BULLETIN

A Publication of the Sleep Research Society, USA
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SRS BULLETIN

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Dear SRS Members,

The world changed abruptly September 11, 2001, profoundly altering the lives of hundreds of millions of free people. For those in our community who lost family and friends, we offer condolences. To take strength from such utter tragedy, let it remind us that the collective passion we share for a scientific understanding of sleep and its role in human health, well-being, and behavioral effectiveness also has relevance to the new challenges we now face as a civilized society under terrorist attack. Examples include the relevance of our scientific knowledge on how to minimize the adverse effects of sleep deprivation on emergency and military personnel; on ways to help public safety and public health personnel remain vigilant 24/7; and on ways to identify and treat insomnia and sleep disturbance associated with stress, depression and trauma. (A number of SRS members were involved in an NIH sponsored workshop on the "Neurobiology of Sleep and Waking: Implications for Insomnia," when the events on the morning of September 11 overtook us. A report from that meeting is currently being completed.)

It is gratifying to see that the sleep research community did not let the events since September 11 paralyze them. Despite the earliest abstract deadline ever imposed for APSS submissions (something we hope to re-evaluate for next year), and both a major sleep meeting and chronobiology meeting within 2 weeks prior to APSS 2002, approximately 750 abstracts were submitted for the APSS meeting in Seattle (Scientific Program June 10-13, 2002; Postgraduate courses and Trainee day, June 8-9, 2002). Please keep the following dates in mind as you prepare for what is shaping up to be an exciting scientific program: the preliminary program will be available March 1, 2002, and the advance (discounted) registration deadline is April 22, 2002. Be sure to book hotel rooms early, as attendance is expected to be high. The meeting will also involve an event hosted by the World Federation of Sleep Research Societies.

SRS continues to grow and develop processes for anticipating future needs. As of November 11, 2001, we totaled 848 members. We must continue to attract established scientists who bring new ideas and techniques to sleep research, and train academically competitive younger scientists if we hope to accelerate and expand discovery. One of the greatest challenges facing sleep research is the need to ensure we have sufficient numbers of scientists trained in the latest techniques of molecular genetics, neuroscience, behavioral science, pharmacology, clinical trials, epidemiology, etc., that are required to ensure we can respond to the growing opportunities created by NIH and other research funding agencies through RFP's. The SRS has a number of new initiatives relevant to this goal. The first J. Christian Gillin Endowment for Junior Faculty was recently awarded to Washington State University. The award will provide $45,000 per annum in salary support for 3 years for a faculty appointment at the assistant professor level, focusing on sleep research. The SRS Board hopes to increase the endowment to a level that will be self-sustaining and permit future awards to be made. To make contributions to the endowment, contact Lance Brink at the SRS National Office (507-285-4384).

Tracking and evaluating the success of our trainees is also critical to our future. To obtain current information on the effectiveness of the trainee support provided by the SRS, the Board of Directors authorized at its November 17-18, 2001 meeting, that Dr. Ron Szymusiak (SRS Program Chair for Trainees) and Dr. Jodi Mindell (SRS Membership Chair) jointly chair a Task Force on Trainee Development. The Task Force will be charged with determining reliable ways to track SRS trainee retention and progression to mature research careers in sleep (faculty positions, publications, grants) during the past 10 years, relative to SRS travel awards and support. The goal is to obtain data that will inform the Society on how to optimally allocate its trainee support relative to the goal of training competitive career scientists in sleep.

SRS planning in all areas relevant to the growth of sleep science is timely. The NIH National Center for Sleep Disorders Research (NCSDR), established within NHLBI in 1993, and working closely with the Trans-NIH Sleep Research Coordinating Committee is in the process of developing a new 5-year strategic research plan. The previous plan, formulated in 1996 is in its terminal year. To this end, a National Sleep Disorders Research Plan Revision Task Force has recently been convened (see announcement in this issue of the Bulletin) and charged with recommending a draft plan to the Sleep Disorders Research Advisory Board of the NCSDR. The Task Force is seeking input on ideas for future sleep research focus from all stakeholders in sleep research, and a preliminary draft of its report will be made publicly available in 2002 for comment and additional suggestions. The resulting strategic research plan will provide a critical roadmap for NIH-initiated sleep research efforts over the next 5 years.

To keep abreast of the growth of SRS activities and involve more members in SRS leadership and management, the Board has recently approved and appointed membership to three new Committees. The Education and Scientific Review Committee (ESRC), to be chaired by Dr. Kingman Strohl, will propose, plan, and develop educational programs and materials concerning sleep research to meet the needs of the SRS membership (excluding trainee activities, which are managed by TEAC). The ESRC will also be responsible for the development of an oversight mechanism for accepting educational and scientific program proposals. The Government Affairs Committee (GAC), currently chaired by Dr. Merrill Mitler, will be responsible for coordinating SRS efforts aimed at continued enhancement of NIH support of sleep research; developing an interactive mechanism by which

the SRS disseminates information and positions on matters concerning research funding; and maintaining communications with the AASM and other entities concerned with sleep research and sleep disorders. The GAC recently recommended support (matched by AASM) for completion of a research survey of medical resident works hours, sleep loss, and medical errors. The issue of resident work hours relative to medical errors and public safety has come under national scrutiny this past year-a bill limiting resident's work hours is currently in committee in Congress. In October, 2001, the SRS, along with AASM, AMA, NCSDR, and AHRQ sponsored a workshop ("Sleep, Fatigue and Medical Training: Optimizing Learning and the Patient Care Environment") on the sleep science relevant to this issue, with recommendations for improving the situation, whether or not legislation is passed. The International Outreach Committee (IOC), currently chaired by Dr. Michael Vitiello, will be responsible for developing the mechanisms for providing assistance to junior and/or financially disadvantaged sleep scientists working in economically disadvantaged countries. The committee is responsible for the application mechanism, recruitment efforts, and evaluating the effectiveness of the program.

Finally, please take note of the following items involving SRS publications. (1) February 1, 2002 is the deadline for applying for the position of Editor for SLEEP. Dr. Robert McCarley chairs the Editor Search Committee. (2) This issue of the Bulletin displays not only a new cover, but also the new (updated) logo for the SRS. (3) Last, but certainly not least, through the diligence and hard work of SRS Publications Chair, Dr. Christine Acebo, and the members of the SRS Web Committee, the new SRS website is scheduled to be up and running by March-be sure to look for it at www.srssleep.org.

--David Dinges

Editor's Column

by Larry D. Sanford, PhD

The American Physiological Society began, in 2000, a Latin American Initiative to foster collaborative efforts between physiologists of the Americas. Drs. Mark R. Opp, University of Michigan, and Deborah Suchecki, Universidad Federal de São Paulo, organized a symposium entitled "Stressor-induced Alterations in Sleep", which was funded under the auspices of this program. The symposium was held October 18 - 19, 2001, in the Department of Psychobiology, Universidad Federal de São Paulo, Brazil. Both North and South American scientists presented their work, and about 50 registered participants attended the two day symposium, many of whom were students. In this, and the next, issue of the SRS Bulletin, we will present the proceedings of that symposium. We congratulate Drs. Opp and Suchecki for their success in competing for these funds and encourage others in the sleep research community to take advantage of opportunities that may arise to organize symposia or small meetings that focus on topics of interest to sleep research in venues and under the sponsorship of scientific organizations other than the APSS or WFSRS.

The cover story of the October issue of the Monitor on Psychology, a publication of the American Psychological Association was entitled, "Sleep: Research and Practice." This article discusses issues involved in several subdisciplines of our field and mentions several of our clinical and research colleagues. It is gratifying to see our field receive this type of attention outside our own publications. The issue is accessible at: http://www.apa.org/monitor/.

This issue marks the end of my first year as editor of the SRS Bulletin. I want to thank those who have contributed to the Bulletin during this time. I also want to thank Lance Brink who has temporarily stepped back in to handle the formatting and organization chores. As always, your comments and suggestions are welcome.

STRESS-INDUCED ALTERATIONS IN SLEEP

Introduction

by Mark R. Opp and Deborah Suchecki

The relationship between sleep and stress has long been recognized. Hippocrates stated millennia ago that lack of sleep is a sign of pain and suffering that can result in mental illness, whereas diurnal somnolence is a sign of illness. These ancient observations spoke to a fundamental relationship between mental and physical health and sleep.

We all are aware – from personal experience – that psychological (emotional) and physical (infection, trauma) stressors disrupt sleep. Despite being a readily recognized association, it is surprising that very few researchers spend time and effort trying to understand this relationship.

Our modern society represents a unique experimental setting for the study of stressor-induced alterations in sleep. Shiftwork, circadian misalignment, stressful life events (possibly leading to psychiatric or psychological disorders), voluntary sleep curtailment and sleep deprivation, and infections are, in one way or another, part of our everyday lives. The purpose of this symposium was to address how these events can alter sleep and the main consequences of this alteration.

We wish to thank the sponsors of the symposium, The American Physiological Society, Associação Fundo de Incentivo à Psicofarmacologia, Fundação de Amparo à Pesquisa do Estdo de São Paulo, and the Department of Psychobiology, Universidad Federal de São Paulo for recognizing the importance of this topic to society.

Sleep and Circadian Rhythms Disturbances and Mood Disorders

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1. Introduction

In the elaboration of hypotheses about mood disorders there is a prevalence of the homeostatic view, i.e., changes in neurotransmitters and their receptors and, in neurohormones and neuropeptides are responsible for mood, sleep and appetite disorders, for the lack of interest and for somatic symptoms, etc. (Green et al., 1995). Throughout the years, several theories based on observations and scientific results were put forward to explain alterations of sleep and its components (NREM and REM sleep), of different hormones (melatonin, cortisol), and of positive results of non-pharmacological therapies (sleep deprivation, phototherapy) (Le Bon et al., 1997). Nonetheless, we are still unable to structure and unify them in one wide theory, which takes into consideration all the above-mentioned alterations and explains the appearance of such disorders, insofar as they occur in cycles.

Two epidemiological studies in the general population (Ford & Kamerov, 1989; Mellinger et al., 1985) presented data indicating a higher probability of insomniac adults suffering of depression (14% to 21%) than individuals without sleep complaints (depression rate under 1% in both studies). In addition to insomnia, the Mellinger and co-workers' study (1985) showed a prevalence of 9.9% of hypersomnia in individuals with mood disorders. Although mood disorders are, in general, easily identified in depressive individuals, the contrary does not seem to be true, since mood disorders are often misdiagnosed in non-psychiatric institutions. It is estimated that more than half of depressive individuals are not diagnosed as such in primary care institutions (Benca et al., 1996).

Approximately two thirds of the individuals with one episode of major depression presents some type of insomnia, where 40% of complaints refer to specific symptoms of difficulty for sleep onset, multiple arousals and terminal insomnia (Hamilton, 1997; Perlis et al., 1997). With a lower frequency, approximately 15%, individuals present excessive somnolence (hypersomnia), in the form of prolonged episodes of nocturnal or daytime sleep (Buysse et al., 1999; DSM-IV, 1995). Regarding sleep, disruptions of the sleep-wake cycle that happen in these disorders include the homeostatic hypotheses (the two-process model, reciprocal interaction/ cycle limitation reciprocal interaction and REM sleep deprivation), as well as circadian rhythm hypotheses (free running, phase advance/phase agreement, amplitude reduction and phase instability). There are several controversies about these hypotheses and the relationships between these disorders, on one hand, and of sleep and circadian rhythm disorders, on the other hand. Many results are the opposite of others. For this reason it is difficult to build a hypotheses or working model that explains the most consistent findings of the many studies performed in this field.

2. Polysomnographic findings

Kupfer and Foster (1972) first published what would become one of the most studied sleep alterations in subjects with mood disorders, i.e., shortening of REM sleep latency. Although we can not consider this alteration as the pathognomonic depression "marker", since it occurs in other psychiatric disorders, it became quite popular for being easily measured when compared to other REM sleep changes (increased density and flatter distribution of REM sleep throughout the night), for the prompt diagnostic associations made with it and for its covariance with other REM sleep abnormalities (Le Bon et al., 1997).

The polysomnographic characteristics of patients with mood disorders may be included in four different categories (Buysse et al., 1999):

- Reduced sleep continuity: prolonged sleep latency, increased

number of awakenings, augmented early awakening and diminished sleep efficiency;
- Diminished slow wave sleep: reduction of stages 3 and 4; decreased delta activity during the period-amplitude or power spectral analysis;
- Augmented REM sleep: increased percentage of REM sleep, increased phasic eye movements during REM sleep;
- Altered temporal characteristics of sleep: reduced REM sleep latency, i.e., length of the first sleep period without rapid eye movements (non-REM sleep [NREM]), reduced electroencephalographic delta activity during the first NREM period, compared to the second one (decreased "sleep delta ratio"), augmented length of the phasic activity of eye movements during the first REM period.

Although each one of these findings is characteristic of depression, most individuals do not present all findings and none of them can be considered a sensitive or specific marker of depressive disorders. In a meta-analysis study of polysomnographic findings, Benca and colleagues (1992) observed that REM sleep-related parameters were consistently more abnormal in major depression individuals than in patients with different diagnosis.

Polysomnographic studies performed in subjects with bipolar disorders presented similar results (Hudson et al., 1988; Linkowski et al., 1986). Benca and colleagues (1992), when referring to polysomnographic alterations, do not consider mood disorders as distinct entities; quite contrary, they pooled these disorders, despite taking a precaution to this approach.

Depressive individuals exhibit elevated nocturnal temperature and reduced amplitude of temperature rhythm. Shortening of the REM sleep latency has been significantly correlated with both flattening of temperature amplitude and higher nocturnal temperature averages (Benca, 2000).

Some of these polysomnographic findings may be related to observations of several studies:

I. Reduction of melatonin secretion, with irregular diurnal peaks, reduced nocturnal peak, and phase advance could be associated to the elevated average of night temperature (Avery et al., 1986; Benca, 2000) and reduced amplitude of temperature rhythm.
II. Reduction of melatonin secretion appears to involve a serotonergic and noradrenergic deficit as the primary cause of pineal dysfunction (Brown, 1985).
III. The effects of sleep deprivation on depression are acute and transitory and may be mediated by mechanisms distinct from those that mediate the gradual improvement obtained with antidepressive drugs (Wirz-Justice & Van den Hoofdakker, 1999). Nonetheless, serotonergic mechanisms appear to be involved, especially with down-regulation of pre-synaptic 5-HT1A receptors. This observation allows an explanation for why the combination of one to three nights of sleep deprivation with the onset of antidepressive therapy accelerates the antidepressive response. In addition, it allows us to quickly notice resiliency to drug therapy (Lustberg & Reynolds, 2000; Wirz-Justice & Van den Hoofdakker, 1999).
IV. Regardless of shortening of REM sleep latency being one of the most recalled alterations by depressive subjects, Buysse and colleagues (1990) do not believe that these individuals would present REM sleep phase advance. On the contrary, REM sleep would be unaltered in relation to the 24h period, and sleep, in fact, would be delayed in relation to the phase, possibly related to the more elevated average nocturnal temperature.
V. Among the affective disorders, patients with seasonal affective disorder exhibit more clearly a connection between symptoms - approximately 90% of these patients present diagnostic criteria for type II bipolar disorder, according to the DSM-IV (Faedda et al., 1993) - and circannual alterations of sunlight, mainly in countries or regions situated in higher latitudes (Booker & Hellekson, 1992). It has been postulated that supersensitivity to lighting variation occurs throughout the year in this disorder (Rosenthal et al., 1984).

3. Conclusion

A question hangs in the air: circadian alterations are partly responsible for affective disorders or they are just an epi-phenomenon? Dagan and co-workers (1988) studied the frequency of sleep phase delay syndrome among adolescent inpatients with various psychiatric diagnostics and found a positive correlation between this syndrome and borderline personality, schizoaffective disorder (mainly affective) and bipolar disorder. On the other hand, none of the inpatients diagnosed with schizophrenia or schizoaffective (mainly schizophrenic) disorder presented sleep phase delay syndrome. Following the same line of reasoning, patients with diagnosis of uni- or bipolar affective disorder and sleep phase delay syndrome, under adequate drug therapy and solution of the affective disorder, were not capable to initiate sleep earlier and to wake up earlier.
This close relationship between mood and circadian rhythm, including the sleep-wake cycle, shall become the subject of intense observations in the following years, with the purpose of, in addition to answering the initial question, making the treatment faster and more personal.

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Wake-Sleep Cycle and Transportation Shift Workers

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1. Introduction

Sleep disorders are abnormalities of sleep regulation or physiological alterations specifically related to sleep (Hauri, 1982). An initial and spontaneous report of sleep-related complaints is frequent in the general population. One of the main influencing aspects of emergence of these disorders is the fact that humans are mostly active during the day and sleep or rest during the night (Rutenfranz et al., 1989).

Sleep disorders have become a concern among physicians and public health administrators with, in the last decades, growing indexes of problems associated to these disorders. It can be noted that most people who complain of sleep problems report excessive daytime somnolence, irritability and stress (Hauri, 1982). Examples are given by the studies carried out by the Department of Psychobiology of UNIFESP/EPM with the population of São Paulo City, in 1987 (Del Giglio, 1988) and 1995 (Palma et al., 1997). The data obtained in 1987 show that approximately 76% of the general population presented a sleep problem; in the 1995 study, 82% of the population presented at least one sleep-related complain. Compared to the first survey, there was an increase of the general complaints related to sleep.

A further example is the epidemiological survey of sleep patterns and complaints among bus drivers (Mello et al., 2000). This survey was carried out by means of a sleep questionnaire, elaborated and validated by the Department of Psychobiology of UNIFESP/EPM (Del Giglio, 1988; Palma et al., 1997), which was applied to 400 male bus drivers. The country's geographical regions were South, Southeast, Midwest and North. The results showed that 60% of the bus drivers presented at least one sleep-related complaint. Compared to the data obtained in the general population of São Paulo City, these findings indicate a reduction of the indexes. However, in a specific population of bus drivers, this index could be considered high, due to their work characteristics, such as irregular schedules, short time off and inappropriate sleep place, which can influence the alertness status that these drivers must show during working hours (Oliveira et al., 1997). Another important finding presented in this study was a 16% report of the drivers assuming that they sleep/nap while driving, with an average of 8 naps per trip. Nonetheless, when questioned whether they know of a colleague who has slept/napped at the wheel, the answer was positive in 56% of the interviewed. This means that the driver does not assume that he sleeps while working, but he reports that a fellow worker does. These data are confirmed in other studies currently being carried out in our

University, using computerized polysomnography and application of a structured interview using the EVAL system.

Sleep disorders can lead to several consequences such as impairment of professional performance, social and marital disruption, mood disorders, severe vehicle or work accidents, increased incidence of cardiovascular disorders and systemic arterial hypertension. These perturbations of the sleep pattern may also result in somnolence, i.e., hypersomnolence may be the cause of sleep disorders elicited by shift work. Disorders such as obstructive sleep apnea-hypopnea syndrome, periodic leg movements, insomnia, and narcolepsy are some of the factors that can lead to a somnolence condition (Gulleminault & Carskadon, 1977).

An essential aspect of the diagnosis is the history of sleep-related complaints, medical and psychiatric interview, family history, use of medication and/or drugs and the subject's psychosocial situation (Tufik et al., 1997). By means of polysomnographic evaluations it is possible to identify a specific disorder; however, under certain circumstances, the sleep diary and complementary exams, such as a detailed clinical evaluation, actigraphy, radiographic tests and otolaryngologic evaluation, may provide important data about the individual's sleep-wake cycle.

2. The relationship between sleep and accidents during working hours

During the last decade, shift and night work positions have grown considerably. Economic, demographic and technological changes are the most likely causes of such increases (Presser, 1999). This new model of work organization is not exclusive of the industrial era, insofar as certain forms of shift work, such as soldiers/security, firemen, police officers and nurses, existed since the beginning of social relations among humans (Rutenfranz, 1989).

According to Harrington (cited in Rutenfranz, 1989), acceptance of shift work varies from 60 to 70% of the workers; however, it is important to emphasize that it may be a health risk. Approximately 20 to 25% of American workers are engaged with some kind of shift work (US Congress, 1991). Many of them often complain of being incapable of sleeping at irregular hours and of somnolence-related problems (Rutenfranz et al., 1985).

Sleep is fundamental for adequate professional performance because attention, motor coordination, mental rhythm and, especially alertness are influenced by fatigue. The sleep need varies from one person to the other and is independent from shift work. However, this factor (work at irregular hours) may alter sleep patterns, thus reducing total sleep time in night workers (Rutenfranz, 1982). These effects of shift work may still be observed regarding the worker's difficulty in sleeping at irregular hours and reduced sleep efficiency during daytime sleep. This could lead to further consequences on physical and mental health, neural disorders, fatigue, irritability, anxiety, depression, sexual problems and stress. These alternating schedules may still increase the risk for gastrointestinal (Costa, 1996) and cardiovascular problems (Scott & La Dou, 1990).

Somnolence in shift workers may be caused by insufficient total sleep time or increase in sleep fragmentation (Monk et al., 1996). Fischer et al. (2000) observed a reduction of the total sleep time in subjects after a night of work. They also showed reduced perception of alertness after the sixth and tenth working hour.

The adverse effects of shift work may vary from person to person, specially regarding some aspects such as age and gender. Youngsters endure shift work better than older people, mainly due to changes of sleep architecture and pattern in elderly (Harma, 1995).

Many studies show a reduction of one hour in total sleep time/day, making up for seven hours/week in shift workers. Nonetheless, polysomnographic evaluation shows that this reduction is approximately 2 hours or more/day (Torsvall et al., 1989). This reduction is even greater in night shift workers (Colligan & Tepas, 1986), in early morning workers (Kecklund et al., 1994) and women who take care of the kids at home (Gadbois, 1981). One of the consequences is the somnolence which contributes significantly to mistakes and increases the risk for work accidents, which can affect industrial operations, petrochemical platforms and transportation systems (Dinges, 1995). Several studies show a higher number of accidents during the night shift than the in day shift (Costa, 1996) because of this problem.

More recent studies show increased risk of accidents as a function of working hours. This augmented risk would occur approximately 9 hours after the onset of the working time. Twelve hours later the risk is doubled, and with 14 hours of continuous work there is a 3-fold increase of risk. Some studies found an increment of accidents and injuries with 5 hour of work (Folkard, 1996). This increased risk of accidents during the night shift could also be due to the time of shift onset. However, this influence is still controversial and requires better controlled studies (Folkard, 1996), because of the natural variability of the worker's biological rhythm.

An important preventive measure is to inform and orient shift workers about the increased risk of accidents and his/her vulnerability in face of alternating shift work (Richardson et al., 1990). The major consequence of the somnolence problem for shift workers is associated with his/her quality of life, decreased production and increased potential risk of accidents and injuries during working hours (Dinges, 1995). Regarding quality of life, this population shows augmented neural signs, use of alcohol, stimulant drugs and hypnotics (Gordon et al., 1985). However, the self-evaluation of quality of life may be subjective, thus being correlated with the education of the subject.

Studies that evaluate the sleep pattern of shift workers indicate that 60 to 70% of the subjects complain of difficulties in sleeping or of excessive somnolence (Rutenfranz et al., 1985). In face of such aspects, other studies suggest that human mistakes are the major determinant of car accidents due to lack of attention, inadequate evaluations and cognitive mistakes, being responsible for 40% of all cases of accidents. Excessive daytime somnolence may trigger a reduction of attention, increasing the risk of accidents (George & Sliley, 1999). There is a literature consensus that excessive daytime somnolence and other sleep-related problems, place drivers, passengers and other people at high risk of acci-

dents (Hansotia, 1997).

Highway accident-related statistics involving commercial and private vehicles are growing every year, becoming a source of worry to public authorities. In the last years, the relationship between vehicle accidents and sleep problems has received special attention (Horne & Reyner, 1995). It is also known that during prolonged stress, vigilance systems associated to attention are abruptly reduced, likely leading to accidents (Florez-Lozano, 1980). Therefore, there is considerable evidence that fatigue and somnolence contribute importantly to industrial and transportation accidents (Lauber & Kayten, 1988; Mitler et al., 1988; Mitler et al., 1994). There is no precise National statistics in Brazil about work or vehicle accidents caused by somnolence because police or administrative reports do not provide this data. However, an ever so growing number of accidents do present specific features that indicate lack of concentration or attention due to excessive somnolence.

Numerous behavioral alterations can lead to a state of tiredness, such as anxiety, tension, lack of confidence, fear, shift in biological rhythms, and these can be accentuated by social-economical conditions, working and/or family situation. Other factors such as the level of lightning, noise, temperature, humidity, and atmosphere ionization are also relevant (Florez-Lozano, 1980) and can increase the incidence of this disorder.

Prolonged working hours reduce attention and increase the risk of working accidents, being influenced by the time and the amount of worked hours (Florez-Lozano, 1980). A study showed that 50% of the sample evaluated showed reduced total sleep time within the 24 h preceding a trip. This finding is in agreement with other studies that demonstrate that sleep deprivation associated with prolonged driving period is frequently linked to increased risk of traffic accidents (Philip et al., 1999). Usually this sleep deprivation occurs exactly in the last third of the night, being the period of REM sleep predominance. This fact generally reflects the reduced attention and concentration capacities in subjects who continue to work, even though they are conscious of their fatigue (Brow, 1994).

The factors previously mentioned, such as sleeping at the wheel, represent a considerable part of car accidents, especially associated with monotonous trips. Some conditions can potentiate the sleeping state, including sleep restriction or deprivation, and circadian alterations evidenced during rotating shift work, without a fixed work schedule. Other drivers are vulnerable to somnolence in the mid- or at the end of the afternoon. Another contributing factor is the length of the work or the trip, which is regulated by public policies. Pathologies can cause somnolence, but there is little evidence supporting the idea that they can increase the statistics (Horne & Reyner, 1999). Nonetheless, the biological aspect must prevail in assembling the work schedule.

Vehicle accidents are multifactorial in nature. In those that occur at night we can say that the likely causes are darkness, reduced vision, consumption of alcohol, excessive speed, cardiac problems, mechanical problems, inadequate highway conditions and sleepiness (Horne & Reyner, 1995; Leger, 1995).

After a long period of work or a period of intense physical and mental weariness the electroencephalographic (EEG) activity indicates increased somnolence and potential increase of inadequate behavior (Akerstedt & Gilbert, 1990; Torvall & Akerstedt, 1988). Usually alpha activity is used to characterize somnolence (Rechstchaffen & Kales, 1968), but there are large inter-individual differences between the EEG and self-perceived somnolence (Akerstedt & Gilbert, 1990).

Other studies have also shown the effects of somnolence on attention, memory, reaction time and problem solving, leading to risk of accidents (Dinges, 1988; Dinges et al., 1994). Nonetheless, somnolence is only one of the factors, among which we can mention alcohol use, sleep restriction/deprivation and inadequate sleep hygiene (Leger, 1995; Philip et al., 1999). One way to evaluate daytime somnolence is through the application of the Epworth Sleepiness Scale (ESS) (Johns, 1991?), Multiple Sleep Latency Test (MSLT) (Hansotia, 1997) and the Test of Maintenance of Vigilance (Noda et al., 1998).

Individual differences such as house factors, age, gender and chronobiology may influence the tolerance of the individual for shift work. Practice of sports and the ability to sleep have been studied in an attempt to find significant effects that may influence sleep or alertness, improving the adaptation to shift work (Härmä, 1995).

Caffeine is used to reduce sleep, but it is less effective than a nap (Akerstedt, 1995; Walsh et al., 1995). When used in the beginning of the night, caffeine in several doses increases alertness and improves psychomotor performance, mainly between 10:30 p.m. and 1:20 a.m. (Rosekind et al., 1995). However, both caffeine and hypnotics reduce, but does not eliminate sleep problems associated with alternating shift work (Walsh et al., 1995).

Another technique is to allow the worker to take naps, which has being taken into consideration as a possible strategy to increase vigilance state in shift workers (Stampi, 1992). The objective is to improve performance and alertness during the night in operational workers who perform the same task for a long period of time (Dinges, 1995; Rosekind et al., 1995). However, it presents a negative effect which is sleep inertia immediately after the nap, when the subject still presents smaller alert and responses (Muzet et al., 1995). Sleep inertia, however, depends on the quantity and quality of the nap (Haslam, 1985; Rosa et al., 1983), of the sleep stage that precedes waking (Stones, 1977), the place of napping (Matsumoto, 1981) and the time of the day it occurred (Balkin & Badia, 1988).

It is evident that the quality and time of sleep are important for an adequate diurnal alertness, and the ideal total sleep time is dependent on individual variations. Since both day- and nighttime sleep fragmentation reduce alertness during the active period it is necessary that during the sleep period, there are long periods without arousals and with maintenance of the sleep architecture (slow wave sleep and paradoxical sleep)(Gilbert, 1995).

Another sleep condition related to increased risk of accidents is obstructive sleep apnea-hypopnea syndrome (OSAHS)(George & Sliley, 1999). OSAHS patients submitted to a driving simulation

for 30 to 90 min show reduced concentration, augmented reaction time and reaction fatigue. They are also 2 to 3 times more involved in vehicle accidents than the general population, indicating once again that this syndrome is a risk factor for car accidents (Barbé et al., 1998). Prevalence of OSAHS in professional drivers is larger than in the general population and may be related to age, obesity and mainly sedentariness. Complaints of snoring and excessive daytime somnolence are also observed in this population. This is why a more specific evaluation of this population is necessary, through detailed clinical and polysomnographic (PSG) assessments.

More studies and orientation are necessary to employers and employees, since a good planning of the working schedule is essential and, consequently, more awareness of the risk of driving or working when sleepy or at the imminence of sleep. These risks may be potentiated in specific times, which are biologically vulnerable (sleep portal) during the day (Horne & Reyner, 1999).

3. Conclusion

It is possible to observe that sleep disorders, especially excessive somnolence, which may be present in a primary or secondary form, may be one of the most likely causes of the high index of vehicle and work accidents.

It is imperative, therefore, to observe the working schedules of professional drives and shift workers in a detailed manner, in order to fit the biological rhythm and chronotype of the individual as best as possible to the work. This procedure will result in reduction of operational costs for the industries and, at the same time, will provide a better quality of life for the worker, by decreasing the incidence of sleep disorders.

It is also important to perform a good clinical evaluation, by a Sleep Medicine expert, a PSG evaluation (Horne & Reyner, 1995), Multiple Latency test (Hansotia, 1997) and Maintenance of Vigilance (Johns, 2000). In addition the individual's biological rhythm and chronotype must be assessed (Benedito-Silva et al., 1990) so the working schedule will suit the worker. It is also extremely important that occupational medicine doctors learn about sleep disorders and the aspects related to chronobiology in order to reduce the loss of quality of life and of health of shift workers.

In face of such a situation it is essential that the industries have alternating working hours, evaluation of the sleep pattern and complaints of shift workers, especially those who work in the night shift, so it is possible to improve the workers professional performance and quality of life. This evaluation may be carried out by means of a specific questionnaire, where the severe cases that are found out can be guided to a sleep laboratory for a specific medical analysis and PSG evaluation (Laubel et al., 1999).

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Modeling the Effects of Sleep Debt: On the Relevance of Inter-Individual Differences

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Modern society is increasingly driven to take advantage of the full 24 hours of the day. Therefore, despite large-scale automation efforts, the demand and desire for wakefulness at all hours of the day steadily increases. However, two fundamental neurobiological processes do not readily adapt to this situation. On the one hand, there is an imperative need for sleep, the fulfillment of which may be postponed but eventually cannot be ignored. On the other hand, the biological clock is driving wakefulness during the day, but not during the night. Thus, living in a society running 24/7 presents considerable challenges for sleep and wake neurophysiology. Nevertheless, the 24-hour society offers unique opportunities for those who have little need for sleep, for those who have low vulnerability for the functional impairment associated with sleep loss, and for those whose circadian phase position or rate of adjustment allows them to be awake at night naturally or to rapidly adapt. Inter-individual differences in sleep need, vulnerability to sleep loss and circadian adaptation remain understudied scientifically and are rarely considered theoretically (e.g., in mathematical models) or practically (e.g., in interventions for sleepiness and fatigue in the workplace). Here we present evidence that inter-individual differences in sleep need and in vulnerability to the effects of sleep loss need to be taken into account when modeling the waking neurobehavioral response to sleep deprivation.

The temporal dynamics of neurobehavioral functioning

To describe and understand the temporal profile of neurobehavioral functioning, researchers have applied the two-process model of sleep–wake regulation [3,4,6]. This model, which was designed to predict the timing and duration of sleep, consists of a *homeostatic* process (process S) and a *circadian* process (process C), which are combined to estimate the timing of the onset and offset of sleep. The homeostatic process represents the drive for sleep that increases progressively during wakefulness, and decreases during sleep (symbolizing the physiological recovery obtained from sleep). Sleep is triggered when the homeostatic drive increases above a certain threshold (unless wakefulness is deliberately maintained). Wakefulness is spontaneously invoked when the homeostatic drive has decreased sufficiently during sleep to fall below another threshold. The circadian process represents the daily oscillatory component in the drive for sleep and wakefulness, which is modeled as circadian variation in the threshold values. It has been suggested that the circadian system actively promotes wakefulness more so than sleep. The circadian drive for wakefulness can be observed as, for instance, the spontaneously enhanced alertness that occurs during the day following a sleepless night. From this perspective, the homeostatic and circadian systems can be viewed as opponent processes [15].

The homeostatic and circadian components of the two-process model can also be utilized to predict waking neurobehavioral alertness [16,17,19]. In this conceptualization, the circadian and homeostatic processes influence neurobehavioral functioning simultaneously. The interaction of the two processes appears to be oppositional during natural diurnal wake periods, such that a relatively stable level of alertness and performance can be maintained throughout the day [7]. At night, however, a rapid breakdown of alertness and performance capability is predicted and observed, as the two processes are now both permissive of sleep. During prolonged sleep deprivation, neurobehavioral deficits occur in response to increasing homeostatic sleep drive as well as circadian-modulated withdrawal of waking drive, making the impairment progressively worse during biological night [29].

A model-based understanding of the interaction of the homeostatic and circadian regulation of sleep and wakefulness can be helpful to elucidate the consequences of stress on the homeostatic and circadian regulatory systems. Obvious stressors on these systems include extension of wakefulness (i.e., sleep deprivation; e.g., [10]), sleep–wake cycle displacement (e.g., shift work; [22]), and circadian displacement (e.g., transmeridian flights; [31]). Under such circumstances, the circadian and homeostatic systems can be found to interact in a way that decreases neurobehavioral function. Alertness and performance may decline considerably, enhancing the probability of accidents in the workplace [8] and on the road [21,25]. However, the predicted magnitude of the performance decline depends on the precise values of at least three independent model parameters:

– the timing and/or rate of adjustment of the circadian process (*circadian phase*);
– the amount of sleep needed per day (*sleep need*);
– the rate of impairment per hour of sleep loss (*vulnerability*).

The *vulnerability* parameter is an often overlooked dimension of the waking neurobehavioral response to sleep deprivation, that is distinct from the *sleep need* parameter. For example, two individuals could each have the same *sleep need* of, say, 8 hours per day, but when they both receive only 4 hours of sleep on a given day, the magnitude of their performance impairment due to the ensuing sleep loss could still be substantially different—thus, the rate of impairment per hour of sleep loss (i.e., *vulnerability*) would be different for the two individuals in this example. The *vulnerability* parameter has thus far not been considered explicitly in models of sleep–wake regulation.

There is evidence for substantial inter-individual differences in the values of each of the three model parameters *circadian phase*, *sleep need* and *vulnerability*. Historically only the variability in *sleep need* has been broadly recognized and focused upon.

Inter-individual differences in circadian phase, sleep need and vulnerability

Considerable variation has been observed among humans in the timing of circadian rhythmicity (i.e., *circadian phase*). This variation finds expression in morningness/ eveningness [14,20]. By means of constant routine experiments, it was shown that the phase positions of the endogenous circadian rhythms of extreme morning-types and evening-types, as measured by core body temperature, differ by more than 2 hours. Possibly due to the interaction with the homeostatic drive for sleep, this results in a difference of at least 4 hours in the timing of peak alertness between morning-types and evening-types [20]. This may explain the finding that evening-types are more tolerant to permanent night work than morning-types [26]. Similarly, in shift work, morning-types appear to have a relative advantage on morning shifts and a disadvantage on night shifts, and vice versa for evening-types (e.g., [2,5]). Furthermore, evening-types appear to adapt more easily to rotating shift work [24].

Inter-individual differences in *sleep need* have been carefully studied in the context of the two-process model of sleep–wake regulation. Using the waking electroencephalogram (EEG) as a physiological marker of sleep homeostasis, it was found that naturally short sleepers tolerate a higher homeostatic pressure for sleep than naturally long sleepers [1]. There may be a genetic basis for this variability in natural *sleep need* [18]. Not everybody who reports to be a naturally short sleeper actually is one, though [23]; many can sustain living on a short sleep schedule for a while but eventually build up a sleep debt (resulting in the need to extend sleep on the weekend, for instance).

Individuals who sleep comparable amounts each night, who have comparable circadian phases, and who are comparable neurobehaviorally when not sleep-deprived, are observed to be differentially affected—by as much as an order of magnitude—in their neurobehavioral functioning when exposed to loss of sleep [11]. These inter-individual differences in *vulnerability* are consistent, as was evident in the three studies that have been done on repeated exposure to sleep loss in the same subjects [28,30,32]. The studies of Wilkinson [32] and Webb and Levy [30] both reported substantial inter-individual differences in the effects of sleep deprivation, that appeared to reliably reflect greater sensitivity of some subjects to deprivation. Neither study actually quantified the stability of inter-individual differences in response to sleep loss, but this issue was addressed in the third study [28]. This study sleep-deprived 10 healthy adults (who were comparable in *sleep need*) on two separate occasions, and found that a significant portion of the variance in vigilance performance deficits (58% of total variance) was stable. This finding indicated that some individuals were consistently more vulnerable to neurobehavioral deficits due to sleep loss than others (i.e., *trait vulnerability*).

Inter-individual differences in the response to chronic sleep loss

Chronic sleep restriction causes cumulative sleep debt, which results in increasing neurobehavioral performance deficits [12]. The magnitude of these deficits depends on the cumulative amount of sleep loss. When fixing time in bed, the actual amount of sleep loss depends on *sleep need*. The magnitude of performance deficits, therefore, depends on *sleep need* as well. It also depends, however, on the *vulnerability* to the effects of a given amount of sleep loss. Thus, there are at least two factors for which inter-individual differences affect the waking neurobehavioral response to chronic sleep restriction (i.e., *differential sleep need* and *differential vulnerability)*. In a laboratory experiment, we studied the effects of chronic sleep restriction in a group of subjects who had a sleep–wake history indicative of equivalence in circadian timing. Using the data obtained for neurobehavioral functioning in this study, we evaluated an additive (i.e., linear) model of sleep debt postulating that, for a given individual, each hour of sleep loss is equally important in determining the performance deficits resulting from chronic sleep restriction. We explicitly estimated inter-individual variability in *sleep need* and *vulnerability* as part of the model.

Data from n=24 subjects (ages 22–36; 5 females) who spent 20 days inside a controlled laboratory environment were used. After 3 baseline days (time in bed 23:30–07:30), subjects were partially sleep deprived for 14 days. Time in bed was restricted to 4h (03:30–07:30; n=9), 6h (01:30–07:30; n=8), or 8h (23:30–07:30; n=7) per day. Neurobehavioral performance was tested every 2h during wakefulness, and included a 10-minute psychomotor vigilance test (PVT). The daily average (09:30–23:30) of PVT lapses (reaction times ? 500 ms) per test bout was used to measure neurobehavioral impairment (IMP), relative to baseline. Sleep was recorded polysomnographically on all baseline nights and on 2 out of every 3 nights throughout the 14-day restriction period, and total sleep time (TST) was assessed using conventional sleep scoring criteria. For the 4 days with no sleep recordings, TST was estimated by linear regression interpolation over the other days of sleep restriction. Finally, cumulative TST (CTST) was computed for each day of restriction.

The additive model of sleep debt

The additive model of sleep debt was formulated as:

$$IMP_D \sim \alpha .(\gamma . D - CTST_D)$$

where D is the day of sleep restriction (1–14), $CTST_D$ is the cumulative total sleep time (in hours) on day D, IMP_D is the neurobehavioral impairment (in PVT lapses) on day D, α is the *vulnerability* parameter (in PVT lapses per hour of sleep loss), and γ is the *sleep need* parameter (in hours). Between-subjects variances for α and γ were estimated in the model by incorporating random effects for these parameters, assuming a bivariate normal distribution. The model was fit to the data using mixed-model regression, for which we applied the SAS procedure NLMIXED [27].

We found that the model had good predictive potential. The residual error variance was only 17.4% of the overall variance in the data. Thus, 82.6% of the variance was explainable by the model assuming that subject-specific values for *sleep need* and *vulnerability* were known. Without subject-specific knowledge, the explained variance dropped dramatically to 21.9%. Considerable variability in both *sleep need* as well as *vulnerability* contributed to the additional variance explained when inter-individual differences were taken into account. These results highlight the importance of inter-individual differences in modeling the effects of

cumulative sleep debt [9]. In this experiment, the estimated *sleep need* was 8.2 hours, and the estimated standard deviation for inter-individual differences in *sleep need* was 2.6 hours. As the subjects in the study were similar in circadian timing, we did not as yet incorporate inter-individual variability for the *circadian phase* parameter in the model.

Conclusion

Despite the success of the two-process model of sleep–wake regulation to describe and predict group data in a variety of experimental protocols, it has proven to be difficult to apply the model to individual subjects reliably. There is mounting evidence that inter-individual differences in variables affecting the model are consistent and substantial. Conceivably, the parameters of the two-process model could be adjusted to match each individual's characteristics. The challenge will be, however, to find objective behavioral or physiological markers of these parameters. For *circadian phase*, core body temperature or melatonin profiles can provide reliable markers. To date, there is no consensus about what biological markers should be used for *sleep need*, or for *vulnerability* to neurobehavioral impairment from sleep loss. When probed with a psychomotor vigilance test, *vulnerability* to neurobehavioral impairment from sleep loss is observed as "wake state instability" [13]. This is the term we use for the mixture of normal performance with lapses and false responses, increasing in frequency with time on task, observed to be a consequence of sleep loss. A quantitative measure of wake state instability (e.g., performance lapses) may serve as a marker of *vulnerability*. It is becoming increasingly clear that inter-individual differences in *vulnerability*, as well as *sleep need* and *circadian phase*, should be taken into account explicitly when studying the waking neurobehavioral consequences of sleep deprivation or circadian misalignment [9]. These stressors do not have the same effects for everyone.

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CLUB HYPNOS EVENTS HELD IN SAN DIEGO & PHILADELPHIA

Club Hypnos at the Association for the Advancement of Behavior Therapy

by Kathy Sexton-Radek, Ph.D.

A Club Hynos reception was held at the Association for the Advancement of Behavior Therapy conference in Philadelphia November 17, 2001. The event followed their special interest group on Insomnia meeting that is coordinated by Kenny Lichstein of Memphis State. Approximately 25 were in attendance. Although the event got off to a few minutes late start because of the length of the meeting, all sleep researchers in attendance seemed to enjoy the time to socialize and snack on some treats. The SRS membership forms were passed out to everyone and many conveyed their thanks for the SRS hosting of a social at the AABT conference. A word of note to all future Club Hypnos coordinators, read through the helpful information sent by Jodi Mindell to make sure all the details of hosting the event are in order-it is all there in the attachment that is sent to you and really helps to identify the steps necessary for a success! Furthermore, many of the sleep researchers at the event knew each other and the new people were readily introduced to members in attendance at the social-it proved to be a wonderful opportunity for networking.

Club Hypnos at Society for Neuroscience 2001

This last November's Club Hypnos reception at the Society for Neuroscience meeting in San Diego again proved to be a great success. This year marked the seventh time this SRS-sponsored social event has been held. The original idea for such an event, conceived by Dr. Adrian Morrison, was to publicize opportunities in sleep research and attract students to the field. Since the Society for Neuroscience meeting held in New Orleans in 1997, Club Hypnos has met sequentially in time with the National Center for Sleep Disorders Research (NCSDR)-sponsored "Neuroscience in Sleep and Circadian Biology Data-Blitz". This cooperation has resulted in increased publicity and visibility for the SRS and Club Hypnos.

I am pleased to report that this year there were 150 visitors to Club Hypnos in San Diego. Not only did several SRS Executive Committee members attend, but Dr. Carl Hunt; the recently appointed Director of the NCSDR also paid a visit. The food provided proved to be a big hit once again and since all the SRS membership brochures I brought were picked up, I am hoping that our central office will soon report that some new members were recruited. In closing I would like to encourage all of you to think about the other scientific meetings you attend and consider if a Club Hypnos social would enhance your meeting experience. As many of you know, the Society for Neuroscience annual meeting is huge and often overwhelming. Through hosting Club Hypnos I have a gained a renewed interest and enthusiasm for this meeting and believe that by providing this SRS "home away from home" we are promoting the field of sleep research.

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SRS Young Investigator Award Winners

Thomas Thannickal, Ph.D.

Dr. Thomas Thannickal completed his Ph.D. degree in 1995 from Mahatma Gandhi University, Kerala, India and joined as Research Officer in the Department of Physiology, All India Institute of Medical Sciences, New Delhi, India. He came to the United States in June of 1999 to work under the supervision of Prof. Jerome Siegel, Department of Psychiatry, UCLA. Dr. Thannickal is studying the neurophysiological basis of human narcolepsy.

Karine Spiegel, Ph.D.

Karine received a Bachelor's degree in Biochemistry in 1990 and a Master's Degree in Animal Physiology in 1991 from the Université Louis Pasteur, Strasbourg, France. Afterward, Karine entered the PhD program in Neuroscience at that university under the direction of Gabrielle Brandenberger. The Laboratory of Dr. Brandenberger was studying the relationships between sleep and physiological parameters, especially endocrine function. In 1995, Karine obtained a Ph.D. in Neuroscience, "summa cum laude" on "Prolactin secretion during the sleep-wake cycle in humans" and became convinced of the primary importance of sleep for physiological functions and health. However, at that time, the consensus was that sleep was for the brain not for the rest of the body. Dr. Spiegel was then offered a post-doctoral fellowship in the laboratory of Dr. Eve Van Cauter in the Department of Medicine at the University of Chicago. It is there that Dr. Spiegel conducted a study that examined the effect of sleep debt on EEG patterns, metabolic, hormonal, cardiovascular, cognitive and immunological functions that lead to this award. In 1997, Dr. Spiegel obtained a 2nd PhD in Biomedical Science and began pursuing postdoctoral training at the "Université Libre de Bruxelles", Bruxelles, Belgium. Karine's current work focuses on the reactivity and the resilience to stress in the presence of a sleep debt in humans. Besides sleep and science, Karine is fascinated with the underwater world and spends as much time as possible free diving and scuba diving.

Sean P.A. Drummond, PhD

Sean first became hooked on sleep research as an undergraduate working with Michael Perlis and Dick Bootzin at the University of Arizona. After graduating, he worked with Sarah Mosko for two years at UC Irvine Medical Center. He then entered graduate school in the San Diego State/UC San Diego Joint Doctoral Program and joined the lab of Chris Gillin. While in graduate school, Sean became more involved in the SRS and the sleep community. He served on the first 3 Trainee Program Committees, serving as Chair of the third. In 1997, he served as the Trainee Member at-Large to the SRS Executive Committee and currently serves on the Committee for Animal Research Ethics. Sean's dissertation, which gave rise to the paper receiving this award, used functional magnetic resonance imaging to examinethe effects of total sleep deprivation on cognitive performance and brain function. Sean completed his Clinical Psychology Internship at the Tucson VA Medical Center and graduated with his PhD in June 2000. He has since returned to UCSD as a post-doc in Greg Brown's lab where he continues to study the effects of sleep deprivation with FMRI and other techniques. Sean would like to thank all of his mentors throughout the years, as each has provided something unique to his scientific development. Of particular note are Chris Gillin who mentored him through his dissertation and has contributed more than any other mentor to Sean's professional growth, as well as Michael Perlis, whose friendship and advice have been invaluable.

Markus Schmidt, MD, PhD

Markus received his undergraduate degree from University of Toronto in 1989 where he did an undergraduate thesis on the "Evolution of Sleep and its Implication for Function" under the direction of Prof Allen Rechtschaffen, University of Chicago. He completed the MD/PhD program at the Medical College of Ohio in 1997 and completed a graduate thesis in the laboratory of Prof. Michel Jouvet in Lyon France on the "Neurophysiology of Sleep-Related Penile Erections". Currently, Dr. Schmidt is finishing residency training in clinical Neurology at the Cleveland Clinic. He will be joining his father, Helmut S. Schmidt, M.D., to practice sleep disorders medicine at the Ohio Sleep Medicine and Neuroscience Institute in Dublin, Ohio this summer.

NIH LOAN REPAYMENT PROGRAMS ANNOUNCED

Two new NIH loan repayment programs have been announced that will be of interest to trainees who have educational loans to pay off. They are the Clinical Research Loan Repayment Program for Extramural Investigators and the Pediatric Research Loan Repayment Program. In the past, NIH has had loan repayment programs to attract researchers to work at NIH, but now the same type of program will apply to extramural research.

To be eligible, applicants will need to be citizens, nationals, or permanent residents of the U.S., have a Ph.D., M.D., Pharm.D., D.O., D.D.S. or equivalent doctoral degree, be affiliated with the NIH by having a postdoctoral National Research Service Award (NRSA), individual postdoctoral fellowship, or institutional postdoctoral research training grant award, or be the recipient of an R03, R21, R01, or various K-awards, among others, and have qualifying educational debt in excess of 20 percent of their annual compensation on the expected date of program eligibility.

The idea is to counter the pressure on individuals to take exclusively clinical positions to pay off their educational loans and instead provide an incentive to be engaged in extramural research. NIH will repay a substantial portion of educational loans (both undergraduate and graduate) for those associated with NIH as described above. The loan programs can repay up to a maximum of $35,000 a year, for two and perhaps three years, dependent upon the total eligible debt. In return, participants will sign a contract agreeing to conduct qualified clinical research for a minimum of two years.

The links below are information sheets in the form of answers to Frequently Asked Questions (FAQ). There are still important details to be worked out and more information will come available in the next few weeks. The program went into effect December 1, 2001 and applications are expected to be available after December 28, 2001. Applications can be requested at the links below and will be sent as soon as they are available.

Clinical Research Loan Repayment Program for Extramural Investigators:
http://lrp.info.nih.gov/extramural/FAQ_CRE.htm

Pediatric Research Loan Repayment Program:
http://lrp.info.nih.gov/extramural/FAQ_PR.htm

WORLD FEDERATION OF SLEEP RESEARCH SOCIETIES CONGRESS IN PUNTE DEL ESTE, URUGUAY

The World Federation of Sleep Research Societies co-sponsored with the Federation of Latin American Sleep Societies an interim congress, "Physiological Basis for Sleep Medicine," in Punte del Este, Uruguay, October 21-25. Professor Ricardo Velluti and his organizing committee did a splendid job in spite of difficulties posed by world events. The scientific program was excellent, in my opinion, and I heard no one disagree. A very friendly and efficient administrative staff ensured a smoothly running program. The intimacy of the venue allowed good interactions among senior scientists and trainees. The latter had the opportunity to socialize with the many Uruguayan students assisting the conference organizers. Adding to the congress's success were the very enjoyable social events: a gaucho lunch on a ranch and a lively farewell dinner and dance. Finally, the Governing Council met and recommended that the Executive Committee elect Adrian Morrison to the interim presidency to guide the federation until the next regular election in 2003 and Dag Stenberg be elected to fill the office of Secretary General until 2003 when the entire Executive Committee will step down.

Adrian R, Morrison, President

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The Sleep Research Society,
The Society for Research on Biological Rhythms
and The National Institutes of Mental Health (USA)
are pleased to announce a jointly-sponsored,one-day conference*

Circadian Rhythms and Sleep; Views to the Future

May 22, 2002, Amelia Island, Florida
in association with the biannual meeting of the
Society for Research on Biological Rhythms

The goals of this program are to:

- Stimulate interactions and mutual appreciation between the scientists in the sleep and circadian rhythms communities.
- Improve communication between scientists working at the molecular, physiological, and clinical levels of inquiry.
- Excite trainees about potential areas of discovery.

Plenary Lectures

- *Sleep: A rhythmic necessity*
  Derk-Jan Dijk (University of Surrey)
- *Future directions in circadian and sleep research*
  Fred Turek (Northwestern University).

Symposium Presentations

- *How does sleep differ in different species*
  Jerome Siegel (UCLA)
- *From SCN to sleep centers in the brain; finding the pathways*
  Kazue Semba (Dalhousie University)
- *What influence do sleep, sleep deprivation and activity have on the circadian pacemaker?*
  Ralph Mistlberger (Simon Fraser University)
- *Gene expression and the sleep-wake cycle.*
  Joan Hendricks (University of Pennsylvania)
- *The rhythm of human sleep and wakefulness*
  Mary Carskadon (Brown University)
- *Circadian rhythm sleep disorders*
  Phyllis Zee (Northwestern University)
- *From clock genes to clinical medicine; the example of FASPS*
  Louis Ptacek (University of Utah)
- *Enlightenment from studies of blind human subjects.*
  Alfred J. Lewy (Oregon Health Sciences University)

*Planning Committee

Robert L. Sack, Co-Chair	Martha Gillette	Michael Vitiello
Israel Lederhendler, Co-Chair	Phyllis Zee	Derk-Jan Dijk
	Orfeu Buxton	

A special program for trainees will be held in conjunction with this conference. Travels awards are available for student members of the SRS and SRBR.

Registration for this conference in included in the SRBR registration fee. **A Trainee Program** will also be held at this meeting. Program and registration materials will be posted on the SRS and SRBR websites as soon as they become available. For additional information, please contact Lance Brink at the SRS National Office at 507-285-4384.

CIRCDIAN RHYTHMS AND SLEEP: VIEWS TO THE FUTURE CONFERENCE—The Sleep Research Society (SRS), The Society for Research on Biological Rhythms (SRBR) and The National Institutes of Mental Health (USA) are planning a one-day conference entitledCircadian Rhythms and Sleep; Views to the Future. It will be held on May 22, 2002, at Amelia Island, Florida in association with the biannual meeting of the SRBR. For registration information, contact Lance Brink, Executive Director of the SRS (lbrink@aasmnet.org).

HELP! RE 9/11/01; RECORDING DREAMS—I'm seeking help with my research from anyone who regularly records his or her dreams.

As most of you know, I have been studying for many years the dreams of people who have experienced a serious trauma.

I believe that all of us experienced a serious trauma on September 11, 2001. Therefore I would be most grateful for help from any of you who have been keeping a dream log or regularly recording your dreams for some time, and who would be willing to share some of them with me.

If this description fits you and you'd like to participate, what I would need is a copy of your last ten dreams recorded before 9/11/01, and your first ten dreams starting the evening of 9/11/01. I need the dreams exactly as they are written. Do not select particular dreams, omit dreams, or alter dreams in any way (except to omit or change people's names if you wish).

To anyone who can help me with this research, I would like to be helpful in turn, in any way I can.

At the moment all I can think of is this: First of all I want to assure you that your dreams will be treated with complete respect and confidentiality. You will not only be helping me, but contributing to the field of dream research and our increasing understanding of the nature and value of dreams. I will be happy to discuss with you the progress and results of studies which involve your dreams. Finally I'll be happy to send you a copy of my recent book Dreams and Nightmares if you'd like one.

If you can do the above, please send dreams to me, or get in touch with me, at Ernest Hartmann M.D., 27 Clark St., Newton MA 02459, or EHDream@AOL.com.

LIFE OF THE SLEEPY PERSON V CONFERENCE : NEW PERSPECTIVES ON DISORDERS OF EXCESSIVE SLEEPINESS—Center for Narcolepsy Research, University of Illinois at Chicago;
March 7-8, 2002
Chicago Marriott O'Hare, Chicago, IL

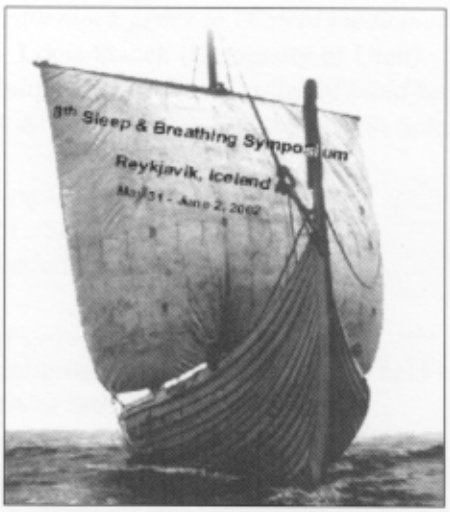
Events: Support group meeting in PM of March 7; next day speakers include Drs. Emmanuel Mignot, Patricia Mercer and Roza Hayduk, and Mr. Barry Taylor, Attorney
For more information: Visit www.uic.edu/depts/cnr or contact the center at 312.996.5176 or email: narcolep@listserv.uic.edu.

SLEEPTECH, LLC NAMES NEW SENIOR MANAGEMENT TEAM (Kinnelon, NJ)—Vyto Kab, Co-Managing Director, SLEEPTECH, LLC, is pleased to announce the following newly created positions. These positions are a direct result of the growth and expansion SleepTech is experiencing as the regional leader in hospital-based sleep disorders diagnostic and treatment services in the Northeast: Kevin Kelly has been named Chief Financial Officer. He comes to us from Saint Barnabas Medical Center where he was also Chief Financial Officer. Mr. Kelly's responsibilities will be focused on new business development and strategic planning. Mark Rose has been named Chief Information Officer. He comes to us from Fitch, Inc., an international rating agency serving capital markets, where he served as Managing Director of Information Technology. Mr. Rose will be responsible for our technological advancement in the industry, including becoming paperless, and our commitment to HIPAA Compliance. For information regarding SLEEPTECH's services, please call 1-800-774-7533 or email info@sleeptech.com.

calendar of events

February 2002

Association Name	Meeting Name	Dates	Location
National Institute on Alcohol Abuse & Alcoholism	Advisory Council Meeting	February 7	NIH
Sleep Research Society	Board of Directors Meeting	February 7	Conference Call
National Heart Lung and Blood Institute	Advisory Council Meeting	February 7—8	NIH
National Institute of Neurological Disorders and Stroke	Advisory Council Meeting	February 14—15	NIH
American Association for the Advancement of Science	Annual Meeting	February 14—19	Boston, MA
National Institute on Drug Abuse	Advisory Council Meeting	February 20—21	NIH



8th Sleep & Breathing Symposium

A Satellite of the 16th Bi-Annual Meeting of the ESRS
May 31 – June 2, 2002
Loftleidir Hotel/Conference Center, Reykjavik, Iceland.
Deadline for registration and abstracts: **February 1, 2002**.
Registration fee discount for attendants of both meetings. Young investigator travel awards available – see the web site. For more information, e-mail Dr. Leszek Kubin at lkubin@vet.upenn.edu or visit:
http://www.uphs.upenn.edu/sleepctr/iceland/index.html

March 2002

Association Name	Meeting Name	Dates	Location
Sleep Research Society	Board of Directors Meeting	March 22-23	TBD
American Academy of Sleep Medicine	Board of Directors Meeting	March 8—10	Naples, FL

April 2002

Association Name	Meeting Name	Dates	Location
National Sleep Foundation	National Sleep Awareness Week	April 1—7	Washington, DC
Society of Behavioral Medicine	Annual Meeting	April 3—6	Washington, DC
Sleep Research Society	Board of Directors Meeting	April 4	Conference Call
American Board of Sleep Medicine	Part 2—Board Exam	April 8	Multiple Locations

May 2002

Association Name	Meeting Name	Dates	Location
Sleep Research Society	Board of Directors Meeting	May 2	Conference Call
National Heart Lung and Blood Institute	Advisory Council Meeting	May 9 - 10	NIH
National Institute of Mental Health	Advisory Council Meeting	May 9 - 10	NIH
American Thoracic Society	International Conference	May 17 - 22	Atlanta, GA
National Institute on Aging	Advisory Council Meeting	May 21 - 22	NIH
National Institute on Nursing Research	Advisory Council Meeting	May 21 - 22	NIH
National Institute Drug Abuse	Advisory Council Meeting	May 22 - 23	NIH
Society for Research on Biological Rhythms	Annual Meeting	May 22 - 26	Amelia Island Plantation, Jacksonville, FL
National Institute of Neurological Disorders and Stroke	Advisory Council Meeting	May 30 - 31	NIH

POSITIONS AVAILABLE

HARVARD NEUROSCIENCE ACADEMIC/POST-DOCTORAL POSITIONS.—Multidisciplinary lab studying the neural control of sleep and wakefulness in animal models. One position is for an experienced in vitro electrophysiologist (Asst. Prof. or postdoc); other postdoc positions in: microdialysis/HPLC & ELISA, in vivo electrophysiology, molecular biology, histology. NIH stipend or better. EOE. Send: CV, brief description of research experience/interest, names & contact information for three references to: R. E. Strecker, M. Thakkar, R. Basheer, R. Tao, & R.W. McCarley, Harvard Medical School, Brockton VAMC (151C), 940 Belmont St., Brockton, MA 02301; email: robert.strecker@rcn.com

NOTICE OF VACANCY #3001

Department of Veterinary and Comparative Anatomy, Pharmacology and Physiology (VCAPP)
College of Veterinary Medicine
Washington State University
Pullman, WA 99164-6520

POSITION: Assistant/Associate Professor within the Neuroscience Program

TERMS OF SERVICE: Tenure-track Appointment

SALARY: Dependent upon qualifications

EFFECTIVE DATE: July 1, 2002

DUTIES: Establish a strong independent and extramurally funded research program in Sleep Research. Serve the Department of VCAPP and the Neuroscience Program by participating in teaching and in departmental and college committees.

QUALIFICATIONS: Required: Ph.D. or equivalent

Preferred: Three (3) years of postdoctoral research experience. Those with a research program in the area of sleep will be given preference. Experience and interest in effectively teaching undergraduate or graduate students. Willingness and ability to engage in collaborative research, interest and training in neurobiology, publications in high-impact, peer-reviewed journals, and demonstrated communication and interpersonal skills.

NEUROSCIENCE PROGRAM: The Program in Neuroscience at Washington State University offers the Bachelor of Science degree and the Neurosciences Graduate Program offers the Master of Science, and Doctor of Philosophy degrees. There are over 40 neuroscience faculty from four colleges and three WSU campuses. There is a strong emphasis on neuroscience research in areas such as alcohol and drug abuse, sleep, ingestive behavior, cell biology, behavior, and neuroendocrinology.

DEPARTMENT: The Department of VCAPP has 25 full-time and 47 adjunct faculty. The department's mission involves teaching, research and service. Courses in anatomy, pharmacology, physiology, toxicology and neuroscience are offered to under-graduate, professional, and graduate students. The department has a strong focus in neuroscience and cardiovascular/muscle biology with a large number of extramurally funded laboratories in these areas. Degrees offered are: M.S. and Ph.D. in Neuroscience as well as in Veterinary Science. Additional interdepartmental programs include Genetics and Cell Biology, Reproductive Biology, Pharmacology-Toxicology, and Bioengineering. VCAPP is also home department for the undergraduate Neuroscience Program.

COLLEGE: The College of Veterinary Medicine at Washington State University offers a 4-year course of study to acquire the Doctor of Veterinary Medicine degree. Graduate programs in veterinary science lead to the M.S. and Ph.D. degrees. The college participates in a regional program with Idaho and Oregon, which is referred to as the Washington-Oregon-Idaho Regional Program in Veterinary Medicine. The College is organized into 3 departments: Veterinary and Comparative Anatomy, Pharmacology and Physiology; Veterinary Microbiology and Pathology; and Veterinary Clinical Medicine and Surgery.

UNIVERSITY: Washington State University, a PAC-10 school, is a land-grant institution with an enrollment of approximately 22,000 students statewide. Branch campuses are located in 3 urban communities across the state. The University has a wide variety of outstanding academic programs, offering more than 150 undergraduate majors, options, and degree programs, and more than 100 masters' and doctoral degrees. Special academic experiences are available through the nationally recognized Honors Program, international education, and research opportunities. The University of Idaho, also a land grant university, is located in Moscow, Idaho, 8 miles from the Pullman campus. These communities offer a friendly, small-town living environment and access to a rich assortment of cultural, recreational and athletic attractions. Pullman combines a mild climate, clean air, good schools, affordable homes, and a low crime rate. Pullman (population 25,000) is about 75 miles south of Spokane, Washington, and lies close to recreational areas in Washington, Oregon, Idaho, Montana and British Columbia.

APPLICATION REVIEW DATE: December 15, 2001 or until filled

APPLICATIONS: Send letter of application, curriculum vitae, list of publications, description of teaching experience, summary of research interests and potential projects, and the names, addresses and phone numbers of three references to:

Sleep Search Committee
Department of VCAPP
Washington State University
Pullman, WA 99164-6520
509-335-6621
bmorton@vetmed.wsu.edu

POSTDOCTORAL OR RESEARCH ASSOCIATE POSITIONS IN

SLEEP RESEARCH

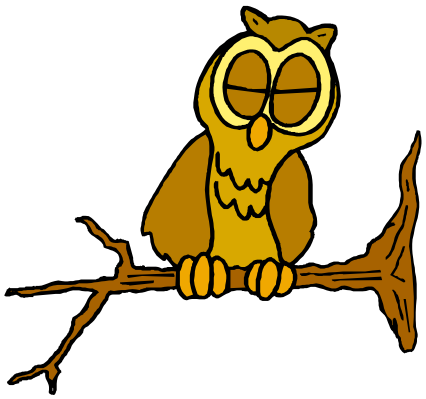
- **Biochemical regulation of sleep**
- **Sleep function, modeling**
- **Sleep neuroendocrinology**
- **Sleep cell biology**
- **Sleep molecular biology**
- **Alcohol and Sleep**

Environment:

- Large well-funded sleep research group composed of 4 individual laboratories plus their lab members.
- Washington State University is a Research I institution.
- Many cultural opportunities in an area with two major universities.
- Small town "Far from the maddening crowd." Great hiking, boating, skiing, fishing, etc. Good for family, safe, quiet, inexpensive living.
- Part of undergraduate and graduate neuroscience programs, the bioengineering program, and the College of Veterinary Medicine.
- Great support staff.

Desired expertise in one or more of these areas:

- Sleep biology
- Cell biology
- Molecular biology
- Neuroendocrinology



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