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J Camosun Psyc Res. (2021). Vol. 3(2), pp. 59-71.
Submitted Winter 2021; accepted July 2021.

What Are the Biological Mechanisms of Dream Abnormalities?

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ABSTRACT

In this paper, we sought to understand both the biological and psychological mechanisms responsible for dream abnormalities, in hopes to use the information to improve our relationships with sleep and dreams and to understand these concepts on a deeper level. Previous research has predicted dream abnormalities by variables such as sleep fragmentation, altered sleep architecture, and the involvement of the prefrontal cortex. In our correlational study, we tested the strength of these relationships by examining naturalistic daily changes in their variables longitudinally over a two-week period. We used Fitbit devices to measure both sleep fragmentation (by the number of sleep interruptions) and REM sleep amounts, and used subjective scales to measure prefrontal cortex activity during REM sleep (by the level of risk-taking and impulsive behaviour in dreams), the lucidity of dreams, and the nightmarish nature of dreams. Though it varied across participants, data pooled in our correlation study showed that there was a significant correlation of nightmares and lucid dreaming with sleep fragmentation, altered sleep architecture, and prefrontal cortex activity during REM sleep. This correlational study supports the role of these biological mechanisms in producing dream abnormalities.

1. Introduction

1.1 Research Problem

In this paper we hope to build a clear relationship between dream abnormalities and both the biological and psychological mechanisms that cause them. A rationale for researching this topic is its usefulness in understanding the basics of sleep and the many myths that exist about dreams and their biological and psychological implications. This topic of research also serves to build upon how we have come to understand REM sleep in the past and how that has adapted over time. To begin to understand dream abnormalities one must understand what is happening while we sleep and why we dream in the first place. Another rationale for wanting to study the biological and psychological mechanisms of dream abnormalities is because it is fascinating that even when the body is not in motion, the mind is still able to function and create images or scenarios. Obtaining the answers to why these types of dream abnormalities occur will be beneficial to an individual and the collective. This type of study can be valuable in many ways, especially for students, as a first step to improve quality of sleep and actualize the impacts of dream abnormalities on day-to-

day living while also navigating a way to understand them and how they work.

1.2 Literature Review

Some of the factors that past research has discovered to be mechanisms of dream abnormalities and lucid dreaming are sleep fragmentation and awakenings. by Gott et al. (2020) outlined how self-reported awakenings result in higher accounts of lucid dreaming and left room for speculation that disrupted sleep cycles have a connection to dream lucidity. In their study, participants were asked to fill out sleep questionnaires on a scale about how disrupted they assessed their own sleep to be, how many times they awoke during the night, and at which level of sleep continuity they believed produced the most lucid dreams. The results of this study lead to a connection between wakeful brain activity and how that may transfer to vivid dreams during REM sleep. Fragmented wake-REM sleep cycles often led to full arousal as a result of attempting to predict lucid dreams, proving to be harmful for the posed hypothesis. Experiences of lucid dreams can be connected to metacognition but more importantly poses the question of what level of arousal elicits vivid lucid dreams without completely disrupting REM sleep. This leads to the necessity for further research that sleep fragmentation can be associated with lucid dreaming and at what level of arousal it is the most effective.

Another mechanism previously found to be a cause of dream abnormalities is altered sleep architecture, which is the structure of the sleep cycle and the five stages of sleep. In an experiment conducted by Simor et al. (2012), participants complete an online questionnaire assessing dream quality as well as other personality factors. The Nightmare Subjects (NMs) were selected based on the International Classification of Sleep Disorders. Subjects with one or more nightmares and/or bad dreams per week were placed in the NMs, whereas subjects with less than two nightmares and/or bad dreams during the last year were inducted into the Control Subjects (CTLs). A developed program was used to determine output sleep architecture variables such as the following: wake time after sleep onset (WASO), sleep efficiency (sleep time/ time in bed), sleep latency (the period between lights off and the first epoch scored as Stage 2), the absolute and relative duration of Non-REM sleep, Stage 1, Stage 2, slow-wave sleep (Stage 3 and 4), REM sleep, and REM latency. NMs had significantly different sleep variables than the CTLs, such as WASO, sleep efficiency, and slow-wave sleep duration. The NMs revealed a worse sleep quality than the CTLs with less sleep efficiency, increased wakefulness, and less slow-wave sleep. However, the NMs had a longer sleep latency, an increase of Stage 1 sleep, and a higher number of nocturnal awakenings in Stage 2 sleep. Based on the results, Simor et al. (2012) suggest that nightmares (a dream abnormality) are associated with altered sleep architecture.

In addition to sleep fragmentation and altered sleep architecture, another mechanism found to be responsible for dream abnormalities is activity of the prefrontal cortex. In the Stumbrys et al. (2013) study), the prefrontal cortex was stimulated in hopes of finding a connection between lucid dreams and the activation of parts of the brain that are considered not as active during REM sleep. In this study, 23 participants were screened for sleep disorders and surveyed on the quality of their sleep and then tested for their sensitivity to transcranial direct current stimulation (tDCS) to ensure significant results. After these first tests, the

experimental period began where the participants were subjected to tDCS stimulation at random, on both the second and third night. Many of the participants awoke after the stimulation to the prefrontal cortex, and lucid dreams were mostly reported by those who already assessed themselves as frequent lucid dreamers. This study, although valuable in affirming the connection between brain activity in specific regions and dream abnormalities, more importantly, demands from future research a way in which dreams can come to be understood beyond subjectivity.

1.3 Hypotheses

Based on the literature review, we predicted the following hypotheses:

- Hypothesis #1: If sleep fragmentation increases during the night then lucid dreaming will increase.
- Hypothesis #2: If REM sleep increases then nightmares will increase.
- Hypothesis #3: If activity in the prefrontal cortex increases during REM sleep then lucid dreaming will increase.

2. Methods

2.1 Participants

The two authors of this paper served as the participants in its studies. The participants ranged in age from 20 to 21 years old, with an average age of 20.5 years, and included one male and one female. The participants were all undergraduate students at Camosun College who completed the current studies as an assignment for Psyc 215 ("Biological Psychology") and were grouped together due to their mutual interest in dream abnormalities. All participants had experiences or associations with dream abnormalities and the amount of sleep that the participants received during these studies fell within their normal ranges.

2.2 Materials and Procedure

We first performed a correlational study to test concurrently all of our hypotheses by examining naturalistic daily changes in their variables longitudinally. Each participant kept a study journal with them at all times over this study's two-week period in order to record self-observations of the following five variables: (1) sleep fragmentation, (2) altered sleep architecture, (3) prefrontal cortex activity during REM sleep, (4) lucid dreaming, and (5) nightmares.

Sleep fragmentation and altered sleep architecture were measured using a FitBit, which automatically tracks sleep patterns, and an iOS application on an apple watch called Pillow Automatic Sleep Tracker (created by Neybox Digital Ltd.). Pillow Automatic Sleep Tracker uses smartwatch sensors to detect and analyze sleep automatically, through bodily movements and heart rates. To measure sleep fragmentation participants recorded how many times their sleep was interrupted as indicated by these devices. After the participants awoke they recorded how many times their device indicated a level of activity that signified some form of awakening during the night. To measure sleep architecture alteration, each participant used their device to track each stage of sleep that they went through. Immediately when the participants awoke in the morning, they wrote down the number of REM sleep episodes they had throughout the night.

In order to measure activity in the prefrontal cortex during sleep, each participant monitored their own dreams for experiences of risk-taking behaviours and impulsivity. Participants were required to rate how risky and impulsive their behaviour

was in their dreams on a scale of 0-10. The prefrontal cortex is responsible for many functions and is known to play a major role in action planning and behaviour management, and its inactivity has been explored in relevant research as a cause for bizarre imagery typically found in dreams (Hobson et al., 1999).

To measure lucid dreaming, participants rated the lucidity of their dreams on a scale from 0-10, 0 being not lucid at all with no control, and 10 being highly lucid with complete control. Participants were also asked to mark down every time they became conscious of the fact that they were dreaming. All answers were recorded in study journals each morning.

To be able to measure nightmares, participants recorded on a scale 0-10 and marked down any indications of nightmares they experienced throughout the night. The scale was rated from 0 being no experience of disturbed dreams and feelings, 5 being a moderate experience of disturbed dreams and feelings, and 10 being an experience of highly disturbed dreams and feelings. Participants' answers were documented in a dream journal each morning they awoke.

To assess the strength and statistical significance of associations between variables predicted by our three hypotheses, we performed Pearson product moment correlations of their predictor variables (sleep fragmentation, sleep architecture, and prefrontal cortex activity) with their outcome variables (lucid dreaming and nightmares). For hypothesis #1, we correlated higher sleep fragmentation and states of arousal with a higher chance of lucid dreaming. For testing Hypothesis #2, we correlated altered sleep architecture of each participant for each night in which the participant had nightmares. For Hypothesis #3, we correlated activity in the prefrontal cortex during REM sleep of each participant in which the participant had lucid dreaming. We performed all of the above correlations separately for each participant as well as using data pooled across all of the participants. For the correlations using pooled data, in addition to using the raw data, we also performed correlations after we had first transformed the data from each participant into *z*-scores in order to standardize differences in averages and variability seen between the participants in their data and thus make them more comparable. A correlation coefficient was considered statistically significant if the probability of its random occurrence (p) was $< .05$ (i.e., less than 5% of the time expected by chance alone).

3. Results

As shown in Table 1, all three variables (sleep fragmentation, altered sleep architecture, and prefrontal cortex activity) were significantly correlated with nightmares and lucid dreaming. Although sleep fragmentation was not significantly correlated with lucid dreaming for one of the participants and when using pooled raw data ($r = .21$, $p = .028$; see Figure 1A), sleep fragmentation was significantly correlated with lucid dreaming using pooled standardized data ($r = .42$, $p = .019$; see Figure 1B). Similarly, although altered sleep architecture was not significantly correlated with nightmares for both participants or when using pooled raw data ($r = .24$, $p = .21$; see Figure 2A), altered sleep architecture was significantly correlated with nightmares using pooled standardized data ($r = .42$, $p = .018$; see Figure 2B). In comparison, while prefrontal cortex activity and lucid dreaming was not significantly correlated for both participants, prefrontal cortex activity was significantly correlated with lucid dreaming using both pooled raw

data (r =.41, p = .022; see Figure 3A) and pooled standardized data (r = .49, p = .005; see Figure 3B). Based on a comparison of the correlation coefficients using either the pooled raw data or the pooled standardized data, the strongest correlation seen was between prefrontal cortex activity and lucid dreaming.

4. Discussion

4.1 Summary of Results

Based on previous research we hypothesised that three variables would predict dream abnormalities (lucid dreaming/nightmares): sleep fragmentation (Hypothesis #1), altered sleep architecture (Hypothesis #2), and activity in the prefrontal cortex (Hypothesis #3). Data pooled across participants in our correlational study showed statistically significant results for all three hypotheses.

4.2 Relation of Results to Past Research

The ability of sleep fragmentation to predict dream abnormalities based on our correlational study aligned well with previous research. While Gott et al. (2020) stated that self-reported awakenings resulted in higher dream lucidity, the devices used in our correlational study made it easier to record sleep fragmentation and numbers of awakenings in a more objective manner. The similarity of both of our conclusions despite using different research designs suggests a generalized relationship exists between sleep fragmentation and lucid dreaming.

The strong relationship found between altered sleep architecture and nightmares in our correlational study is consistent with past research. Simor et al. (2012) found that nightmares are caused by altered sleep architecture. While the Simor et al. (2012) study a large number of sleep variables in laboratory conditions (WASO, sleep efficiency, sleep latency, slow-wave sleep, and REM sleep), our correlational study recorded participants' number of REM sleep episodes that occurred in a natural setting. The fact that we found the same relationship between altered sleep architecture and dream abnormalities (nightmares) despite these differences in methodology speaks to the universality of its relationship.

In addition, the relationship between prefrontal cortex activity and lucid dreaming was strong in our correlational study and was in line with previous research. While Stumbreys et al. (2013) conducted a tDCS study to stimulate the prefrontal cortex and the effects of activation of this part of the brain that is usually inactive during sleep, we took an approach more similar to Hobson et al. (2000), who looked at dream themes and dream behaviour to predict brain activity. Despite these different methods of research, our findings of similar conclusions reveal a universal relationship between prefrontal cortex activity and lucid dreaming.

4.3 Implications of Results

Throughout this correlational study, there was a clear relationship between biological and psychological factors and dream abnormalities. The practical application of this correlational study further extends the knowledge of how biological mechanisms of sleep related to lucid dreaming and nightmares, which can provide insight into our personal sleep habits. Both lucid dreaming and nightmares were significantly associated with sleep cycles and patterns, leaving room for further research that can blur the divide between our objective understanding of sleep and our subjective experiences of it. The findings of this study

shed a positive light on the research question as sleep fragmentation, sleep architecture, and prefrontal cortex activity are biological mechanisms that showed positive correlations with dream abnormalities.

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Table 1

Correlation coefficient (r) values, with number of daily trials (n) per correlation in brackets.

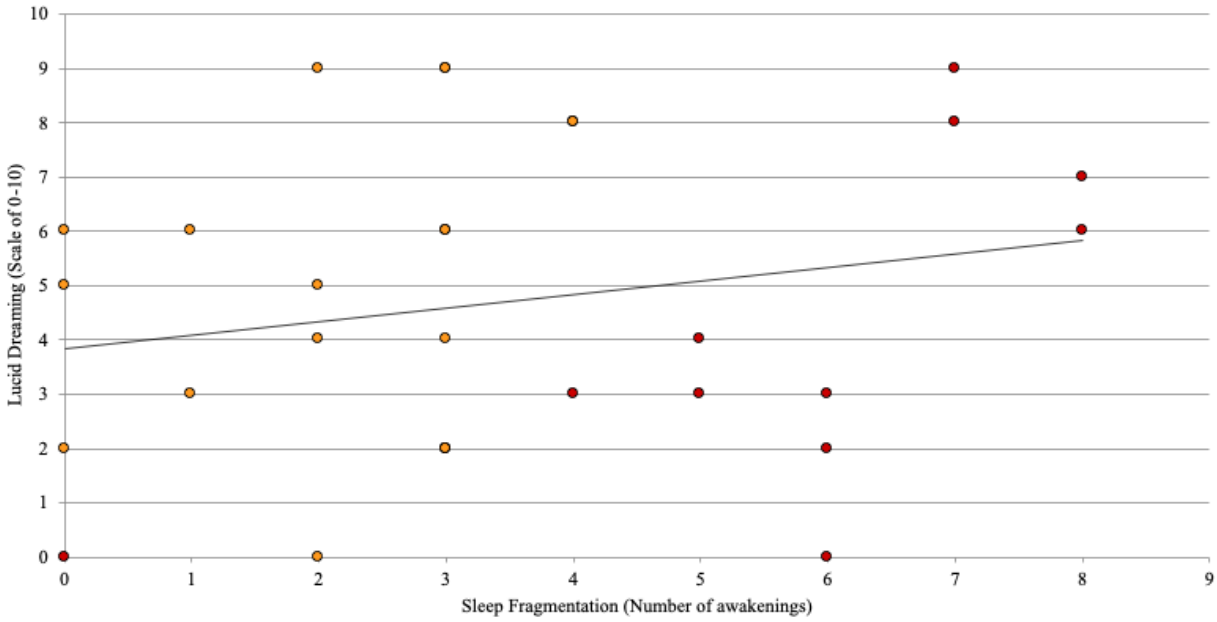
Variables correlated	Participant #1	Participant #2	Pooled raw data	Pooled standardized data
Sleep Fragmentation & Lucid Dreaming	.52(15) *	.32(15)	.21(30)	.42(30) *
Altered Sleep Architecture & Nightmares	.55(15) *	.30(15)	.24(30)	.42(30) *
Prefrontal Cortex Activity & Lucid Dreaming	.62(15) *	.36(15)	.41(30) *	.49(30) *

* $p < .05$.

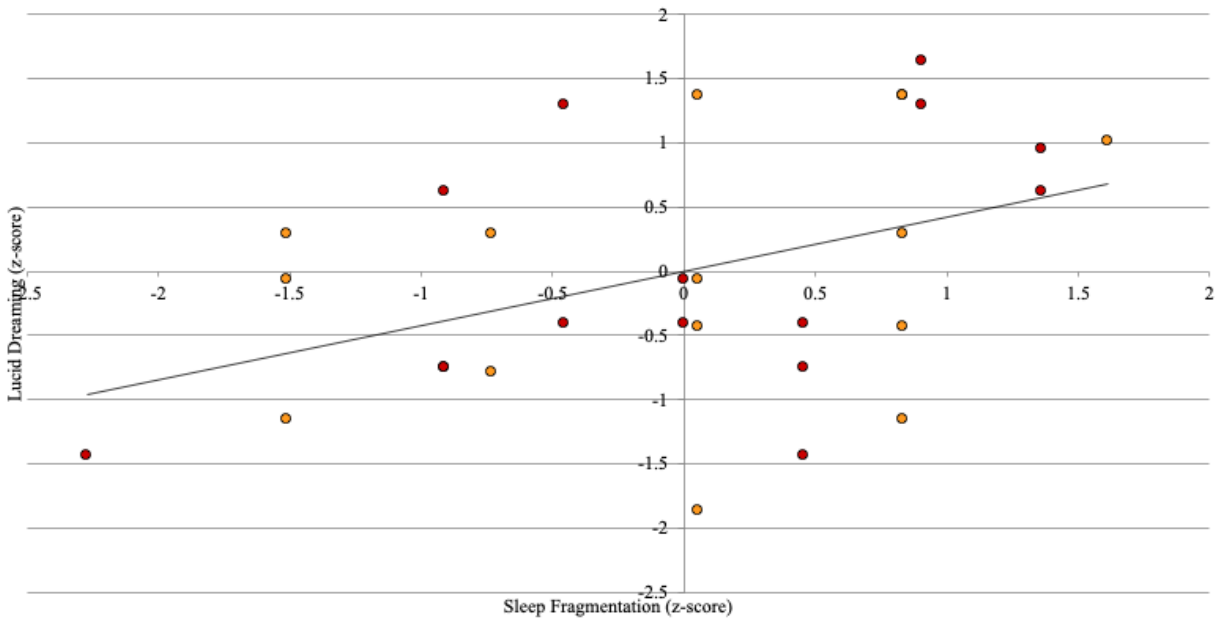
Figure 1

Scatterplot of sleep fragmentation and lucid dreaming using pooled (A) raw and (B) standardized data across participants.

A.



B.

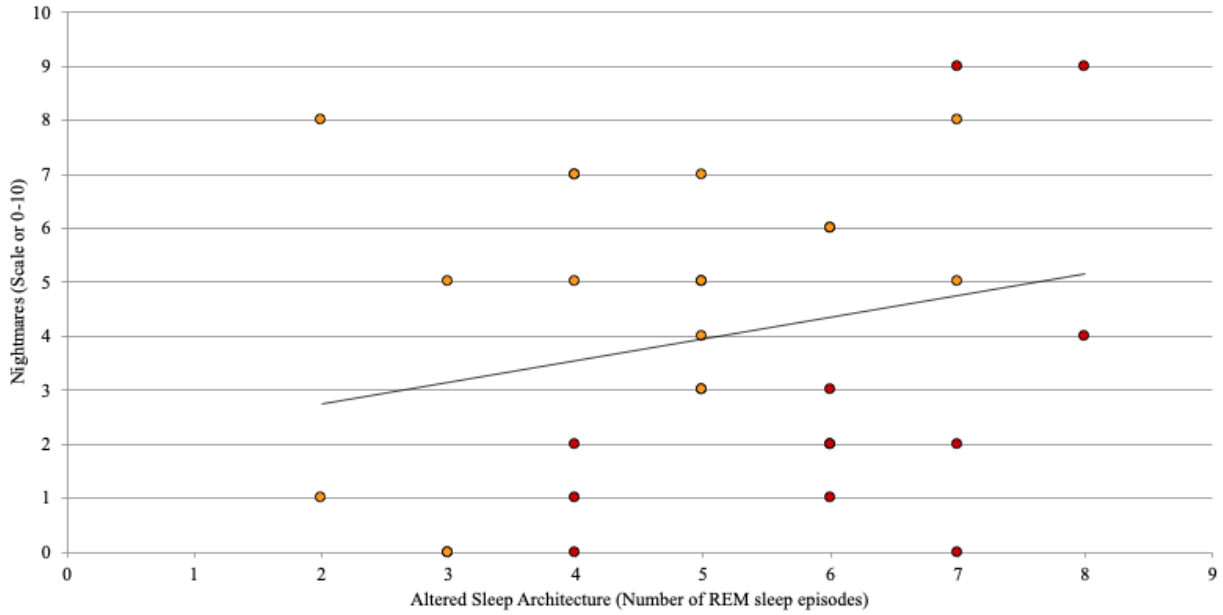


Marker color indicates which participant data is from: red = participant #1 and orange = participant #2. Some data might not be visible in the figure due to overlapping markers.

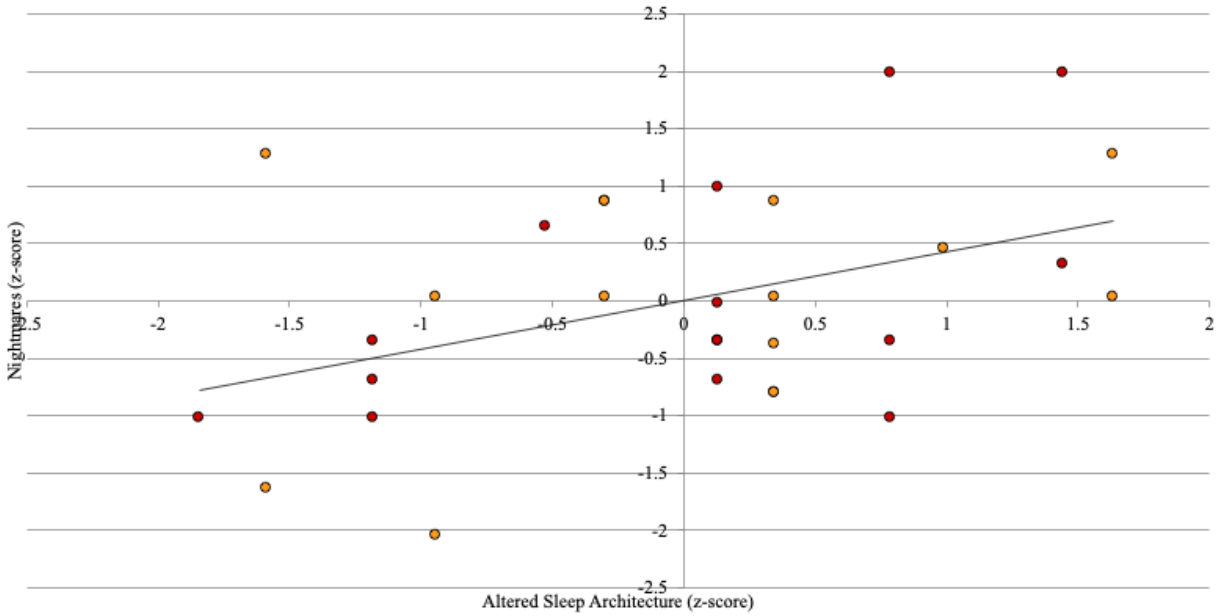
Figure 2

Scatterplot of altered sleep architecture and nightmares using pooled (A) raw and (B) standardized data across participants.

A.



B.

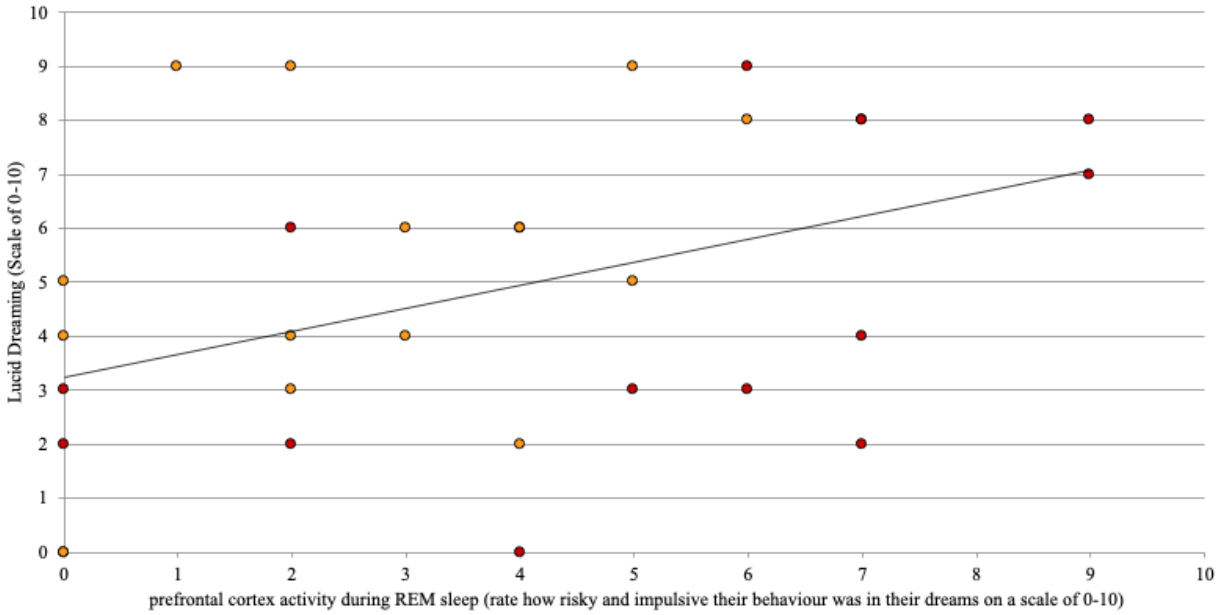


Marker color indicates which participant data is from: red = participant #1 and orange = participant #2. Some data might not be visible in the figure due to overlapping markers.

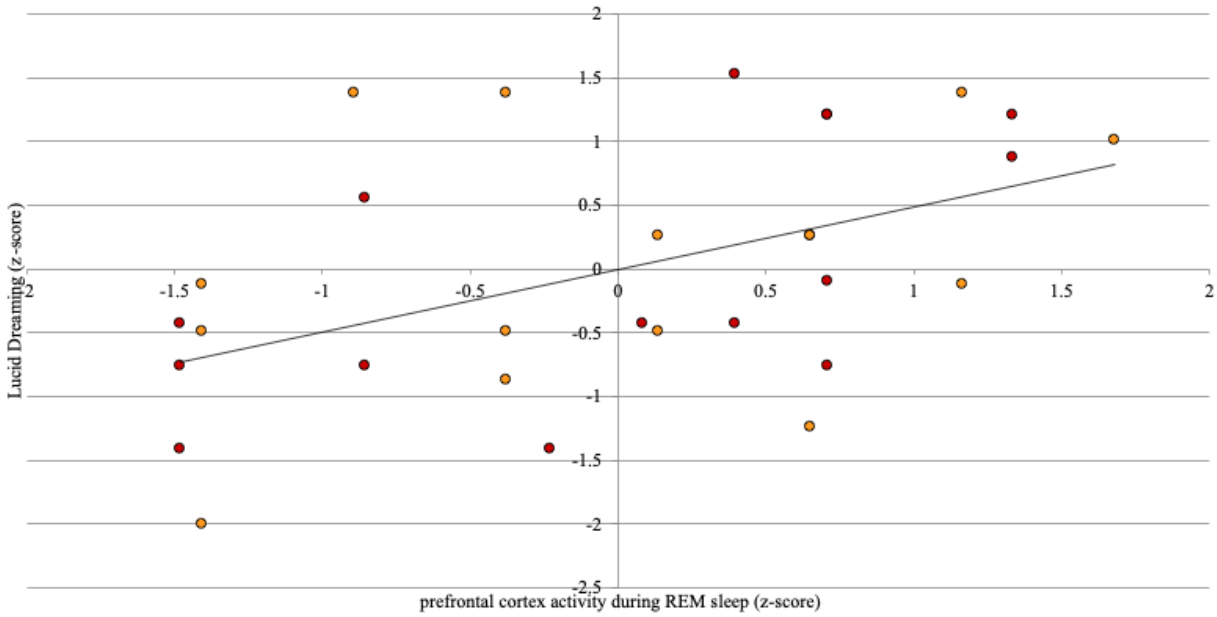
Figure 3

Scatterplot of prefrontal cortex activity and lucid dreaming using pooled (A) raw and (B) standardized data across participants.

A.



B.



Marker color indicates which participant data is from: red = participant #1 and orange = participant #2. Some data might not be visible in the figure due to overlapping markers.