Sleepiness as motivation: a potential mechanism for how sleep deprivation affects behavior

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Abstract

Study Objectives: To determine how sleepiness and sleep deprivation drive the motivation to engage in different behaviors.

Methods: We studied the sleepiness of 123 participants who had been randomized to sleep deprivation or normal sleep, and their willingness to engage in a range of everyday behaviors.

Results: Self-reported sleepiness was a strong predictor of the motivation to engage in sleep-preparatory behaviors such as shutting one's eyes (OR = 2.78, 95% CI: 2.19–3.52 for each step up on the Karolinska Sleepiness Scale) and resting (OR = 3.20, CI: 2.46–4.16). Sleepiness was also related to the desire to be cared for by a loved one (OR = 1.49, CI: 1.22–1.82), and preparedness to utilize monetary and energy resources to get to sleep. Conversely, increased sleepiness was associated with a decreased motivation for social and physical activities (e.g. be with friends OR = 0.71, CI: 0.61–0.82; exercise OR = 0.65, CI: 0.56–0.76). Sleep deprivation had similar effects as sleepiness on these behaviors. Neither sleepiness nor sleep deprivation had strong associations with hunger, thirst, or food preferences.

Conclusions: Our findings indicate that sleepiness is a dynamic motivational drive that promotes sleep-preparatory behaviors and competes with other drives and desired outcomes. Consequently, sleepiness may be a central mechanism by which impaired alertness, for example, due to insufficient sleep, contributes to poor quality of life and adverse health. We propose that sleepiness helps organize behaviors toward the specific goal of assuring sufficient sleep, in competition with other needs and incentives. A theoretical framework on sleepiness and its behavioral consequences are likely to improve our understanding of several disease mechanisms.

Key words: sleepiness; sleep deprivation; motivation; behavior; health

Statement of Significance

We show that sleep deprivation and self-reported sleepiness (1) increase willingness to engage in sleep-preparatory behaviors, (2) reduce willingness to engage in physical and social behaviors, and (3) induce a preparedness to utilize resources toward being allowed to sleep. Through this reduction in motivation to engage in physical and social activities—activities that increase the quality of life and protect against health problems—too much sleepiness may act as a mechanism contributing to cardio-metabolic and mood disorders. Sleepiness is common in many patient groups, further indicating its role as a mediator between insufficient sleep and increased risk for developing several afflictions. This manuscript thus provides a new theoretical framework around self-reported sleepiness as a mechanistic risk factor for adverse health outcomes.
Introduction

Primary motivational drives, such as hunger and thirst, promote behaviors that meet biological needs. Sleep is an essential biological need regulated by homeostatic and circadian processes [1], but little is known about sleep-related motivations. The primary consequence of sleep deprivation is sleepiness [2], a phenomenon strongly influenced by external stimuli, incentives, and desired outcomes [3], similar to how the flexibility of motivational drives, emotions, and survival circuits helps the organism respond to challenges and opportunities in a dynamic environment [4]. The present study proposes a new model where sleepiness functions as such a motivational drive, assuring that basic requisites for sleeping are obtained.

Traditionally, sleepiness has been defined as a physiological inability to stay awake, commonly measured as the time it takes to fall asleep when lying in a bed in a dark environment [5]. However, humans, primates, and many other animals do not enter sleep indiscriminately, but instead engage in a number of sleep-preparatory behaviors before sleeping [6]. Typically, such behaviors relate to safety (e.g. finding a safe site in a tree/burrow/cave, sleeping together), comfort and hygiene (e.g. shelter from elements, avoidance of light or biting insects, making the bed), social dynamics and huddles (e.g. kin relationship, conservation of body heat), and obtaining a particular sleep position (e.g. lying in a certain position, shutting one’s eyes) [6, 7]. Many of these behaviors ensure that sleep occurs in an acceptable setting. The fact that most of them are robust across species and cultures suggests that preparing for sleep is driven by a motivating mechanism to assure the safety and adequacy of one’s surroundings. Here, we investigated the potential role of sleepiness as such a mechanism, coordinating these preparatory behaviors.

Insufficient sleep is related to an increased risk for adverse health outcomes [8], and proposed mechanisms include metabolic, immunological, and cognitive alterations. The primary consequence of insufficient sleep—either caused by acute sleep loss, repeated partial sleep restriction [2], or disruption of slow-wave sleep [9]—is sleepiness, and it seems possible that any sleepiness-related alterations of motivation and behavior could be a previously unexplored pathway between insufficient sleep and health problems. For example, previous findings indicate that sleep loss has a larger effect on the experience of exertion than on physiological effort, as indicated by heart rate and metabolic rate, while exercising [10]. It seems possible that through decreased motivation, sleepiness results in avoidance of, and reduced tolerance to, physical activity.

We hypothesized that sleepiness would be associated with an increased desire for safety-seeking and utilization of resources toward sleeping, and a decreased inclination toward engaging in other, competing, behaviors. To test these hypotheses, participants were randomized to one night of sleep loss or a normal night’s sleep. The following day they rated their sleepiness and how much they wanted to engage in different behaviors. With an exploratory aim, we also assessed eating behaviors and preferences. While the literature shows that sleep deprivation increases energy expenditure, hunger, and preference for high-energy foods [11], eating and drinking could also be seen as a competitive behavior to the initiation of sleep.

Methods

Participants

To cause variation in sleepiness, 123 participants (61 women, mean age 25.3 ± 6.3 years) were randomized to normal sleep at home (N = 60; 29 women, mean age 25.1 ± 6.8 years), or a night of total sleep deprivation (N = 63; 32 women, mean age 25.5 ± 5.7 years), verified by continuous monitoring in the laboratory. Inclusion criteria were fluent Swedish, and a habitual sleep needs of 7–9 hours. Participants were free from sleep disorders and other physical and mental disorders, not allowed to have worked shifts the previous 3 weeks, habitually drink more than three cups of coffee or tea per day, smoke, take medication that affects sleep/sleepiness, or have studied psychology. The sample was a subset of a larger sleep-deprivation study, see Ref. [12] for the detailed study protocol. The study was approved by the Regional Ethical Review Board in Stockholm (dnr: 2012/2189-31/3 and 2014/1766-32). Participants provided written informed consent.

Participants spent three nights sleeping 7–9 hours before being informed of their sleep condition for the fourth night. For the sleep group, participants were instructed to spend one more night sleeping 7–9 hours at home and coming to the lab at 10:00 the following day. Participants in the sleep-deprivation group instead came to the lab at 22:00 that evening and stayed awake throughout the night. Breakfast was served 06:30–07:30 in the sleep-deprivation condition, whereas normal-sleep participants had breakfast at home at a time of their choosing, before coming to the lab. Lunch was served in the lab at 12:00. Ratings were collected at 11:00. No strenuous exercise or alcohol was allowed during the day prior to, or the morning of, coming to the lab, and no caffeine intake or naps were allowed during the lab day.

Sleep

Participants’ sleep was measured with actigraphy (Motionwatch 8, Cammtech, UK) and sleep diaries for three to four nights in their own homes. The average sleep times across the first 3 nights were 470 ± 39 min (mean ± SD) for the normal-sleep group and 470 ± 35 min for the sleep-deprivation group. During the fourth night, when the sleep-deprivation group was awake in the lab, the normal-sleep group slept for an average of 474 ± 50 min.

Sleepiness and motivation

At 11:00, participants used the newly constructed motivation scale of sleepiness (MOSS) to indicate their motivation to engage in 24 different behaviors on 5-graded scales (see supplement for full questionnaire and distributions). They also rated their self-reported sleepiness on the Karolinska Sleepiness Scale (KSS), see Figure 11. The KSS is a well-validated scale related to physiological markers of sleepiness, reduced performance, and accidents, and responses are strongly affected by sleep loss [13], making it widely used in experimental sleep-deprivation studies as well as in field studies. The MOSS was developed for the present study to measure behavioral dimensions of sleep preparation, sleep, social activities, physical activities, resource utilization, and food preferences.

In a previous study [14], we found that two nights of partial sleep deprivation increased sleepiness with approximately 3 units on the KSS scale, with a pooled standard deviation of approximately 1.6, indicating an effect size of $d = 1.9$ and 100% power to
detect such an effect in this study. The sample size of $n = 123$ allowed for detection of true associations between sleepiness and motivation of $r = 0.32$ with 95% and $r = 0.25$ with 80% power at $p < .05$. All independent variables (i.e. sleep deprivation and sleepiness ratings) and dependent variables (MOSS items) collected for the aim of this article have been reported. No participants or observations were excluded from the analyses.

Statistics

Data were analyzed using an ordinal (proportional odds) logistic regression model. This model assumes a continuous latent dependent variable that has been measured on an ordered category scale with $K$ responses, and fits multiple intercepts $\zeta_{1,2,...,K-1}$ representing cut-points between the observed response categories, where:

$$-\infty = \zeta_0 < \zeta_1 < \ldots < \zeta_{K-1} = \infty$$

The probability of response $K$ is then the normal logistic model below, where $x_0$ is the combined linear predictor excluding the intercepts:

$$\logit[P(Y \leq k|x)] = \zeta_k - x$$

Figure 1. (A–G) Expected means of the responses (1 = not at all, 5 = very/very much) as a function of self-reported sleepiness (Karolinska Sleepiness Scale (KSS) 1 = very alert, 9 = very sleepy). (H) Probability of paying money or walking any distance as a function of sleepiness. (I) Distribution of observed sleepiness for the two groups. Expected means were calculated from predicted probabilities based on estimates from ordinal (proportional odds) logistic regression models.
Two different models were fitted. The first model estimated the association of responses with sleep deprivation and the second model estimated the association of responses with sleepiness. The exponentiated estimates describe the odds ratio (OR) of an increase of at least one step on the MOSS-scale for the sleep-deprived group compared to the group with normal sleep, or an increase in the MOSS-scale response for every one unit increase of rated sleepiness (KSS: 1–9) for all participants combined. Expected means of the responses were subsequently calculated from the predicted probabilities. A likelihood ratio test was used to assess statistical significance, by comparing the model fitting sleep deprivation or sleepiness as a predictor, with a model fitting only the intercepts. All analyses were performed in R using the polr function from the package MASS [15].

Results

The effects of sleep deprivation are detailed in Table 1 (mean values are reported in Supplementary Table 1). In addition to being sleepier, sleep-deprived participants expressed more preference for sleep preparation, resting, sleeping, and care seeking (being cared for by a partner, but not by a parent). The sleep-deprived group also showed less interest in engaging in social activities with others, and instead wanted to be alone. Sleep-deprived participants were also prepared to utilize more resources in order to be able to sleep, with regard to both paying for every one unit increase of rated sleepiness (KSS: 1–9) for all participants combined. Expected means of the responses were subsequently calculated from the predicted probabilities. A likelihood ratio test was used to assess statistical significance, by comparing the model fitting sleep deprivation or sleepiness as a predictor, with a model fitting only the intercepts. All analyses were performed in R using the polr function from the package MASS [15].

Table 1. Effects of sleep deprivation on the motivation to engage in different behaviors and activities, and their associations with sleepiness

| Group       | Dependent variable | Effect of sleep deprivation | Association with sleepiness |
|-------------|--------------------|-----------------------------|----------------------------|
| Sleepiness  | Sleepiness         | OR 31.66 12.94, 77.5 .000*** | OR  — — — — — — — — — — |
| Sleep preparation | Shut eyes        | 9.63 4.54, 20.4 .000*** 2.78 2.19, 3.52 .000*** |
|              | Quiet              | 2.47 1.29, 4.72 .006** 1.42 1.22, 1.66 .000*** |
|              | Be home            | 9.04 4.28, 19.1 .000*** 1.80 1.52, 2.15 .000*** |
| Resting and sleeping | Rest            | 18.00 7.87, 41.2 .000*** 3.20 2.46, 4.16 .000*** |
|              | Sleep own bed      | 24.82 10.43, 59.1 .000*** 3.05 2.35, 3.96 .000*** |
|              | Sleep any bed      | 14.96 6.84, 32.8 .000*** 2.58 2.03, 3.28 .000*** |
| Care seeking | Partner            | 5.59 2.15, 14.5 .000*** 1.49 1.22, 1.82 .000*** |
|              | Parent             | 1.51 0.78, 2.91 .221 1.19 1.03, 1.39 .019* |
| Physical activities | Food shopping     | 0.19 0.09, 0.41 .000*** 0.77 0.66, 0.89 .001*** |
|              | Walk               | 0.35 0.18, 0.67 .011** 0.79 0.69, 0.91 .001* |
| Social activities | Be with stranger  | 0.16 0.08, 0.34 .000*** 0.65 0.56, 0.76 .000*** |
|              | Date               | 0.19 0.09, 0.39 .000*** 0.61 0.51, 0.72 .000*** |
|              | Be alone           | 5.52 2.76, 11.05 .000*** 1.62 1.37, 1.91 .000*** |
|              | Be with friends    | 0.12 0.06, 0.24 .000*** 0.71 0.61, 0.82 .000*** |
| Hunger and thirst | Hungry            | 3.84 0.97, 3.53 .003 1.16 1.00, 1.34 .045* |
|                | Water              | 0.58 0.30, 1.10 .095 0.93 0.81, 1.07 .329 |
|                | Meal               | 1.24 0.66, 2.33 .510 1.04 0.91, 1.20 .542 |
| Food preferences | Fruit             | 0.94 0.50, 1.77 .850 1.00 0.87, 1.15 .973 |
|                | Sweets/candy       | 1.12 0.59, 2.14 .726 1.08 0.94, 1.25 .264 |
|                | Steak/protein      | 0.90 0.48, 1.69 .738 1.00 0.86, 1.15 .945 |
| Utilization of resources | Pay             | 5.41 2.48, 11.8 .000*** 1.66 1.34, 2.05 .000*** |
|                | Walk               | 2.15 1.12, 4.11 .020 1.20 1.03, 1.40 .017* |

Ordinal (proportional odds) logistic regression. The table shows odds ratios (OR) for a one-unit increase on the response scale after sleep deprivation (left) and with an increase of one unit of sleepiness on the Karolinska Sleepiness Scale (KSS) (right), with 95% confidence intervals, p-values based on likelihood ratio tests.

*p < .05 "p < .01 "p < .001.
sooner, although this effect size was small, likely due to the fact that even alert participants chose to walk at least some distance.

Although sleepiness was related to a slight increase in hunger, there was no association between sleepiness and thirst or food preferences (Figure 1F and G).

As indicated in Figure 1, not all variables were associated with sleepiness. A more detailed analysis is presented in the supplementary appendix (Supplementary Figure S1), showing the probability distribution of the responses as a function of sleepiness. The weakest associations (Supplementary Figure panels P–U) show a relatively constant probability distribution of the responses across all levels of sleepiness, while the strongest associations, such as shutting the eyes and resting (Supplementary Figure panels A–F) clearly indicate how probabilities for higher ratings increase with higher sleepiness levels.

Discussion
These data indicate that sleepiness is more than just the propensity to fall asleep. In line with our hypothesis, sleepiness seems to function as a dynamic motivational drive, affecting daily activities, promoting sleep-preparatory behaviors, and influencing the timing of sleep. Thus, sleepiness can be seen as an integral mechanism assuring that sleep occurs in an adequate and safe environment. Many animals have particular sleep sites and engage in sleep-preparatory behavior [6, 7], and it is likely that such a mechanism would be conserved across a large range of species.

Although sleepiness contributes to sleep occurring during optimal times and in an adequate environment, it could also be viewed as a potential mechanism behind the detrimental effect of sleep loss on health, including mental and cardio-metabolic health. The finding that sleepiness reduced the motivation to engage in physical and social activities has important implications, as such activities contribute to the quality of life and protect against ill-health [16, 17]. Along these lines, viewing sleepiness as motivation may help explain why less sleep relates to wanting to be alone, social withdrawal, and loneliness [18–20], as well as why many mental disorders, often comorbid with sleep problems [21], are characterized by a reduced motivation to engage socially with others. Sleepiness might thus be a mechanism behind how short and disturbed sleep, as well as excessive daytime sleepiness unrelated to sleep loss, increase the risk for negative health outcomes and a sedentary lifestyle. Furthermore, the motivation to be at home and sleep in one’s own bed when sleepy could be a potential risk factor for accidents. For example, shift workers driving home from a night shift might be highly motivated to get there as soon as possible, decreasing the likelihood of safety behaviors such as stopping for a nap or coffee, or planning for an alternative way to get home.

Targeting self-reported sleepiness directly (e.g. through light, sleep treatment, or caffeine) might aid health-protective behaviors, improve adherence with treatment schemes, and reduce health risks. This kind of intervention may be particularly successful in cases where there is a strong relationship between excessive sleepiness and health problems, such as depression [22, 23]. Our findings highlight the need for future studies in the fields of human biology, psychology, and medicine to investigate sleepiness from a motivational perspective, and to elucidate its potential role in competing with other behaviors. From one perspective, it seems likely that some motivational drives, such as socializing with others [24] and breeding [25], cause individuals to down-prioritize sleep duration despite becoming sleepier. There is a clear need for studying the importance of sleepiness as a signal for decision making in competition with other motivational drives, and why individuals’ choices vary under different circumstances.

A crucial aspect in the current study was the focus on how much the participants wanted to engage in different behaviors rather than monitoring what behaviors they actually engaged in. It will be important to carry out studies of how sleepiness and other motivational drives together predict actual behavior in real-life situations, both at work and at home. Other ways of manipulating sleepiness, for example, with repeated sleep restriction, naps, or caffeine, would also help delineate how motivation and behavior are affected. Furthermore, future work will have to define how sleepiness and fatigue relate to one another from a motivational viewpoint. Although from a theoretical point of view these concepts are strongly separated from each other, they are often used interchangeably [26]. The definitions of fatigue vary, with some including sleepiness as one of its dimensions (e.g. Ref. [27]), while others have used definitions and shown data indicating that sleepiness and fatigue are independent manifestations of insufficient sleep [28]. Limitations of the current study include the restricted sample of healthy young adults and the one-time assessment, that participants in the normal-sleep condition could eat what and when they wanted the morning before coming into the lab, and that hunger was measured at a time when participants were not very hungry. Measures of how sleepiness relates to behavioral changes and actual food intake at all times awake will also be an important future step. Furthermore, there is a need to study sleepiness as a motivational drive in more diverse and less healthy groups, preferably in a longitudinal manner.

In conclusion, this study shows for the first time that sleepiness can function as a motivational drive and is an important step in optimizing one’s sleep. If the reduced desire to engage in physical and social activities when sleep translates to actual reductions in these behaviors, it has implications for how insufficient sleep contributes to negative health outcomes [8, 22] as well as how these could be counteracted. We propose that factors reducing sleepiness may, besides countering deteriorations in performance, also aid individuals in managing a healthier lifestyle.

Supplementary Material
Supplementary material is available at SLEEP online.

Authors’ Contributions
J.A., G.K., M.L., and T.S. developed the study concept and designed the research; T.S., and J.A. collected the data; M.I., and J.A. analyzed the data and drafted the manuscript. All authors interpreted the data, revised the manuscript and accepted the final version for submission.

Open Practices Statement
The experiment in this article was not formally preregistered. The MOSS is provided in the supplemental information. MOSS, statistics, data, and code are available at https://osf.io/ pa2ng/?view_only=a04b6093c4074cdeba0759a048e1792

Axelsson et al. | 5
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References
1. Borbély AA. A two process model of sleep regulation. Hum Neurobiol. 1982;1(3):195–204.
2. Lo JC, et al. Effects of partial and acute total sleep deprivation on performance across cognitive domains, individuals and circadian phase. PLoS One. 2012;7(9):e45987.
3. Horne J. Sleepiness as a need for sleep: when is enough, enough? Neurosci Biobehav Rev. 2010;34(1):108–118.
4. LeDoux J. Rethinking the emotional brain. Neuron. 2012;73(4):653–676.
5. Carskadon MA, et al. Guidelines for the multiple sleep latency test (MSLT): a standard measure of sleepiness. Sleep. 1986;9(4):519–524.
6. Anderson JR. Sleep, sleeping sites, and sleep-related activities: awakening to their significance. Am J Primatol. 1998;46(1):63–75.
7. Zepelin H, Siegel JM, Tobler I. Mammalian sleep. In: Kryger MH, Roth T, Dement WC, eds. Principles and Practice of Sleep Medicine. Vol. 4. Philadelphia: Elsevier Saunders; 2005: 91–100.
8. Kecklund G, et al. Health consequences of shift work and insufficient sleep. BMJ. 2016;355:i5210.
9. Groeger JA, et al. Dissociating effects of global SWS disruption and healthy aging on waking performance and daytime sleepiness. Sleep. 2014;37(6):1127–1142.
10. Martin BJ. Effect of sleep deprivation on tolerance of prolonged exercise. Eur J Appl Physiol Occup Physiol. 1981;47(4):345–354.
11. Markwald RR, et al. Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. Proc Natl Acad Sci USA. 2013;110(14):5695–5700.
12. Holding BC, et al. Sleep deprivation and its effects on communication during individual and collaborative tasks. Sci Rep. 2019;9(1):3131.
13. Akerstedt T, et al. Subjective sleepiness is a sensitive indicator of insufficient sleep and impaired waking function. J Sleep Res. 2014;23(3):240–252.
14. Sundelin T, et al. Negative effects of restricted sleep on facial appearance and social appeal. R Soc Open Sci. 2017;4(5):160918.
15. Venables WN, et al. Modern Applied Statistics with S. New York: Springer; 2002.
16. Kyu HH, et al. Physical activity and risk of breast cancer, colon cancer, diabetes, ischemic heart disease, and ischemic stroke events: systematic review and dose-response meta-analysis for the Global Burden of Disease Study 2013. BMJ. 2016;354:i3857.
17. Leigh-Hunt N, et al. An overview of systematic reviews on the public health consequences of social isolation and loneliness. Public Health. 2017;152:157–171.
18. Ben Simon E, et al. Sleep loss causes social withdrawal and loneliness. Nat Commun. 2018;9(1):3146.
19. Cacioppo JT, et al. Loneliness and health: potential mechanisms. Psychosom Med. 2002;64(3):407–417.
20. Totten D, et al. Associations of sleep with everyday mood, minor symptoms and social interaction experience. Sleep. 1994;17(5):466–475.
21. Baglioni C, et al. Sleep and mental disorders: a meta-analysis of polysomnographic research. Psychol Bull. 2016;142(9):969–990.
22. Buyse D. Sleep health: can we define it? Does it matter? Sleep. 2014;37(1):9–17.
23. Chellappa SI, et al. Chronobiology, excessive daytime sleepiness and depression: is there a link? Sleep Med. 2009;10(5):505–514.
24. Bassner M, et al. American time use survey: sleep time and its relationship to waking activities. Sleep. 2007;30(9):1085–1095.
25. Lesku JA, et al. Adaptive sleep loss in polygynous pectoral sandpipers. Science. 2012;337(6102):1654–1658.
26. Chen J, et al. Distinguishing sleepiness and fatigue: focus on definition and measurement. Sleep Med Rev. 2006;10(1):63–76.
27. Ahsberg E. Dimensions of fatigue in different working populations. Scand J Psychol. 2000;41(3):231–241.
28. Hossain JL, et al. Subjective fatigue and subjective sleepiness: two independent consequences of sleep disorders? J Sleep Res. 2005;14(3):245–253.