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Abstract

Objectives: Chronic sleep restriction is highly prevalent in modern society and is in its clinical form, insufficient sleep syndrome, one of the most prevalent diagnoses in clinical sleep laboratories, with substantial negative impact on health and community burden. It reflects every-day sleep loss better than acute sleep deprivation, but its effects and particularly the underlying mechanisms remain largely unknown for a variety of critical cognitive domains, as for example risky decision-making.

Methods: We assessed financial risk-taking behavior after 7 consecutive nights of sleep restriction and after one night of acute sleep deprivation compared to a regular sleep condition in a within-subject design. We further investigated potential underlying mechanisms of sleep loss induced changes in behavior by high-density electroencephalography recordings during restricted sleep.

Results: We show that chronic sleep restriction increases risk-seeking, while this was not observed after acute sleep deprivation. This increase was subjectively not noticed and was related to locally lower values of slow wave energy during preceding sleep, an electrophysiological marker of sleep intensity and restoration, in electrodes over the right prefrontal cortex.

Interpretation: This study provides for the first time evidence that insufficient sleep restoration over circumscribed cortical areas leads to aberrant behavior. In chronically sleep restricted subjects, low slow wave sleep intensity over the right prefrontal cortex - which has been shown to be linked to risk behavior – may lead to increased and subjectively unnoticed risk-seeking.

Introduction

The insufficient sleep syndrome is a very prevalent sleep-wake disorder which negatively impacts health and causes major community burden[1, 2]. In affected subjects, excessive daytime sleepiness is caused by behavior, i.e., chronic sleep restriction (SR), and not by a medical pathophysiology[3]. Accordingly, the current diagnostic criteria require that a patient's sleep time is usually shorter than expected for age[3]. Chronic SR generally affects large parts of modern society[4-6]. Apart from sleepiness, regular sleep durations of less than 7 hours per night causes impairments in cognitive performance involving vigilant attention, cognitive processing speed, and working memory[7].

However, the majority of experimental studies on the behavioral effects of sleep loss focused on the effects of acute sleep deprivation (SD)[8], a term used to indicate the full absence of sleep for an entire night or a longer consecutive period of time. In contrast to chronic SR, however, acute SD does not reflect a major socio-medical problem. It is currently unclear whether effects observed after SD also apply to chronic SR[8-10]. As a consequence, the effects and underlying mechanisms of chronic SR, despite reflecting every-day sleep loss much better than acute SD, remain largely unknown for a variety of critical cognitive domains.

Decision-making in chronically sleep-restricted people has hardly been studied so far but is crucial for activities of daily living and particularly essential for economic and political leaders[11, 12]. Over 40% of managers of companies and enterprises sleep 6 hours or less per night[13]. These figures clearly indicate that we need insights into the effects of insufficient sleep on decision-making, including financial risk-taking. Some studies investigated risk-taking after acute sleep deprivation. The results on the influence on risk-taking have been mixed though[14-21], probably due to different paradigms and protocols. However, up to date no paper assessed physiological brain measures related to risk-taking after SR.

Therefore, the aim of our study was to assess the effects of both chronic SR and acute SD on risk-taking, i.e., on individual risk-preferences, and to investigate underlying mechanisms of potential changes after SR. Slow oscillating brain activity, namely slow waves during non-rapid eye movement (NREM) sleep are thought to reflect sleep intensity and the restorative function of sleep[22]. Slow waves are not equally distributed over the cortex but show local differences that vary between individuals[23] and that can be assessed using high-density EEG[24]. Given that cognitive domains known to be impaired after sleep loss are processed in specific cortical areas, we hypothesized that local differences in sleep intensity during the short nights of SR would relate to eventual changes in risk-taking after SR.

Methods

Experimental Design

We evaluated risk-taking in a controlled cross-over and within-subject protocol that included SR (7 nights during which time in bed was restricted to 5 hours per night) and SD (40 hours of continuous wakefulness), compared to a regular sleep condition (RegS) in a counterbalanced design (Fig 1). Risk-taking was assessed twice a day (afternoon and evening - to mitigate potential diurnal fluctuations of behavior) after RegS, after SR and after SD. Both SR and SD were preceded by one week of regular sleep-wake rhythm at home (eight hours in bed per night adapted to each subject's habitual bedtime). The interval between SR and SD was at least two weeks. SD was performed in the sleep laboratory under constant supervision. The first 4 nights and days of the SR protocol were performed at home, the last 3 nights in the sleep laboratory, which was achieved by delaying the bedtime by two hours and advancing the time of getting up by one hour. Compliance outside the laboratory was ensured by wrist actigraphy[25] (on the non-dominant wrist; light sensor data included, ActiWatch, Respironics), sleep diaries, and phone calls (mean ± s.e.m. actigraphy derived time in bed

during SR nights at home: 4.94 ± 0.01 h, in the sleep lab: 5.01 ± 0.03 h). Daytime napping was not allowed throughout the protocol. All actograms were therefore checked by an expert and no indication of unplanned sleep has been detected in any of the subjects. Subjects were further asked to abstain from caffeine, alcohol, and medication intake, starting 3 days prior to SR and SD and lasting throughout the protocol. Smokers were requested not to increase their habitual cigarette consumption, and to not smoke at least 30 minutes prior to each assessment. This 30-minute restriction also applied to food intake.

To evaluate repetitive test effects, we performed a task validation with another group of male subjects. This task validation group, which was different from the participants in the main experiment, maintained a regular sleep-wake rhythm with eight hours in bed per night for two weeks in total, controlled by wrist actigraphy and sleep diaries. Behavioral assessments took place during the second week at the same times as during RegS followed by SR in the main experiment (cf. Fig 1B). All study-related restrictions were identical to the main experiment.

Participants

Fourteen healthy, right-handed, male participants, aged between 18 and 28 years (21.9 ± 3.0, mean ± s.d.) were recruited from a student population. The sample size was chosen in accordance to previous studies investigating sleep loss-induced changes in behavior (e.g.[10]) and alterations of risk-taking behavior due to brain stimulation (e.g.[26]). The reported regular sleep duration ranged from seven to eight hours per night (7.7 ± 0.4 hours per night, mean ± s.d.). Female subjects were not included in this study, as for example sleep deprivation-induced cognitive impairments have been shown to vary in response to the phase of the menstrual cycle and the intake of oral contraceptives[27]. Confounds related to different time points of the menstrual cycle would have been unavoidable due to the lengthy study protocol.

Subjects were carefully screened for exclusion criteria that may potentially affect brain functioning, sleep physiology, or risk-taking. These encompassed clinically relevant diseases, regular medication intake, history of a seizure or moderate to severe traumatic brain injury, history of sleep-wake disorders or complaints (including excessive daytime sleepiness and irregular sleep-wake rhythm), drug or alcohol abuse, long (>9 hours per night) or short sleepers (<7 hours per night), recent travelling across more than two time zones, more than five drinks or food items containing caffeine per day, more than ten cigarettes per day, and subjects studying mathematics, physics, computer science, economics, or psychology as these students might be familiar with behavioral test settings or their analyses. A screening night was further performed to exclude any undiagnosed sleep disorders, to assess sleep efficiency, and to let the subjects adapt to the lab environment. The same inclusion and exclusion criteria applied to the subjects recruited for the task validation group (n = 14, aged between 18 and 29 years; 22.5 ± 3.1). Sleep disturbances were only excluded based on questionnaires but no sleep EEG was recorded in the inclusion procedure. Although, we cannot fully exclude the presence of any undiagnosed sleep disorders without a sleep EEG in the subjects recruited for task validation, none of the well-established questionnaires indicated disturbed sleep (range of scores Pittsburgh Sleep Quality Index: 0 – 4; Sleep Apnea scale of the Sleep Disorders Questionnaire (SAS-SDQ): 17 – 28; Ullanlinna Narcolepsy Score: 3 – 8; Epworth Sleepiness Scale: 0 – 10).

All subjects received fixed and variable (depending on task outcomes) monetary compensation for study participation. The local ethics committee approved the study (cantonal ethics commission Zurich, KEK-ZH-Nr. 2012-0496; registration at clinicaltrials.gov: NCT02305225) and written informed consent was obtained from all participants.

Assessment of Risk-Taking

To determine individual risk-preferences we used a binary probabilistic decision task adapted from Levy et al.[28]. On every trial, subjects had to choose between a specified amount of money paid out with a given probability or a lower amount of money paid out for sure (Fig 1). This paradigm is a standard procedure in behavioral economics that is routinely used to measure the individual propensity for risk-taking[29, 30]. Both options were displayed simultaneously on either side of the computer screen (spatial position of options was randomized across trials). We used four different levels of probability in the risky options (20%, 40%, 60% and 80%) and all of these risky offers were matched with a certain monetary amount to keep the expected value (EV = p * v, with p = probability, v = value) fixed at 20 Swiss Francs (100, 50, 33.30 and 25 Swiss Francs, respectively). Each probabilistic offer was paired with 22 linearly increasing certain choice alternatives ranging from 1.75 to 38.30 Swiss Francs. These ranges had been optimized prior to the study in an independent pilot trial (in 9 healthy, young students who did not participate in the study itself) to capture the risk attitudes of the targeted participant population. All pairs of options were repeated four times per session in a random order, adding to a total of 352 trials. Maximal time given to indicate the choice by left or right key press was 8 seconds, therefore ensuring spontaneous, realistic answers, but giving enough time to process the displayed information. The next trial appeared 1 second after the preceding choice, or after 8 seconds if no answer had been given (mean ± s.e.m number of trials without response was 0.0 ± 0.0 after RegS, 0.3 ± 0.2 after SR and 3.2 ± 1.7 after SD). During the task, the subject was monitored by video surveillance. After each session, subjects were asked to rate how often they thought to have chosen the risky option using a visual analogue scale, ranging from “never risky” to “always risky”. To ensure that decisions were taken in an incentive-compatible fashion, we randomly selected one trial at the end of some sessions (determined by coin flip) and played out the trial according to the

subject's decision (for risky options a lottery with the given probability was played by open dice toss).

The total risk premium was calculated for each session as the main outcome measure of a subject's risk-preference. It indicates by how many percent a subject undervalues a risky option compared to its objective expected value (EV). The risk premium of a specific probability was defined in % as (EV - CE) / EV * 100, where the certainty equivalent (CE) is the amount of money that a subject treated as equally desirable as the risky option. We calculated the CE by sorting trials of each probability level according to their certain amount (ascending) and determining the choice reversal point at which subjects switched from the probabilistic option to the certain alternative. If a subject's behavior is risk-neutral then CE equals EV, resulting in a risk premium of 0%. Risk aversion is expressed as a positive value, reflected by a smaller CE than EV. Conversely, risk-seeking is expressed as a negative value, reflected by a larger CE than EV. Thus, the total risk premium refers to the extent of "irrational" risk-taking behavior, either in terms of not choosing risky options despite higher EVs (risk-averse) or choosing risky options despite lower EVs (risk-seeking) compared to offered amount in the safe option.

We assessed the consistency of observed choices with the inferred risk-preference by counting how often the subject's choices were inconsistent with the estimated CE. The number of deviations (x) was transformed by $\sqrt{x} + \sqrt{(x+1)}$ to approximate a normal distribution of the data (analogue to the transformed frequency of lapses in vigilance – see below).

To evaluate potential repetition effects, we administered the risk task repeatedly to a task validation group (another group of male subjects) at the same time points as during SR in the main experiment (6 repetitions in total over the course of 8 days, cf. Fig 1B) but while adhering to a regular sleep-wake rhythm of 8 hours per night. We excluded two subjects from

the task validation group, one due to acute illness and one due to missing the last assessment. We found that the risk premium was different from the following assessments up to the third repetition of the task and was stable afterwards in the task validation group when looking at the single assessments. When investigating the change of averages across two test sessions – equivalent to the two test sessions per condition in the main experiment – only the average over the first two assessments was significantly different from the following ones (Fig 1C). Results were comparable when examining the first five assessments including the subject in which data for the sixth assessment was missing (data not shown). As a result, we included two habituation test sessions in the main experiment before starting the first sleep manipulation (either before SR or SD, Fig 1B). Along the counter-balanced design, half of the subjects in the experiment performed SR before RegS followed by SD and the other half performed first SD before RegS followed by SR (cf. Fig 1B), allowing also to test for potential order effects.

Assessment of Vigilance and Sleepiness

To exclude that changes in risk-preferences are merely a result of impaired vigilance we also acquired the psychomotor vigilance test[31] (PVT-192, Ambulatory Monitoring Inc.). The PVT is a sustained visual vigilance reaction-time task lasting ten minutes. We investigated the number of lapses which corresponds to trials for which the subject was not able to respond within 500 ms. The number of lapses (x) was transformed by $\sqrt{x} + \sqrt{(x+1)}$ to approximate normal distribution of the data[32]. In addition, we assessed subjective excessive daytime sleepiness before and after the seven nights of SR with the Epworth sleepiness scale.

Assessment of Sleep

Sleep was recorded in the sleep laboratory at the Department of Neurology, University Hospital Zurich, using a high-density EEG net (Electrical Geodesics Inc. Sensor Net for long-term monitoring) consisting of 128 electrodes. Impedances of all electrodes were kept below 50 kΩ, and data was recorded with a sampling rate of 500 Hz. Offline data processing, including filtering (0.5 Hz high-pass, 40 Hz low-pass filter), artefact and bad quality channel rejection, sleep stage scoring, re-referencing of data to the average of all electrodes, spectral analysis and interpolation of previously excluded channels was conducted in MATLAB as reported previously (e.g.[33]). We quantified the extent of slow waves during sleep by slow wave energy (SWE), i.e., the summed the whole-night mean spectral power in the range of 0.75 – 4.5 Hz of all artefact-free NREM 2 and NREM 3 epochs. As in previous studies assessing topographical differences[23, 33], we normalized the obtained power values resulting in individual topographical distributions indicating relative SWE values at every electrode (expressed as % of average SWE over all electrodes of a subject). Without the excluded channels below the ears (which were excluded from further analysis to avoid artefacts induced by facial and neck muscles), our topography finally consisted of 109 electrodes.

Statistical Analysis

We performed a mixed analysis of variance (ANOVA) for the risk premium and subjective risk-taking measures after testing for normal distribution (Shapiro-Wilk test) with within-subject factors condition (RegS, SR, SD) and daily time point (1, 2) and the between-subject factor order (SD-RegS-SR, SR-RegS-SD) to test for any potential interactions. As our main interest was to contrast the two sleep conditions to the condition of RegS, we performed a-priori defined simple contrasts, comparing the SR and SD conditions to RegS, in case of a significant main or interaction effect. We excluded one subject from the analysis of subjective

risk-taking measures due to missing data in the SD condition (order of conditions in this subject: SD, RegS, SR). However, results were not different for SR and RegS when all subjects were included (data not shown).

For the consistency of choices in the risk-task and the lapses in the PVT, we performed a repeated-measure Friedman's ANOVA with all six assessments (both time points at RegS, SR and SD) as factor levels since the variables were not normally distributed in at least one condition even after transformation. To stay consistent with the parametric procedure, we performed a-priori defined post-hoc comparisons according to the contrasts in the parametric procedure with the Wilcoxon Signed Ranks Test. Having only one factor did not allow us to restrict the number of post-hoc comparisons in case of a significant result in Friedman's ANOVA. Hence, in this case we controlled for multiple testing by applying Bonferroni correction to *P*-values (multiplying the *P*-values by 5, corresponding to the number of performed post-hoc comparisons). Correspondingly, we calculated 99% confidence intervals (CI) for non-parametric comparisons (while in comparisons without Bonferroni correction of *P*-values, 95% CI are reported). To test for any order effects, we performed a post-hoc between-group comparison on the above mentioned differences using the Mann-Whitney U-test with the same Bonferroni correction.

Frequency distributions across categories were compared using Pearson's chi-square test and related changes with McNemar's test.Relationships between measures were assessed by Pearson's correlation coefficients, which were tested for significance by permutation testing (5000 permutations of the subject order in the data) whenever one variable were not normally distributed. Electrodes were only considered significant when forming a cluster of at least 5 neighboring electrodes for SWE data (as with 109 electrodes tested and an alpha level of 0.05 this number corresponds to the number of expected false-positives). By restricting the possible location of the electrodes, the actual probability of false-positives is much lower.

All statistical analyses were performed using SPSS (IBM SPSS Statistics 22.0) or MATLAB (R2014a).

Results

Effects of SR and SD on Risk-Preference

The mixed analysis of variance (ANOVA) for the risk-preferences revealed a significant effect for the factor sleep condition only (F (2, 24) = 3.72, P = 0.04, η_p^2 = 0.24, all other factors and interactions: all P > 0.25, η_p^2 < 0.12). Planned contrasts comparing risk-preferences both after 7 nights of SR and after SD to RegS revealed that only SR resulted in significantly increased risk-seeking (Table 1, Fig 2A). Most subjects were risk-aversive after RegS, which is in agreement with the well-documented risk-avoidance for gains[34] (Fig 2B). This was no longer observed after SR. Compared to RegS, the clear majority of subjects, i.e., 11 out of 14 showed an increase in risk-seeking after SR (Fig 2B), and 6 of them changed from being risk-aversive after RegS to being risk-seeking after SR (red dots in Fig 2B; risk premium in those subjects after RegS: 6.3 ± 3.3%, after SR: -12.3 ± 6.2%, difference from RegS to SR: -18.6 ± 6.4%). That is, subjects moved towards a more risk-seeking attitude after SR. The extent of increase in risk-seeking after SR was independent of the individual baseline level of risk-preference after RegS (r (12) = 0.04, P = 0.90, two-sided). This means that both risk-averse and risk-seeking subjects increased their preference for risk after SR. Furthermore, individual preferences after SR were not closer to risk-neutrality (i.e., zero) than after RegS (absolute difference to risk-neutrality after RegS: 14.4 ± 4.0%, after SR: 17.5 ± 4.2%; difference from RegS to SR: +3.2 ± 3.8%, t (13) = -0.82, P = 0.43, two-sided paired-samples t-test, r = 0.22, CI (-11.4%, 5.1%)). Hence, SR does not lead to risk-neutral behavior but rather to a significant increase in risk-seeking. Assessing the subjective ratings of risk-taking behavior revealed that participants were not aware of their increased risk-seeking after SR, as

the mixed ANOVA showed no significant effect for either factor (all $P > 0.26$, $\eta_p^2 < 0.13$, Table 1, Fig 2C). Hence, altered risk-taking behavior did not reflect a conscious intentional reaction to the SR and was not even perceived by the subjects.

Effects of SR and SD on Vigilance and Consistency of Choices

After chronic SR, subjective excessive daytime sleepiness as assessed with the Epworth sleepiness scale increased markedly compared to pre-SR conditions (median [interquartile range] pre-SR: 5.5 [3.8, 7.3], after SR: 10.5 [6.8, 13.5], z = -3.1, median difference from pre-SR to after SR: +4.5, $P = 0.002$, two-sided, $r = 0.59$, CI (2.0, 7.0)).

Following a significant result in Friedman's ANOVA for the number of lapses in the PVT (χ^2 (5) = 35.94, $P = 0.000$), planned post-hoc comparisons revealed that sustained attention was only significantly impaired after SD but not after SR (Table 1, Fig 3A; all other planned comparisons were non-significant: all $P > 0.55$, $r < 0.30$). The impairment in vigilance was further not associated with changes in risk-preferences after SR or SD (SR: r (12) = 0.19, $P = 0.51$; SD: r (12) = -0.04, $P = 0.87$, both P-values: two-sided, permutation testing). Parallel to vigilance, we evaluated how consistent the choices in the risk task were with the inferred risk-preference (i.e., how stable the estimated risk-preferences were expressed across trials for each subject). Following a significant result in Friedman's ANOVA (χ^2 (5) = 24.50, $P = 0.000$), post-hoc comparisons revealed that choice consistency was significantly reduced after SD but no significant change was observed after SR (Fig 3B, Table 1; all other planned comparisons were non-significant: all $P > 0.07$, $r < 0.47$). Furthermore, no association between changes in consistency with changes in risk-preferences was detected (SR: r (12) = -0.24, $P = 0.41$; SD: r (12) = -0.31, $P = 0.26$, both P-values: two-sided, permutation testing). This again indicates that SR merely affected risk-preferences but not general behavioral variability that may reflect attentional lapses.

Association of Changes in Behavior with the Restorative Function of Sleep

To examine whether the observed significant shift towards risk-seeking was associated with differences in local sleep intensity we correlated all-night SWE during the last night of SR (Fig 4A) with the increase in risk-seeking after SR (risk premium after RegS minus risk premium after SR). Production of less SWE in a right prefrontal cluster of 6 electrodes was linked to increased risk-seeking the following day (Fig 4B). Low levels of right prefrontal SWE were highly specific for predicting an increase in risk-seeking after SR (Fig 4C).

Discussion

This study provides first-time evidence that chronic SR as it occurs in the prevalent insufficient sleep syndrome negatively affects decision-making behavior in association with locally decreased sleep restoration over circumscribed cortical areas. In chronically sleep restricted subjects, low slow wave sleep intensity over the right prefrontal cortex - which has been shown to be linked to risk behavior – may lead to increased and subjectively unnoticed financial risk-seeking - while no major effects on vigilance or the consistency of choices with the subject's risk-preference were detected. On the other hand, SD did not significantly affect a subject's preference for risk but lead to reduced choice consistency paralleled by significantly impaired vigilance. While these latter effects of SD are in line with previous results[18-20] and may mostly reflect behavioral instability, the divergent findings imply that not all aspects of acute SD can be generalized to other forms of sleep loss such as chronic SR. The increase in risk-seeking after SR was subjectively not noticed, indicating a misperception of altered decision-making, which fits the previously reported underestimation of cognitive

impairments after SR[10]. Up to date, only one study investigated impulsive-risk taking after mild chronic sleep restriction[35], but found no effects on risk-taking, yet on impulsive action. However, it has been shown before that sleep deprivation leads to increased effort discounting[36], which can mask effects on risk-preferences when no forced choice paradigm is used, as it is the case in the task applied in that study, i.e., in the balloon analogue risk task (BART)[37]. Furthermore, the study did not include any physiological brain measures - including sleep parameters per se – which made it impossible to investigate by what mechanisms chronic sleep loss might have altered decision-making. Considering that the cause for the insufficient sleep syndrome, i.e., chronic SR is a highly prevalent condition in modern societies, with 30% or more of the population in various countries reporting inadequate sleep durations[4-6], our findings further emphasize the importance of investigating the effects of chronic SR.

We found that lower SWE over the rPFC during SR relate systematically to the increase in risk-seeking after SR. Hypo-activity of the rPFC during rest is a dispositional indicator of risk-seeking[38]. Hence, insufficient restoration during chronic SR might have an impact on rPFC function, resulting in effects similar to those of experimental rPFC disruption[26]. While it has been shown before that consequent increases in slow waves are linked to plastic processes occurring during learning[39], we show here that the local extent of slow waves during restricted sleep periods is linked to sleep-loss induced changes in behavior. To the best of our knowledge, such a direct link between local electrophysiological correlates of the restorative function of sleep and changes in behavior resulting from insufficient sleep has not been shown before.

Slow waves result from synchronous activity of neuronal populations[40, 41]. The intensity of slow waves varies due to differences in strengths of connections or their relative regional density[22]. The topography of slow waves is highly stable within an individual and

thus is thought to reflect individual traits of functional anatomy[23]. Hence, SWE might not only indicate how much restoration is obtained during the night but could also reflect stable degrees of a brain structure's functional integrity, with higher levels of integrity making a structure more robust against function deterioration when challenged by SR. Future studies manipulating slow waves in the rPFC could help to determine whether functional deterioration after sleep loss is determined by the extent of sleep-related restoration or by more stable neuro-anatomical differences.

Our results imply that the insufficient sleep syndrome might come along with further, until now unrecognized alterations of behavior, beside the defining and up-to now identified secondary symptoms, like for example dysphoria or reduced motivation[3], with potentially negative consequences. As the current findings are based on a carefully selected study sample including only healthy, young men, future studies are needed to assess whether the findings generalize to other parts of the population. A further increase in risk-seeking due to insufficient sleep might be particularly problematic in individuals who already have a high preference for risk per se. While over 70% of business students report to obtain subjectively insufficient or highly insufficient sleep[42], they are also more risk-seeking than the general public[43] and the economic crisis in 2008 is at least partly attributed to risky business decisions[12]. Nevertheless, the prevalence of short sleep durations due to extended working hours and commute time is increasing in modern society[6, 8, 44]. An alarming additional result is our observation that sleep-restricted subjects do not notice their increased drive for risk-taking. While we cannot exclude that individuals in positions that require high-impact decision-making may be more resilient to the effects of sleep restriction, our results suggest that all of us, but particularly leaders of companies and countries, are well advised to work and make decisions only when fully sleep-satiated.

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Author contributions

Study concept and design: CRB, RP, RH, AM.

Data acquisition and analysis: AM, EM, JL, MS, CCR, SW, RP, EW.

Drafting the manuscript and figures: AM, CRB, RP, RH, SW, CCR.

Conflicts of Interest

The authors declare no competing financial interests.

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Figure 1: Study Setup. (**A**) Schematic study design. Risk-taking behavior was assessed in the same subjects after 7 nights of regular sleep (8 hours in bed per night) and compared to behavior after 7 nights of sleep restriction (5 hours in bed per night), and after a night of sleep deprivation (one night without sleep). High-density EEG recordings were performed during sleep. In the risk task subjects had to choose between two options represented by two bars (in this example left reflects the safe and right the risky option). Numbers in the bar: money value in Swiss Francs. Green bar height: probability level of receiving the money. Red bar height: probability level of getting nothing (0.00). When the right bar was chosen there was a 40% chance to win 50 Swiss Francs and a 60% chance of getting nothing, when the left green bar was chosen the chance was 100% to receive 24.35 Swiss Francs in this trial. (**B**) Detailed study design. Regular sleep (RegS, green), sleep restriction (SR, orange) and sleep deprivation (SD, blue) in a counterbalanced cross-over design (upper and lower line show the two possible sequences of conditions), with repeated behavioral tests (red and pink rectangles). Black bars: nights with 8h (broad bars) and 5h (thin bars) time in bed. H (gray): assessments performed to habituate a subject to the tests. Red rectangles: data points analyzed in this manuscript, including 2 assessments per day (approx. 2pm and 7.30pm) after the last night of SR, during SD and RegS. Pink rectangles: data points not included in the analysis. (**C**) Risk premium values over six repeated assessments for task validation (n = 12, means ± s.e.m.). Bars indicate means ± s.e.m. The single assessments are shown in dark grey, the average over two assessments in light grey. Repeated-measure analysis of variance (ANOVA) across all assessments and the average over two assessments was significant in both cases (Assessment 1 to 6: F (2.91, 32.02) = 13.87, P = 0.000; Average over two sessions (3 averages): F (1.47, 16.19) = 16.80, P = 0.000; Huynh-Feldt correction for degrees of freedom). Planned contrasts (Helmert) were performed for all assessments (first to following: F (1, 11) = 23.40, P = 0.001, second to following: F (1, 11) = 9.56, P = 0.01, third to following: F (1, 11) = 10.73, P = 0.01, fourth to following: F (1, 11) = 0.28, P = 0.61.; fifth to

sixth: F (1, 11) = 0.004, P = 0.95) and for the averages across two assessments (first to following: F (1, 11) = 19.68, P = 0.001, second to third: F (1, 11) = 0.84, P = 0.38).

Figure 2: Changes in risk-taking behavior following sleep restriction (SR) and (SD) compared to regular sleep (RegS). (**A**) Changes in risk-preferences. * $P < 0.05$; n.s.: $P > 0.05$, two-sided. For exact test values see table 1. (**B**) Difference plot of risk premium values after RegS and after SR. Deviation from the diagonal indicates the amount and direction of change in each subject. Different colors indicate whether a subject was risk-aversive or risk-seeking after RegS and after SR: The majority of subjects was risk-aversive after RegS (red and orange dots; χ^2 (1) = 4.57, $P = 0.03$, sample proportion ± s.e. = 0.79 ± 0.11, CI (0.57, 1.00)) but not after SR (orange and purple dots; χ^2 (1) = 0.29, $P = 0.59$, sample proportion ± s.e. = 0.43 ± 0.13, CI (0.17, 0.69), change of proportion from RegS to SR: χ^2 (1) = 2.29, $P = 0.13$, difference in sample proportion ± s.e. = -0.35 ± 0.22, CI (-0.78, 0.07)). Compared to RegS, the clear majority of subjects showed an increase in risk-seeking after SR (points above diagonal; χ^2 (1) = 4.57, $P = 0.03$, sample proportion ± s.e.m. = 0.79 ± 0.11, CI (0.57, 1.00)). (**C**) Changes in subjectively reported percentage of choosing the probabilistic option. No planned contrasts performed. Zero represents no change compared to RegS, bars indicate means ± s.e.m.

Figure 3: Changes in vigilance and choice consistency following sleep restriction (SR) and sleep deprivation (SD) compared to regular sleep (RegS). (**A**) Vigilance, i.e., transformed number of lapses in the psychomotor vigilance test (PVT). (**B**) Consistency of choices over all trials, i.e., transformed number of trials in which the choice deviated from the inferred risk-preference. Box plots indicate medians (horizontal line), upper and lower quartiles (box), and extrema (whiskers); outliers are shown as black dots. *$P < 0.05$, n.s.: $P > 0.05$, two-sided testing with post-hoc Bonferroni correction. For exact test statistics see Table 1.

Figure 4: Slow wave energy (SWE) topography and the correlation of SWE with the increase in risk-seeking after sleep restriction (SR) relative to regular sleep (RegS). (**A**) Whole-night SWE topography (average of all subjects) during the last night of SR. SWE values at every electrode were normalized in relation to average SWE over all electrodes of a subject. Dark blue to dark red colors indicate minimal (42%) to maximal (170%) SWE. (**B**) Topographical distribution of Pearson's correlation coefficients between normalized SWE during the last night of SR and the increase in risk-seeking after SR. White dots: significant cluster of electrodes (all $P < 0.05$, permutation testing, two-sided). (**C**) Relationship between averaged SWE in the cluster and the increase in risk-seeking after SR (r (12) = -0.76, P = 0.001, Pearson's correlation coefficient, permutation testing, two-sided). Dotted lines: no change in risk-seeking or average SWE (relative to the other electrodes of a subject). Red: subjects with increased risk-seeking and below average SWE. Blue: subjects without increased risk-seeking and above average SWE.

Table 1: Assessed variables and statistical comparisons.

	Mean ± s.e.m			Mean difference ± s.e.m. and planned contrasts	
	RegS	SR	SD	SR vs. RegS	SD vs. RegS
Risk premium	7.8 ± 5.2%	-2.3 ± 6.4%	7.6 ± 5.5%	**-10.1 ± 4.1%** F(1, 12) = 6.04 **P = 0.03** r = 0.58 CI[a] (1.1, 19.1)	-0.2 ± 3.0% F(1, 12) = 0.01 P = 0.94 r = 0.02 CI[a] (-6.3, 6.8)
Subjective risk frequency	60.0 ± 5.3%	60. 5 ± 3.7%	58.5 ± 4.2%	+0.5 ± 5.2% CI[a] (-10.9, 11.8)	-1.5 ± 3.2% CI[a] (-8.4, 5.4)
	Median [interquartile range]			Median difference and planned comparisons	
	RegS	SR	SD	SR vs. RegS	SD vs. RegS
PVT lapses	2.0 [1.0, 2.5]	2.1 [1.5, 5.2]	4.5 [2.8, 7.4]	+ 1.1 z = -1.87 P = 0.31[c] r = 0.35 CI[b] (-0.7, 3.6)	**+ 3.1** z = -3.18 **P = 0.01**[c] r = 0.60 CI[b] (1.3, 5.2)
Choice consistency	10.2 [8.2, 11.6]	10.9 [10.1, 12.0]	12.4 [10.0, 15.3]	+ 1.2 z = -2.10 P = 0.18[c] r = 0.40 CI[b] (-0.4, 2.9)	**+ 2.6** z = -2.92 **P = 0.02**[c] r = 0.55 CI[b] (0.8, 4.8)

RegS: regular sleep, SR: sleep restriction, SD: sleep deprivation, PVT: psychomotor vigilance test, [a] 95% confidence interval (CI), [b] 99% CI, [c] Bonferroni corrected P-values; All P-values are based on two-sided testing, bold values indicate *P*-values < 0.05. Planned simple contrasts were conducted following a significant effect in the mixed analysis of variance (ANOVA). Planned comparisons, i.e., Wilcoxon Signed Ranks Tests, were performed for non-normally distributed data following a significant result in a repeated-measures Friedman's ANOVA.