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# Local slow-wave activity over the right prefrontal cortex reveals individual risk preferences

Mirjam Studler<sup>a\*</sup>, Lorena R.R. Gianotti<sup>a\*</sup>, Katharina Koch<sup>a</sup>, Jan Hausfeld<sup>a,b</sup>, Leila Tarokh<sup>c,d</sup>,  
Angelina Maric<sup>e</sup>, Daria Knoch<sup>a</sup>

<sup>a</sup>Department of Social Neuroscience and Social Psychology, Institute of Psychology,  
University of Bern, Fabrikstrasse 8, 3012 Bern, Switzerland

<sup>b</sup>CREED, Faculty of Economics and Business, University of Amsterdam, Postbus 15867,  
1001 NJ Amsterdam, Netherlands

<sup>c</sup>University Hospital of Child and Adolescent Psychiatry and Psychotherapy, University of  
Bern, Bolligenstrasse 111, 3000 Bern, Switzerland

<sup>d</sup>Translational Research Center, University Hospital of Psychiatry and Psychotherapy,  
University of Bern, Bolligenstrasse 111, 3000 Bern, Switzerland

<sup>e</sup>Department of Neurology, University Hospital Zurich, University of Zurich,  
Frauenklinikstrasse 26, 8091 Zürich, Switzerland

\*Both authors contributed equally to this work

Corresponding Authors:

Daria Knoch  
Department of Social Neuroscience and  
Social Psychology  
Institute of Psychology  
University of Bern  
Fabrikstrasse 8  
CH-3012 Bern  
daria.knoch@unibe.ch  
Telephone: +41 31 684 5432

Lorena R.R. Gianotti  
Department of Social Neuroscience and  
Social Psychology  
Institute of Psychology  
University of Bern  
Fabrikstrasse 8  
CH-3012 Bern  
lorena.gianotti@unibe.ch  
Telephone: +41 31 684 4043

## Highlights

- Portable high-density EEG is performed at participants' home from a group of healthy good sleepers.
- Individual fingerprints in deep sleep EEG topography relate to individual differences in risk preferences.
- Lower slow-wave activity over the right prefrontal cortex is associated with higher individual risk propensity.
- Slow-wave activity over the right prefrontal cortex might serve as a dispositional indicator of self-regulatory ability.

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**Abstract**

In everyday life, we have to make decisions under varying degrees of risk. Even though previous research has shown that the manipulation of sleep affects risky decision-making, it remains unknown whether individual, temporally stable neural sleep characteristics relate to individual differences in risk preferences. Here, we collected sleep data under normal conditions in fifty-four healthy adults using a portable high-density EEG at participants' home. Whole-brain corrected for multiple testing, we found that lower slow-wave activity (SWA, an indicator of sleep depth) in a cluster of electrodes over the right prefrontal cortex is associated with higher individual risk propensity. Importantly, the association between local sleep depth and risk preferences remained significant when controlling for total sleep time and for time spent in deep sleep, i.e., sleep stages N2 and N3. Moreover, the association between risk preferences and SWA over the right prefrontal cortex was very similar in all sleep cycles. Because the right prefrontal cortex plays a central role in cognitive control functions, we speculate that local sleep depth in this area, as reflected by SWA, might serve as a dispositional indicator of self-regulatory ability, which in turn reflects risk preferences.

Keywords: Risk preferences, sleep, slow-wave activity, prefrontal cortex, neural trait, individual differences.

## 1. Introduction

There is abundant evidence that characteristic electroencephalographic (EEG) oscillations of the sleeping brain show large inter-individual variation and remarkable trait-like stability within an individual across nights (Botella-Soler et al., 2012; Kerkhof & Lancel, 1991; Ong et al., 2019; ). This is, for example, the case for slow-wave activity (SWA), a measure used to quantify the occurrence of slow waves, which form the major EEG hallmark of deep sleep. Therefore, SWA is generally recognized as a physiological marker for sleep depth. SWA is homeostatically regulated, reflecting the changes in sleep pressure resulting from previous sleep-wake history (Borbély & Achermann, 1999). However, even when considering the relatively large increases in SWA following sleep deprivation, the variance in SWA levels between individuals is larger (De Gennaro et al., 2005; Gander et al., 2010; Tarokh et al., 2015). Interestingly, the topographic distribution of SWA shows local differences, varies between individuals (Finelli et al., 2001), and is therefore unique to each person (Markovic et al., 2018; Rusterholz & Achermann, 2011). These trait-like characteristics of the sleeping brain correlate with individual differences in cognitive performance (e.g. Anderson & Horne, 2003; Walker, 2009; Wilckens, Hall, et al., 2016). However, the current literature on trait-like features of sleep neurophysiology and cognitive performance has mainly focused on the role of sleep in sustained attention and vigilance, learning and memory, and executive functions (Lowe et al., 2017), but has largely neglected a very important function for successful navigation in daily life, namely functional decision-making.

A particularly important type of decision-making concerns decisions made under risk. In our daily life, we constantly face situations where we have to decide between options with different levels of risk (Reyna & Zayas, 2014). Recent work suggests that risk preference is a

stable individual trait (Frey et al., 2017) that varies greatly between individuals (Mata et al., 2016; Mishra & Lalumière, 2011).

Past studies on the association between sleep and risky decision-making have primarily used sleep deprivation (i.e., complete sleep loss) and sleep restriction to modulate sleep quantity (i.e., total sleep time), revealing that sleep loss increases risky decisions and leads to suboptimal decision-making (Killgore et al., 2006, 2012; Venkatraman et al., 2011). Studies on the effects of sleep loss and risky decision-making provide much information about the effects of artificially limited sleep on behaviour. However, complete sleep loss is rather rare in our everyday life. There are a few studies that investigate the effects of partial and not total sleep deprivation on risky decision-making (e.g., Maric et al., 2017; Salfi et al., 2020). Maric et al. (2017) examined the effects of chronic sleep restriction (7 nights of 5 hours time in bed) on the topographic distribution of slow-wave oscillations and on risk-taking behaviour. The authors report increased risk-taking after sleep restriction as compared to baseline values. Interestingly, individuals who showed the greatest increase in risk-taking behaviour following sleep restriction manifested the lowest normalized SWA over a right prefrontal (PFC) cluster of electrodes. One unresolved question is, however, whether the topographic distribution of SWA explains the variance in risk preference without experimental manipulation of sleep duration (i.e., without sleep deprivation or sleep restriction). Thus, this study aims to assess the topographic distribution of SWA under normal sleep conditions (i.e., habitual sleep of 7-8 hours per night) and link these individual differences to risk preferences. Based on Maric et al.'s results (2017), we hypothesized that lower SWA in the right PFC is associated with increased risk-taking behaviour. Further support for the relevance of the right PFC in explaining interindividual differences in risk preferences stems from studies using a

neural trait approach during wakefulness, namely resting-state EEG (Gianotti et al., 2009; Studer et al., 2013).

## 2. Material and Methods

### 2.1 Participants

We performed polysomnography (PSG) at participants' home from self-reported good sleepers with a habitual sleep duration of 7-8 hours per night and measured risk-taking in our laboratory. An *a priori* power analysis was conducted with G\*Power 3.1.9.7 (*F* tests, Linear multiple regression, Fixed model,  $R^2$  deviation from zero; Faul et al., 2007) based on the criterion of  $\alpha = 0.005$ ; effect size  $f^2 = 0.25$ . The effect size was estimated according to relevant previous studies on neural traits and economic preferences (Baumgartner et al., 2013; Gianotti et al., 2009, 2018; Knoch et al., 2010). The power analysis indicated that 58 participants in total would ensure 80% statistical power. We recruited 60 healthy participants and excluded five of them because of non-compliance with the study protocol (see Procedure) and one because they did not understand the behavioural task. The remaining 54 participants (42 females) were 21.11 years old (SD = 2.04 years). All participants gave written informed consent and were informed of their right to discontinue participation at any time. Participants received 160 Swiss francs (CHF 1  $\approx$  USD 1) for participating, in addition to the money earned in the risk-taking task. This experiment is part of a bigger study, which was approved by the local ethics committee and conducted according to the principles expressed in the Declaration of Helsinki.

## 2.2 Procedure

Before the experiment, we performed a detailed screening for inclusion criteria. All participants fulfilled the following inclusion criteria: self-reported good sleepers with a habitual sleep duration of 7-8 hours per night (Pittsburgh Sleep Quality Index  $< 5$ ; Buysse et al., 1989), normal sleepiness index (Epworth Sleepiness Scale  $< 10$ ; Johns, 1991), no extreme chronotype (Munich Chronotype Questionnaire  $> 2$  &  $< 7$ ; Roenneberg et al., 2003), no current or past history of neurological, psychiatric, or sleep disorders, no drug nor alcohol abuse, no regular medication intake, normal weight, and no traveling across more than two time-zone within the last 30 days before the experiment. Additionally, we queried participants about their regular caffeine, alcohol, and nicotine consumption. As women's risk-taking behaviour (e.g., Bröder & Hohmann, 2003) and sleep quality (e.g., Baker & Driver, 2004) might be influenced by their menstrual cycle phase, we assessed this for each of the subjects using the forward counting method. Naturally cycling women were not invited during their fertile days and during the first 2 days of their menstruation. Women on hormonal contraception were not invited during their pill-free intervals.

A week before the experiment took place, participants came to our laboratory to receive detailed study instructions. We told participants to keep a regular sleep-wake rhythm adjusted to their habitual bedtimes (sleep duration of 7-8 hours). Daytime napping was not allowed throughout the week. Participants were also told to limit their caffeine consumption to two units/day (1 unit = caffeine content of one cup of coffee) and their alcohol consumption to one standard drink/day (1 standard drink = 1 beer (350ml) = 10g ethanol). Smokers were asked to not change their habitual nicotine consumption. Each participant was given a tri-axial accelerometer (GENEActiv, activinsights Ltd., Kimbolton, Huntingdon, UK) to wear on their non-dominant hand. Actigraphy is a validated objective measure of sleep behaviour



(e.g., de Souza et al., 2003; Marino et al., 2013) and delineates sleep from waking based on motion and was used to confirm adherence to the study protocol. Since it was necessary to make sure that participants did not remove the actigraph and give it to another person to circumvent the study protocol rules, we used single-use straps. Sleep diaries and consumption diaries were also used to confirm adherence to the study protocol. Additionally, participants were given a chest harness with a sham amplifier to simulate the wearing of the mobile PSG system. As the amplifier can be attached to the harness at different positions, we asked participants to sleep with the chest harness and the sham amplifier to find the optimal amplifier position for the recording night.

On the day of the experiment, participants were asked to refrain from extensive exercise or visiting the sauna to avoid post sweating. The experiments started at 4.30 pm in our behavioural laboratory with the collection of the behavioural data, followed by hook up of the portable PSG system. Participants then went home and continued with their habitual routine. Shortly before bedtime, experimenters visited participants at home to check and, if needed, correct the impedances of the electrodes, and start the recording.

### ***2.3 Risk-taking task***

Risk-taking was assessed using a newly developed task implemented in Z-tree (Fischbacher, 2007). In this task, participants were asked to decide how many meters they want to drive with a toy car on a 50 meter road. Every meter driven earned them additional 0.1 MU (1 MU = CHF 1). However, participants were also aware that a wall will appear at a random distance on the road (somewhere between 0 and 50 meters). This means that there are 51 different positions of where the wall will be placed each with a chance of 1/51. If participants chose to drive more meters than the distance to the wall, the car crashed and

participants lost both the money earned for the meters driven (0.1 MU per meter) and an initial endowment of 5 MU, resulting in 0 MUs payment for this task. The payoff-relevant decision was made by stating the meters they want to drive. The payoffs for any meter  $x$ , which is between 0 and 50, is as follows:

$\frac{51-x}{51}$  chance of earning  $(x * 0.1 + 5)$  MUs, and  $\frac{x}{51}$  chance of earning 0 MUs.

For example, a participant choosing 0 meters, will receive 5 MUs for sure. In comparison, a participant choosing 50 meters, has a  $1/51$  chance to receive  $50 * 0.1 + 5$  MUs, and a  $50/51$  chance to receive 0 MUs. Thus, driving 50 meters without hitting the wall would result in 10 MUs, which doubles the initial 5 MUs.

Risk-taking behaviour was operationalized as the number of driven meters. Every additional meter can yield more MUs, but comes at the risk of not receiving any MU. An expected-value maximizer would choose to drive either 0 or 1 meters in our task. Therefore, more meters driven imply a higher willingness to take risks.

This task bears some resemblance to the Balloon Analogue Risk Task (BART, Lejuez et al., 2002): Every additional action (meter/pump) increases the payoff, but comes at the risk of losing all. In comparison to the BART-task, our task involves a single choice capturing risk preferences which is not conditional on where the wall is being placed, i.e., participants can state to drive 40 meters even if the wall is placed at 20 meters. In other risk tasks, such as the BART, the number of pumps is limited by whether the balloon exploded. Because of this truncation in the data, only the average number of pumps of a large sample of choices can be used as indicator of risk preferences. Avoiding the truncation of the data, as in our risk task, guarantees the possibility to play it one-shot.

Since this task was new for the participants, they had the possibility to use a simulator for 2 minutes. Here, they could do the task as often as they wanted without any monetary consequences.

#### **2.4 Polysomnography**

High-density portable EEG (61 electrodes), electrooculogram, and submental electromyogram were continuously recorded during the nighttime sleep episode. The signals were recorded with a sampling rate of 500Hz (third order low-pass filter at 131Hz). The electrode at the position FCz was used as recording reference and the electrode at position CPz served as ground. Impedances were kept below 25k $\Omega$ . Data were offline bandpass filtered between 0.5-40 Hz and down-sampled to 250 Hz. For each participant, lights-off and wake-up times were determined according to his or her habitual sleep time.

Sleep was visually scored according to standard criteria (Berry et al., 2018). The following sleep parameters were extracted from sleep stage scoring: total sleep time (i.e., the objective sleep quantity), sleep efficiency (proportion of total time in bed spent asleep), sleep latency (time from lights out to sleep onset), wake after sleep onset (length of periods of wakefulness occurring after sleep onset), percentage of total sleep time spent in each sleep stage (N1, N2, N3 and REM). Sleep cycles were defined according to an adaptation of Feinberg and Floyd's criteria (Feinberg & Floyd, 1979; Jenni & Carskadon, 2004; Kurth et al., 2010).

Bad channels were individually identified by visual inspection of the spectrograms. Power density spectra were calculated for 30-s epochs using Fast Fourier Transformation (5-s subepochs, Hanning window, no overlap). Artifacts were excluded semi-automatically, whenever power exceeded a threshold based on a moving average over epochs for the frequency bands 0.8-4.6 and 20-40 Hz (Buckelmüller et al., 2006). Data were re-referenced to

the average reference. Slow-wave activity (SWA) in the range between 0.8-4.6 Hz in sleep stages N2 and N3 was computed for further analyses. To reduce confounds without regional specificity, individual SWA distribution maps were normalized to the mean values across all electrodes before statistical analyses, yielding normalized SWA distribution maps (e.g., Finelli et al., 2001).

### *2.5 Statistics*

The goal of this study was to assess whether individual differences in the local distribution of SWA during a night of sleep under normal conditions explain differences in risk preferences. To achieve this goal, we computed Spearman's rank order correlations between normalized SWA distribution map and risk-taking. To correct for multiple comparisons, statistical nonparametric mapping (SnPM) using a suprathreshold cluster analysis was applied (Huber et al., 2004; Nichols & Holmes, 2001). For each permutation, the maximal cluster size of neighboring electrodes reaching an  $r$  value above the critical value was counted and used to build a cluster size distribution. The 95<sup>th</sup> percentile was defined as the critical cluster size threshold. All statistical analyses were performed with the software packages MATLAB (Mathworks) and R.

## **3. Results**

On average, participants drove 16.48 meters ( $SD = 9.70$ , range: 0-40). As illustrated in **Figure 1** they showed large inter-individual variability. Furthermore, sleep parameters were within the expected range for this age group (see **Table 1**).

----- Figure 1 -----

----- Table 1 -----

We then investigated whether individual differences in the local distribution of SWA (see **Figure 2A**) during a night of sleep under normal conditions explain individual differences in risk preferences. We found robust and significant negative associations in a cluster of five electrodes placed on the right PFC (Fp2, AF8, AF4, F6, F4,  $p < 0.05$ , corrected for multiple testing, see **Figure 2B**). The correlation between mean SWA in the significant cluster and risk-taking behaviour resulted in a rho-correlation coefficient of  $-0.38$  ( $df = 52$ ),  $p = 0.004$ ,  $R^2 = 0.14$  (see **Figure 2C**). Removing one participant with a value of 2.99 in the mean SWA in the significant cluster did not affect the result ( $\rho(51) = -0.37$ ,  $p = 0.007$ ,  $R^2 = 0.14$ ). Crucially, partialling out participants' total sleep time or time spent in deep sleep, i.e. sleep stages N2 and N3, did not affect the relation between SWA in the right PFC and risk-taking behaviour ( $\rho(51) = -0.39$ ,  $p = 0.004$ ,  $R^2 = 0.15$ ;  $\rho(51) = -0.39$ ,  $p = 0.004$ ,  $R^2 = 0.15$ ). Thus, the negative correlation between SWA in the right PFC and risk-taking behaviour is independent of the quantity of sleep. Moreover, partialling out participants' age and gender also did not affect the relationship between SWA in the right PFC and risk-taking behaviour ( $\rho(50) = -0.39$ ,  $p = 0.004$ ,  $R^2 = 0.15$ ).

----- Figure 2 -----

SWA declines across the night and do so to a different degree at different cortical areas (Rusterholz & Achermann, 2011). Thus, averaging SWA over an entire night of sleep (i.e., across all sleep cycles) might lead to a loss of information. For this reason, we correlated

SWA in the right PFC cluster (see **Figure 2B**) with risk-taking behaviour separately for all sleep cycles. Not all participants had a fifth sleep cycle, therefore we present analyses from the first four cycles only. As illustrated in **Figure 3**, correlation analyses demonstrated a highly similar pattern for each of the four cycles compared to the whole night (see **Figure 2B**). To ensure that the main result was not driven by SWA in the first sleep cycle, we have excluded this cycle in an additional analysis and correlated SWA of the second, third and fourth sleep cycles pooled together with risk-taking behaviour. The result shows, again, a significant negative correlation between SWA in the PFC and risk-taking behaviour ( $\rho = -0.41$ ,  $p = 0.022$ ,  $R^2 = 0.17$ ).

----- Figure 3 -----

#### 4. Discussion

The topography of slow-wave activity (SWA) during sleep shows a high degree of inter-individual variability and a remarkable consistency across multiple nights within individuals. Some authors described this sleep characteristic as an electrophysiological “fingerprint” (Buckelmüller et al., 2006; De Gennaro et al., 2005). Here we examine if this EEG sleep trait explains individual differences in risk preferences. Because SWA is a physiological (and thus objective) marker for sleep depth, and because risky decision-making includes crucial decisions within a wide range of consequences in various contexts, identifying the association between sleep traits and risk preference is of great interest. We were particularly interested in whether individual differences in the local distribution of SWA during a night of sleep under normal conditions explain differences in risk preferences. We therefore applied whole-brain

corrected analyses in a sample of 54 healthy self-reported good sleepers with a habitual sleep duration of 7-8 hours. Using a portable multi-channel PSG that does not require constant supervision by a technician, we were able to measure brain activity during a typical night of sleep at participants' homes. We found that normalized SWA in the right PFC is associated with an individual's propensity to engage in risk-taking behaviour: Individuals with a high-risk preference showed less SWA in the right PFC than those with a low-risk preference. Our findings were highly specific to the right lateral PFC; in particular, we found no significant SWA correlations with risky decision-making in other brain regions.

Previous studies that looked at the brain during wakefulness also found the right PFC to be involved in risky decision-making. For example, functional imaging studies suggest that the right PFC may be particularly critical for the regulation of risk-taking behaviour (Fishbein et al., 2005; Mohr et al., 2010; Rao et al., 2008; Rogers et al., 1999; Schonberg et al., 2012; Yamamoto et al., 2015). Neuro-modulation studies further support these findings, which showed a causal involvement of the right PFC in risk-taking behaviour (e.g., Knoch, Gianotti, et al., 2006; Tulviste & Bachmann, 2019).

As mentioned above, evidence from a chronic sleep restriction EEG study also demonstrated a link between the right PFC and risk-taking behaviour (Maric et al., 2017). They found a negative correlation between slow-wave oscillations in the right PFC and changes in risk-taking behaviour after restricting time in bed to five hours for seven consecutive nights. Even though we specifically recruited a homogeneous group of good sleepers with a habitual sleep duration of 7-8 hours, we controlled for the total sleep time to exclude any possible confounds. Our results clearly show a significant association between a regional measure of sleep depth, namely SWA in the right PFC, and risk preferences.

The SWA across a night has been associated with activation levels during wakefulness; that is, higher SWA leads to increased activation in the PFC (Wilckens et al., 2016). Further support for the link between SWA and prefrontal activation during wakefulness stems from studies showing that sleep deprivation most strongly affects the cortical metabolic rates in prefrontal regions (e.g., Wu et al., 2006). Moreover, several studies show that executive functioning related to the PFC is particularly vulnerable to sleep loss (e.g., Groeger et al., 2014; Killgore et al., 2008; Thomas et al., 2000; for an overview see Harrison & Horne, 2000). If we focus on trait-like features of resting-state EEG studies during wakefulness and risk preferences, two studies revealed that task-independent baseline activation in the right PFC explains individual differences in risk preferences (Gianotti et al., 2009; Studer et al., 2013). Specifically, individuals with lower baseline activation in the right PFC are more prone to display riskier behaviour. Although very speculative, we suggest that the amount of SWA across a night determines the level of baseline activation in the PFC in wakefulness, which in turn explains individual differences in risk-taking behaviour. In our risk-taking task, each additional meter should give additional points. Thus, behaving riskier can yield a higher outcome. However, driving any meter comes at the risk of not winning anything. Note that this design does not allow for subjects to show risk-averse behaviour, which is a limitation we want to acknowledge. However, since we are interested in individual differences, and driving more meters implies a higher risk of not receiving anything, we believe our task allows us to differentiate between participants.

A large body of research indicates that higher levels of both baseline and task-related activation in the lateral PFC correlates with increased self-regulation, inhibitory control, or executive functions in general (e.g., Diamond, 2013; Gianotti et al., 2012; Heatherton & Wagner, 2011; Schiller et al., 2014). Hence, it seems reasonable to assume that higher SWA



in the lateral PFC during sleep is critical for restoring self-regulatory abilities, which are fundamental not only in mitigating risk-taking, but also in other important decision-making processes such as instance delay discounting (e.g., Figner et al., 2010; Gianotti et al., 2012; McClure et al., 2004; Turner et al., 2019), norm compliance (e.g., Gianotti et al., 2018; Spitzer et al., 2007; Strang et al., 2015; Yamagishi et al., 2016), costly punishment behaviour (e.g., Knoch et al., 2010; Knoch, Pascual-Leone, et al., 2006; Steinbeis, 2018), health-related behaviour (e.g., Friese et al., 2016; George & Koob, 2013; Goldstein & Volkow, 2011), and pro-environmental behaviour (e.g., Baumgartner et al., 2019).

It is known that SWA typically decreases towards morning (Achermann et al., 1993); therefore, one could ask whether the restoration of self-regulatory abilities occurs precisely at the beginning of the sleep. Our analyses clearly demonstrate that this is not the case. We found that the association between SWA and risk preferences was very similar in all sleep cycles.

There is a saying that people need a good night's sleep before making important decisions. However, what does a good night of sleep mean? Most people would probably say that a good night of sleep means sleeping long and deep enough. Here we demonstrate that sleep depth is a decisive factor. Importantly, it depends on where this happens. Our results show that sleep depth in the right PFC has a significant impact on risk-taking behaviour. Hence, it depends on how the brain sleeps locally. Recent evidence shows that brain stimulation techniques, such as transcranial magnetic stimulation, transcranial direct current stimulation, and auditory closed-loop stimulation enable the modulation of SWA (e.g., Bellesi et al., 2014; Ngo et al., 2013; Sousouri et al., 2021). Thus, these techniques might be promising tools for boosting SWA specifically in the right PFC to improve self-regulatory abilities and consequently functional decision-making.

**Data and code availability statement**

The dataset analyzed in the present study as well as scripting and plotting code are available from the corresponding authors via email on reasonable request.

**Declaration of Competing Interest**

The authors have no conflict of interest to disclose.

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Journal Pre-proof

**Table****Table 1:** Mean with 95% CIs for total sleep time, sleep efficiency, wake after sleep onset, and duration of sleep stages for total sample (N=54).

	Total sleep time [min]	Sleep efficiency [%]	Wake after sleep onset [min]	Duration of sleep stages (% of total sleep time)			
				N1	N2	N3	REM
Mean	438 .8	93.1	21.4	6.5	50.9	24	18.6
95% CIs	430.2-447.5	92.1-94.0	18.0-24.8	5.7-7.4	49.4-52.3	22.7-25.3	17.5-19.6



## Figure Captions

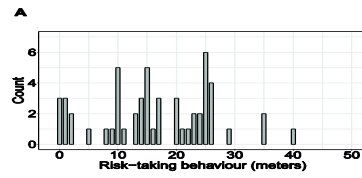
**Fig. 1:** Barplot depicting the distribution of the risk-taking behaviour among all participants.

**Fig. 2:** Topographical distribution of normalized SWA (0.8-4.6 Hz) and its correlation with risk-taking behaviour. **(A)** Topographical distribution of SWA (average over all subjects). SWA values at every electrode were normalized in relation to average SWA over all electrodes of a subject. Dark blue to dark red colors indicate minimal (45%) to maximal (162%) SWA. **(B)** Statistical topographical distribution of rho-coefficients between normalized SWA and risk-taking behaviour. Blue areas indicate negative correlation, red areas indicate positive correlation. White dots indicate electrodes with significant correlations ( $p < 0.05$ , corrected for multiple testing with a suprathreshold cluster analysis). Black dots indicate the position of the 59 electrodes. **(C)** Scatterplot of the negative correlation between mean normalized SWA in the significant cluster over the right PFC and risk-taking behaviour (including regression line and confidence interval 95%).

**Fig. 3:** Relationship between SWA and risk-taking behaviour for sleep cycle 1 **(A)**, sleep cycle 2 **(B)**, sleep cycle 3 **(C)**, and sleep cycle 4 **(D)**. On the left side, statistical topographical distributions of rho-coefficients between normalized SWA and risk-taking behaviour. Blue areas indicate negative correlations, red areas indicate positive correlations. White dots indicate electrodes with significant correlations ( $p < 0.05$ ) in the cluster of five electrodes identified in the main analysis (see Figure 2). On the right side, scatterplots of the negative

correlations between mean normalized SWA in the significant cluster over the right PFC and risk-taking behaviour (including regression lines and confidence intervals 95%).

**Figure 1 (1 column)**



**Figure 2 (2 columns)**

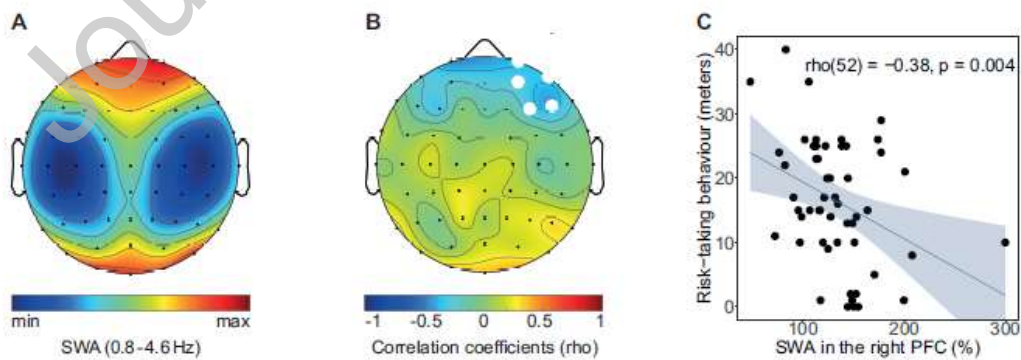


Figure 3 (1.5 columns)

