



Chronobiological aspects of sleep restriction modulate subsequent spontaneous physical activity

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ABSTRACT

Purpose: First evidence suggests that chronobiological aspects of sleep restriction affect metabolic conditions. Our aim was to investigate whether spontaneous free-living physical activity likewise is affected by chronobiological timing of short sleep.

Methods: In an experimental randomized, balanced cross-over design, eleven healthy, normal-weight (BMI: $23.9 \pm 0.4 \text{ kg/m}^2$) men were evaluated. Physical activity was assessed by tri-axial wrist actigraphy after (i) four-hour sleep during the first night-half of the night ('late night sleep loss'), (ii) four-hour sleep during the second night-half ('early night sleep loss'), and (iii) eight-hour regular sleep ('regular sleep'), from 7:00 to 24:00 (17 h). Feelings of tiredness and activity were measured by semi-quantitative questionnaires.

Results: Physical activity differed between sleep conditions ($P < 0.05$) with the lowest physical activity after 'late night sleep loss'. Accordingly, less time was spent in high-intensity physical activity after 'late night sleep loss' as compared to the 'early night sleep loss' and 'regular sleep' conditions (both $P < 0.05$). Perceived feelings of tiredness were higher after both short sleep conditions as compared to 'regular sleep' (both $P < 0.05$).

Conclusions: Sleep restriction during the second half of the night elicits stronger effects on spontaneous physical activity than sleep restriction during the first half of the night despite identical sleep duration, but the impact of longer period awake needs to be evaluated in further research. In sum, these data indicate that not only short sleep *per se* but also chronobiological aspects modulate physical activity pattern.

1. Introduction

In our modern societies with a westernized lifestyle including a 24/7 pattern of social functioning, short sleep duration and disrupted sleep (i.e., a disturbed sleep pattern with altered sleep stages organization, decreased sleep efficiency, and increased wake time during the night) are common behaviours. Consequences of this shortened and disturbed sleep pattern are focused by epidemiological as well as experimental studies (for review see [1]). Epidemiological data indicate that impaired quantity and quality of sleep are associated with increased body mass index (BMI), obesity and adverse metabolic conditions such as

type 2 diabetes [2–5]. Obesity is a chronic disease caused by a positive energy balance that is driven by increased energy intake and vice versa lower physical activity [6]. With regard to energy intake, experimental studies provide evidence that sleep curtailment led to elevated feelings of hunger and appetite [7,8] as well as increased caloric intake [9,10].

Moreover, physical inactivity is also a common behaviour [11] that might contribute to a positive energy balance and finally contribute to obesity. However, data on the relationship between sleep duration, scattered sleep pattern and physical activity are inconsistent. In a Finnish cohort, the most prevalent profile in men was sleep of normal range (i.e. 7–9 h) and moderate to high leisure time physical activity. In

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women, the most prevalent cluster was 'physically active and good sleeper' (i.e. 8–9 h). Overall, sufficient sleep was associated with higher leisure time physical activity, while poorer sleep was linked to physical inactivity [12]. Two further epidemiological studies reported on an association between either short sleep duration [13] or long sleep duration [14] and reduced physical activity. In children aged 9–11 years from 12 countries, Chaput and coworkers [15] found that short sleep duration and poor sleep efficiency were associated with higher levels of moderate to vigorous physical activity. However, this at a first glance unexpected results are in line with other studies. These data suggest that more active children show less total sleep time than less active children [15]. Experimental studies have also drawn a mixed picture on the link between sleep restriction and physical activity. While we have previously reported [16] that physical activity was lower after 4 h of sleep compared to 8 h regular bedtime, other studies found no effects of acute sleep restriction on physical activity and energy expenditure [9,17] or even (partly) increased physical activity after sleep restriction [10]. It is likely that different experimental protocols and different techniques for measurement of physical activity and energy expenditure, e.g. by accelerometry [10,16], 24 h heart rate monitoring and pedometry [9], or doubly labeled water [17] contribute to these heterogeneous data.

There is evidence that chronobiological aspects of sleep restriction affect metabolic conditions [16,18]. Thus, one may speculate that spontaneous free-living physical activity likewise is affected by the timing of sleep restriction. In order to investigate such chronobiological aspects of the timing of sleep restriction on free-living physical activity, we studied two conditions of acute short sleep, i.e. restricted to 4 h in the first half of the night, and 4 h in the second half of the night, both compared to 8 h sleeping as control condition. We hypothesized that short sleep decreases free-living physical activity with greater reduction of physical activity after short sleep during the first half of the night.

2. Material and methods

2.1. Subjects

The study was carried out in 16 healthy young men. Due to technical problems of physical activity measurements ($n = 4$) and general study drop out ($n = 1$), data of 11 men aged 19 to 31 years (mean \pm SEM: 24.3 ± 0.9 years) with a BMI between 21.7 and 25.7 kg/m^2 ($23.9 \pm 0.4 \text{ kg/m}^2$) were included. Exclusion criteria were chronic or acute illness, current medication of any kind, smoking, elevated alcohol ($>50 \text{ g}$ per day) and caffeine ($>300 \text{ mg}$ per day) consumption, use of prescription drugs, diabetes in first-degree relatives, shift work, travel across time zones during the past four weeks, and short habitual sleep duration ($<6 \text{ h}$ per day). Furthermore, abnormal findings on physical examination or routine laboratory testing (complete blood counts, comprehensive metabolic panel, thyroid function, lipid composition) were also exclusion criteria. Physical activity, sleep behavior, nutrition and subjective feelings during the day before experimental visits were assessed by a structured interview on each in-lab visit to ensure participants adherence to the above listed criteria. The study protocol was approved by the local ethics committee of the University of Luebeck according to the Declaration of Helsinki on research involving humans, and all participants gave written informed consent.

2.2. Study design

All subjects were tested in a randomized counter-balanced cross-over design on three conditions spaced at least four weeks apart: (i) 1 night with 4 h sleep during the first half of the night ('late night sleep loss'; bedtime: 22:30–03:00), (ii) 1 night with 4 h sleep during the second half of the night ('early night sleep loss'; bedtime: 02:15–06:45), and (iii) 1 control night with regular 8 h sleep ('regular sleep'; bedtime:

22:15–06:45). This study was part of a larger study investigating the effects of sleep restriction on glucose homeostasis [19].

On each experimental night, subjects arrived at the research unit at 19:15 and had a standardized light dinner (380 kcal) at 20:15. Participants went to bed at 22:15 in the 'regular sleep' and at 22:30 in the 'late night sleep loss' condition, respectively, while they remained awake in a sitting position until 02:15 in the 'early night sleep loss' condition. During sleep restriction conditions, participants were allowed to read and to watch non-arousing movies while being monitored constantly by the experimenters. Light intensity in the laboratory was 300 lx. Subjective feelings of tiredness and activity were assessed hourly by a semi-quantitative questionnaire anchored from 0 (none) to 9 (severe) between wake-up time and the end of the laboratory experiment (11:30) when subjects left the research unit [20].

Physical activity was assessed by triaxial wrist accelerometry (Acti-Watch; Cambridge Neurotechnology; Cambridge; UK) on the subjects' non-dominant hand during each experimental day in the laboratory (from 07:00 to 11:30) and the consecutive day under free-living conditions (from 11:30 to 24:00), in total 17 h. Physical activity was measured as activity counts (AC) and the sampling interval was 1 min with a subsequent averaging of AC to 5-min intervals.

2.3. Statistical analyses

Separate analyses of physical activity were done for the (i) 17h-wake time period (07:00–24:00) and the following averaged subsumed intervals: (ii) 'lab time' (07:00–11:30) vs. 'free-living' conditions (11:30 to 24:00), (iii) 6 h-time intervals during free-living conditions, i.e. 'afternoon' (12:00–18:00) vs. 'evening' (18:00–24:00). Total AC comprises the sum of activity registered on the respective day or time interval. Intensity of physical activity was calculated and intra-individually graded into low (AC below 33rd percentile), moderate (AC within 33rd and 66th percentile), and high (AC above 66th percentile) intensities with reference to the recording under the 'regular sleep' condition.

All values are expressed as means \pm SEM. Analyses were performed with SPSS 22.0 for Mac (SPSS Inc, Chicago, IL). Figures were prepared by using GraphPad Prism 7 for Mac (San Diego, CA). Analyses of physical activity were based on analysis of variance (ANOVA) for repeated measurements, including the factors 'condition' (for 'late night sleep loss' vs. 'early night sleep loss' vs. 'regular sleep') and 'time' for different time intervals (e.g. 'lab time' vs. 'free-living'; 'afternoon' vs. 'evening'; 6 h time intervals); and 'activity level' (for 'low', 'moderate', and 'high' intensity physical activity). Helmert contrast tests were used to explore first- vs. second-level differences (e.g., 'regular sleep' vs. 'short sleep' and 'early wake up' vs. 'late wake up', respectively). Pairwise comparisons of single time-points were performed by paired Student's *t*-test. A *P* value < 0.05 was considered significant.

3. Results

3.1. Total physical activity

Cumulative physical activity during the study period was significantly different between conditions ($P < 0.05$ for ANOVA main effect 'condition'; Fig. 1) with lower physical activity after 'late night sleep loss' than 'early night sleep loss' and 'regular sleep' ($P < 0.05$ for Helmert contrast test). However, physical activity was not influenced by short sleep *per se* ($P < 0.05$ for Helmert contrast test). Subsequently carried out pairwise comparisons revealed that physical activity was lower after 'late night sleep loss' than 'early night sleep loss' and 'regular sleep' (both $P < 0.05$, respectively, for pairwise comparison), while physical activity was well comparable between 'early night sleep loss' and 'regular sleep' ($P = 0.724$ for pairwise comparison).

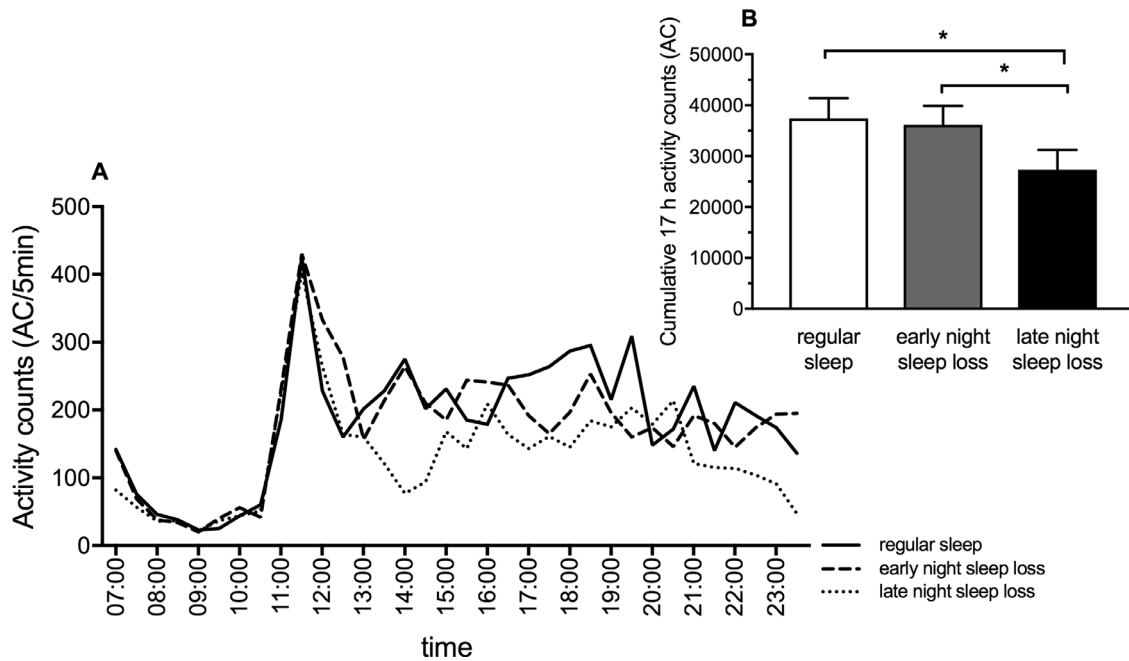


Fig. 1. Activity counts over time (A) and cumulative physical activity counts (B) (from 7:00 to 24:00) after ‘regular sleep’ (solid line; open column), ‘early night sleep loss’ (dashed line; grey column), and ‘late night sleep loss’ (dotted line; black column). * $P < 0.05$. AC: activity counts.

3.2. Physical activity during lab time and under free-living conditions

Physical activity was distinctly lower during lab time than under free-living conditions ($P < 0.001$ for ANOVA main effect ‘time’; Fig. 2), and differed between conditions ($P < 0.05$ for ANOVA main effect ‘condition’). Further analyses revealed that subjects displayed lower free-living physical activity after ‘late night sleep loss’ than ‘early night sleep loss’ and ‘regular sleep’ ($P < 0.05$ for Helmert contrast test). Again, there was no effect of shortened sleep *per se* ($P = 0.148$ for Helmert contrast test). Pairwise comparisons support that subjects displayed lower free-living physical activity after ‘late night sleep loss’

than ‘early night sleep loss’ and ‘regular sleep’ (both $P < 0.05$) with no difference between ‘early night sleep loss’ and ‘regular sleep’ ($P = 0.702$).

However, due to an overall restricted physical activity, there was no difference between sleep conditions during lab time (all $P \geq 0.229$ for pairwise comparisons; Fig. 2).

Analysing the 6 h time intervals under free-living conditions revealed no differences in physical activity between the afternoon and the evening with respect to a general time of day effect ($P = 0.417$ for ANOVA main effect ‘time’; Fig. 3) or the preceding sleep condition ($P = 0.769$ for ANOVA ‘condition’*‘time’ interaction). However, as

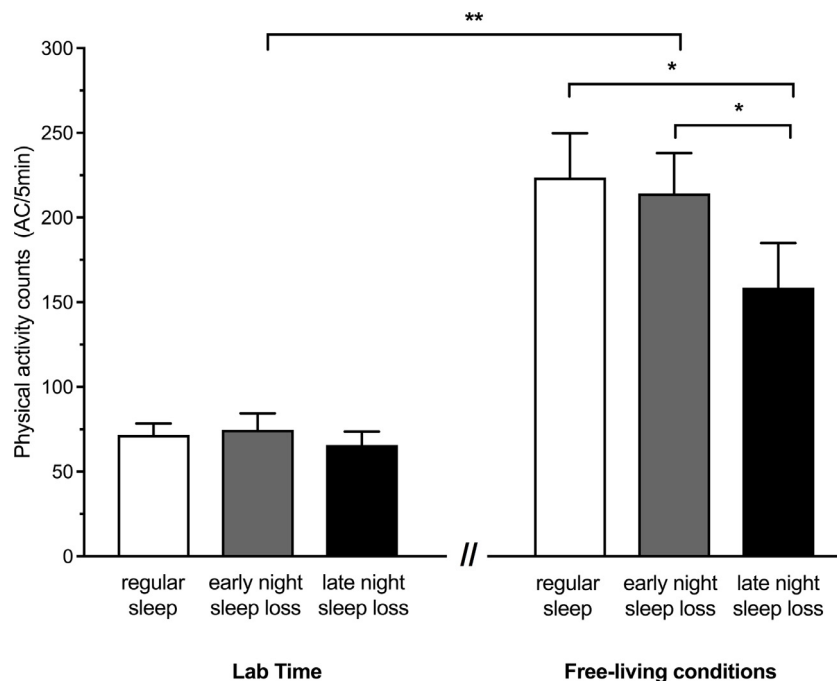


Fig. 2. Physical activity during lab time (07:00–11:30; left) and under free-living conditions (11:30–24:00; right) after ‘regular sleep’ (open columns), ‘early night sleep loss’ (grey columns), and ‘late night sleep loss’ (black columns); * $P < 0.05$; ** $P < 0.001$. AC: activity counts.

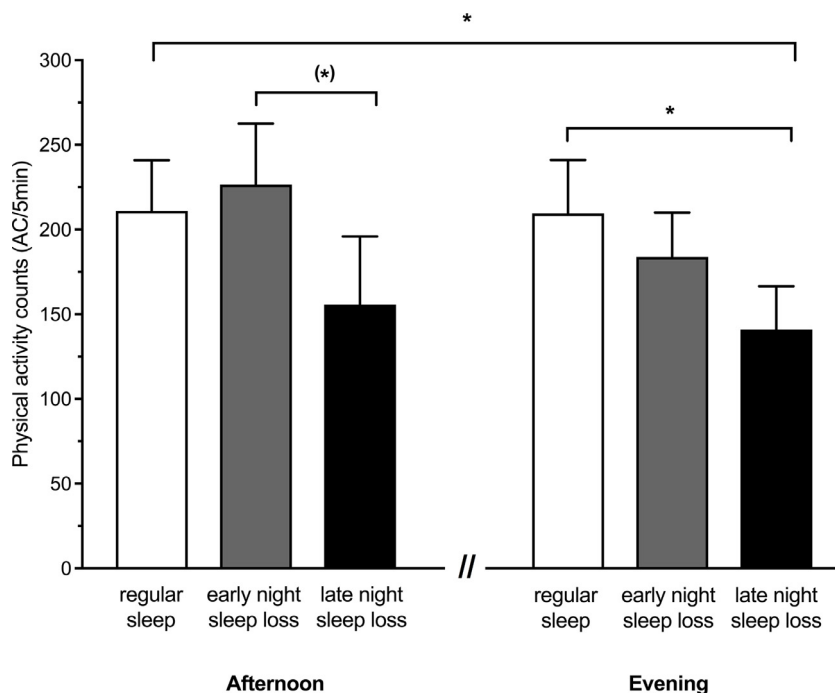


Fig. 3. Physical activity during free-living conditions divided into 6 h intervals, i.e. afternoon (12:00–18:00; left), and evening (18:00–24:00; right), after ‘regular sleep’ (open columns), ‘early night sleep loss’ (grey columns), and ‘late night sleep loss’ (black columns); ^(*) $P < 0.1$; * $P < 0.05$. AC: activity counts.

shown above, physical activity differed between conditions ($P < 0.05$ for ANOVA main effect ‘condition’) with lowest physical activity after ‘late night sleep loss’ ($P < 0.05$ for pairwise comparison ‘early night sleep loss’ vs. ‘regular sleep’ in the evening; $P = 0.053$ for pairwise comparison ‘early night sleep loss’ vs. ‘late night sleep loss’ in the afternoon).

3.3. Intensity of physical activity

During lab time participants spent most of time with low intensity physical activity ($P < 0.001$ for ANOVA main effect ‘activity level’) independent of the preceding sleep condition ($P = 0.479$ for ANOVA ‘activity level’*‘condition’ interaction; Fig. 4). There was also no significant difference in physical activity intensity pattern under free-living conditions ($P < 0.001$ for ANOVA main effect ‘activity level’;

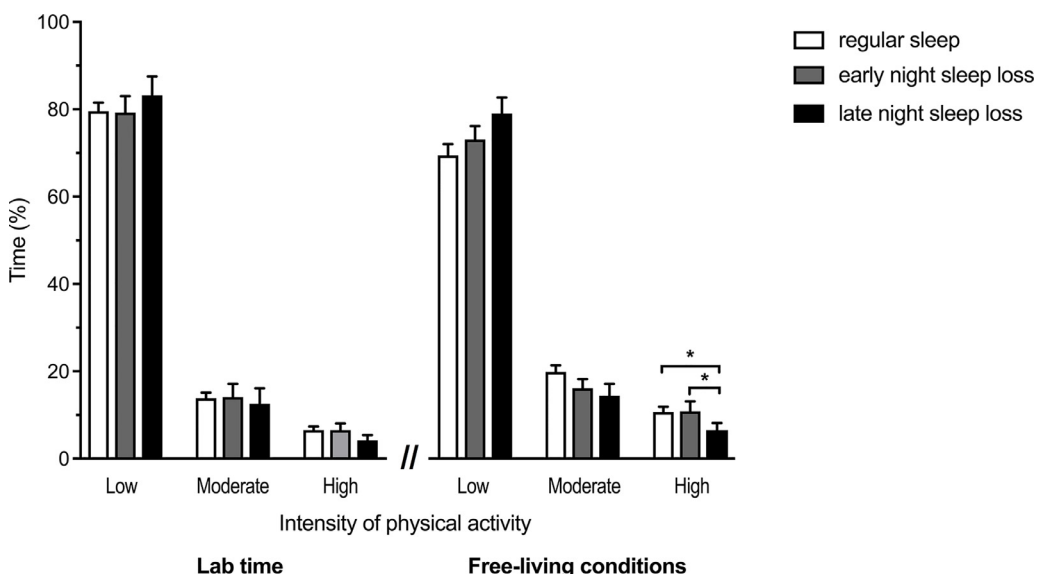


Fig. 4. Time spending in different physical activity intensities as characterized by low, middle and high levels during lab time (07:00–11:30; left) and under free-living conditions (11:30–24:00; right) after ‘regular sleep’ (open columns), ‘early night sleep loss’ (grey columns), and ‘late night sleep loss’ (black columns); * $P < 0.05$ for pairwise comparisons.

$P = 0.131$ for ANOVA ‘activity level’*‘condition’ interaction; Fig. 4). However, further analysis revealed less episodes of high intensity physical activity after ‘late night sleep loss’ as compared to the ‘early night sleep loss’ and ‘regular sleep’ condition ($P < 0.05$ for pairwise comparisons).

3.4. Ratings of perceived activity and tiredness

Perceived activity levels were similar between the 3 conditions during the time in the lab ($P = 0.381$ for ANOVA main effect ‘condition’; $P = 0.379$ for ANOVA ‘condition’*‘time’ interaction; Fig. 5). Of note, comparing values rated at equivalent time points after respective waking up time, i.e. from 03:00 to 07:00 for ‘late night sleep loss’; from 07:00 to 11:00 for ‘early night sleep loss’ and ‘regular sleep’, respectively) revealed also similar results ($P = 0.320$ for ANOVA main effect

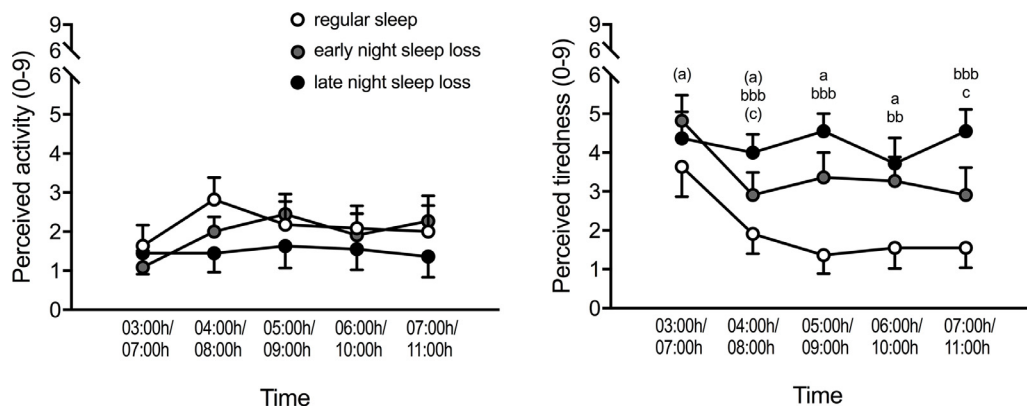


Fig. 5. Perceived activity (left) and tiredness (right) at equivalent time points after waking up after 'regular sleep' (open circles), 'early night sleep loss' (grey circles), and 'late night sleep loss' (black circles). ^(a) $P < 0.1$, ^a $P < 0.05$ for 'regular sleep' vs. 'early night sleep loss'; ^{bb} $P < 0.01$, ^{bbb} $P < 0.001$ for 'regular sleep' vs. 'late night sleep loss'; ^(c) $P < 0.1$, ^c $P < 0.05$ for 'late night sleep loss' vs. 'early night sleep loss'.

'condition'; $P = 0.347$ for ANOVA 'condition'*'time' interaction).

In contrast, ratings of perceived tiredness differed significantly between conditions ($P < 0.001$ for ANOVA main effect 'condition'; Fig. 5) with highest values after 'late night sleep loss' and intermediate values after 'early night sleep loss'. Perceived tiredness decreased across the time ($P < 0.05$ for ANOVA main effect 'time') in all experimental sleep conditions ($P = 0.306$ for ANOVA 'condition'*'time' interaction). Again, comparing values rated at equivalent time points after waking up revealed similar results ($P < 0.001$ for ANOVA main effect 'condition'; $P < 0.01$ for ANOVA main effect 'time'; $P = 0.213$ for ANOVA 'condition'*'time' interaction). Perceived tiredness was distinctly increased after shortened sleep than regular sleep at all equivalent time points (all $P < 0.01$ for Helmert contrast test), but not directly after awakening ($P = 0.123$). Feelings of perceived tiredness were also affected by early wake up than regular wake up (all $P < 0.01$ for Helmert contrast test at time points 04:00/08:00, 05:00/09:00, 07:00/11:00). Subsequent pairwise analysis of single equivalent time points revealed that subjects mostly reported more tiredness after both short sleep conditions as compared to 'regular sleep'.

4. Discussion

The main finding of the present study was a reduced physical activity after one night after 'late night sleep loss' compared to 'regular sleep' and in part after one night after 'early night sleep loss'. Furthermore, perceived feelings of tiredness were elevated by 'short sleep' *per se*.

This study clearly shows for the first time that the effects of acute sleep loss on physical activity are far more pronounced if sleep is curtailed during the second half of the night than during the first half of the night. However, our results need to be discussed in relation to timing of sleep loss in the 24-hour period, in addition to changes on length of time awake. Previous studies by Schmid et coll. [16] and Spiegel et coll. [8] suggested that earlier wake up will lead to stronger effects of sleep loss on human metabolism and appetite regulation. One may speculate that the effects of 'late night sleep loss' on subsequent spontaneous physical activity are modulated by the extended wake time period and probably higher homeostatic sleep pressure [21] that – in turn – may inhibit spontaneous physical activity. The graded homeostatic sleep pressure might be best reflected by subjective tiredness ratings that were increased in the morning after both short sleep conditions and in particular after 'late night sleep loss' as compared to 'regular sleep'. However, differences in tiredness did not translate into respective perception of subjective activity, which seems surprising at the first glance.

Spontaneous physical activity will be governed by several potential neuromodulators like other physiological functions (for review [22]). It is tempting to speculate that reduced spontaneous physical activity after acute sleep loss is mediated by altered ghrelin and leptin concentrations. So far, there is evidence from animal studies that leptin

modulates dopaminergic pathways to elicit spontaneous physical activity in rodents [23,24]. Central ghrelin administration increases spontaneous physical activity and dopamine concentrations in the nucleus accumbens and ghrelin was also shown to potentiate the cocaine-stimulated spontaneous physical activity [25,26]. However, results obtained from animal studies cannot be easily transferred one to one to humans.

In the present study, in contrast to our previous findings [16], physical activity was not reduced after 'early night sleep loss'. Several aspects might explain these – at first glance – contradictory outcomes. First, due to different experimental designs participants of the present study stayed in the lab until 11:30 (vs. 08:00 in the study by Schmid et coll. [16]) and thus, spontaneous physical activity was curtailed during this period that is characterized by relatively high levels of spontaneous physical activity [27]. On the other hand, physical activity was assessed until 24:00 (vs. 20:00 in the study by Schmid et coll. [16]) and comprises a period (i.e. from 20:00 to 24:00) that is characterized by physiologically decreasing physical activity pattern. These differences between the both studies become obvious when comparing total physical activity. In the present study, total physical activity after 'regular sleep' was around 10'000 AC less than reported by Schmid et coll. [16], while physical activity after 'early night sleep loss' was more or less comparable in both studies, i.e. approximately 40'000 AC, and physical activity after 'late night sleep loss' was lowest. Of note, same accelerometers and calculation procedure were used in both studies so that a technical bias is unlikely. Second, the sample size of the present study was smaller (11 vs. 15 subjects). This made it harder to detect statistical differences as compared with our previous published results [16]. Insofar, the present data do not necessarily contradict previous results [16]. With regard to the statistical approach, we used the Helmert contrast test since, as compared to usual post hoc tests, it allows a comparison of one level of a variable (e.g. 'late night sleep loss') to the mean of subsequent levels (e.g. wake up at 7:00 in 'earlynight sleep loss' and 'regular sleep' conditions). We are convinced that this approach, after running the ANOVA is appropriate and helps to evaluate the effects of both short sleep and wake-up time.

Other studies found no effects of sleep restriction on physical activity [9,17] or show an uneven picture with partly increased physical activity after sleep restriction [10]. Different definitions of physical activity, i.e. by body movement parameters (e.g. activity counts, steps) or energy expenditure related parameters (e.g. physical activity level, activity related energy expenditure) and thus, different techniques used (e.g. accelerometry and pedometry for body movement parameters, and doubly labelled water and 24h-heart rate monitoring for energy expenditure related parameters, respectively) might contribute to these inconsistent findings. Different study designs especially with regard to sleep restriction regimen, and study groups (e.g. men vs. women) could also lead to this heterogeneous picture. Of note, the technique used in the present study, i.e. accelerometry, is an objective and generally accepted method to assess free-living physical activity [28] that provides

detailed intensity, frequency, and duration regarding movement data [29]. However, accelerometers cannot account for all activities, such as cycling, stair climbing, and weight-lifting activities [29] that also could contribute to inconsistency between studies.

The fact that physical performance displays a circadian rhythm in humans is an interesting issue regarding our findings. Both aerobic and anaerobic fitness, motor skills, as well as functions relevant for athletic performance such as sensorimotor and cognitive performance, show highest levels in the late afternoon and early evening hours [30,31]. Therefore, acute sleep loss in the second half of the night could be particularly detrimental for exercising at a time-of-day when physical performance is enhanced and thus, vulnerable for physical activity lowering effects. Overall, this result complements at least in part the picture of the impact of circadian rhythms for optimal physical functioning in humans.

As mentioned above, the sample size of the present study is small which limits statistical power. However, the used cross over design partly compensates this limitation and the originally larger planned sample size, i.e. 16 subjects, would not lead to different results as calculated by post hoc power analysis (G*Power, Version 3.1.9.2 for Mac). With regard to the study design and sample size, subjects were not uniformly distributed to experimental conditions (i.e. 11 subjects, 3 conditions). Post hoc analyzes revealed that distribution was equal to conditions (Chi square = 0.756). Therefore, we believe that this aspect didn't influence present results. Furthermore, it was not possible to blind subjects to the experimental condition, at least, if they had undergone the first visit. However, subjects had no idea whether there was any hypothesis if one short sleep condition would be more deleterious than the other. However, we believe that this unblinded study design had any effects on results. Since we focused on daytime physical activity, physical activity during the night period was not compared with physical activity during sitting while staying awake in both short-sleep conditions. However, since participants were seated during the 4 h while awake in both experimental conditions we believe that this issue did not affect our present results. Lastly, we cannot separate time-of-day from prior wake effects, i.e., more prolonged wakefulness during 'late night sleep loss' as compared to 'early night sleep loss' and control condition due to the study design. Therefore, we cannot completely exclude that the longer time of prior wake in 'late night sleep loss' interacts with physical activity performance and thus, not only chronobiological aspects be influencing our results.

In summary, early waking up after a short night of sleep elicits stronger effects on subsequent spontaneous physical activity and perceived tiredness than going to bed late at night, despite identical sleep duration. Thus, our data indicate that not only short sleep, but also chronobiological aspects modulate physical activity pattern in men.

These novel data highlight and extend recommendations for sleep hygiene in our 24h-society, since short and - in particular desynchronized - sleep not only impairs metabolic conditions but also lowers spontaneous free-living physical activity. Besides its effects on energy balance, physical inactivity as well as cardiorespiratory fitness is associated with health status [32]. A complete preservation of sleep duration and circadian synchrony in our globalized 24 h-society seems hardly feasible. Further research including larger cohorts is required in order to develop sleep recommendations for example for shift workers so that the detrimental effects of sleep disturbance could be attenuated.

Declaration of Competing Interest

None.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.physbeh.2019.112795.

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