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Report

Social Jetlag and Obesity

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Summary

Obesity has reached crisis proportions in industrialized societies [1]. Many factors converge to yield increased body mass index (BMI). Among these is sleep duration [2–10]. The circadian clock controls sleep timing through the process of entrainment. Chronotype describes individual differences in sleep timing, and it is determined by genetic background, age, sex, and environment (e.g., light exposure) [11–14]. Social jetlag quantifies the discrepancy that often arises between circadian and social clocks, which results in chronic sleep loss [11, 15]. The circadian clock also regulates energy homeostasis [16], and its disruption—as with social jetlag—may contribute to weight-related pathologies [17–19]. Here, we report the results from a large-scale epidemiological study, showing that, beyond sleep duration, social jetlag is associated with increased BMI. Our results demonstrate that living "against the clock" may be a factor contributing to the epidemic of obesity. This is of key importance in pending discussions on the implementation of Daylight Saving Time and on work or school times, which all contribute to the amount of social jetlag accrued by an individual. Our data suggest that improving the correspondence between biological and social clocks will contribute to the management of obesity.

Results and Discussion

The circadian clock controls processes—from gene expression to sleep—to occur at distinct times over the course of a 24 hr day. Despite this circadian control, humans often use alarm clocks and/or medication to align their sleep and wake times with social obligations (e.g., work and school schedules or other social events [15]). To quantitatively characterize these individual differences in daily timing on a population level, we use a simple, internet-based questionnaire (the Munich ChronoType Questionnaire, MCTQ; [20]) that assesses sleep and wake behavior on work and free days. After applying quality control criteria (see Experimental Procedures), the database contained more than 65,000 complete entries of primarily central European participants. The MCTQ parameters relevant for this study are sleep duration, sleep timing (chronotype), and the discrepancy between biological and social timing (social jetlag). The quantification of chronotype is based on the midpoint of sleep on free days (MSF), corrected for "oversleep" on free days (MSF_{sc}), and social jetlag is the difference between midsleep times on free days and on workdays (MSF-MSW; see [15], Figure S1A available online, and Supplemental Experimental Procedures).

The weekly average of sleep duration drastically shortens during puberty and adolescence [Figure 1A; age effect, $F_{(1,\ 64,107)} = 2{,}392$, $p < 0.0001$]. Our results show that sleep is profoundly influenced by social time, predominantly by work schedules. The strong social influence on sleep becomes apparent when sleep duration is analyzed separately for work and free days (Figure 1B) with significantly longer sleep on free days than during the workweek [$F_{(1,64,106)} = 960.63$, $p < 0.0001$]. This discrepancy is most pronounced in young adults and thereafter decreases steadily with age [interaction between age and type of day, $F_{(1,64,108)} = 2{,}625.38$, $p < 0.0001$].

The differences in sleep between work and free days not only pertain to sleep duration but also to sleep timing (i.e., at what local time people sleep; Figure S1A). Although one can sleep outside the temporal window provided by the circadian clock (e.g., naps), sleep is more efficient when coinciding with this window [21]. Around 80% of the regularly working individuals represented in our database use an alarm clock on workdays. This premature interruption results in sleep loss (especially in the later chronotypes), because the circadian clock strongly influences when one can fall asleep. To compensate for this sleep debt accumulated over the workweek, people commonly oversleep on free days (Figure S1A). Because the difference in sleep timing between work and free days resembles the situation of traveling across several time zones to the West on Friday evenings and "flying" back on Monday mornings, the phenomenon of regular, weekly changes in sleep timing was coined social jetlag [15]. The symptoms of jetlag (e.g., problems in sleep, digestion, and performance) are manifestations of a misaligned circadian system. In travel-induced jetlag, they are transient until the clock re-entrains. In contrast, social jetlag is chronic throughout a working career. One-third of the population represented in our database suffers from 2 hr or more of social jetlag, and 69% report at least 1 hr of social jetlag (Figure S1B).

Chronotype (assessed by sleep times) becomes progressively later throughout puberty and adolescence [13]. Thereafter, chronotype progressively advances until the elderly become as early as children [13]. These developmental changes in circadian timing, in combination with the fact that school start times are not tuned to the generally late chronotype of teenagers [22], lead to a peak of social jetlag at around the end of adolescence (Figure S1C). This is why teenagers show the largest discrepancy in sleep duration between free days and workdays compared to all other ages (Figure 1B). Although social jetlag is most acute during adolescence, it typically continues throughout active work life until retirement. Insufficient sleep on workdays is more common among later chronotypes [correlation between chronotype and sleep duration on workdays, SD_w: $r_{(64110)} = -0.12$; $p < 0.0001$], as is (chronic) sleep loss over the workweek [$r_{(64110)} = 0.14$; $p < 0.0001$].

Using our large epidemiological database, we revisited the reported correlation between body mass index (BMI) and

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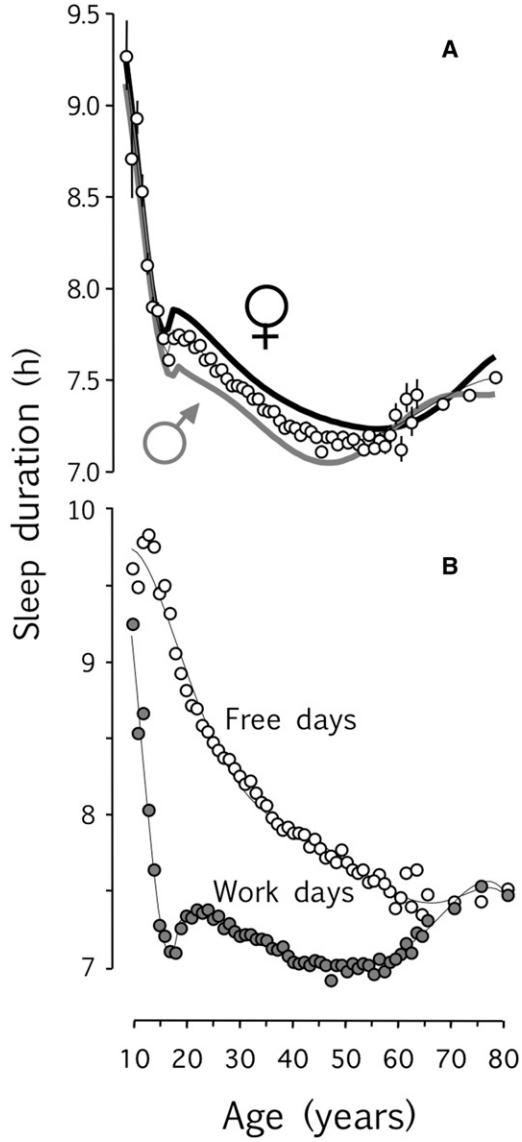

Figure 1. Average Sleep Duration as a Function of Age

One year bins were used for age groups 10–65, >65; the bins are made in 5 year steps. For details of computing sleep timing and duration, see "Evaluation of the MCTQ" in Supplemental Information; for parameters of the respective fits, see Table S4. Vertical lines represent SEM (±SEM; in most cases they are smaller than the respective symbols).

(A) Age and sex dependencies of sleep duration. A univariate ANOVA—with age and sex as covariates—shows that these two variables influence average sleep duration: age, $F_{(1,\ 64,107)}$ = 2,392.7, $p < 0.0001$, partial $\eta 2$ = 0.04, and sex, $F_{(1,\ 64,107)}$ = 1,028.3, $p < 0.0001$; partial $\eta 2$ = 0.02. Curves are polynomial fits (black, both sexes; red, females; blue, males).

(B) Average values for sleep duration separately for free days (open circles) and for workdays (gray dots). A repeated-measures ANOVA with the covariates age, chronotype, and the between-subject factors type of day and sex shows a significant difference between sleep duration on work and free days, $F_{(1,64.108)}$ = 960.6, $p < 0.0001$, partial $\eta 2 = 0.015$. Sex, age, and chronotype have an impact on sleep duration on work and free days (p values < 0.0001, with age having the strongest impact; effect size partial $\eta 2 = 0.08$).

Table 1. Predicting Group Assignment to the Higher BMI Group

	Predictor variables	B	SE	OR	95% CI
Step 0	Constant	−0.737	0.008	0.479	
Step 1	Sex	0.641	0.018	1.899	1.833 to 1.967
	Age	4.247	0.081	69.904	59.621 to 81.962
	Social jetlag	1.194	0.139	3.300	2.512 to 4.334
	Chronotype (MSFsc)	−1.316	0.116	0.268	0.194 to 0.371
	Average sleep duration	−4.881	0.259	0.008	0.005 to 0.013
	Constant	−3.399	0.352	0.033	

We used a logistic regression model approach (enter method) with a sample size of 20.735 for BMI $\geq$ 25 and 43.308 for the lower BMI group. B, unstandardized regression coefficients; SE, standard error; OR, odds ratio; CI, confidence intervals. R^2 = 0.093 (Cox & Snell) and = 0.13 (Nagelkerke).

average sleep duration [2–9]. The BMI distribution of the database population concurred with previous reports (inset in Figure S2 and [23, 24]); it increases with age [univariate ANOVA, $F_{(1,\ 64,107)}$ = 5,965.85, $p < 0.0001$] until about the age of 52 (coinciding with the average age of menopause in women; Figure S2). As was shown previously [6, 25], short sleep is associated with higher BMI (partial correlation between average sleep duration and BMI, controlling for age and sex, $r = -0.082$, $p < 0.0001$), although we (as others [4]) failed to see a significant increase with long sleep (Figures S3A and S3B). The correlation between BMI and sleep duration in our data set is highly significant for both work and free days, but the correlation coefficient on free days is double that on workdays (see legend to Figures S3A and S3B); this is a first indication that social jetlag (i.e., sleeping outside the circadian sleep window) may be a factor contributing to developing obesity.

Based on these initial observations, we examined the potential role of social jetlag in predicting whether or not a person was overweight. To that end, we divided the database population (inset in Figure S2) into two groups, normal (BMI < 25) and overweight/obese (BMI $\geq$ 25). We then analyzed whether a participant belonged to the overweight group by using age, sex, and average sleep duration, as well as chronotype and social jetlag, as explanatory variables with a logistic regression approach (see Experimental Procedures for details of data preprocessing). The variables in the model accounted for 13% of the variance in the data set (model summary: $\chi^2_{(5)}$ = 6,248.93, $p < 0.000$). As expected, the probability to be in the overweight group increased significantly with age and was modulated by sex (for odds ratios and confidence intervals, see Table 1). In agreement with previous reports, longer sleep decreases the probability of being overweight (see also Figures S3A and S3B). Over and above the impact of sleep duration, social jetlag significantly increased the probability of belonging to the group of overweight participants (for odds ratios and confidence intervals, see Table 1).

Based on these results, we used a hierarchical, multiple regression approach (enter method, replicated with stepwise entry) to investigate the relationship between BMI and social jetlag within the normal and overweight individuals. Although social jetlag does not explain the variance in weight in the normal BMI group [Table S1A, model summary: $F_{(5,43302)}$ = 1267,68, $p < 0.000$], it is positively associated with weight increase in the overweight group [Figure 2 and Table S1B, model summary: $F_{(5,20729)}$ = 72,38, $p < 0.000$]. In the normal BMI group, both chronotype and sleep duration predict BMI equally well (equivalent standardized regression coefficients in the model), whereas chronotype adds no additional explanatory power to the regression model in the overweight group (Table S1B). Overall, our results indicate that sleep timing is an equally important predictor for BMI as is sleep duration.

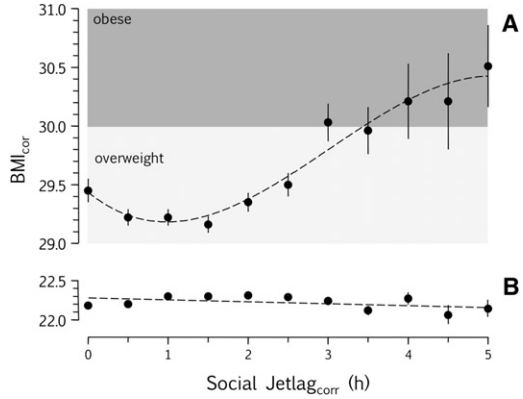

Figure 2. To Further Investigate the Association between BMI and Social Jetlag, We Used a Hierarchical Multiple Regression Analysis

Variables were entered in a four step model, as summarized in Tables S1A and S1B, separately for overweight (BMI $\geq$ 25; n = 20,731; A) and normal (BMI < 25; n = 43,308; B) participants. The models included age, sex, and average sleep duration as variables along with social jetlag and chronotype. Analyses confirmed previous findings, showing that age, sex, and sleep duration are important factors in predicting BMI in both weight groups. For individuals in the normal group, social jetlag is not a predictor for BMI (B), whereas chronotype shows a negative relationship with BMI (Table S1A, step 4) with an effect size comparable to that of sleep duration (cf. β-coefficients). In contrast, living against the clock increases BMI in the overweight group, as exemplified by the positive association between BMI and social jetlag (Table S1B; step 3 of the regression model). In this case, the influence of social jetlag is more than half of the impact of sleep duration, whereas chronotype has no additional explanatory power. For illustration purposes, social jetlag and BMI were corrected for age and sex (SJLcorr; BMIcorr) according to the fits shown in Figures S1 and S2 (see also Table S4 for correction parameters). Vertical lines represent SEM of the 15 min binned averages.

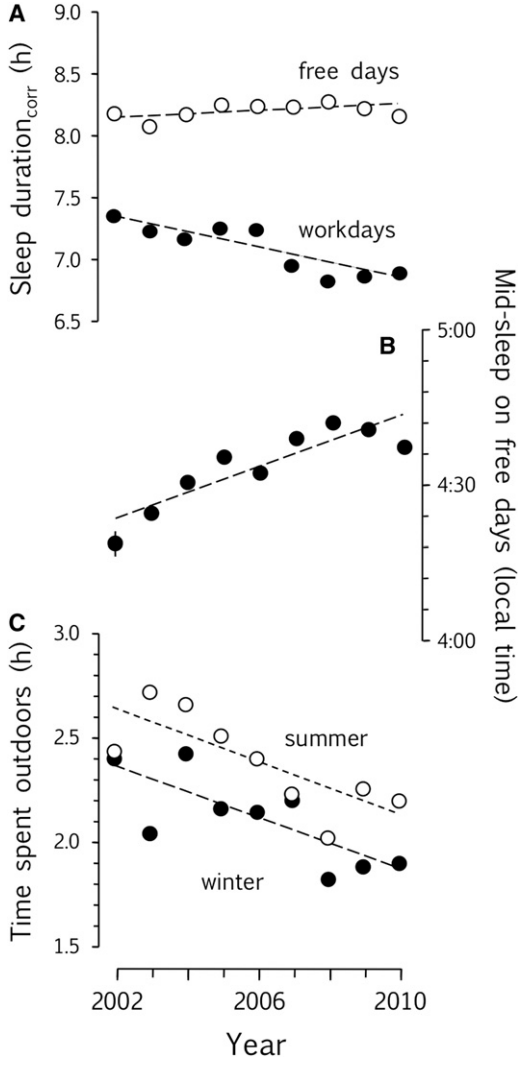

Figure 3. Sleep Timing, Sleep Duration, and Light Exposure over the Past 8 Years

Data was collected from 2002 to 2010, but to avoid imbalance in the annual groups, year 2002—though shown in the figures—was excluded from the analyses because the sample size was comparably low with <1,000 cases, i.e., $\approx$1% of the total population (see also Table S3). Binned data is shown $\pm$SEM (in most cases they are smaller than the respective symbols). Analyses were conducted with univariate ANOVAs including age and sex as covariates (or the nonparametric correspondent, Kruskal-Wallis Test, for light exposure). Effect sizes are evaluated by partial correlations (or Spearman's ρ).

(A) Sleep duration changed significantly over the years [workdays: filled symbols, $F_{(7,63,427)} = 275$, $p < 0.001$; free days: open symbols, $F_{(7,63,427)} = 31.7$, $p < 0.001$], but especially on work days ($r = -0.14$, $p < 0.001$ versus free days: $r = 0.042$, $p < 0.001$, n = 63,433).

(B) Average chronotype changed from 2003 to 2010, $F_{(7,\ 63,427)} = 51.9$, $p < 0.001$. Although small, this annual progression in "lateness" is significant ($r = 0.07$, $p < 0.001$, n = 63,334).

(C) Average weekly light exposure ("time spent outdoors without a roof above the head") during winter [October–March, filled symbols; chi-square$_{(7)}$ = 525.35, $p < 0.001$; Spearman's $\rho = -0.15$, $p < 0.001$, n = 17,764] and summer [April–September, open symbols; chi-square$_{(7)}$ = 1,355.73, $p < 0.001$; Spearman's $\rho = -0.18$, $p < 0.001$, n = 37,710].

The observed differences between the relationships of social jetlag and BMI in normal and overweight individuals suggest different underlying mechanisms that need to be further investigated.

Epidemiological reports on population-wide sleep duration over the past decades are mixed [26–32]. The MCTQ database has continuously collected entries since 2002 (see Table S2), allowing the systematic investigation of trends in chronotype, social timing, and sleep behavior. Our results support the finding that sleep duration has decreased over the past decades but also show that this is only due to a shortening of workday sleep (Figure 3A). What could be causing this trend? According to the social jetlag hypothesis (Figure S1), a differential shortening of sleep on work days would be predicted if chronotype (phase of entrainment) was progressively delayed over the years with similar work onset times (correlation of yearly bins in sleep-end: $r = -0.21$; $p = 0.56$). Average chronotype is indeed delayed over the past decade in the population represented by our database (Figure 3B). Circadian theory includes various explanations for how entrained phase is influenced, whereby zeitgeber strength is of primary importance. Thus, the progressive delay could be explained by decreasing zeitgeber strength [12, 13]. In industrial areas, people generally experience a weaker zeitgeber because they are exposed to less light during the day (working indoors) and more light pollution during the night. Whereas indoor intensities rarely exceed 400 lux, light intensities from the open sky can range between 10,000 and >100,000 lux, depending on time of day and cloud cover. Under weak zeitgebers, the phase of entrainment becomes later for most chronotypes [33]. Our data show that people indeed spent less time outdoors over the course of our data collection

[both in winter and in summer; $\chi 2_{(7)} > 530$, $p < 0.0001$; Figure 3C].

Previous studies have concluded that short sleep duration is a risk factor for obesity and metabolic disease [34–40]. We suggest here that sleep timing is a vital factor for understanding the relationship between sleep and metabolic pathogenesis. Associations between sleep, circadian timing, and metabolic pathologies have long been a concern for shift workers, those members of society who suffer from the most extreme form of social jetlag. The situation, where people have to be active and try to sleep outside their circadian window, has been simulated in carefully controlled laboratory studies called forced desynchrony. These simulations result in an imbalanced glucose metabolism that normally is associated with metabolic syndrome or type II diabetes [19]. Social jetlag is a small but chronic version of shift work or forced desynchrony that is broadly experienced throughout the population. Presumably due to weak zeitgebers, chronotype has become so late in industrialized countries that sleep timing has become incompatible with traditional work times. A majority of the population is active on workdays before the end of the circadian sleep window is reached and generally fails to fall asleep before this window opens. Chronic sleep loss is a consequence of this situation. It is as though the majority of the population is working the early shift. Here, we identify this discrepancy between biological and social timing as one of many factors contributing to the epidemic of overweight and obesity.

Experimental Procedures

The MCTQ [20] (www.thewep.org) is an online survey on the temporal aspects of human behavior. The online version of the MCTQ has received ethical approval by the Ethics Committee of the Ludwig Maximilian University, Munich, Germany, and the data collection as well as anonymization procedures are in line with the principles of the Helsinki Declaration for Ethical Principles for Medical Research Involving Human Subjects (last updated Seoul, 2008). Assessment of chronotype is based on the midpoint of sleep on free days (MSF), which is then corrected for "oversleep" on free days (MSF_{sc}; see [15] and Supplemental Experimental Procedures). The discrepancy between biological and social timing, social jetlag, was computed by subtracting the MSW (midpoint of sleep on work days) from MSF. BMI was determined by using the standard formula: weight/height^2, kg/m^2.

The data set was cleaned for unrealistic (extreme) values in sleep times or biometric information, for those who indicated not to work regular weekly schedules (i.e., all or no free days), and for those who use alarm clocks on free days (for cleaning protocols, see Supplemental Information, specifically Table S2).

BMI scores, MSF_{sc}, social jetlag, and sleep offset are not normally distributed (by visual inspection; the extremely large sample size makes a reliable quantitative approach to normality unreliable [41]). For parametric statistics continuous variables were $\log_{10}$ transformed.

The results of the all regression analyses were reproduced with different methods (enter, stepwise, backward); the indices of multicollinearity—that may bias the models' validity, because we have reported correlations between the predictor variables—were within the accepted range (tolerance < 1 and variance inflation factor, VIF, range: 1.03 to 1.6).

Supplemental Information

Supplemental Information includes three figures, four tables, and Supplemental Experimental Procedures and can be found with this article online at doi:10.1016/j.cub.2012.03.038.

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