The Negative Effects of Sleep Deprivation on Firefighters and Paramedics

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Introduction

While it's commonly known that the chronic, intermittent stress experienced by firefighters and paramedics is detrimental to their health and well being, more and more attention is being paid to medical research that shows chronic sleep deprivation can have even more detrimental effects on first responders.

This paper outlines the negative effects of chronic sleep deprivation, and the fire station alerting technology that helps mitigate sleep deprivation for first responders: **Zoned Fire Station Alerting**.

The number one reason why fire-EMS departments deploy zoned fire station alerting is to mitigate sleep deprivation for its firefighters and paramedics.

A chronically exhausted first responder is at risk of many health problems due to sleep deprivation.

Negative Effects of Sleep Deprivation on First Responders

Medical research published in one of the most respected medical research databases in the United States reveals the following harmful effects of sleep deprivation:

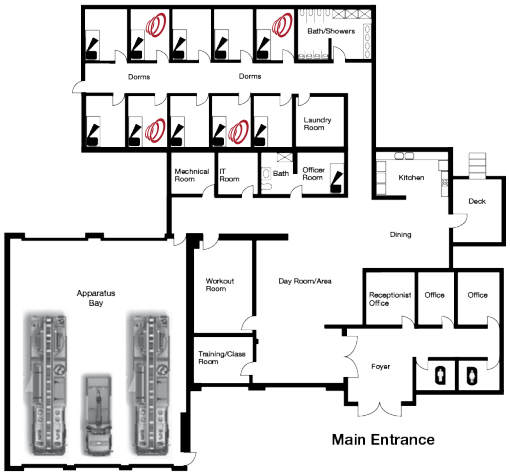
- General decreased brain activity
- Impaired working memory
- Higher risk of errors
- Impaired learning ability
- Impaired immune system
- High blood pressure
- Depression & heightened anxiety
- Impaired cardiovascular system function
- Impaired endocrine system function
- Genetic damage in blood & brain cells
- Accelerated cellular aging
- Sudden cardiac death

For more information on the specific medical research on the ill effects of sleep deprivation, please see Addendum A in this document for the abstracts from this medical research.

High blood pressure is one of many negative side effects of sleep deprivation in first responders.

Zoned Fire Station Alerting Developed Specifically to Mitigate Sleep Deprivation for First Responders

Zoned Fire Station Alerting automatically routes dispatch information and alerting directly to the unit assigned to handle the emergency, allowing responders not assigned to the call to sleep through the night.



To address and help mitigate sleep deprivation for firefighters and paramedics, zoned fire station alerting has been developed in recent years.

What is zoned fire station alerting? Zoned fire station alerting occurs when a fire station alerting system automatically routes dispatch information and alerting functions directly to and only to the unit assigned to handle the call, typically for calls that happen after everyone in the fire station has gone to sleep.

By only alerting the unit assigned to handle the call, the other first responders in the fire station get to sleep through the night (unless they are dispatched to handle a different emergency).

In simple terms, this means automated zoned dispatches are not vocalized on the fire station's PA system which wakes up all the responders in the fire station. Instead, the dispatch and related alerting functions such as tones and automated lighting route directly to zoned areas within the fire station, to responders in their individual dorm rooms, or to responders sleeping in group dorm rooms, who are assigned to handle the emergency.

Zoned Fire Station Alerting Ideal for Multi-Unit Fire Stations

Multi-unit fire stations benefit the most from zoned fire station alerting. For example, if Fire Station A has both a ladder unit and a medical unit, it's common that the medical unit will be dispatched for more runs in the middle of the night, while the ladder unit may be dispatched fewer times.

In a traditional fire station alerting scenario, the unit not assigned to handle the call would be alerted and wake up every time the assigned unit is dispatched. This creates unnecessary sleep deprivation, which takes a heavy toll on first responders.

Many firefighters & paramedics say that, once they wake up to alarms from a deep sleep, it often takes more than an hour for them to fall asleep again.

Zoned Fire Station Alerting Comes in Many Forms

There are many ways to configure zoned fire station alerting within individual fire stations. Considerations include the age of the fire station, the physical construction of the fire station and how it limits or facilitates the wiring requirements to create individual zones, and more.

The ideal time to deploy zoned fire station alerting is when a new fire station is being built.

Zoned fire station alerting can be configured by general AREAS within the fire station, to individual responders in their own dorm rooms, to individual first responders in group dorm rooms where focused speakers concentrate sound near the responders assigned to handle the call, as well as other special zoning configurations.

The bottom line is that zoned fire station alerting effectively and speedily alerts first responders assigned to handle the call, while letting other responders in the fire station sleep through the night.

Medical Research on the Negative Effects of Sleep Deprivation

The following is an overview of medical research studies that highlight the negative effects of sleep deprivation. The negative effects of sleep deprivation on responders are as harmful, or worse, than the negative effects of stress.

That's why it's so important to deploy fire station alerting technologies to mitigate sleep deprivation for firefighters and paramedics, i.e. Zoned Fire Station Alerting.

Distinct effects of acute and chronic sleep loss on DNA damage in rats.

Andersen ML[1], Ribeiro DA, Bergamaschi CT, Alvarenga TA, Silva A, Zager A, Campos RR, Tufik S.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/19258023

Learning Point

Sleep deprivation can cause genetic damage in blood and brain cells.

Author information—Abstract

The aim of this investigation was to evaluate genetic damage induced in male rats by experimental sleep loss for short-term (24 and 96 h) and long-term (21 days) intervals, as well as their respective recovery periods in peripheral blood, brain, liver and heart tissue by the single cell gel (comet) assay. Rats were paradoxically deprived of sleep (PSD) by the platform technique for 24 or 96 h, or chronically sleep-restricted (SR)

for 21 days. We also sought to verify the time course of their recovery after 24 h of rebound sleep. The results showed DNA damage in blood cells of rats submitted to PSD for 96 h. Brain tissue showed extensive geno-toxic damage in PSD rats (both 24 and 96 h), though the effect was more pronounced in the 96 h group. Rats allowed to recover from the PSD-96 h and SR-21 days treatments showed DNA damage as compared to negative controls. Liver and heart did not display any geno-toxicity activity. Corticosterone concentrations were increased after PSD (24 and 96 h) relative to control rats, whereas these levels were unaffected in the SR group. Collectively, these findings reveal that sleep loss was able to induce genetic damage in blood and brain cells, especially following acute exposure. Since DNA damage is an important step in events leading to genomic instability, this study represents a relevant contribution to the understanding of the potential health risks associated with sleep deprivation.blood pressure.

The effect of sleepiness on performance monitoring: I know what I am doing, but do I care?

Murphy TI[1], Richard M, Masaki H, Segalowitz SJ.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/16489998

Author information—Abstract

The behavioral, cognitive, and psychophysiological effects of extended wakefulness are well known. As time awake increases, errors become more common and are often attributed to lapses in attention. Such lapses can be reflected in the error-related negativity (Ne/ERN), a negative electroencephalogram deflection occurring after errors and is thought to be related to error detection or response conflict. Following the Ne/ERN, a positive deflection (error positivity, Pe) is also observed and is thought to reflect further evaluation of the error. To elicit Ne/ERNs, the Eriksen Flanker Task was administered to 17 women (aged 19-45 years) at two levels of alertness (4 and 20 h awake). After extended wakefulness, participants reported being subjectively sleepier and performing worse, but showed no significant difference in subjective effort. Across alertness conditions, they reported a similar number of subjective errors which closely matched an objective analysis of the errors. The Ne/ERN was not significantly reduced by sleepiness in contrast to the Pe which was

Learning Point

Once sleep deprived, people have trouble correcting their errors, even if they have a desire to do so.

reduced. Behavioral slowing after errors was larger in the alert than in the sleepy condition. These results show that after **20 h of wakefulness, individuals are reacting to their errors. However, further evaluation of the error, and remediation of these errors may be impaired despite continued effort.**

Sleep loss results in an elevation of cortisol levels the next evening.

Leproult R[1], Copinschi G, Buxton O, Van Cauter E.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/9415946

Author information—Abstract

Sleep curtailment constitutes an increasingly common condition in industrialized societies and is thought to affect mood and performance rather than physiological functions. There is no evidence for prolonged or delayed effects of sleep loss on the hypothalamo-pituitary-adrenal (HPA) axis. We evaluated the effects of acute partial or total sleep deprivation on the nighttime and daytime profile of cortisol levels. Plasma cortisol profiles were determined during a 32-hour period (from 1800 hours on day 1 until 0200 hours on day 3) in normal young men submitted to three different protocols: normal sleep schedule (2300-0700 hours), partial sleep deprivation (0400-0800 hours), and total sleep deprivation. Alterations in cortisol levels could only be demonstrated in the evening following the night of sleep deprivation.

Learning Points

Even after resuming a full night's sleep, the sleep-deprived person will still have elevated cortisol levels the following evening.

After normal sleep, plasma cortisol levels over the 1800-2300-hour period were similar on days 1 and 2. After partial and total sleep deprivation, plasma cortisol levels over the 1800-2300-hour period were higher on day 2 than on day 1 (37 and 45% increases, p = 0.03 and 0.003, respectively), and the onset of the quiescent period of cortisol secretion was delayed by at least 1 hour. **We conclude that even partial acute sleep loss delays the recovery of the HPA from early morning circadian stimulation and is thus likely to involve an alteration in negative glucocorticoid feedback regulation. Sleep loss could thus affect the resiliency of the stress response and may accelerate the development of metabolic and cognitive consequences of glucocorticoid excess.**

Systemic bacterial invasion induced by sleep deprivation.

Everson CA[1], Toth LA.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/10749778

Author information—Abstract

Profound sleep disruption in humans is generally believed to cause health impairments. Through comparative research, specific physical effects and underlying mechanisms altered by sleep deprivation are being elucidated. **Studies of sleep-deprived animals previously have**

Learning Point

When people are sleep deprived, bacteria from the intestines can migrate through the body causing systemic infection.

shown a progressive, chronic negative energy balance and gradual deterioration of health, which culminate in fatal bloodstream infection without an infectious focus. The present study investigated the conditions antecedent to advanced morbidity in sleep-deprived rats by determining the time course and distribution of live microorganisms in body tissues that are normally sterile. The tissues cultured for microbial growth included the blood, four major organs, six regional lymph nodes, the intestine, and the skin. **The principal finding was early infection of the mesenteric lymph nodes by bacteria presumably trans-located from the intestine and bacterial migration to and transient infection of extra-intestinal sites.** Presence of pathogenic microorganisms and their toxins in tissues constitutes a septic burden and chronic antigenic challenge for the host. Bacterial translocation and pathogenic sequelae provide mechanisms by which sleep deprivation appears to adversely affect health.

Neural basis of alertness and cognitive performance impairments during sleepiness. I. Effects of 24 h of sleep deprivation on waking human regional brain activity.

Thomas M[1], Sing H, Belenky G, Holcomb H, Mayberg H, Dannals R, Wagner H, Thorne D, Popp K, Rowland L, Welsh A, Balwinski S, Redmond D.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/11123521

Learning Point

Sleep deprivation causes decreases in overall brain activity.

Author information—Abstract

Profound The negative effects of sleep deprivation on alertness and cognitive performance suggest decreases in brain activity and function, primarily in the thalamus, a subcortical structure involved in alertness and attention, and in the prefrontal cortex, a region subserving alertness, attention, and higher-order cognitive processes. To test this hypothesis, 17 normal subjects were scanned for quantifiable brain activity changes during 85 h ofsleep deprivation using positron emission tomography (PET) and (18)Fluorine-2-deoxyglucose ((18)FDG), a marker for regional cerebral metabolic rate for glucose (CMRglu) and neuronal synaptic activity. Subjects were scanned prior to and at 24-h intervals during the sleep deprivation period, for a total of four scans per subject. During each 30 min (18)FDG uptake, subjects performed a sleep deprivation-sensitive Serial Addition/Subtraction task. Polysomnographic monitoring

confirmed that subjects were awake. Twenty-four hours of sleep deprivation, reported here, resulted in a significant decrease in global CMRglu, and significant decreases in absolute regional CMRglu in several cortical and subcortical structures. No areas of the brain evidenced a significant increase in absolute regional CMRglu. Significant decreases in relative regional CMRglu, reflecting regional brain reductions greater than the global decrease, occurred predominantly in the thalamus and prefrontal and posterior parietal cortices. Alertness and cognitive performance declined in association with these brain deactivations. **This study provides evidence that short-term sleep deprivation produces global decreases in brain activity**, with larger reductions in activity in the distributed cortico-thalamic network mediating attention and higher-order cognitive processes, and is complementary to studies demonstrating deactivation of these cortical regions during NREM and REM sleep.

Effects of sleep deprivation and exercise on cognitive, motor performance and mood.

Scott JP[1], McNaughton LR, Polman RC.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/16403541

Learning Point

When sleep deprived, resting reaction times are significantly slower.

Author information—Abstract

This study examined the effect of 30 hours of sleep deprivation and intermittent physical exercise, on both cognitive and psychomotor function as well subjective ratings of mood. Six subjects with the following physical characteristics participated in the study (Mean +/- S.D.): age 22 +/- 0.3 years, height 180 +/- 5 cm, body mass: 77 +/- 5 kg, VO2peak 44 +/- 5 ml kg(-1) min(-1). Three subjects engaged in normal sedentary activities while three others cycled on a cycle ergometer at 50% VO2peak for 20 min out of every 2 h during 30 h of sleep deprivation. One week later sleep deprivation was repeated with a cross over of subjects. Every 4 h, subjects completed simple and two-choice reaction time tasks at both rest and during exercise, a computerized tracking task, a number cancellation task, and an assessment of subjective mood state as measured by the POMS questionnaire. A 3 x 4 repeated measures ANOVA revealed that resting but not exercising reaction times were significantly slower with sleep deprivation. Sleep deprivation was also associated with significantly greater negative disturbances to

subjective vigor, fatigue and depression assessed by the Profile of Mood States questionnaire. Compared to those who have been deprived of sleep alone, individuals that performed 5 h of intermittent moderate exercise during 30 h of sleep deprivation appeared to be more vulnerable to negative mood disturbances and impairment in reaction times. This could result in greater risk of accident due to a reduced capacity to respond quickly.

Immune, inflammatory and cardiovascular consequences of sleep restriction and recovery.

Faraut B[1], Boudjeltia KZ, Vanhamme L, Kerkhofs M.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/21835655

Author information—Abstract

In addition to its effects on cognitive function, compelling evidence links sleep loss to alterations in the neuroendocrine, immune and inflammatory systems with potential negative public-health ramifications. The evidence to suggest that shorter sleep is associated with detrimental health outcomes comes from both epidemiological and experimental sleep deprivation studies. This review will focus on the post-sleep deprivation and recovery changes in immune and inflammatory functions

Learning Points

Sleep deprivation causes low-level inflammation that does not abate once a person has "recovered" from their loss of sleep.

in well-controlled sleep restriction laboratory studies. **The data obtained indicate non-specific activation of leukocyte populations and a state of low-level systemic inflammation after sleep loss. Furthermore, one night of recovery sleep does not allow full recovery of a number of these systemic immune and inflammatory markers.** We will speculate on the mechanism(s) that link(s) sleep loss to these responses and to the progression of cardiovascular disease. The immune and inflammatory responses to chronic sleep restriction suggest that chronic exposure to reduced sleep (<6 h/day) and insufficient time for recovery sleep could have gradual deleterious effects, over years, on cardiovascular pathogenesis with a heightened risk in women and in night and shift workers. Finally, we will examine countermeasures, e.g., napping or sleep extension, which could improve the recovery processes, in terms of alertness and immune and inflammatory parameters, after sleep restriction.

Learning Points

Sleep deprivation causes widespread cognitive, nervous system, and endocrine changes, and reduced cognitive functioning.

Neurophysiological effects of sleep deprivation in healthy adults, a pilot study.

Klumpers UM[1], Veltman DJ[1], van Tol MJ[2], Kloet RW[3], Boellaard R[3], Lammertsma AA[3], Hoogendijk WJ[1].

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/25608023

Author information—Abstract

ITotal sleep deprivation (TSD) may induce fatigue, neurocognitive slowing and mood changes, which are partly compensated by stress regulating brain systems, resulting in altered dopamine and cortisol levels in order to stay awake if needed. These systems, however, have never been studied in concert. At baseline, after a regular night of sleep, and the next morning after TSD, 12 healthy subjects performed a semantic affective classification functional magnetic resonance imaging (fMRI) task, followed by a [11C] raclopride positron emission tomography (PET) scan. Saliva cortisol levels were acquired at 7 time points during both days. Affective symptoms were measured using Beck Depression Inventory (BDI), Spielberger State Trait Anxiety Index (STAI) and visual analogue scales. After TSD, perceived energy levels, concentration, and speed of thought decreased significantly, whereas mood did not. During fMRI, response speed decreased for neutral words and positive targets, and accuracy decreased

trendwise for neutral words and for positive targets with a negative distracter. Following TSD, processing of positive words was associated with increased left dorsolateral prefrontal activation. Processing of emotional words in general was associated with increased insular activity, whereas contrasting positive vs. negative words showed subthreshold increased activation in the (para)hippocampal area. Cortisol secretion was significantly lower after TSD. Decreased voxel-by-voxel [11C]raclopride binding potential (BPND) was observed in left caudate. **Total Sleep Deprivation induces widespread cognitive, neurophysiologic and endocrine changes in healthy adults, characterized by reduced cognitive functioning, despite increased regional brain activity.** The blunted HPA-axis response together with altered [11C]raclopride binding in the basal ganglia indicate that sustained wakefulness requires involvement of additional adaptive biological systems.

Learning Points

Sleep deprivation lowers emotional empathy.

The effects of sleep deprivation on emotional empathy.

Guadagni V[1], Burles F, Ferrara M, Iaria G.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/25117004

Author information—Abstract

Previous studies have shown that sleep loss has a detrimental effect on the ability of the individuals to process emotional information. In this study, we tested the hypothesis that this negative effect extends to the ability of experiencing emotions while observing other individuals, i.e. emotional empathy. To test this hypothesis, we assessed emotional empathy in 37 healthy volunteers who were assigned randomly to one of three experimental groups: one group was tested before and after a night of total sleep deprivation (sleep deprivation group), a second group was tested before and after a usual night of sleep spent at home (sleep group) and the third group was tested twice during the same day (day group). Emotional empathy was assessed by using two parallel versions of a computerized test measuring direct (i.e. explicit evaluation of empathic concern) and indirect (i.e. the observer's reported physiological arousal) emotional empathy. The results revealed that the post measurements of both direct and indirect emotional empathy of participants in the sleep deprivation group were significantly lower than

those of the sleep and day groups; post measurement scores of participants in the day and sleep groups did not differ significantly for either direct or indirect emotional empathy. These data are consistent with previous studies showing the negative effect of sleep deprivation on the processing of emotional information, and extend these effects to emotional empathy. The findings reported in our study are relevant to healthy individuals with poor sleep habits, as well as clinical populations suffering from sleep disturbances.

Coping with sleep deprivation: shifts in regional brain activity and learning strategy.

Hagewoud R[1], Havekes R, Tiba PA, Novati A, Hogenelst K, Weinreder P, Van der Zee EA, Meerlo P.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/21102988

Author information—Abstract

STUDY OBJECTIVES:

dissociable cognitive strategies are used for place navigation. Spatial strategies rely on the hippocampus, an area important for flexible integration of novel information. Response strategies are more rigid and involve the dorsal striatum. These memory systems can compensate for each other in case of temporal or permanent damage. Sleep deprivation has

Learning Point

When sleep deprived, the brain compensates for negative impacts to memory by using striatal memory, which is not as effective.

adverse effects on hippocampal function. However, whether the striatal memory system can compensate for sleep-deprivation-induced hippocampal impairments is unknown.

DESIGN:

with a symmetrical maze paradigm for mice, we examined the effect of sleep deprivation on learning the location of a food reward (training) and on learning that a previously nonrewarded arm was now rewarded (reversal training).

MEASUREMENTS AND RESULTS:

five hours of sleep deprivation after each daily training session did not affect performance during training. However, in contrast with controls, sleep-deprived mice avoided a hippocampus-dependent spatial strategy and preferentially used a striatum-dependent response strategy. In line with this, the training-induced increase in phosphorylation of the transcription factor cAMP response-element binding protein (CREB) shifted from hippocampus to dorsal striatum. Importantly, although sleep-deprived mice performed well during training, performance during reversal training was attenuated, most likely due to rigidity of the striatal system they used.

CONCLUSIONS:

together, **these findings suggest that the brain compensates for negative effects of sleep deprivation on the hippocampal memory**

system by promoting the use of a striatal memory system. However, effects of sleep deprivation can still appear later on because the alternative learning mechanisms and brain regions involved may result in reduced flexibility under conditions requiring adaptation of previously formed memories.

Blood pressure increases during a simulated night shift in persons at risk for hypertension.

McCubbin JA[1], Pilcher JJ, Moore DD.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/20878512

Author information—Abstract

BACKGROUND:

Shift work with sleep disruption is a systemic stressor that may possibly be associated with blood pressure dysregulation and hypertension.

PURPOSE:

We hypothesize that rotation to a simulated night shift with sleep deprivation will produce blood pressure elevations in persons at risk for development of hypertension.

METHOD:

We examined the effects of a simulated night shift on resting blood pressure in 51 diurnal young adults without current hypertension. Resting blood pressure was monitored throughout a 24-h period

Learning Point

Sleep deprivation can cause shifts in blood pressure, particularly for those people with a family history of high blood pressure.

of total sleep deprivation with sustained cognitive work. Twelve participants (23.5%) reported one or more parents with a diagnosis of hypertension. Ten participants were classified as prehypertensive by JNC-7 criteria. Only two prehypertensive subjects reported parental hypertension.

RESULTS:

Results indicate that, as the night shift progressed, participants with a positive family history of hypertension showed significantly higher resting diastolic blood pressure than those with a negative family history of hypertension ($p = 0.007$). Prehypertensive participants showed elevated blood pressure throughout the study.

CONCLUSION:

These data suggest that **rotation to a simulated night shift with sleep deprivation may contribute to blood pressure dys-regulation in persons with a positive family history of hypertension.**

The impact of sleep loss on hippocampal function.

Prince TM[1], Abel T.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/24045505

Author information—Abstract

Hippocampal cellular and molecular processes critical for memory consolidation are affected by the amount and quality of sleep attained. Questions remain with regard to how sleep enhances memory, what parameters of sleep after learning are optimal for memory consolidation, and what underlying hippocampal molecular players are targeted by sleep deprivation to impair memory consolidation and plasticity. In this review, we address these topics with a focus on the detrimental effects of post-learning sleep deprivation on memory consolidation. Obtaining adequate sleep is challenging in a society that values "work around the clock." **Therefore, the development of interventions to combat the negative cognitive effects of sleep deprivation is key.** However, there are a limited number of therapeutics that are able to enhance cognition in the face of insufficient sleep. The identification of molecular pathways implicated in the deleterious effects of sleep deprivation on memory could potentially yield new targets for the development of more effective drugs.

Learning Point

Sleep deprivation attacks the hippocampal area of the brain and impairs memory.

Learning Points

Sleep deprivation does not necessarily affect blood sugar levels directly, but it affects weight and metabolism by increasing appetite.

Short sleep duration, glucose dysregulation and hormonal regulation of appetite in men and women.

St-Onge MP[1], O'Keeffe M, Roberts AL, RoyChoudhury A, Laferrère B.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/?term=Short+sleep+duration%2C+glucose+dysregulation+and+hormonal+regulation+of+appetite+in+men+and+women

Author information—Abstract

STUDY OBJECTIVE:

To determine the hormonal effects of reducing sleep duration under controlled feeding conditions.

DESIGN:

Randomized, crossover study.

PARTICIPANTS:

Twenty-seven normal weight, 30- to 45-yr-old men and women habitually sleeping 7-9 hr/night.

INTERVENTION:

PARTICIPANTS WERE STUDIED UNDER TWO SLEEP CONDITIONS: short (4 hr in bed) or habitual (9 hr in bed) sleep. A controlled diet was provided for each 4-day study period.

MEASUREMENTS AND RESULTS:

Fasting blood samples were obtained daily and frequent blood samples were obtained throughout day 4. The main outcomes measures included glucose, insulin, leptin, ghrelin, adiponectin, total glucagon-like peptide 1 (GLP-1) and peptide YY(3-36) (PYY(3-36)) concentrations. Body weights were reduced by 2.2 ± 0.4 lb and 1.7 ± 0.4 lb during the habitual and short sleep phases, respectively (both $P < 0.0001$). There was no effect of sleep duration on glucose, insulin, and leptin profiles (all $P > 0.05$). Ghrelin and GLP-1 responses differed by sex. Short sleep increased fasting ($P = 0.054$) and morning (08:00-12:00) ($P = 0.042$) total ghrelin in men but not women. The reverse was observed for GLP-1: afternoon levels (12:30-19:00) were lower ($P = 0.016$) after short sleep compared with habitual sleep in women but not men.

CONCLUSIONS:

These data suggest that, in the context of negative energy balance, short sleep does not lead to a state of increased insulin resistance, but may predispose to overeating via separate mechanisms in men and women.

Poor sleep as a potential causal factor in aggression and violence.

Kamphuis J[1], Meerlo P, Koolhaas JM, Lancel M.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/22305407

Learning Points

Sleep deprivation increases aggressive behavior & violence.

Author information—Abstract

Clinical observations suggest that sleep problems may be a causal factor in the development of reactive aggression and violence. In this review we give an overview of existing literature on the relation between poor sleep and aggression, irritability, and hostility. Correlational studies are supporting such a relationship. Although limited in number, some studies suggest that treatment of sleep disturbances reduces aggressiveness and problematic behavior. In line with this is the finding that **sleep deprivation actually increases aggressive behavior in animals and angriness, short-temperedness, and the outward expression of aggressive impulses in humans.** In most people poor sleep will not evoke actual physical aggression, but certain individuals, such as forensic psychiatric patients, may be particularly vulnerable to the emotional dys-regulating effects of sleep disturbances. The relation between sleep problems and aggression may be mediated by the negative effect of sleep loss on prefrontal cortical functioning. This most likely contributes to loss of control over emotions, including loss of the regulation of aggressive impulses to

context-appropriate behavior. Other potential contributing mechanisms connecting sleep problems to aggression and violence are most likely found within the central serotonergic and the hypothalamic-pituitary-adrenal-axis. Individual variation within these neurobiological systems may be responsible for amplified aggressive responses induced by sleep loss in certain individuals. It is of great importance to identify the individuals at risk, since recognition and adequate treatment of their sleep problems may reduce aggressive and violent incidents.

Unstable sleep and higher sympathetic activity during late-sleep periods of rats: implication for late-sleep-related higher cardiovascular events.

Kuo TB[1], Lai CT, Chen CY, Lee GS, Yang CC.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/22957846

Author information—Abstract

We proposed that the higher incidence of sleep fragmentation, sympathovagal imbalance and baroreceptor reflex impairment during quiet sleep may play a critical role in late-sleep-related cardiovascular events. Polysomnographic recording was performed through wireless transmission using freely moving Wistar-Kyoto rats over 24 h. The low-frequency power of arterial

Learning Point

Sleep deprivation generates a higher incidence of cardiovascular events during the later stage of a full sleep cycle (beta, alpha, theta & delta stages, followed by REM sleep and then waking up).

pressure variability was quantified to provide an index of vascular sympathetic activity. Spontaneous baroreflex sensitivity was assessed by slope of arterial pressure-RR linear regression. As compared with early-light period (Zeitgeber time 0-6 h), rats during the late-light period (Zeitgeber time 6-12 h) showed lower accumulated quiet sleep time and higher paradoxical sleep time; furthermore, during quiet sleep, the rats showed a lower δ% of electroencephalogram, more incidents of interruptions, higher σ% and higher β% of electroencephalogram, raised low-frequency power of arterial pressure variability value and lower baroreflex sensitivity parameters. During the light period, low-frequency power of arterial pressure variability during quiet sleep had a negative correlation with accumulated quiet sleep time and δ% of electroencephalogram, while it also had a positive correlation with σ% and β% of electroencephalogram and interruption events. However, late-sleep-related raised sympathetic activity and sleep fragmentation diminished when an α1-adrenoceptor antagonist was given to he rats. **Our results suggest that the higher incidence of sleep fragmentation and sympathovagal imbalance during quiet sleep may play a critical role in late-sleep-related cardiovascular events.**

Histone Acetylation Regulation in Sleep Deprivation-Induced Spatial Memory Impairment.

Duan R[1], Liu X[1], Wang T[1], Wu L[1], Gao X[1], Zhang Z[2].

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/27161370

Learning Point

Spatial memory impairment occurs when someone is sleep deprived.

Author information—Abstract

Sleep disorders negatively affect cognition and health. Recent evidence has indicated that chromatin remodeling via histone acetylation regulates cognitive function. This study aimed to investigate the possible roles of histone acetylation in sleep deprivation (SD)-induced cognitive impairment. Results of the Morris water maze test showed that **3 days of sleep deprivation can cause spatial memory impairment** in Wistar rats. SD can also histone decrease acetylation levels, increase histone deacetylase 2 (HDAC2) expression, and decrease histone acetyltransferase (CBP) expression. Furthermore, SD can reduce H3 and H4 acetylation levels in the promoters of the brain-derived neurotrophic factor (Bdnf) gene and thus significantly downregulate BDNF expression and impair the activity of key BDNF signaling pathways (pCaMKII, pErk2, and pCREB). However, treatment with the HDAC inhibitor trichostatin A attenuated all the negative effects induced by SD. Therefore, BDNF and its histone acetylation regulation may play important roles in SD-induced

spatial memory impairment, whereas HDAC inhibition possibly confers protection against SD-induced impairment in spatial memory and hippocampal functions.

Effects of 72hours total sleep deprivation on male astronauts' executive functions and emotion.

Liu Q[1], Zhou R[2], Liu L[3], Zhao X[4].

Learning Points

Sleep deprivation affects one's ability to process information, and influences people to shift to a negative mood.

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/26112064

Author information—Abstract

BACKGROUND:

To provide evidence for better understanding stressful situations, the present study was designed to investigate the specific physiological and psychological responses under stressful situations like social isolation and sleep deprivation.

METHODS:

Twelve healthy male adults (age: 18-30years old) who participated in our study were randomized to the 72 hours of social isolation and 72 hours of sleep deprivation experimental conditions. Performances (event-related potentials and physiological activities) on the Go/Nogo task which reflected the executive functions were accessed at baseline (pretest) and after 72-hour of the experiment (posttest).

RESULTS:

The results showed that compared to the social isolation, the participants got strengthened heart rate (HR), weakened HR variability and smaller amplitude of the P300 under the sleep deprivation condition; moreover, they had lower positive emotion and higher negative mood in the post test.

CONCLUSIONS:

The present study indicated that sleep deprivation specifically influenced the intensity of task-relevant information processing, mood and vagal tone.

Learning Points

Mitigating sleep deprivation into middle age improves cognitive functioning and helps to protect against age-related cognitive declines (dementia/Alzheimers).

Sleep, cognition, and normal aging: integrating a half century of multidisciplinary research.

Scullin MK[1], Bliwise DL[2].

Weblink: https://www.ncbi.nlm.nih.gov/pubmed/25620997

Author information—Abstract

Sleep is implicated in cognitive functioning in young adults. With increasing age, there are substantial changes to sleep quantity and quality, including changes to slow-wave sleep, spindle density, and sleep continuity/fragmentation. A provocative question for the field of cognitive aging is whether such changes in sleep physiology affect cognition (e.g., memory consolidation). We review nearly a half century of research across seven diverse correlational and experimental domains that historically have had little crosstalk. Broadly speaking, sleep and cognitive functions are often related in advancing age, though the prevalence of null effects in healthy older adults (including correlations in the unexpected, negative direction) indicates that age may be an effect modifier of these associations. We interpret the literature as suggesting that **maintaining good sleep quality, at least in young adulthood and middle age, promotes better cognitive functioning and serves to protect against age-related cognitive declines.**

For more information on how Locution Systems' PrimeAlert® Zoned Fire Station Alerting can reduce sleep deprivation in first responders, please contact Locution Systems at: 303.301.7300, info@locution.com, or www.locution. Mountain Time USA.

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