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Review Article

The Significance of Rapid-Eye-Movement Sleep for Regulating Emotions and Obtaining Long-term Memory

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Abstract

Humans spend a great deal of time sleeping and dreaming. While the benefits of good sleep hygiene are well-known, fewer appreciate the importance of dreaming. This paper investigates the necessity of rapid-eye-movement (REM) sleep (dreaming) by examining scientific research articles' methods and results. These scientific experiments examine the genetic drawback of REM sleep and the outcomes of REM sleep deprivation on emotional regulation and long-term memory. REM sleep deprivation is becoming a modern epidemic and many substances consumed during the day can adversely affect sleep. Nicotine, antidepressants, sleep medication, and cannabis consumption can all lead to REM sleep deprivation, which negatively influences emotional regulation and long-term memory. Paradoxically, these substances are often used to help one fall asleep faster.

Keywords: ***REM sleep deprivation, Memory, Long-Term, Emotional Regulation, Nicotine, Antidepressive Agents, Cannabis.***

Introduction

Sleep is composed of multiple stages and scientists determine these by measuring the brain wave activities. When we are awake, these waves have higher amplitude and frequency while during sleep, they display lower amplitude and frequency [1]. Full-night sleep follows this cycle of stages multiple times (approximately three to four times): three stages of non-REM sleep (N1, N2, and N3) which are then followed by one stage of REM sleep [2]. Out of these stages, the rapid-eye-movement (REM) sleep stage has a unique distinction as people dream during this stage while this does not occur in the other ones. REM sleep is also known as rapid eye movement because our eyes roll backward and the cerebral cortex is activated when the brain proceeds to this stage of sleep [3]. In REM sleep, the brain wave patterns mimic the waking brain, but the body's muscles become paralyzed to avoid harm [2]. All sleeping patterns and stages have unique characteristics; these characteristics are essential and significant, and the REM sleep stage is specifically important for building strong long-term memory, maintaining emotional processing, and more. Multiple experiments have been performed on humans and rats to examine these aspects of REM sleep, some of which will be explored in this paper.

Sleep deprivation adversely affects emotional regulation and long-term memory according to amygdala and hippocampus activities. Interestingly, genetic factors play a significant role in brain wave patterns during REM sleep. This conclusion is drawn from a study on monozygotic and dizygotic twins [4]. Monozygotic twins have the same DNA sequences in their genes, while dizygotic twins have similarities in their DNA sequences equivalent to siblings. In the study, sleep patterns were recorded using an electroencephalogram, showing that monozygotic (MZ) twins had more similarities in their brain wave patterns than dizygotic (DZ) twins [4]. In addition, emotional regulation is essential to everyday activities and for good mental health. REM sleep regulates emotional processes by activating the hippocampus and amygdala which are the workers of the brain for controlling emotions during the day [5-6]. Furthermore, a fear-conditioning experiment performed on rats deprived of REM and non-REM sleep to test their long-term memory consolidation showed that the rats that were deprived of REM sleep displayed less freezing response. This result indicates that REM sleep is essential for building long-term memory [7]. From these experiments, we can conclude that the REM sleep wave patterns are both inheritable and important for building long-term memory and emotional regulation.

Like sleep deprivation generally, REM sleep deprivation is becoming a major health concern, and both can have negative physical and mental outcomes. However, knowledge about REM sleep deprivation is poorly understood among the general public. Many common substances that people use daily can suppress the REM sleep cycle [3,8]. Alcohol, cannabis, caffeine, narcotic pain medications, antidepressants, and lithium consumption can all cause REM sleep deprivation [9]. Many people consider alcohol and cannabis helpful for sleep since they suppress the nervous system; however, they actually harm the quality of sleep by disrupting REM sleep. Surprisingly, prescription sleep medication also acts in a similar manner, but its list of side effects rarely includes REM sleep suppression [1]. People often try to cure their sleep deprivation without realizing they are harming their REM sleep cycle because

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they are not aware of the side effects of sleep medications. Society must be more cautious about REM sleep deprivation than they are about sleep deprivation in general.

Methods

This literature review followed the methods described by Mohamed Shaffril Samsuddin *et al*. (2021) [10]. Inclusion criteria included English language journal articles published from 1975 to 2022 which summarized the general outcomes of REM sleep deprivation on cognitive performance, long or short-term memory, and emotional regulation while the genetic variance is considered for a deeper understanding of REM sleep stages. In this literature review, the primary author used the manual searching method on three main databases: ProQuest, Google Scholar, and Google Engine Search (**Table 1**).

Table 1: Search Strategies.

Database	Search Strategy
ProQuest	(Genetic Variance) AND (Emotional Regulation OR Anxiety Levels) AND (Long-term Memory OR Short-term Memory OR Freezing Behavior) AND (Cognitive Skills OR Anagram Word Puzzles) AND (Nicotine Consumption OR DREAM Proteins)
Google Scholar	(REM Sleep) AND (Emotional Regulation OR Emotional Reactivity Task) AND (REM Sleep Deprivation OR Antidepressant Drug Consumption) AND (REM Sleep Deprivation OR Cannabis Consumption)
Google Engine Search	(Sleeping Stages OR Brain Wave Activity) AND (Sleeping Phases OR REM Sleep Stage) AND (REM Sleep Deprivation OR Drug Consumption OR Sleep Disorders) AND (REM Sleep OR Brain Wave Frequencies)

Following the searches, the primary author reviewed and selected studies to include in the literature review based on the criteria that are given in the article. Each article was separated into REM sleep or REM sleep deprivation categories and then divided into sub-categories or themes for organization and summarization purposes. The sub-categories were determined prior to data collection included emotional regulation, genetic variance; cognitive skills, memory, and the modern epidemic of REM sleep deprivation. No assessment of the methodological quality of the articles was conducted.

Experimental Methods

Experiment 1: Twin Study

A previous study conducted by Admczyk *et al*. (2015) sought the significance of genetic drawbacks to REM sleep brain wave regulation and duration [4]. The experimenters hypothesized that the brain waves' frequency and amplitude will be similar as the genetic similarities increase such as the DNA sequence of the monozygotic twins. To support this hypothesis, the experiment considered 32 monozygotic (17-43 years, 16 male, 16 female pairs) and 14 dizygotic (18-26 years, 7 male, 7 female pairs) twin pairs who had been living together their whole lives [4]. Brain waves were followed by EEG during this experiment and the analysis of these brain waves determined the time that twins entered REM sleep as well as the duration of this stage. Phasic REM sleep parameters (EOG potentials of opposite polarities synchronized for the time period within 40 ms-70 ms) were used to understand when participants entered the REM sleep automatically [4]. Genetic variance analysis was performed on all twin partners to identify the genetic differences and similarities among the participants. The Intraclass Correlation Coefficients (ICC) analysis helped to determine the difference between the resemblance of twin pairs and night-to-night stability in this experiment; the ICC values needed to be positive to be valid [4]. Finally, to identify the similarities of EEG power spectra with respect to their morphology, hierarchical cluster analysis was performed on the data collected from the twin participants [4]. The participants spent three nights in the sleep laboratory: the first night was for the adaptation without any sleep disturbances while the other two nights introduced REM sleep disturbance for analyzing the results. Most of the twin partners (both monozygotic and dizygotic) were recorded with EEGs at the same time during this experiment. Genetic variance analysis was performed on all twin partners to identify the genetic differences and similarities among the participants [4].

Experiments 2 and 3: Emotional Regulation Studies

Study conducted by da Silva Rocha-Lopes *et al*. (2018) investigates the emotional regulation and REM sleep deprivation was performed on rats [5]. This study aimed to find the correlation between deprived REM sleep and its consequences manifested as anxiety behavior and adrenal levels in rats. Twenty-one male Wistar rats were used in the experiment: 11 rats in the control group (CTL) and 20 rats in the REM sleep restriction group (REMSR), which under-maintained room conditions (22±2°C and 12 h light/dark cycle) [5]. The REMSR group had a unique cage submerged in water to deprive rats of the REM sleep stage. In total, the rats spent 16 hours a day for 21 days in the cage. The animals were weighed and their body length was measured every 3 days for 21 days total after they returned to their sleep cages from restricted cages for recovery. Afterward, a sucrose negative contrast test (SNCT) was submitted on 11 CTL and 10 REMSR rats to identify the impairment in the hedonic/motivational profile after sleep deprivation. To evaluate anxious behavior caused by sleep deprivation, rats were exposed to an elevated plus maze (EPM) at the end of the SNCT [5]. The relative adrenal weight was used to determine the anxiety level corresponding to the adrenal glands' weight and body mass ratio. Plasma levels of corticosterone and adrenaline in the bloodstream were tested to examine the anxiety levels of the rats after they had been in the maze. The brains of each rat were dissected to examine the hypothalamus (5-HT1A serotonergic and β1 noradrenergic receptors), amygdala, hippocampus (ventral and dorsal), and frontal cortex activation for further investigation of the physiological changes in these sites of the brain [5].

Study conducted by Lagarde *et al*. (2012) aimed to understand the correlation between REM sleep deprivation and emotional regulation in humans [6]. Twenty right-handed approved male participants (aged between 21 and 35 years with no sleep and medical disorders) were randomly divided into two groups: one awakened during REM sleep, the other awakened during non-REM sleep as a control. The control group was awakened during a non-REM sleep period to indicate that sleeplessness does not affect emotional regulation. Standard polysomnography (PSG), EEG,

electrooculogram (EOG), and electromyogram (EMG) recorded the participants during baseline, experimental, and recovery nights to understand the effect of deprived REM sleep on emotional regulation [6]. The REM sleep-deprived group was awakened whenever the PSG showed slow brain wave activity which is when the sleep spindles and K complexes were not visible on EEG reading [6]. The experimental task involved a reactivity task to observe the emotional reactions of the REM-D and NREM-I participants. The Emotional Reactivity task involved the presentation of sixty pictures: forty negative valance and twenty non-valence images to observe the active parts of the brain according to the negative emotions. Participants reacted to this task at the same time period each day under an MR scanner to observe the reactivity of different parts of the brain [6]. According to the defense responses of the participants, they were labeled as high emotional reactivity trials (HER) and non-defending responses as low emotional reactivity trials (LER). The number of HER and LER was recorded to understand the outcomes of deprived REM sleep while a one-variable t-test was performed on these data to show mathematical analysis of results collected during the experiment. fMRI signals of occipital and temporal areas (emotional processing parts of the brain) and the ventrolateral prefrontal cortex (involved in top-down emotion regulation which is a part of the limbic system) have been used to indicate the effects of the awakening periods (during REM or non-REM) in humans [6].

Experiments 4 and 5: Cognitive Skills and Memory Studies

Flexible cognitive processes such as problem-solving and creative ability are fundamental and require a healthy sleep schedule. An experiment was performed by Mohamed Shaffril *et al*. (2021) to understand the cognitive effects of REM and N-REM sleep deprivation [10]. Six male and ten female native English-speaking undergraduate students with no history of mental illness or neurological, psychiatric, sleep, drug, or alcohol problems and with normal or corrected vision were used in the experiment [10]. The study consisted of two groups of randomly selected participants: one was awakened during REM sleep and the other during non-REM sleep. Participants solved computerized anagram tests four times across a 10-hour time period: at 10 p.m. before sleep (pre), once followed 10 minutes into non-REM sleep, followed 10 minutes into the REM sleep, at morning approximately 8 a.m. (post), and 30 minutes after awakening. REM and non-REM sleep periods were confirmed by polysomnographic data. The specific cognitive flexibility test involved solving anagram word puzzles composed of five letters. For example, 'OSEOG' is 'GOOSE' and more words (a total of thirty-two words which is divided into four sets of eight) were shown to participants. Sixteen subjects had 10 seconds to respond to these questions and they were tested 4 four times a day according to their experimental group. If they could not answer the question in 10 seconds, the program would automatically skip to the next question. The recorded data was used to analyze the results of the experiment with Pearson's correlation analysis to understand the outcomes of the cognitive skills of the participants according to their deprived sleep schedule. Pearson's correlation analysis was used on the Stanford Sleepiness Scale scores of subjects to correlate the percent correct and reaction times according to the difference in the state of awakened: REM or NREM. Evaluation of sleepiness of subjects prior to the PRE and POST sleep trials was obtained with Stanford Sleepiness Scales [10].

Study performed by Lee *et al*. (2011) looked at the effect of REM sleep deprivation on long-term and short-term memory [7]. Light-dark (LD) 24 rats were kept constantly under a light-dark cycle consisting of 12 hours of light and 12 hours of darkness while forced desynchronized (FD) rats were under a 22-hour light-dark cycle under 11 hours of light and 11 hours of darkness. After 10 to 15 days 22 hour LD cycle rats became desynchronized which was confirmed by the appearance of two statistically rhythmic locomotive activity. Recording of the electrical activity of the cerebral cortex was followed by placing implemented EEG electrodes over frontal and parietal cortices as the rats slept [7]. Low-amplitude EEG waves were characterized as wakefulness with active locomotive activity while REMS was characterized as high-amplitude EEG (theta) waves and lack of locomotive activity. Two groups of participants were used in this experiment: non-sleep-deprived and REM sleep-deprived groups of rats trained on a contextual fear conditioning task by placing them into a conditioning chamber. The animals had a chance to explore the conditioning chamber for two minutes. The rats' movements were recorded with night shot mode after the foot shock was delivered. This technique was used to set a memory for these experimental rats. To test memory performance, experimenters observed freezing behavior (defined as cessation of all but respiratory movement). Experimenters tested the memory performance of two groups of rats and freezing behavior was continuously recorded. Data was quantified and presented as a percentage of time that an animal spent frozen [7].

Results

The Outcome of Experiment 1: Twin Study

The REM sleep architecture, REM EEG spectral power, and structural organization of REMs were compared between a group of 32 monozygotic and 14 dizygotic same-gender healthy twins [1]. The genetic variance test was used to determine the similarities of genetic sequences between the pairs of twins. The genetic variance test showed that monozygotic twins had a low variance while dizygotic twins had higher levels of variance in their genetic sequences. According to the genetic variance analysis of REM sleep architecture, genetics significantly affect REM sleep duration (the duration is similar in monozygotic twins while in dizygotic twins it is more dissimilar) [1]. Moreover, participants of the same age and gender showed similar structures of REM sleep: this finding was especially true in female twins. To find the similarities of EEG frequencies, ICC tests were performed. The mean ICC for all EEG frequency bins which was collected during a two-night period was 0.91 for monozygotic twins and 0.45 for dizygotic twins. These numbers indicate that monozygotic twins showed more similar EEG frequencies rather than dizygotic twin participants. Furthermore, there was a substantial genetic influence on the REM EEG power spectrum (δ to high σ, β2 to γ band) which was found by cluster analysis in this experiment [1]. In conclusion, the experiment showed a significant effect of genetic variability on the REM sleep waves, which was determined by the similarities between MZ twins were relatively higher than the DZ twins.

The Outcomes of Experiments 2 and 3: Emotional Regulation Studies

The time spent in the open-armed cages correlated with the anxiety level and behavior in rats (less time shows a higher level of anxiety). REM sleep-deprived group of rats spent less time in open arms rather than the control group which resulted in a significant difference in anxiety levels with higher in the REMS-restricted group [5]. On the other hand, weight gain was used to indicate the anxiety outcome, and post hoc analysis showed that REMS-restricted rats were lighter than the control group. The levels of some specific neurotransmitters increased in the hippocampus and

amygdala, the two main parts of the brain responsible for emotional regulation in the REM sleep-restricted group. In the hippocampus of REMS-r adolescents showed higher levels of noradrenaline in the ventral hippocampus while in the amygdala the levels of 5-HT increased to lower the metabolism in the dorsal hippocampus [5]. In order to evaluate the neurotransmitter release changes in the amygdala and hippocampus, Western blotting assays were performed to find the expression of 5-HT1A and β1 receptors. REM sleep restriction causes a reduction in the expression of 5-HT1A receptors in the dorsal hippocampus compared to the non-restricted group. Furthermore, negative energy balance was observed in the chronic REMS-r group but the levels of sucrose did not change at all [5]. Because the restricted group was more anxious than the other group, they performed less exploration in the elevated maze and had trouble regulating their emotional state. Also, their anxiety altered their growth state by showing no weight gain and an increase in naso-anal length. This experiment showed chronic REM sleep deprivation during the prepubertal period impaired physical development and led to anxiety-like behavior in rats. The anxiety was indicated by heavier adrenal glands and higher basal corticosterone plasma levels. Higher corticosterone basal levels, heavier adrenals, and induced anxiogenic profile showed that this was a stressful protocol for the REMS-r group [5]. According to this experiment, lack of REM sleep can cause anxiety behavior which can result in weight loss and a higher level of noradrenaline levels in the body.

The experiment performed by Lagarde *et al*. (2012) examined the impact of selective REM-D on emotional reactivity to threatening visual stimuli and associated brain activity changes. After REM sleep deprivation occurred in one group and another had non-REM sleep deprivation, they performed low (LER) and high (HER) emotional reactivity trials [6]. The results of the emotional reactivity trials showed that REM sleep deprivation has an effect on emotional regulation. According to ANOVA's statistical result, the procedure successfully reduced from 21.01% to 4.03% REM sleep only in the REM-deprived group while the other group significantly had the same REM sleep duration as usual. On the other hand, the non-REM-deprived group had a significant decrease in Stages 1 and 2 of the sleep cycles while REM-D had no difference in these sleep cycles. The REM-deprived group showed an increase in their high emotional reactivity trials (HER) compared to their results before they had a deprived sleep (before the mean was 26.3 after the mean was 31.6) [6]. However, there was no significant increase or decrease for HER trials in non-REM-deprived sleep. Both groups had some kind of deprivation in their sleep schedule which resulted in a decrease in reaction times for HER responses. HER minus LER trials showed increased activation of the right inferior frontal gyrus in both groups which can result in high emotional reactions that can cause activation in this brain area unrelated to sleep deprivation [6]. The comparison of HER and LER before and after sleep deprivation showed that HER trials increased the activity in the left middle occipital gyrus and right inferior frontal gyrus in the REM-deprived group. Corresponding to the results from this experiment, defensive responses were enhanced after one night of controlled sleep for the REM-D group while the NREM group showed no change [6]. In conclusion; threatening stimuli after REM-D in humans showed an enhanced emotional reactivity which explains that lack of REM sleep causes humans to have trouble regulating their emotions.

The Outcomes of Experiments 3 and 4: Cognitive Skills and Memory Studies

Problem-solving skills can derive from different stages of sleep: REM, NREM, and WAKE, which is the main topic this experiment investigated. Experimenters hypothesized that the non-REM sleep-deprived group would be less successful solving anagrams compared to the WAKE state and REM-deprived group [11]. This hypothesis was supported by the experimental results: REM awakenings solved 32% number of anagrams higher than NREM awakenings. There was no significant difference in WAKE and REM sleep awaken participants' solving percentages while for NREM waken group showed less success in this test [11]. However, there was no significant difference in mean reaction time in both groups and before they had a deprived sleep (WAKE=4.2s, REM=4.3s, NREM=4.5s). Participants awakened during REM sleep solved 15-35 percent more puzzles than they solved in the waking stage, which also supports the hypothesis of this experiment. Additionally, the number of solved anagrams was lower in the NREM awakened group compared to the number of anagrams solved before they had sleep restrictions. In contrast, the neurophysiological profiles were found to be considerably different in the REM and WAKE stages. Significantly, the percentage of solved puzzles was the same in early and late REM sleep deprivation (REMa and REMb), while the early non-REM deprived group (NREMb) was less successful than the late non-REM deprived (NREMa) group. According to these results, the participants awakened during REM sleep showed an increase in cognitive ability indicated by the number of solved anagram questions rather than when they did not experience any sleep restrictions and when they experienced non-REM sleep restriction [11]. Awakening during REM sleep showed significantly higher numbers of solved anagrams and puzzles compared to the non- REM deprived group and WAKE baseline levels which showed a higher capability of cognitive skills.

The experiment performed by Lee (2011) probed the effects of deprived REM sleep on long-term and short-term memory in rats by using fear conditioning as a memory [7]. REM deprivation had a significant effect on long-term memory while there were no significant differences in short-term memory. Furthermore, the study showed that REM sleep deprivation negatively correlates with long-term memory. Contextual fear conditioning procedures were performed on adolescent rats to test the effects of sleep deprivation on long-term memory. This training only required one session which lasted several months. In addition, the experiment used shocks, which resulted in freezing behavior in rats. Moreover, this experiment used a type of fear conditioning known as the hippocampus-dependent memory type to determine the sleep deprivation effects on memory. The NREM-deprived group of rats showed an increase in freezing behavior which correlated with survival rate which is indicated by exponential coefficients analysis. To test the long-term memory effect on these rats, testing occurred at 12 days following training, and the freezing behavior was analyzed without the presence of a shock chamber for 4 minutes [7]. LD 24 rats expressed 45% freezing, negative control group 12%, aligned rats 49%, and misaligned rats significantly express less freezing than 24 LD and aligned rats. Percent of time spent as a freezing behavior was recorded for NREM, REM, and WAKE bout. Exponential coefficients were used to indicate the survival fitting of aligned, misaligned, and LD 24 control rats for indicating survival rate. As the NREM survival increased, rats increased their freezing behavior to protect themselves from the shock. In contrast, the REM-deprived rat group showed less response as a freezing behavior when they were in the maze [7]. This result indicates that REM sleep is significant in building long-term memory because the deprived group did not show freezing behavior when they entered the maze. This experiment showed that long-term memory consolidation NREM sleep duration following REM sleep is essential for positively regulating the hippocampus.

Modern Epidemic (REM Sleep Deprivation)

REM sleep is a crucial phase of the sleep cycle, integral to cognitive functions such as learning, memory consolidation, emotional regulation, and metabolic processes. Deprivation of REM sleep disrupts these functions, leading to significant impairments in daily life [9]. Many people lack the awareness of the harm associated with REM sleep deprivation. In the 1960s, researchers became interested in REM deprivation and found that selective deprivation of dreaming caused subjects to gain weight and demonstrate irritation, anxiety, tension, delusions, difficulties in concentration, and hallucinations [12]. This type of sleep deprivation can be caused by commonly used substances or sleep disorders such as obstructive sleep apnea and narcolepsy [9]. Many common substances are the main drivers of REM sleep deprivation: caffeine, alcohol, cannabis, nicotine, opioid or narcotic pain medications, benzodiazepine medications, antidepressant medications, Lithobid (lithium), and sleep medications [1,9,12,13]. Alcohol and cannabis are commonly used to hasten sleep by acting as nervous system depressants; however, although they may cause sleepiness, they also suppress REM sleep[1].

Alcohol consumption (one glass per day for women and two drinks per day for men) can trigger the compensatory adrenergic surge which disturbs dreaming. Additionally, cannabis has also become popular for aiding sleep. Cannabis has been used as a psychoactive and medicinal botanical for centuries and the effects can vary depending on the different strain. Furthermore, researchers recently discovered that cannabis increases deep sleep while suppressing the REM sleep which creates a REM rebound upon discontinuation [12].

In European countries and all around the world, cigarette consumption is high and represents a major public health concern. One recent study on rats showed that nicotine consumption can significantly increase the hippocampus DREAM protein expression in CA1, CA2, CA3, and DG regions which results in significant REM sleep deprivation. Nicotine consumption leads to impairments in learning and memory performance since it causes this suppression in the expression of the DREAM protein [13].

Discussion

Sleep works to maintain metabolism, memory, and cognitive skills, and most brain functions require energy that is produced during sleep. Humans typically spend nearly one-third of the day asleep-about 8 hours-although it varies from person to person. The human body experiences two different sleeping phases: REM and non-REM, which are divided into three sections (N1, N2, and N3) [2]. During sleep, brain activity continues constantly; however, the frequency of brain waves can deviate during different stages. This paper has examined multiple scientific studies that demonstrate the negative consequences of REM sleep deprivation. Moreover, brain waves' frequencies and amplitudes can be used to determine the stage of sleeping to examine REM sleep or non-REM sleep. To understand the changes in brain waves during sleep, electroencephalograms (EEG) have been used in the experiments discussed. In summary, healthy humans experience REM sleep and non-REM sleep (stages 1, 2, and 3) when they are asleep, and each sleep cycle shows different types of frequencies and amplitudes of brain waves [14]. REM sleep produces human dreams and brain waves mimic the patterns seen in waking brains. Additionally, during REM sleep muscle paralysis occurs to prevent harm [2]. This stage usually occurs every 90 minutes, which explains why people dream but do not necessarily remember those dreams [14].

This paper has examined a total of five studies to understand the consequences of REM sleep deprivation and to show the impact of genetics on dreaming. Deprivation in REM sleep can negatively affect cognitive skills, long-term memory, and emotional regulation. According to experiments, REM sleep is significant for regulating emotions by positively affecting the amygdala and hippocampus [5,6]. In REM sleep deprivation, the regulation of the amygdala and hippocampus slows, making it harder to maintain emotional responses, which can become more intense [5,6]. Moreover, human brains continue being active during dreaming which is an inherited self-defense mechanism [11]. Participants who awakened immediately following REM sleep showed higher success in solving anagrams than when they were awake and when they were awakened after non-REM sleep [11]. Fear conditioning has been used to set a memory for the rats to understand the effects of deprived sleep on long- term and short-term memory. According to this study, regulation of hippocampus reactivity is important for building long-term memory and REM sleep is significant for the regulation of this process [7]. Additionally, the twin study explained that the REM sleep cycle and the pattern showed similarities between peers (same age and gender), and in the monozygotic twins, it was almost identical [4]. Meanwhile, the negative consequences of REM sleep deprivation remain poorly understood in the general population. Commonly used substances can cause REM sleep deprivation, although they might hasten sleep. The most common examples of these substances include alcohol, nicotine, cannabis, antidepressants, and sleep medications [1,9,12,13]. Full awareness of the negative effects of alcohol, cannabis, antidepressants, and nicotine use should be fully understood among the general population to avoid the effects of REM sleep deprivation.

Conclusion

In conclusion, these findings explain that dreaming can be seen as subconscious unnecessary images; however, they are significant in building your long-term memory, emotional regulation, and cognitive abilities. Furthermore, dream deprivation is slowly becoming a common health problem and a modern epidemic due to the high consumption of daily substances that cause impairment in cognitive skills, memory performance, and emotional regulation.

Declarations

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Ethics of Study

None

Conflict of interest

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