Journal Scan

U. C. Ojha
Senior Specialist & Head - Department of Respiratory Medicine, E.S.I Hospital, New Delhi

Indian J Sleep Med 2016; 11.4, 161-169

1. Sleep Disord. 2015;2015:607148.

Residual Effects of Sleep Medications Are Commonly Reported and Associated with Impaired Patient-Reported Outcomes among Insomnia Patients in the United States.

Fitzgerald T(1), Vietri J(2).

Author information: (1)Merck & Co., Whitehouse Station, NJ 08889, USA. (2)Kantar Health, 20121 Milan, Italy.

Study Objective: To measure the association of symptoms attributed to residual effects of sleep medication (e.g., drowsiness, difficulty concentrating, and impaired memory) on self-reported functioning and satisfaction with these medications. Methods. Individuals using prescription medications for insomnia were invited to complete an Internet-based survey. Respondents were compared according to the presence of self-reported residual effects; relationships between severity of these effects and outcomes were modeled using regression.

Measures included the Brief Insomnia Questionnaire, Work Productivity and Activity Impairment Questionnaire, and SATMED-Q. Subgroup analyses were conducted with patients aged ≥65 years. Approximately 80% reported experiencing ≥1 residual effect. The severity of residual effects was associated with increased residual effect-related work impairment, including absenteeism (RR = 1.46, $p < 0.001$), presenteeism (RR = 1.12, $p < 0.001$), overall work impairment (RR = 1.13, $p < 0.001$), and nonwork activity impairment (RR = 1.11, $p < 0.001$). More severe residual symptoms were also associated with increased difficulty in home management (Beta = .31, $p < 0.001$), ability to work (Beta = .31, $p < 0.001$), social relationships, (Beta = .32, $p < 0.001$), close personal relationships (Beta = .30, $p < 0.001$), and lower medication satisfaction (Beta = -.37, $p < 0.001$). Conclusions. Individuals using medications for insomnia commonly experience symptoms considered as residual effects, and these symptoms are associated with greater interference of sleep-related problems at work, at home, and with social relationships.

2. Front Neurol. 2015 Dec 21;6:263.

Fifty Percent Prevalence of Extracampine Hallucinations in Parkinson's Disease Patients.

Wood RA(1), Hopkins SA(2), Moodley KK(1), Chan D(3).

Author information: (1)Department of Medicine, Brighton and Sussex Medical School , Brighton, UK. (2)Department of Medicine for the Elderly, Addenbrooke's Hospital, Cambridge, UK. (3)Department of Clinical Neurosciences, University of Cambridge, Cambridge, UK.

Extracampine hallucinations (EH), the sense of a presence or fleeting movement in the absence of an associated visual percept, have been reported in Parkinson's disease (PD) patients but their prevalence, characteristics, and temporal relationship to visual hallucinations (VH) remain unclear. Given that, VH are predictive of cognitive impairment in PD, improved understanding of EH may have significant prognostic implications. The objective of this study is to evaluate the prevalence and characteristics of EH in a large unselected population with PD and to assess the temporal relationship between EH, VH, and memory decline. Cross-sectional data were collected from 414 PD patients using a questionnaire circulated via an online patient

community. Data were obtained regarding the occurrence, timing, and characteristics of VH and EH and symptoms of PD, disease duration, disease severity, and medication history. About 50.4% of respondents reported EH and 15.5% reported VH. EH were typically experienced alongside, rather than behind, the individual ($p < 0.001$) without clear lateralization ($p = 0.438$) and were more likely to be of unfamiliar presences ($p < 0.001$). The occurrence of EH was associated with Hoehn and Yahr score ($p = 0.002$) but not disease duration ($p = 0.158$). EH onset was associated with VH onset ($p = 0.046$) and occurred after the onset of anosmia ($p < 0.001$), cognitive decline ($p = 0.002$), and sleep disturbance ($p =0.002$). The reported prevalence of EH in PD patients was threefold greater than that of VH, with similar timings of onset, suggesting that EH are under-recognized and under-reported. Further work is needed to determine whether EH are predictive of cognitive decline.

3. Brain Sci. 2015 Dec 29;6(1). pii: E1.

REM-Enriched Naps Are Associated with Memory Consolidation for Sad Stories and Enhance Mood-Related Reactivity.

Gilson M(1)(2), Deliens G(3)(4)(5), Leproult R(6)(7), Bodart A(8)(9), Nonclercq A(10), Ercek R(11), Peigneux P(12)(13).

Author information: (1)UR2NF-Neuropsychology and Functional Neuroimaging Research Unit, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. Medhi.Gilson@ulb.ac.be. (2)UNI-ULB Neuroscience Institute, Université Libre de Bruxelles (ULB), avenue F.D. Roosevelt 50, Bruxelles, Belgium. Medhi.Gilson@ulb.ac.be. (3)UR2NF-Neuropsychology and Functional Neuroimaging Research Unit, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. gaetane.deliens@ulb.ac.be. (4)UNI-ULB Neuroscience Institute, Université Libre de Bruxelles (ULB), avenue F.D. Roosevelt 50, Bruxelles, Belgium. gaetane.deliens@ulb.ac.be. (5)CO3-Consciousness, Cognition & Computation Group, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. gaetane.deliens@ulb.ac.be. (6)UR2NF-Neuropsychology and Functional Neuroimaging Research Unit, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. rleproul@ulb.ac.be. (7)UNI-ULB Neuroscience Institute, Université Libre de Bruxelles (ULB), avenue F.D. Roosevelt 50, Bruxelles, Belgium. rleproul@ulb.ac.be. (8)UR2NF-Neuropsychology and Functional Neuroimaging Research Unit, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. alice.bodart@ulb.ac.be. (9)UNI-ULB Neuroscience Institute, Université Libre de Bruxelles (ULB), avenue F.D. Roosevelt 50, Bruxelles, Belgium. alice.bodart@ulb.ac.be. (10)LISA-Laboratories of Image, Signal processing and Acoustics, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. anoncler@ulb.ac.be. (11)LISA-Laboratories of Image, Signal processing and Acoustics, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. rercek@ulb.ac.be. (12)UR2NF-Neuropsychology and Functional Neuroimaging Research Unit, avenue F.D. Roosevelt 50, Bruxelles 1050, Belgium. Philippe.Peigneux@ulb.ac.be. (13)UNI-ULB Neuroscience Institute, Université Libre de Bruxelles (ULB), avenue F.D. Roosevelt 50, Bruxelles, Belgium. Philippe.Peigneux@ulb.ac.be.

Emerging evidence suggests that emotion and affect modulate the relation between sleep and cognition. In the present study, we investigated the role of rapid-eye movement (REM) sleep in mood regulation and memory consolidation for sad stories.In a counterbalanced design, participants (n = 24) listened to either a neutral or a sad story during two sessions, spaced one week apart. After listening to the story, half of the participants had a short (45 min) morning nap. The other half had a long (90 min) morning nap, richer in REM and N2 sleep. Story recall, mood evolution and changes in emotional response to the re-exposure to the story were assessed after the nap. Although recall performance was similar for sad and neutral stories irrespective of nap duration, sleep measures were correlated with recall performance in the sad story condition only. After the long nap, REM sleep density positively correlated with retrieval performance, while re-exposure to the sad story led to diminished mood and increased skin conductance levels. Our results suggest that REM sleep may not only be associated with the consolidation of intrinsically sad material, but also enhances mood reactivity, at least on the short term.

4. Front Neurosci. 2015 Dec 8;9:445.

Learning, Memory, and Executive Function in New MDMA Users: A 2-Year Follow-Up Study.

Wagner D(1), Tkotz S(1), Koester P(1), Becker B(1), Gouzoulis-Mayfrank E(1), Daumann J(1).

Author information: (1)Department of Psychiatry and Psychotherapy, University of Cologne Cologne, Germany.

3,4-Methylenedioxymethamphetamine (MDMA) is associated with changes in neurocognitive performance. Recent studies in laboratory animals have provided additional support for the neurodegeneration hypothesis. However, results from animal research need to be applied to humans with caution. Moreover, several of the studies that examine MDMA users suffer from methodological shortcomings. Therefore, a prospective cohort study was designed in order to overcome these previous methodological shortcomings and to assess the relationship between the continuing use of MDMA and cognitive performance in incipient MDMA users. It was hypothesized that, depending on the amount of MDMA taken, the continued use of MDMA over a 2-year period would lead to further decreases in cognitive performance, especially in visual paired association learning tasks. Ninety-six subjects were assessed, at the second follow-up assessment: 31 of these were non-users, 55 moderate-users, and 10 heavy-users. Separate repeated measures analyses of variance were conducted for each cognitive domain, including attention and information processing speed, episodic memory, and executive functioning. Furthermore, possible confounders including age, general intelligence, cannabis use, alcohol use, use of other concomitant substances, recent medical treatment, participation in sports, level of nutrition, sleep patterns, and subjective well-being were assessed. The Repeated measures analysis of variance (rANOVA) revealed that a marginally significant change in immediate and delayed recall test performances of visual paired associates learning had taken place within the follow-up period of 2 years. No further deterioration in continuing MDMA-users was observed in the second follow-up period. No significant differences with the other neuropsychological tests were noted. It seems that MDMA use can impair visual paired associates learning in new users. However, the groups differed in their use of concomitant use of illicit drugs. Therefore, performance differences between the groups cannot completely ascribed to the use of MDMA.

5. PLoS One. 2015 Dec 15;10(12):e0144720.

Coupling of Thalamocortical Sleep Oscillations Are Important for Memory Consolidation in Humans.

Niknazar M(1)(2), Krishnan GP(1), Bazhenov M(1), Mednick SC(2).

Author information: (1)Department of Cell Biology & Neuroscience, University of California Riverside, 900 University Ave, Riverside, CA, 92521, United States of America. (2)Department of Psychology, University of California Riverside, 900 University Ave, Riverside, CA, 92521, United States of America.

Sleep, specifically non-rapid eye movement (NREM) sleep, is thought to play a critical role in the consolidation of recent memories. Two main oscillatory activities observed during NREM, cortical slow oscillations (SO, 0.5-1.0 Hz) and thalamic spindles (12-15 Hz), have been shown to independently correlate with memory improvement. Yet, it is not known how these thalamocortical events interact, or the significance of this interaction, during the consolidation process. Here, we found that systemic administration of the GABAergic drug (zolpidem) increased both the phase-amplitude coupling between SO and spindles, and verbal memory improvement in humans. These results suggest that thalamic spindles that occur during transitions to the cortical SO Up state are optimal for memory consolidation. Our study predicts that the timely interactions between cortical and thalamic events during consolidation, contribute to memory improvement and is mediated by the level of inhibitory neurotransmission.

6. EBP Briefs. 2015 May;10(1):1-21.

Sleep Disorders as a Risk to Language Learning and Use.

McGregor KK, Alper RM.

CLINICAL QUESTION: Are people with sleep disorders at higher risk for language learning deficits than healthy sleepers?

METHOD: Scoping Review.

STUDY SOURCES: PubMed, Google Scholar, Trip Database, ClinicalTrials.gov.

SEARCH TERMS: sleep disorders AND language AND learning; sleep disorders language learning -deprivation -epilepsy; sleep disorders AND verbal learning.

NUMBER OF INCLUDED STUDIES: 36.

PRIMARY RESULTS: Children and adults with sleep disorders were at a higher risk for language problems than healthy sleepers. The language problems typically co-occurred with problems of attention and executive function (in children and adults), behavior (in children), and visual-spatial processing (in adults). Effects were typically small. Language problems seldom rose to a level of clinical concern but there were exceptions involving phonological deficits in children with sleep-disordered breathing and verbal memory deficits among adults with sleep-disordered breathing or idiopathic REM sleep behavior disorder.

CONCLUSIONS: Case history interviews should include questions about limited sleep, poor-quality sleep, snoring, and excessive daytime sleepiness. Medical referrals for clients with suspected sleep disorders are prudent.

7. Front Neurol. 2015 Nov 24;6:241.

Sleep and Motor Learning: Implications for Physical Rehabilitation After Stroke.

Gudberg C(1), Johansen-Berg H(2).

Author information: (1)Oxford Centre for Functional MRI of the Brain (FMRIB), Nuffield Department of

Clinical Neurosciences, University of Oxford, John Radcliffe Hospital, Oxford, UK; Sleep and Circadian Neuroscience Institute (SCNi), Nuffield Department of Clinical Neurosciences, University of Oxford, Sir William Dunn School of Pathology, Oxford, UK. (2)Oxford Centre for Functional MRI of the Brain (FMRIB), Nuffield Department of Clinical Neurosciences, University of Oxford, John Radcliffe Hospital, Oxford, UK.

Sleep is essential for healthy brain function and plasticity underlying learning and memory. In the context of physical impairment such as following a stroke, sleep may be particularly important for supporting critical recovery of motor function through similar processes of reorganization in the brain. Despite a link between stroke and poor sleep, current approaches to rehabilitative care often neglect the importance of sleep in clinical assessment and treatment. This review assimilates current evidence on the role of sleep in motor learning, with a focus on the implications for physical rehabilitation after stroke. We further outline practical considerations for integrating sleep assessment as a vital part of clinical care.

8. Front Hum Neurosci. 2015 Nov 19;9:624.

Automatic Sleep Spindle Detection and Genetic Influence Estimation Using Continuous Wavelet Transform.

Adamczyk M(1), Genzel L(2), Dresler M(3), Steiger A(1), Friess E(1).

Author information: (1)Max Planck Institute of Psychiatry Munich, Germany. (2)Centre for Cognitive and Neural Systems, University of Edinburgh Edinburgh, UK. (3)Max Planck Institute of Psychiatry Munich, Germany; Donders Institute for Brain, Cognition and Behaviour Nijmegen, Netherlands.

Mounting evidence for the role of sleep spindles in neuroplasticity has led to an increased interest in these non-rapid eye movement (NREM) sleep oscillations. It has been hypothesized that fast and slow spindles might play a different role in memory processing. Here, we present a new sleep spindle detection algorithm utilizing a continuous wavelet transform (CWT) and individual adjustment of slow and fast spindle frequency ranges. Eighteen nap recordings of ten subjects were used for algorithm validation. Our method was compared with both a human scorer and a commercially available SIESTA spindle detector. For the validation set, mean agreement between our detector and human scorer measured during sleep stage 2 using kappa coefficient was 0.45, whereas mean agreement between our detector and SIESTA algorithm was 0.62. Our algorithm was also applied to sleep-related memory consolidation data previously analyzed with a SIESTA detector and confirmed previous findings of significant correlation between spindle density and declarative memory consolidation. We then applied our method to a study in monozygotic (MZ) and dizygotic (DZ) twins, examining the genetic component of slow and fast sleep spindle parameters. Our analysis revealed strong genetic influence on variance of all slow spindle parameters, weaker genetic effect on fast spindles, and no effects on fast spindle density and number during stage 2 sleep.

9. BMC Neurol. 2015 Dec 3;15:251.

Correlation of Tc-99 m ethyl cysteinate dimer single-photon emission computed tomography and clinical presentations in patients with low cobalamin status.

Tu MC(1)(2), Lo CP(3)(4), Chen CY(5)(4)(6), Huang CF(7)(4).

Author information: (1)Department of Neurology, Taichung Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taichung, Taiwan. tmctmc30@yahoo.com.tw. (2) School of Medicine, Tzu Chi University, Hualien, Taiwan. tmctmc30@yahoo.com.tw. (3)Department of Radiology, Taichung Tzu Chi Hospital, Buddhist Tzu Chi Foundation, Taichung, Taiwan. (4)School of Medicine, Tzu Chi University, Hualien, Taiwan. (5)Department of Nuclear Medicine, Taichung Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taichung, Taiwan. (6)Graduate Institute of Medical Imaging and Radiological Sciences, Central Taiwan, University of Science and Technology, Taichung, Taiwan. (7)Department of Neurology, Taichung Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Taichung, Taiwan.

BACKGROUND: Cobalamin (Cbl) deficiency has been associated with various neuropsychiatric symptoms of different severities. While some studies dedicated in structural neuroimaging credibly address negative impact of low Cbl status, functional imaging reports are limited. We herein retrospectively review the correlation of Tc-99 m ethyl cysteinate dimer single-photon emission computed tomography (Tc-99 m-ECD SPECT) and clinical presentations among patients with low serum cobalamin (Cbl) status (<250 pg/ml).

METHODS: Twelve symptomatic patients with low serum Cbl status were enrolled. Clinical presentations, Tc-99 m-ECD SPECT, and neuropsychological tests were reviewed.

RESULTS: Dysexecutive syndrome (67 %), forgetfulness (50 %), attention deficits (42 %), and sleep disorders (33 %) constituted the major clinical presentations. All patients (100 %) had temporal hypoperfusion on the Tc-99 m-ECD SPECT. Five patients (42 %) had hypoperfusion restricted within temporal regions and deep nuclei; seven patients (58 %) had additional frontal hypoperfusion. In patients with hypoperfusion restricted

within temporal regions and deep nuclei, psychiatric symptoms with spared cognition were their main presentations. Among patients with additional frontal hypoperfusion, six of seven patients (86 %) showed impaired cognitive performances (two of them were diagnosed as having dementia). Among ten patients who finished neuropsychological tests, abstract thinking (70 %) was the most commonly affected, followed by verbal fluency (60 %), short-term memory (50 %), and attention (50 %). Anxiety and sleep problems were the major clinically remarkable psychiatric features (33 % both). Four Tc-99 m-ECD SPECT follow-up studies were available; the degree and extent of signal reversal correlated with cognitive changes after Cbl replacement therapy. CONCLUSIONS: Our TC-99 m-ECD SPECT observations provide pivotal information of neurobiological changes within basal ganglia and fronto-temporal regions in conjunction with disease severity among patients with Cbl deficiency.Hypoperfusion within thalamus/basal ganglia and temporal regions may be seen in the earlier state of Cbl deficiency, when psychiatric symptoms predominate. Hypoperfusion beyond thalamus/basal ganglia and involving frontal regions appears when cognitive problems, mostly dysexecutive syndrome, are manifested. Symmetric hypofrontality of SPECT in the context of dysexcutive syndrome serves as a distinguishing feature of non-amnestic mild cognitive impairment attributed to Cbl deficiency. Concordant with TC-99 m-ECD SPECT findings, the psychiatric symptoms and dysexcutive syndrome undergird impaired limbic and dorsolateral prefrontal circuits originating from basal ganglia respectively.

10. Curr Sleep Med Rep. 2015 Mar;1(1):1-8.

Distinct functional states of astrocytes during sleep and wakefulness: Is norepinephrine the master regulator?

O'Donnell J(1), Ding F(1), Nedergaard M(1).

Author information: (1)Center for Translational Neuromedicine, University of Rochester Medical School, Rochester, NY 14642, USA.

Astrocytes are the chief supportive cells in the central nervous system, but work over the past 20 years have documented that astrocytes also contribute to complex neural processes, such as working memory. Recent discoveries of norepinephrine-mediated astrocytic Ca2+ responses have raised the possibility that astrocytic activity in the adult brain is driven by global responses to changes in behavioral state. Moreover, analysis of the interstitial space volume suggests that astrocytes may undergo changes in cell volume in response to activation of norepinephrine receptors. This review will focus on what is known about astrocytic functions within the nervous system, and how these functions interrelate with rapid changes in behavioral state mediated by norepinephrine signaling.

11. MEDtube Sci. 2015 Mar;3(1):35-40.

The Neuroprotective Aspects of Sleep.

Eugene AR(1), Masiak J(2).

Author information: (1)Division of Clinical Pharmacology/Anesthesia Research, Department of Molecular Pharmacology and Experimental Therapeutics, Gonda 19, Mayo Clinic, 200 First Street SW, Rochester, Minnesota 55905, USA. (2)Neurophysiology Unit, Department of Psychiatry, Medical University of Lublin, ul. Gluska 1, Lublin 20-439, Poland.

Sleep is an important component of human life, yet many people do not understand the relationship between the brain and the process of sleeping. Sleep has been proven to improve memory recall, regulate metabolism, and reduce mental fatigue. A minimum of 7 hours of daily sleep seems to be necessary for proper cognitive and behavioral function. The emotional and mental handicaps associated with chronic sleep loss as well as the highly hazardous situations which can be contributed to the lack of sleep is a serious concern that people need to be aware of. When one sleeps, the brain reorganizes and recharges itself, and removes toxic waste byproducts which have accumulated throughout the day. This evidence demonstrates that sleeping can clear the brain and help maintain its normal functioning. Multiple studies have been done to determine the effects of total sleep deprivation; more recently some have been conducted to show the effects of sleep restriction, which is a much more common occurrence, have the same effects as total sleep deprivation. Each phase of the sleep cycle restores and rejuvenates the brain for optimal function. When sleep is deprived, the active process of the glymphatic system does not have time to perform that function, so

toxins can build up, and the effects will become apparent in cognitive abilities, behavior, and judgment. As a background for this paper we have reviewed literature and research of sleep phases, effects of sleep deprivation, and the glymphatic system of the brain and its restorative effect during the sleep cycle.

12.J Int Neuropsychol Soc. 2015 Nov;21(10):802-15.

Age Moderates the Association of Aerobic Exercise with Initial Learning of an Online Task Requiring Cognitive Control.

O'Connor PJ(1), Tomporowski PD(1), Dishman RK(1).

Author information: (1)Department of Kinesiology, University of Georgia, Athens,Georgia.

The aim of this study was to examine whether people differed in change in performance across the first five blocks of an online flanker task and whether those trajectories of change were associated with self-reported aerobic or resistance exercise frequency according to age. A total of 8752 men and women aged 13-89 completed a lifestyle survey and five 45-s games (each game was a block of ~46 trials) of an online flanker task. Accuracy of the congruent and incongruent flanker stimuli was analyzed using latent class and growth curve modeling adjusting for time between blocks, whether the blocks occurred on the same or different days, education, smoking, sleep, caffeinated coffee and tea use, and Lumosity training status ("free play" or part of a "daily brain workout"). Aerobic and resistance exercise were unrelated to first block accuracies. For the more cognitively demanding incongruent flanker stimuli, aerobic activity was positively related to the linear increase in accuracy [B=0.577%, 95% confidence interval (CI), 0.112 to 1.25 per day above the weekly mean of 2.8 days] and inversely related to the quadratic deceleration of accuracy gains (B=-0.619% CI, -1.117 to -0.121 per day). An interaction of aerobic activity with age indicated that active participants younger than age 45 had a larger linear increase and a smaller quadratic deceleration compared to other participants. Age moderates the association between self-reported aerobic, but not self-reported resistance, exercise and changes in cognitive control that occur with practice during incongruent presentations across five blocks of a 45-s online, flanker task.

13.Child Dev Perspect. 2015 Sep;9(3):183-189.

Sleep as a window into early neural development: Shifts in sleep-dependent learning effects across early childhood.

Gómez RL(1), Edgin JO(1).

Author information: (1)The University Of Arizona.

Sleep is an important physiological state for the consolidation and generalization of new learning in children and adults. We review the literature on sleep-dependent memory consolidation and generalization in infants and preschool children and place the findings in the context of the development of the neural systems underlying memory (hippocampus and its connections to cortex). Based on the extended trajectory of hippocampal development, transitions in the nature of sleep-dependent learning are expected. The studies reviewed here show shifts in the nature of sleep-dependent learning across early childhood, with sleep facilitating generalization in infants but enhancing precise memory after 18-24 months of age. Future studies on sleep-dependent learning in infants and young children must take these transitions in early brain development into account.

14.Front Neurol. 2015 Oct 23;6:224.

The Role of Sleep and Sleep Disorders in the Development, Diagnosis, and Management of Neurocognitive Disorders.

Miller MA(1).

Author information: (1)Warwick Medical School, University of Warwick , Coventry , UK.

It is becoming increasingly apparent that sleep plays an important role in the maintenance, disease prevention, repair, and restoration of both mind and body. The sleep and wake cycles are controlled by the

pacemaker activity of the superchiasmic nucleus in the hypothalamus but can be disrupted by diseases of the nervous system causing disordered sleep. A lack of sleep has been associated with an increase in all-cause mortality. Likewise, sleep disturbances and sleep disorders may disrupt neuronal pathways and have an impact on neurological diseases. Sleep deprivation studies in normal subjects demonstrate that a lack of sleep can cause attention and working memory impairment. Moreover, untreated sleep disturbances and sleep disorders such as obstructive sleep apnoe (OSA) can also lead to cognitive impairment. Poor sleep and sleep disorders may present a significant risk factor for the development of dementia. In this review, the underlying mechanisms and the role of sleep and sleep disorders in the development of neurocognitive disorders [dementia and mild cognitive impairment (MCI)] and how the presence of sleep disorders could direct the process of diagnosis and management of neurocognitive disorders will be discussed.

16. Biomed Res Int. 2015;2015:817595.

Cortical Structural Connectivity Alterations in Primary Insomnia: Insights from MRI-Based Morphometric Correlation Analysis.

Zhao L(1), Wang E(2), Zhang X(2), Karama S(3), Khundrakpam B(1), Zhang H(4), Guan M(2), Wang M(2), Cheng J(5), Shi D(2), Evans AC(1), Li Y(2).

Author information: (1)McConnell Brain Imaging Centre, Montreal Neurological Institute, McGill University, Montreal, QC, Canada H3A 2B4. (2)Department of Radiology, Henan Provincial People's Hospital, People's Hospital of Zhengzhou University, Henan 450003, China. (3)McConnell Brain Imaging Centre, Montreal Neurological Institute, McGill University, Montreal, QC, Canada H3A 2B4 ; Douglas Mental Health University Institute, McGill University, Montreal, QC, Canada H4H 1R3. (4)Department of Neurology, Henan Provincial People's Hospital, People's Hospital of Zhengzhou University, Henan 450003, China. (5)MRI Division, First Affiliated Hospital of Zhengzhou University, Zhengzhou, Henan 450052, China.

The etiology and maintenance of insomnia are proposed to be associated with increased cognitive and physiological arousal caused by acute stressors and associated cognitive rumination. A core feature of such hyperarousal theory of insomnia involves increased sensory processing that interferes with the onset and maintenance of sleep. In this work, we collected structural magnetic resonance imaging data from 35 patients with primary insomnia and 35 normal sleepers and applied structural covariance analysis to investigate whether insomnia is associated with disruptions in structural brain networks centered at the sensory regions (primary visual, primary auditory, and olfactory cortex). As expected, insomnia patients showed increased structural covariance in cortical thickness between sensory and motor regions. We also observed trends of increased covariance between sensory regions and the default-mode network, and the salience network regions, and trends of decreased covariance between sensory regions and the frontoparietal working memory network regions, in insomnia patients. The observed changes in structural covariance tended to correlated with poor sleep quality. Our findings support previous functional neuroimaging studies and provide novel insights into variations in brain network configuration that may be involved in the pathophysiology of insomnia.

17.Nat Commun. 2015 Oct 28;6:8729.

Auditory feedback blocks memory benefits of cueing during sleep.

Schreiner T(1)(2), Lehmann M(1)(3), Rasch B(1)(2)(4).

Author information: (1)Department of Psychology, University of Zurich, 8050 Zurich, Switzerland. (2)Department of Psychology, University of Fribourg, 1701 Fribourg, Switzerland. (3)Psychiatric University Hospital Zurich, Clinic of Affective Disorders and General Psychiatry, 8032 Zurich, Switzerland. (4)Zurich Center for Interdisciplinary Sleep Research (ZiS), 8091 Zurich, Switzerland.

It is now widely accepted that re-exposure to memory cues during sleep reactivates memories and can improve later recall. However, the underlying mechanisms are still unknown. As reactivation during wakefulness renders memories sensitive to updating, it remains an intriguing

question whether reactivated memories during sleep also become susceptible to incorporating further information after the cue. Here we show that the memory benefits of cueing Dutch vocabulary during sleep are in fact completely blocked when memory cues are directly followed by either correct or conflicting auditory feedback, or a pure tone. In addition, immediate (but not delayed) auditory stimulation abolishes the characteristic increases in oscillatory theta and spindle activity typically associated with successful reactivation during sleep as revealed by high-density electroencephalography. We conclude that plastic processes associated with theta and spindle oscillations occurring during a sensitive period immediately after the cue are necessary for stabilizing reactivated memory traces during sleep.

18. BMC Med Genet. 2015 Oct 24;16:96.

Genetics, sleep and memory: a recall-by-genotype study of ZNF804A variants and sleep neurophysiology.

Hellmich C(1), Durant C(2), Jones MW(3), Timpson NJ(4), Bartsch U(5), Corbin LJ(6).

Author information: (1)School of Physiology and Pharmacology, University of Bristol, Bristol, UK. charlotte.hellmich@googlemail.com. (2)Clinical Research and Imaging Centre (CRICBristol), University of Bristol, Bristol, UK. Claire.Durant@bristol.ac.uk. (3)School of Physiology and Pharmacology, University of Bristol, Bristol, UK. Matt.Jones@bristol.ac.uk. (4)MRC Integrative Epidemiology Unit at University of Bristol, Bristol, UK. N.J.Timpson@bristol.ac.uk. (5)School of Physiology and Pharmacology, University of Bristol, Bristol, UK. Ullrich.Bartsch@bristol.ac.uk. (6)MRC Integrative Epidemiology Unit at University of Bristol, Bristol, UK. laura.corbin@bristol.ac.uk.

BACKGROUND: Schizophrenia is a complex, polygenic disorder for which over 100 genetic variants have been identified that correlate with diagnosis. However, the biological mechanisms underpinning the different symptom clusters remain undefined. The rs1344706 single nucleotide polymorphism within ZNF804A was among the first genetic variants found to be associated with schizophrenia. Previously, neuroimaging and cognitive studies have revealed several associations between rs1344706 and brain structure and function. The aim of this study is to use a recall-by-genotype (RBG) design to investigate the biological basis for the association of ZNF804A variants with schizophrenia. A RBG study, implemented in a population cohort, will be used to evaluate the impact of genetic variation at rs1344706 on sleep neurophysiology and procedural memory consolidation in healthy participants.

METHODS/DESIGN: Participants will be recruited from the Avon Longitudinal Study of Parents and Children (ALSPAC) on the basis of genotype at rs1344706 (n = 24). Each participant will be asked to take part in two nights of in-depth sleep monitoring (polysomnography) allowing collection of neurophysiological sleep data in a manner not amenable to large-scale study. Sleep questionnaires will be used to assess general sleep quality and subjective sleep experience after each in-house recording. A motor sequencing task (MST) will be performed before and after the second night of polysomnography. In order to gather additional data about habitual sleep behaviour participants will be asked to wear a wrist worn activity monitor (actiwatch) and complete a sleep diary for two weeks.

DISCUSSION: This study will explore the biological function of ZNF804A genotype (rs1344706) in healthy volunteers by examining detailed features of sleep architecture and physiology in relation to motor learning. Using a RBG approach will enable us to collect precise and detailed phenotypic data whilst achieving an informative biological gradient. It would not be feasible to collect such data in the large sample sizes that would be required under a random sampling scheme. By dissecting the role of individual variants associated with schizophrenia in this way, we can begin to unravel the complex genetic mechanisms of psychiatric disorders and pave the way for future development of novel therapeutic approaches.