Journal Scan

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

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Sleep and cognitive function Neurology. 2009 Nov 24; 73(21):e99-e103.

Education research : cognitive performance is preserved in sleep deprived neurology residents.

Reimann M, Manz R, Prieur S, Reichmann H, Ziemssen T.

ANF Laboratory, Department of Neurology, University Hospital Carl Gustav Carus, Dresden University of Technology, Dresden, Germany.
Email: manjareimann@uniklinikum-dresden.de

OBJECTIVE: To test the hypotheses that sleep deprivation in neurology residents is associated with performance deficits and that vigilance and cognitive performance is more compromised after overnight on-call duty compared to night shift.

METHODS: Thirty-eight neurology residents of a university teaching hospital participated in a prospective single-blind comparison study. Residents were recruited according to their working schedule and divided into 3 groups: 24 hours overnight on-call duty, night shift, and regular day shift (controls). All participants underwent serial measurements of sleepiness and cognitive performance in the morning directly after or before their shift. Pupillary sleepiness test and Paced Auditory Serial Addition Test were applied. Perceived sleepiness was assessed by a questionnaire.

RESULTS: Sleepiness was increased in residents after night shift and overnight call compared to controls while the type of night duty was not associated with the extent of sleepiness. Sleep-deprived residents did not show any performance deficits on the Paced Auditory Serial Addition Test. Cognitive performance was not associated with sleepiness measures.

CONCLUSIONS: Night shift and overnight call duty have a similar impact on alertness in neurology residents. Sleep deprived neurology residents may be able to overcome sleep loss-related performance difficulties for short periods.

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Sleep deprivation differentially impairs cognitive performance in abstinent methylenedioxymethamphetamine ("Ecstasy") users.

McCann UD, Wilson MJ, Sgambati FP, Ricaurte GA.

Department of Psychiatry and Neurology, The Johns Hopkins School of Medicine, Baltimore, MD 21224, USA. Email: umccann@jhmi.edu

Methylenedioxymethamphetamine (MDMA; "Ecstasy") is a popular recreational drug and brain serotonin (5-HT) neurotoxin. Neuroimaging data indicate that some human MDMA users develop persistent deficits in brain 5-HT neuronal markers. Although the consequences of MDMA-induced 5-HT neurotoxicity are not fully understood, abstinent MDMA users have been found to have subtle cognitive deficits and altered sleep architecture. The present study sought to test the hypothesis that sleep disturbance plays a role in cognitive deficits in MDMA users. Nineteen abstinent MDMA users and 21 control subjects participated in a 5 d inpatient study in a clinical research unit. Baseline sleep quality was measured using the Pittsburgh Sleep Quality Inventory. Cognitive performance was tested three times daily using a computerized cognitive battery. On the third day of admission, subjects began a 40 h sleep deprivation period and continued cognitive testing using the same daily schedule. At baseline, MDMA users performed less accurately than controls on a task of working memory and more impulsively on four of the seven computerized tests. During sleep deprivation, MDMA users, but not

controls, became increasingly impulsive, performing more rapidly at the expense of accuracy on tasks of working and short-term memory. Tests of mediation implicated baseline sleep disturbance in the cognitive decline seen during sleep deprivation. These findings are the first to demonstrate that memory problems in MDMA users may be related, at least in part, to sleep disturbance and suggest that cognitive deficits in MDMA users may become more prominent in situations associated with sleep deprivation.

Ind Health. 2009 Oct;47(5):518-26.

Individual differences in vulnerability to sleep loss in the work environment.

Van Dongen HP, Belenky G.

Sleep and Performance Research Center, Washington State University Spokane, P.O. Box 1495, Spokane, WA 99210, USA. Email: hvd@wsu.edu

There are considerable individual differences in cognitive performance deficits resulting from extended work hours and shift work schedules. Recent progress in sleep and performance research has yielded new insights into the causes and consequences of these individual differences. Neurobiological processes of sleep/wake regulation underlie trait individual variability in vulnerability to performance impairment due to sleep loss. Trait vulnerability to sleep loss is observed in the laboratory and in the work environment, even in occupational settings where (self-)selection pressures are high. In general, individuals do not seem to accurately assess the magnitude of their own vulnerability. Methods for identifying workers who are most at risk of sleep loss-related errors and accidents would therefore be helpful to target fatigue countermeasure interventions at those needing them most. As yet, no reliable predictors of vulnerability to sleep loss have been identified, although candidate genetic predictors have been proposed. However, a Bayesian forecasting technique based on closed-loop feedback of measured performance has been developed for individualized prediction of future performance impairment during ongoing operations. Judiciously selecting or monitoring individuals in specific tasks or occupations, within legally and ethically acceptable boundaries, has the potential to improve operational performance and productivity, reduce errors and accidents, and save lives. Trait individual variability in responses to sleep loss represents a major complication in the application of one-size-fits-all hours of service regulations—favoring instead modern fatigue risk management strategies, because these allow flexibility to account for individual vulnerability or resilience to the performance consequences of extended work hours and shift work schedules.

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Medical and genetic differences in the adverse impact of sleep loss on performance: ethical considerations for the medical profession.

Czeisler CA.

Division of Sleep Medicine, Department of Medicine, Brigham andWomen's Hospital, Harvard Medical School, 221 Longwood Avenue, Suite 438, Boston, MA 02115, USA. Email: charles_czeisler@hms.harvard.edu

The Institute of Medicine recently concluded that-on average-medical residents make more serious medical errors and have more motor vehicle crashes when they are deprived of sleep. In the interest of public safety, society has required limitations on work hours in many other safety sensitive occupations, including transportation and nuclear power generation. Those who argue in favor of traditional extended duration resident work hours often suggest that there are inter- individual differences in response to acute sleep loss or chronic sleep deprivation, implying that physicians may be more resistant than the average person to the detrimental effects of sleep deprivation on performance, although there is no evidence that physicians are particularly resistant to such effects. Indeed, recent investigations have identified genetic polymorphisms that may convey a relative resistance to the effects of prolonged wakefulness on a subset of the healthy population, although there is no evidence that physicians are over-represented in this cohort. Conversely, there are also genetic polymorphisms, sleep disorders and other inter-individual differences that appear to convey an increased vulnerability to the performance-impairing effects of 24 hours of wakefulness. Given the magnitude of inter-individual differences in the effect of sleep loss on cognitive performance, and the sizeable proportion of

the population affected by sleep disorders, hospitals face a number of ethical dilemmas. How should the work hours of physicians be limited to protect patient safety optimally? For example, some have argued that, in contrast to other professions, work schedules that repeatedly induce acute and chronic sleep loss are uniquely essential to the training of physicians. If evidence were to prove this premise to be correct, how should such training be ethically accomplished in the quartile of physicians and surgeons who are most vulnerable to the effects of sleep loss on performance without unacceptably compromising patient safety? Moreover, once it is possible to identify reliably those most vulnerable to the adverse effects of sleep loss on performance, will academic medical centers have an obligation to evaluate the proficiency of both residents and staff physicians under conditions of acute and chronic sleep deprivation? Should work-hour policy limits be modified to ensure that they are not hazardous for the patients of the most vulnerable quartile of physicians, or should the limits be personalized to enable the most resistant quartile to work longer hours? Given that the prevalence of sleep disorders has increased in our society overall, and increases markedly with age, how should fitness for extended duration work hours be monitored over a physician's career? In the spirit of the dictum to do no harm, advances in understanding the medical and genetic basis of inter-individual differences in the performance vulnerability to sleep loss should be incorporated into the development of work-hour policy limits for both physicians and surgeons.

Semin Neurol. 2009 Sep; 29(4):320-39. Epub 2009 Sep 9.

Neurocognitive consequences of sleep deprivation.

Goel N, Rao H, Durmer JS, Dinges DF.

Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104-6021, USA.

Sleep deprivation is associated with considerable social, financial, and health-related costs, in large measure because it produces impaired cognitive performance due to increasing sleep propensity and instability of waking neurobehavioral functions. Cognitive functions particularly affected by sleep loss include psychomotor and cognitive speed, vigilant and executive attention, working memory, and higher cognitive abilities. Chronic sleep-restriction experiments– which model the kind of sleep loss experienced by many individuals with sleep fragmentation and premature sleep curtailment due to disorders and lifestyle – demonstrate that cognitive deficits accumulate to severe levels over time without full awareness by the affected individual. Functional neuroimaging has revealed that frequent and progressively longer cognitive lapses, which are a hallmark of sleep deprivation, involve distributed changes in brain regions including frontal and parietal control areas, secondary sensory processing areas, and thalamic areas. There are robust differences among individuals in the degree of their cognitive vulnerability to sleep loss that may involve differences in prefrontal and parietal cortices, and that may have a basis in genes regulating sleep homeostasis and circadian rhythms. Thus, cognitive deficits believed to be a function of the severity of clinical sleep disturbance may be a product of genetic alleles associated with differential cognitive vulnerability to sleep loss. Thieme Medical Publishers.

Sleep. 2009 Aug 1; 32(8):1100-3.

White matter differences predict cognitive vulnerability to sleep deprivation.

Rocklage M, Williams V, Pacheco J, Schnyer DM.

Department of Psychology, University of Texas at Austin, Austin, TX 78712, USA. mrocklage@mail.utexas.edu

OBJECTIVES: In other disciplines, white matter (WM) differences have been linked to cognitive impairments. This study sets out to clarify whether similar microstructural differences in WM tracts predict a person's cognitive vulnerability to the effects of total sleep deprivation (TSD).

DESIGN: Participants completed a simple visual-motor task both before and after 24 h of TSD. Using a median split on the percent change in accuracy from pre-TSD to post-TSD, participants were separated into susceptibility groups. A diffusion tensor MR imaging (DTI) scan was acquired from each participant, and fractional anisotropy (FA) was calculated, examined across the brain, and compared between susceptibility groups.

SETTING: University of Texas at Austin.

PARTICIPANTS: Thirty-two West Point cadets (9 females, 23 males) between 19 and 25 years of age.

RESULTS: Participant susceptibility to TSD was correlated with lower FA values in multiple regions of white matter, including the genu of corpus callosum and ascending and longitudinal white matter pathways. Significantly higher FA values in those less vulnerable to TSD, indicating increased neural connectivity and WM organization, may moderate the cognitive effects of sleep deprivation.

CONCLUSIONS: Differences in distributed WM pathways reflect, and may contribute to, a person's ability to function effectively when sleep deprived. The widespread nature of this effect supports previous views that TSD has a global effect on brain functioning.

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Donepezil improves episodic memory in young individuals vulnerable to the effects of sleep deprivation.

Chuah LY, Chong DL, Chen AK, Rekshan WR 3rd, Tan JC, Zheng H, Chee MW.

Cognitive Neuroscience Laboratory, Duke-NUS Graduate Medical School, Singapore.

STUDY OBJECTIVES: We investigated if donepezil, a long-acting orally administered cholinesterase inhibitor, would reduce episodic memory deficits associated with 24 h of sleep deprivation.

DESIGN: Double-blind, placebo-controlled, crossover study involving 7 laboratory visits over 2 months. Participants underwent 4 functional MRI scans; 2 sessions (donepezil or placebo) followed a normal night's sleep, and 2 sessions followed a night of sleep deprivation.

SETTING: The study took place in a research laboratory.

PARTICIPANTS: 26 young, healthy volunteers with no history of any sleep, psychiatric, or neurologic disorders.

INTERVENTIONS: 5 mg of donepezil was taken once daily for approximately 17 days.

MEASUREMENTS AND RESULTS: Subjects were scanned while performing a semantic judgment task and tested for word recognition outside the scanner 45 minutes later. Sleep deprivation increased the frequency of non-responses at encoding and impaired delayed recognition. No benefit of donepezil was evident when participants were well rested. When sleep deprived, individuals who showed greater performance decline improved with donepezil, whereas more resistant individuals did not benefit. Accompanying these behavioral effects, there was corresponding modulation of task-related activation in functionally relevant brain regions. Brain regions identified in relation to donepezil-induced alteration in nonresponse rates could be distinguished from regions relating to improved recognition memory. This suggests that donepezil can improve delayed recognition in sleep-deprived persons by improving attention as well as enhancing memory encoding.

CONCLUSIONS: Donepezil reduced decline in recognition performance in individuals vulnerable to the effects of sleep deprivation. Additionally, our findings demonstrate the utility of combined fMRI-behavior evaluation in psychopharmacological studies.

J Sleep Res. 2009 Jun; 18(2):167-72.

The effect of 40 h constant wakefulness on task-switching efficiency.

Bratzke D, Rolke B, Steinborn MB, Ulrich R.

Cognitive and Biological Psychology, Psychologisches Institut, University of Tübingen, Friedrichstrasse 21, Tübingen 72072, Germany.
Email: daniel.bratzke@uni-tuebingen.de

This study investigated efficiency of switching between different tasks in 12 male participants (19-30 years) during 40 h of constant wakefulness. As index of task-switching efficiency, switch costs in reaction time were assessed every 3 h under controlled behavioural and environmental conditions. Overall reaction times and switch costs showed a temporal pattern consistent with the assumption of a combined influence of a sleep homeostatic and a circadian process. An additional analysis indicated that the variation in switch costs could not be attributed to interference of the current task with persisting activation from preceding tasks. We therefore conclude that sleep loss and the circadian system affect the ability to prepare the current task rather than automatic processing of irrelevant stimulus information.

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Error correction maintains post-error adjustments after one night of total sleep deprivation.

Hsieh S, Tsai CY, Tsai LL.

Cognitive Electrophysiology Laboratory, Institute of Allied Health Sciences, National Cheng Kung University, Tainan, Taiwan.

Previous behavioral and electrophysiologic evidence indicates that one night of total sleep deprivation (TSD) impairs error monitoring, including error detection, error correction, and posterror adjustments (PEAs). This study examined the hypothesis that error correction, manifesting as an overtly expressed self-generated performance feedback to errors, can effectively prevent TSD-induced impairment in the PEAs. Sixteen healthy right-handed adults (seven women and nine men) aged 19-23 years were instructed to respond to a target arrow flanked by four distracted arrows and to correct their errors immediately after committing errors. Task performance and electroencephalogram (EEG) data were collected after normal sleep (NS) and after one night of TSD in a counterbalanced repeated-measures design. With the demand of error .correction, the participants maintained the same level of PEAs in reducing the error rate for trial N + 1 after TSD as after NS. Corrective behavior further affected the PEAs for trial N + 1 in the omission rate and response speed, which decreased and speeded up following corrected errors, particularly after TSD. These results show that error correction effectively maintains posterror reduction in both committed and omitted errors after TSD. A cerebral mechanism might be involved in the effect of error correction as EEG beta (17-24 Hz) activity was increased after erroneous responses compared to after correct responses. The practical application of error correction to increasing work safety, which can be jeopardized by repeated errors, is suggested for workers who are involved in monotonous but attention-demanding monitoring tasks.

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Neurobehavioral performance in young adults living on a 28-h day for 6 weeks.

Lee JH, Wang W, Silva EJ, Chang AM, Scheuermaier KD, Cain SW, Duffy JF.

Division of Sleep Medicine, Department of Medicine, Brigham and Women's Hospital, Boston, MA 02115, USA.

OBJECTIVES: Performance on many cognitive tasks varies with time awake and with circadian phase, and the forced desynchrony (FD) protocol can be used to separate these influences on performance. Some performance tasks show practice effects, whereas the Psychomotor Vigilance Task (PVT) has been reported not to show such effects. We aimed to compare performance on the PVT and on an addition test (ADD) across a 6-week FD study, to determine whether practice effects were present and to analyze the circadian and wake-dependent modulation of the 2 measures.

DESIGN AND SETTING: A 47-day FD study conducted at the Brigham and Women's Hospital General Clinical Research Center.

PARTICIPANTS: Eleven healthy adults (mean age: 24.4 years, 2 women).

MEASUREMENTS AND RESULTS: For 2 baseline days and across 6 weeks of FD, we gave a test battery (ADD, PVT, self-rating of effort and performance) every 2 hours. During FD, there was a significant ($P < 0.0001$) improvement in ADD performance (more correct calculations completed), whereas PVT performance (mean reaction time, fastest 10% reaction times, lapses) significantly ($P < 0.0001$) declined week by week. Subjective ratings of PVT performance indicated that subjects felt their performance improved across the study ($P < 0.0001$), but their rating of whether they could have performed better with greater effort did not change across the study ($P > 0.05$).

CONCLUSIONS: The decline in PVT performance suggests a cumulative effect of sleep loss across the 6-week study. Subjects did not accurately detect their declining PVT performance, and a motivational factor could not explain this decline.

J Occup Health. 2009; 51(5):412-22. Epub 2009 Jul 15.

Recovery of cognitive performance and fatigue after one night of sleep deprivation.

Ikegami K, Ogyu S, Arakomo Y, Suzuki K, Mafune K, Hiro H, Nagata S.

Department of Mental Health, Institute of Industrial Ecological Sciences, University of Occupational and Environmental Health, Kitakyushu, Fukuoka, Japan. Email : kikegami@med.uoeh-u.ac.jp

OBJECTIVES: The aim of this study was to investigate how subjective sleepiness, mood states, simple and high-order cognitive performance change after one night of sleep deprivation (SD) and recover to after 7 h normal recovery sleep opportunity during three recovery days.

METHODS: Ten healthy subjects participated in this study. We measured their subjective sleepiness, mood states and their performances of 2 simple tasks and 4 high-order cognitive tasks twice a day for 5 days, on the baseline day, post-vigil day and 3 recovery days after SD. This study was conducted considering each participant's motivation for task, learning effect and diurnal variation of performance.

RESULTS: The performances of simple tasks such as addition or short-term memory were not reduced after SD and were the poorest on the baseline day, and improved gradually; however the high-order cognitive performances were at their lowest on the post-vigil day and needed 2 recovery sleep opportunities to return to the baseline level. Fatigue and confusion in mood states and subjective sleepiness were also at their lowest after SD. Subjective sleepiness nearly recovered to the baseline level on the 1st recovery day, but fatigue and confusion reached the baseline levels on the 2nd recovery day.

CONCLUSION: These results suggest that cognitive deterioration and the recovery process may differ between simple task performance and high-order cognitive task performance, which needed 2 ordinary sleep opportunities to recover to the baseline level, and the change of subjective mood states were also different for each mood.

Behav Sleep Med. 2009; 7(3):136-63.

Failure to find executive function deficits following one night's total sleep deprivation in university students under naturalistic conditions.

Pace-Schott EF, Hutcherson CA, Bemporad B, Morgan A, Kumar A, Hobson JA, Stickgold R.

Department of Psychiatry, Harvard Medical School, Beth Israel-Deaconess Medical Center, Boston, MA 02215, USA. Email: epacesch@bidmc.harvard.edu

Young adult male students participated in a naturalistic, group-design experiment to ascertain the effects of one night's total sleep deprivation (TSD) on performance of diverse executive function tasks presented as an extended, multitask battery. On the majority of component tasks in this battery, performance has been reported to be impaired following one night's TSD when tasks are administered in isolation. However, participants sleep deprived 35 to 39 hr showed few performance deficits among tests in this battery when compared with non-sleep-deprived controls. Sleep-deprived participants showed only poorer recognition memory and overconfidence in incorrect temporal judgments. Behavioral and physiological adaptation to chronically sleep-restricting lifestyles may confer resistance to the cognitive effects of sleep deprivation in high-functioning young adults.

Sleep. 2009 Feb 1; 32(2):205-16.

Sustaining executive functions during sleep deprivation: A comparison of caffeine, dextroamphetamine, and modafinil.

Killgore WD, Kahn-Greene ET, Grugle NL, Killgore DB, Balkin TJ.

Department of Behavioral Biology, Walter Reed Army Institute of Research, Silver Spring, MD, USA. Email: killgore@mclean.harvard.edu

OBJECTIVES: Stimulant medications appear effective at restoring simple alertness and psychomotor vigilance in sleep deprived individuals, but it is not clear whether these medications are effective at restoring higher order complex cognitive capacities such as planning, sequencing, and decision making.

DESIGN: After 44 hours awake, participants received a double-blind dose of one of 3 stimulant medications or placebo. After 45-50 hours awake, participants were tested on computerized versions of the 5-Ring Tower of Hanoi (TOH), the Tower of London (TOL), and the Wisconsin Card Sorting Test (WCST).

SETTING: In-residence sleep-laboratory facility at the Walter Reed Army Institute of Research.

PARTICIPANTS: Fifty-four healthy adults (29 men, 25 women), ranging in age from 18 to 36 years. Interventions: Participants were randomly assigned to 1 of 3 stimulant medication groups, including caffeine, 600 mg (n = 12), modafinil, 400 mg (n = 12), dextroamphetamine, 20 mg (n = 16), or placebo (n = 14).

MEASUREMENTS AND RESULTS: At the doses tested, modafinil and dextroamphetamine groups completed the TOL task in significantly fewer moves than the placebo group, and the modafinil group demonstrated greater deliberation before making moves. In contrast, subjects receiving caffeine completed the TOH in fewer moves than all 3 of the other groups, although speed of completion was not influenced by the stimulants. Finally, the modafinil group outperformed all other groups on indices of perseverative responding and perseverative errors from the WCST.

CONCLUSIONS: Although comparisons across tasks cannot be made due to the different times of administration, with in task comparisons suggest that, at the doses tested here, each stimulant may produce differential advantages depending on the cognitive demands of the task.

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Control and function of the homeostatic sleep response by adenosine A1 receptors.

Bjorness TE, Kelly CL, Gao T, Poffenberger V, Greene RW.

Department of Psychiatry, University of Texas Southwestern, Dallas, Texas 75390, USA.

During sleep, the mammalian CNS undergoes widespread, synchronized slow-wave activity (SWA) that directly varies with previous waking duration (Borbély, 1982; Dijk et al., 1990). When sleep is restricted, an enhanced SWA response follows in the next sleep period. The enhancement of SWA is associated with improved cognitive performance (Huber et al., 2004), but it is unclear either how the SWA is enhanced or whether SWA is needed to maintain normal cognitive performance. A conditional, CNS knock-out of the adenosine receptor, AdoA(1)R gene, shows selective attenuation of the SWA rebound response to restricted sleep, but sleep duration is not affected. During sleep restriction, wild phenotype animals express a rebound SWA response and maintain cognitive performance in a working memory task. However, the knock-out animals not only show a reduced rebound SWA response but they also fail to maintain normal cognitive function, although this function is normal when sleep is not restricted. Thus, AdoA(1)R activation is needed for normal rebound SWA, and when the SWA rebound is reduced, there is a failure to maintain working memory function, suggesting a functional role for SWA homeostasis.

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Sleep, recovery, and performance: the new frontier in highperformance athletics.

Samuels C.

Centre for Sleep and Human Performance, Calgary, Alberta, Canada. chuck@centreforsleep.com

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The relationship of sleep to post-exercise recovery (PER) and athletic performance is a topic of great interest because of the growing body of scientific evidence confirming a link between critical sleep factors, cognitive processes, and metabolic function. Sleep restriction (sleep deprivation), sleep disturbance (poor sleep quality), and circadian rhythm disturbance (jet lag) are the key sleep factors that affect the overall restorative quality of the sleep state. This article discusses these theoretic concepts, presents relevant clinical cases, and reviews pilot data exploring the prevalence of sleep disturbance in two groups of high-performance athletes.