in Japan (Nishiura, 2014) [63]	3	1687	M	Japan	19–39	Self-reported	Objectively measured (BMI)	No association
Nurses' Health Study (Cespedes, 2016) [67]	14	59,031	F	USA	55–83	Self-reported	Self-reported (weight gain)	Increase in sleep duration associated with weight gain (moderate strength)

One possible explanation for the inconsistent pattern seen in observational studies is that sleep duration could have an age-dependent effect on obesity. Studies in children or adolescents show clearer negative linear associations [73, 74] whereas studies with mostly middle-aged participants such as the Wisconsin Sleep Cohort [17], the Nurses' Health Study [3], the Hordaland Study [32], and the SHE Study [49] tend to find U-shape associations (Tables 1 and 2). It has been speculated in a previous review by Marshall and co-workers that the association could be U-shaped also in children but that this is hampered by the fact that children cannot be exposed to long sleep duration due to a combination of the already greater sleep needs in this group compared with adults, and by a ceiling effect [73]. Hypothetically, long sleep duration effect might emerge at, for instance, 18 h of sleep per night, which very few children show. Marshall et al. therefore suggested that theoretically, the U-shape pattern emerges in adults as the need for sleep decreases; short sleep continues to cause obesity but in addition, long sleep is now also a possible risk factor for obesity/weight gain [73]. In contrast, a meta-analysis by Wu et al. did not show a relationship between long sleep duration and future obesity [75].

Studies with older participants, such as the JACC Study [41], Massachusetts Male Ageing Study [42], older members of the NHANES 1 [4], the Sleep Heart Health Study [40], and the 45 and up study [48], find no significant association between sleep length and obesity. In the SHE study, a population-based longitudinal study comprising 6461 women at baseline and 4903 at follow-up, we have shown that the relationship between reported sleep duration and measures of obesity was strongest in younger women [49, 64]. Also in a subsample of 400 women from the SHE study, where sleep was objectively measured using PSG, the relationship between sleep and central obesity was strongest in younger women [54] (Tables 1 and 2). Together, these findings indicate that the mechanisms underlying the development of obesity may start early in life. In addition, Magee et al. have suggested that one possible explanation for this is that physiological and behavioral mechanisms underlying this relationship are strongest at younger ages [76]. Another possible explanation for the lack of significant associations between short sleep and weight gain in studies in older people is that individuals who are short sleepers do not continue to gain weight linearly over time of their short sleeping. In other words, if a pattern of short sleep duration has started some years before the start of a study and this change in sleep duration results in a certain net increase in calories per day, by the time the study begins, the subject will have reached a weight threshold and is not likely to gain any more weight during the study [76].

Some longitudinal studies have shown short sleep duration as a risk factor for obesity and weight gain [4, 5, 59], and one study in older women has shown increased sleep duration to be associated with increased change in weight [67]. However, in general, the weight gain shown is small or modest and the clinical value of this is therefore questionable. In addition, none of the three longitudinal studies [50, 68, 69••] using objective measures to assess sleep duration, published to date, has shown a relation between sleep and obesity or weight gain. In addition, a systematic review by Patel et al. states that although short sleep duration appears to be independently associated with weight gain, major study design limitations preclude definitive conclusions [77].

In answering the question whether (short) sleep duration causes obesity and weight gain, intervention studies testing the relationship between sleep duration and obesity are helpful. Although several in-lab RCTs have studied this [70], only one has had weight change as a primary outcome [71]. In addition, to date, there is only one study proposing to test the hypothesis that increased sleep duration would reduce obesity in real-life short sleepers [72•]. However, there have been reports from this study that substantial improvements in sleep and also biochemical parameters were seen between screening and randomization (i.e., prior to any intervention), and these improvements have potentially changed the true study baseline which could have affect the outcome [72•]. Nonetheless, results from this study are of great interest to the sleep field and the question of whether short sleep duration causes obesity.

Some previous reviews have discussed the importance of getting reliable measures of both sleep duration and obesity in order to truly assess the relationship between the two [74, 78]. Polysomnography is considered a gold standard for assessing sleep duration. However, it is a costly and time-consuming method and may also be subject to first-night effect although this effect seems to be less of an issue for home-based PSG [79]. As an option, actigraphy may be used for examining sleep in large populations and is a relatively inexpensive method that can monitor sleeping patterns over long periods of time. Actigraphy has also been shown to have good agreement with PSG [80]. Self-report measures are easy to use also in large studies and may also assess subjective sleep quality, but they vary in accuracy and may be subject to age and gender biases [74, 78]. In addition, reliable measures of obesity are of great importance if the relationship between sleep and obesity is to be fully understood. Laboratory methods, such as for instance dual energy X-ray absorptiometry (DEXA) and imaging techniques (CT and MRI), provide good estimates of body composition. However, they are not feasible for use in large-scale field studies. The easily measured BMI and waist circumference (WC) are convenient for use in the field, but they do not provide exact estimates of percentage body fat and give limited information on body composition [74, 78]. A possible option that has been suggested also as a compliment to BMI and WC measures in large-scale studies is bioelectrical impedance devices which are relatively inexpensive and correspond well with laboratory measures [74, 78]. As a result, the choice of method for assessing sleep duration as well as obesity will likely impact the outcome of a particular study.

There has also been an interest in other explanations for the association between sleep duration and obesity such as sleep fragmentation and measures of sleep quality that have been studied or included as mediation factors. In the Rotterdam Study, the association between sleep duration and obesity became non-significant after adjustment for sleep fragmentation [55]. Other studies have suggested sleep variability rather than sleep duration as the factor linking sleep and obesity [61]. In addition, although most studies have been designed to test the hypothesis that short sleep duration is a risk factor for incident obesity, the reverse causation cannot be ruled out. If obesity per se causes short sleep duration that could explain the association seen in many cross-sectional studies, longitudinal studies have failed to identify such a relationship. Furthermore, a recent review [81•] on secular trends on sleep duration shows that, although we have a general belief in today's society that we are sleep deprived, measurements on sleep duration in different countries of the world do not unanimously show decreased sleep duration with some countries having increased, others having decreased, and yet others having had a stable sleep duration over time.

A bi-directional relationship between sleep duration and obesity has also been suggested [82] where sleep problems may be due to obesity and, in turn, influence sleep duration [82]. However, in most of the previous studies on sleep duration, sleep problems such as sleep apnea or frequent awakenings have not been included or analyzed. In addition, a previous review [76] on longitudinal studies assessing the association of sleep duration and subsequent weight gain or incident obesity in adults suggests that the inconsistent results of these studies may in part be due to lack of appropriate inclusion of confounders or lack of examination of mediating or moderating effects, such as sleep problems. In the five studies that at the time of the review had included sleep problems as a confounder, two [68, 83] found no association between sleep duration and weight gain, one [36] found a U-shaped curve but only in women, and two found short sleep to be associated with only a modest weight gain [3, 4]. Magee and Hale argue that there may also be time-varying covariates that are important for finding changes in weight over time [76]. Factors such as smoking status, alcohol intake, diet and energy intake, medications, and psychological problems may all be related to weight gain or loss, and therefore, changes over time in these variables may affect the possible relation between sleep length and obesity.

The most recent longitudinal study on sleep duration and obesity, using an objective measure of sleep duration [69••], has addressed some of the issues raised in the review by

Magee and Hale [76], such as including information on sleep problems. Apart from obesity (BMI) and sleep duration (both self-reported and objectively measured), the study assessed sleep difficulty (normal sleep, poor sleep, insomnia) and emotional stress and also controlled for confounding factors and examined mediating and moderating effects [69••]. In this study, Vgontzas et al. showed that subjective short sleep duration was associated with incident obesity; however, after controlling both for complaints of poor sleep and for level of emotional stress, this association became non-significant. In addition, consistent with previous studies on objective sleep [50, 68], there was no association between objective short sleep duration and incident obesity. Poor sleep and emotional stress were, however, strong predictors of incident obesity and there was an additive role between the two. Those with poor sleep and incident obesity had the greatest emotional stress and the shortest subjective sleep duration. The study also showed that emotional stress was stronger in the young and the middle-aged whereas complaints of poor sleep predicted incident obesity in all age groups [69••].

In the light of the results from Vgontzas et al. [69••], it is interesting to find that Chaput et al. have shown the association between short sleep duration and weight gain in adults to be dependent on the so-called disinhibited eating behavior (i.e., a tendency to overeat and to eat opportunistically) [84]. They found that in short sleepers with high disinhibition eating behavior, the incidence of obesity over 6 years was 2.5 times higher than in short sleep sleepers with a low disinhibition eating behavior. In addition, two recent studies have also found emotional eating behavior, a marker of distress, to influence the relationship between short sleep duration and weight gain [85] and also food consumption [86•] in women. Together with the results on emotional stress [69••], this points in the direction of something else than sleep duration per se being the mediator of the relationship between sleep and obesity.

Conclusion

Sleep duration and obesity are no doubtable strongly related, although the direction and also the nature of this association are still not fully elucidated. Therefore, it is still too early and a too easy solution to suggest that changing the sleep duration will cure the obesity epidemic. Also, based on the fact that we as a society probably are not as sleep deprived as previously believed, that the weight gain associated with short sleep is modest, and that factors such as emotional stress and eating behavior are likely mediators of the relationship between sleep duration and obesity, it is unlikely that it is as simple as sleeping longer to reduce weight. It might be that we should instead aim at reducing emotional stress or identifying "bad" eating behavior in order to reduce obesity in short (and long?) sleepers. Recommendations should still be to get sufficient sleep due to the negative effects of short sleep duration on neurobehavioral function and the associated increased risk of motor vehicle accidents. However, as the prevalence of poor sleep in the general population is high and given the novel results on emotional stress and poor sleep as mediating factors in the relationship between sleep and obesity, detection and management of these should become the target of future clinical efforts as well as future research. Future studies ought to use reliable measures not only on sleep duration (objective and subjective) and obesity (possibly combining measures of BMI and WC with other body composition measures) but also on stress, and mental and physical health.

Compliance with Ethical Standards

Conflict of Interest Jenny Theorell-Haglöw and Eva Lindberg declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent References [46, 54, 64] were performed by the authors and human subjects were involved. All participants gave informed consent. The authors have not performed any animal studies.

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