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**Meet-Up in Minneapolis!**

**SLEEP 2014**

**MINNEAPOLIS, MN**

May 31 - June 4, 2014 • Minneapolis Convention Center

**View our video about Minneapolis!**
Dear Colleagues,

I am delighted to have the opportunity to write, in this first of three presidential updates in the SRS Bulletin, on the various activities of our organization. First, I want to tell you what a great honor it is to be able to serve you as the President of the SRS. I look forward to an active and successful year ahead for the society. I would also like to take this opportunity to thank Ronald S. Szymusiak, PhD for the tremendous work he did as SRS President last year. I look forward to his continuing service over the next year as the Chair of the Government Relations Committee and a member of the Board of Directors.

I would also like to thank all members who attended the very successful SLEEP 2013, the premier meeting in our field. Thanks to the hard work of the APSS staff, a stimulating program and your participation, the meeting was a great success.

Also deserving gratitude are the many SRS Committee members and APSS Program Committee members who volunteer their time throughout the year in service of the society and take time at the SLEEP meeting to attend meetings of their respective committees. In June, several members of the standing committees concluded their terms of service.

The SRS thanks the following members for their loyal service:

**Educational Programs Committee**
Jessica Payne, PhD
Kathleen Sexton Radek, PhD

**Membership and Communications Committee**
Kathryn Reid, PhD
Celyne Bastien, PhD
Helen Burgess, PhD

**Research Committee**
Noor Alam, PhD
Nalaka Gooneratne, MD
Karine Scheuermaier, MD

**Trainee Education Advisory Committee (TEAC)**
Allan I Pack, PhD, MBChB
Megan Ruiter Petrov, PhD – Trainee Member-at-Large
Monique LeBourgeois, PhD
Rachel Manber, PhD

June was a very busy month, and with dedication to our mission of reaching out and increasing education and public awareness about sleep and circadian health, we partnered with the American Academy of Sleep Medicine, to submit a proposal to the [Centers for Disease Control](https://www.cdc.gov), to launch an effort in response to their call for proposals for outreach in the area of “Sleep and Sleep Disorders”. We have recently learned that the CDC will partner with the American Academy of Sleep Medicine and the SRS to support a new “Sleep and Sleep Disorders Awareness Program”. We look forward to working together, to move many of our joint missions forward, with the common goals of educating the public, researchers and practitioners, about the importance of attaining and maintaining sleep and circadian health. This is an exciting new opportunity that will lead to new levels of awareness of the importance of sleep and circadian health, and will feed back to enrich genetic, basic, clinical-translational, patient centered outcomes and virtually all levels of research and technology development pertinent to our field.

I would like to turn our attention to the tasks ahead for the SRS. The [Government Relations Committee](https://www.greenshowroom.com) was established in year 2009 during the presidency of Dr. Clif Saper, and has continued with its efforts, chaired each year by the past president (this year, Dr. Ron Szymusiak). In light of the current grant funding environment, it is my goal to continue the SRS efforts in this important area. This will include continuing our systematic approach to educating Congressional Leaders and NIH Officials about the value and relevance of sleep and circadian research.

Following the APSS meeting in Baltimore, Dr. Phyllis Zee and I visited several Congressional Offices in Washington DC, along with Priyanka Surio and Dale Dirks of the Health and Medicine Counsel of Washington. While on the Hill, we met Senators Dick Durbin and Mark Kirk, of Illinois (see photo on the following page). We thanked them for their support of the NIH and CDC, and encouraged them to continue their efforts to strengthen medical research. We also shared with them our concern about the sleep health of the population, and the importance of sleep and circadian factors for health and safety, and the need for public education in this area.

In times of economic challenge, the SRS has worked to manage finances, while keeping the needs of our members a priority. Through extensive budget review and strategic planning, I will continue to work with our committees and Board of Directors to develop additional revenue streams and provide the same level of services and resources to our members.

I will work closely with the [Educational Programs Committee](https://www.aps.org), to continue developing new educational products and courses that will further the mission of the SRS and be a valuable resource for investigators in our field.

I will work closely with the [Educational Programs Committee](https://www.aps.org) to continue developing new educational products and courses that will further the mission of the SRS and be a valuable resource for investigators in our field. I will continue to work with the [Membership and Communications Committee](https://www.aps.org) to grow our...
membership. We encourage you to engage collaborators from other fields, to join the SRS and to attend our meeting, and to help to grow sleep and circadian research discovery by expanding our collaborative sphere. Our field is an interdisciplinary one that has enormous potential for research growth in bench to bedside, in efficacy testing and education outreach. It is exceedingly important at this time, to strengthen bridges and networks. All of us have important roles to play in this mission.

The SRS will continue to foster our early stage investigators by maintaining support for the tremendously popular Trainee Symposia Series. The Trainee Symposia Series, central to the SRS mission, is a unique opportunity for early career stage scientists to engage in collegial interaction with senior scientists with successful careers across the broad spectrum of sleep and circadian research. This year I will be reaching out to you, our membership, to help support Trainee Day activities, so that we may continue to develop a stronger pipeline of investigators and educators, for the future of our field.

The coming year will be filled with challenges, but also many new opportunities for growth as a field, and I am very pleased to have the opportunity to work with you in this mission. Your input and involvement in the SRS is appreciated and necessary for a healthy organization. Please contact the SRS national office if you wish to volunteer on a committee or have suggestions to improve the society. The national office can be contacted at bmanning@srsnet.org or (630) 737-9763.

Sincerely,

Janet Mullington, PhD
SRS President

From left to right: Senator Durbin, Dr. Mullington, Dr. Zee, Senator Kirk
By Jamie Zeitzer, PhD

Welcome to the Fall issue of the Sleep Research Society Bulletin. I have been passed the mantle of editorship from Dr. Helen Burgess, who has provided us with wonderful information for the past three years. Kudos! I look at myself as the caretaker of the Bulletin, which should be a conduit for information flow among our vibrant membership. Is was wonderful to see so many of you at the recent SLEEP conference in Baltimore and the weather, thankfully, was a bit more cooperative this time around. In this issue, we have a recap of the conference for those of you who were unable to make it or missed a session or two. Dr. Roth gave an excellent keynote address and has summarized his talk on the current state-of-the-science in insomnia. We have included a listing of the SRS 2013 awardees and an interview with Dr. Amita Sehgal, winner of the Outstanding Scientific Achievement award for her work on the molecular biology and genetics of sleep in Drosophila. Dr. Gehrman provides us with a recap of the annual trainee day which was, again, a tremendous success. I hope that all members try to utilize this service of the society by themselves attending or having their students attend. We are also including a recap of a very exciting session on local sleep, given by Drs. Huber, Nir, Vyazovskiy, and Van Dongen. The room was filled and the talks were thought provoking to say the very least. In addition to our summary of SLEEP 2013, Jared Saletin provides a perspective on networking and mentorship in the Trainee corner, we profile two labs - Dr. Carol Everson (Medical College of Wisconsin) and Dr. Dieter Riemann (Freiburg University Medical Center) - and two early career investigators - Dr. Aric Prather (University of California, San Francisco) and Dr. Teresa Arora (University of Birmingham, UK). Our SRS lobbyist write in about goings on around the Capital (short version: all is not gloomy in regards to future budgets) and we have piece by Dr. Claire Caruso describing the work being done on sleep at CDC/NIOSH (National Institute for Occupational Safety and Health). Finally, we include a touching tribute to Dr. Franz Halberg, a pioneer in the circadian field, written by several close colleagues of his, who passed away earlier this year.

Again, if there is something you would like to see in a future issue of the Bulletin, please let me know (jzeitzer@stanford.edu).
Networking, Connecting and Finding New Mentors

As trainees, we have all had people that have influenced our trajectories, our science, and our goals. These mentors have been instrumental in getting us where we are, whether we are undergraduates, graduate students, medical students, residents, post-doctoral fellows, interns, or even faculty. Often, early in our careers, these mentors emerge easily – through the structured formations of our own training, or by the luck of circumstance. Having secondary or tertiary mentors as we progress can provide new opinions, perspectives, opportunities and networks to explore. Nurturing these multiple mentor relationships can yield fruitful results and make you more integrated into, and aware of, the field.

However, while we often find ourselves with default “initial mentors” – graduate advisors, for instance – finding secondary mentorship is a daunting process and often involves the dreaded act of networking. The Sleep Research Society and the faculty that make up our field both sincerely care about our development and are often incredibly willing and wanting to help us achieve our goals. With that generosity as a foundation there are some things we as trainee’s can do to help us secure rich mentor relationships.

1) “Just show up”  
Many people find meeting, interacting, and networking with faculty nerve-wracking, stress-inducing, and downright difficult. Often it is. However, the hardest part to networking is simply getting into the room. Once you’re in the room the nerves quickly dissipate. Remember that the faculty in our field are genuinely interested in you and want to see you succeed. The recent SLEEP 2013 Trainee Series Symposia Career Fair is a great example of this. Faculty lined the halls of the Baltimore Hilton not of need or obligation to be present but simply out of a desire to be helpful to future trainees. Just by entering the room, before any conversations were initiated, a wonderful opportunity to meet new mentors and share ideas was presented. Showing up is always the hardest part.

Another example of where simply showing up can make a difference is in attendance of events like SLEEP 2013. While some may only attend conferences and meetings when they have something to present, I think they are missing a wonderful opportunity. Conferences are more than just chances to present work, however important those chances are. Rather, conferences are networking opportunities, both with faculty and other trainees. Beyond conferences, joining committees such as the Trainee Series Subcommittee (plans each year’s trainee symposia) can be wonderful opportunities to network and a classic example of the “just show up” philosophy. By volunteering you’ve opened a broad network you may never have had before. By attending conferences whether you have data or not, volunteering for committees, and simply being present for opportunities to present themselves, you’ve taken advantage of the second major trick in this article...

2) “Make opportunities, don’t wait for them.”  
It is true that the faculty in the sleep field are incredibly generous with their time and equally willing to devote that time to the advancement of your career. However, they are very busy people, with labs, deadlines and obligations of their own. This means that establishing a new mentorship relationship with them takes initiative from the trainee. Many of us have a tendency to wait for opportunities to present themselves. Often we subscribe to email mailing lists waiting for new jobs, or post doc opportunities, and attend career fairs and ask for openings. While these direct strategies may sometimes succeed, mentor-mentee relationships emerge more commonly not out of direct request, but out of a mutual, organic interest to work together – an interest that can be energized and instigated by the trainee.

By engaging faculty about your research, your goals, your interests you can open the door that they did not previously know exists, let alone was open. Opportunities organically emerge over time, especially when they are sought out. This philosophy of taking agency in making new opportunities extends beyond seeking mentors or job opportunities. It is a good philosophy for all aspects of training. Rather than waiting to be asked to write a review with your advisor, propose one. Rather than only looking for funding opportunities from traditional sources, seek out foundations that may be interested in your research. The sky is the limit when you want to reach for it.

3) “Don’t let perfect be the enemy of good.”  
Let’s face it. We are all perfectionists. It is a trait that helps us do better science. As good as perfection can be, it can sometimes get in the way, particularly when it comes to mentors. No mentor-mentee relationship is perfect. By passing up opportunities because one aspect does not seem perfect, we can rob ourselves of the chance of something quite good and beneficial. Just because the post doc announcement focuses on a project that is not quite right for you, does not mean that PI may not need someone with your skills. Go ahead and email them anyway. You never know the success you may have unless you try.

4) “Pay it forward”  
We are all here because someone once introduced us to another person, and so on. As you progress through your training use your experiences to better your peers’ mentorship relationships and careers. This is critical for late stage graduate students and post-docs. Your network by now is far greater than it was when you entered the field. When new students enter your lab become their secondary mentor. Open their doors.

Science is social discipline as the number of alumni-network dinners and events at SLEEP demonstrate each year. While being a trainee can often feel like a lonely experience focused on your data, finding new mentors, new networks, and new sources of support will only serve to strengthen your success and ultimately, the science you will perform.

Jared Saletin, PhD  
University of California, Berkeley, CA
Insomnia is not simply a report of difficulty sleeping at night, but also associated daytime impairment and/or distress, a frequency criterion of at least three times per week and a chronicity criterion of at least four months, and importantly this difficulty must be presented despite adequate opportunity and circumstance to sleep. Given these diagnostic criteria, “transient insomnia” is a misnomer as it is not insomnia but rather sleep disturbance associated with inadequate opportunity or circumstance for sleep. As such, it is more akin to sleep deprivation or sleep fragmentation rather than insomnia. The prevalence of insomnia using either the ICSD-II or DSM-IV is around 20%. The clearest risk factors are female gender and the presence of a psychiatric or medical disorder. While the prevalence is typically reported to go up with age, this is not the case when full insomnia diagnostic criteria are applied. While the prevalence of disturbed sleep goes up with age, the likelihood of reporting impaired daytime function or distress does not, thereby lowering the prevalence of insomnia in the elderly.

Morbidity
In looking at the next day consequences of insomnia, researchers have traditionally utilized at a variety of parameters associated with sleep deprivation. The majority of these studies produced negative results. This is not unexpected as the effects of sleep loss are primarily sleepiness-mediated and patients with insomnia are not sleepy. They are, in fact, more alert than age- and sex-matched normal sleepers. Insomniacs do, however, often report impairments in daytime function related to disrupted mood, increased fatigue and cognitive decline. Large-scale studies have also documented the effects of insomnia on daytime functioning. For instance, various studies find that patients with chronic insomnia have significantly higher risks for falls and accidents. In addition, adults with insomnia miss twice as many workdays as those without. In fact, insomnia may be the greatest predictor of absenteeism in the workplace. In terms of specific morbidities, patients with insomnia have been shown in multiple studies to be at risk for incident depression. Prospective studies performed in a variety of populations with follow-up periods from 1 to 40 years have shown insomnia to represent about a 3-5 fold risk of incident depression. Another area that has received much attention is hypertension risk. In most, but not all studies, insomnia has been shown to have an increased risk of hypertension. In a population based study, both insomnia reports as well as polysomnographic evidence of short sleep durations were shown to be a risk factor for hypertension. Importantly individuals with both insomnia as well as short sleep duration had a greater risk for hypertension than individuals with either factor alone. A similar interaction between short sleep duration and insomnia has been shown for other morbidities as well. There is little question that there is significant morbidity associated with insomnia in a variety of domains. The issues are what aspects of insomnia mediate them and does treating insomnia reverse them or ideally prevent them.

Pathophysiology
Three different hypotheses have been pursued as the pathological process underlying insomnia. First it was thought that insomnia reflected an impaired homeostat in that insomniacs have a decreased pressure for sleep. It has been shown, however, that in acute and chronic sleep deprivation challenges, insomniacs show...
the same increase in homeostatic drive measure as normal controls in terms of degree of sleepiness and sleep recovery the day after deprivation. Another hypothesis was that insomnia was a circadian rhythm abnormality. While some circadian rhythm disorders present with nocturnal insomnia symptoms and insomniacs tend to show a delayed dim light melatonin onset (DLMO), these findings are not likely to explain the broad insomnia population. The most accepted view is that insomnia is disorder of hyperarousal. Insomniacs do not have an abnormal sleep (homeostat) or circadian system but rather an overactivated wake system. This is evidenced in many different ways. Cognitively, patients often complain they cannot shut their brain down. In terms of physiological measures, there is evidence of increase brain metabolism in arousal areas, increased fast EEG activity, increased autonomic activity, metabolic and hypothalamic-pituitary-adrenal axis activity. The most consistent finding has been elevated alertness as measured by the multiple sleep latency test (MSLT). In normal subjects, decreased sleep at night results in decreased alertness on the MSLT while in insomniacs, decreased sleep at night relates to increased alertness.7

Two important facts about hyperarousal needs to be emphasized. First, hyperarousal is not only present when in bed awake, not even during the night while asleep, but is present 24 hours a day. Stress challenges during the day result in a greater stress response in insomniacs relative to controls. Second, not all insomniacs show signs of hyperarousal. There are clearly individual differences in degree of hyperarousal among insomniacs. One hypothesis is that the degree of hyperarousal relates to the amount of wakefulness an insomniac experiences. If this is the case, the question arises as to whether hyperarousal leads to more wake or does more wake lead to hyperarousal. More generally, is insomnia a cause or consequence of hyperarousal. To address this, we developed a questionnaire to assess the degree to which an individual reacts to daytime stressors with disturbed sleep (FIRST). Research with this scale has been in three general areas: 1) basically normal volunteers who have essentially normal sleep have normal FIRST scores and insomniacs have high FIRST scores; 2) challenging sleep with the use of caffeine as well as the first night effect results in impaired sleep in normal volunteers with high FIRST scores relative to those with low scores; and 3) two prospective studies demonstrated that high FIRST scores are risk factors for incident insomnia. Interestingly in the second of these studies, high FIRST was also found to be a risk for depression, but only if the subject first develops insomnia.9

Therapeutics
There have been two important changes in the approach to insomnia therapeutics. First in terms of pharmacological treatments, insomnia drugs are targeting wake transmitter systems. Traditionally, sleep promoting agents from barbiturates to benzodiazepine receptor agonists have targeted receptors involved in sleep promotion. Specifically, it has been the GABA-A complex generally and benzodiazepine receptors on the GABA-A complex specifically. The rational being that insomnia was an impairment of the sleep system and these drugs would compensate for that. More recently, however, drugs for insomnia are being developed that target receptors involved in the wake-promoting system. Specifically, they are antagonists targeting receptors for serotonin, histamine, and orexin. This parallels the change in our thinking of insomnia as a disorder of the arousal system rather than sleep systems. The potential advantage of these medications has yet to be clearly defined. One potential difference might be the balance between efficacy in the latter part of the night and residual effects. With the benzodiazepine receptor antagonists, duration of action was generally determined by drug concentrations in blood and, therefore, short acting drugs were used to avoid residual effects. However, in the case of orexin and histamine H1 antagonists, higher endogenous levels of these transmitters in the morning allows for lower blood drug levels in the later part of the night, potentially producing longer sleep maintenance without residual effects.

Turning to clinical endpoints in terms of insomnia per se, there is a movement away from simply looking at quantitative measures of nocturnal insomnia symptoms such as time to fall asleep, sleep duration, sleep efficiency and wake time during sleep. Rather the overall effect on insomnia is being evaluated with measures such as the Insomnia Severity Index as well as Clinical Global Impression Scale. Another direction is to look at the effects on the daytime symptoms of insomnia. The most interesting therapeutic innovations have been in the area of treating comorbid insomnias. Both behavioral and pharmacological trials have shown that treating insomnia does not only make the insomnia better but it also improves the comorbid condition. Specifically cognitive behavioral therapy for insomnia (CBT-I) and benzodiazepine receptor antagonists have been shown to improve pain in insomnia comorbid with a variety of pain conditions, as well as improving depression in insomnia comorbid with depression.10 As this research moves forward, it is critical to determine the degree to which the improvement in the comorbid condition is a direct effect of the therapy versus being mediated through the improvement in sleep.

Moving forward more research is needed in all of the areas discussed above, but three new directions are needed: 1) physiological and genetic markers of sleep reactivity; 2) identification and treatment for subjects with high FIRST scores to prevent insomnia; and 3) in areas in which comorbid conditions were attenuated by treating insomnia, like depression, studies are needed to determine whether treating insomnia prevents incident or relapse or depression.

References
2013 Distinguished Scientist Award

Award Description: The Distinguished Scientist Award is the highest award presented by the Sleep Research Society (SRS). Established in 1989, the award recognizes significant, original and sustained scientific contributions of a basic, clinical or theoretical nature to the sleep and circadian research field. This award honors a single individual for research contributions made over an entire career.

Recipient: Eve Van Cauter, PhD, is the Frederick H. Rawson Professor of Medicine at the University of Chicago, where she directs the Sleep, Metabolism and Health Center (SMAHC). She is a member of the Editorial Board of the Journal of Clinical Endocrinology and Metabolism (JCEM) and an Associate Editor of the journal Sleep. She received her master’s degree in physics in 1970, master’s degree in actuarial sciences in 1972 and PhD in biophysics in 1977 from the University Libre de Bruxelles.

Dr. Van Cauter’s initial research focus was the analysis and modeling of biological rhythms, including the development of computer algorithms quantifying circadian and pulsatile variations of hormones and their relationship to sleep stages and other physiological variables. In 1982, she joined the Section of Endocrinology in the Department of Medicine at the University of Chicago. Her research has been continuously funded by the National Institutes of Health (NIH) and other federal research grants, and she has led a large multidisciplinary program project funded by the National Institute on Aging (NIA).

During the past decade, Dr. Van Cauter’s research has focused on the impact of decreases in sleep duration and quality on markers of health and the interaction of sleep loss with the aging process. Her group identified sleep loss and poor sleep quality as novel risk factors for obesity and diabetes. It is widely considered that this work opened up a new field of enquiry of high relevance to the current epidemic of obesity and diabetes and the increased prevalence of age-related chronic diseases. Her groundbreaking article “Impact of sleep debt on metabolic and endocrine function,” published in Lancet in 1999, has been cited more than 700 times. In recent years, her efforts have focused on the impact of obstructive sleep apnea on the risk and severity of type 2 diabetes.

Dr. Van Cauter has received the Robert Wallemgh Prize for medical research, the Hoechst Belgium Prize for research in biological psychiatry, the Soroptimist Prize on the occasion of the 50th anniversary of the Belgian Soroptimist Association, the Pharmacy & Upjohn International Award for Excellence in Published Clinical Research in the Journal of Clinical Endocrinology.

and Metabolism (JCEM), the Pfizer Lectureship in Sleep, the Gerald D. Aurbach Award of the Endocrine Society, and the SRS Outstanding Scientific Achievement Award.

2013 Mary A. Carskadon Outstanding Educator Award

Award Description: Established in 2005, the Mary A. Carskadon Outstanding Educator Award is presented to honor excellence in the field of education related to sleep medicine and sleep research.

Recipient: Meir Kryger, MD, FRCPC, is Professor of Medicine at the Yale University School of Medicine and on the staff of the VA Connecticut Health System. He was previously Professor of Medicine at the University of Manitoba where he was Director of the Sleep Disorders Centre at St. Boniface Hospital Research Centre, the first clinical laboratory to study patients with sleep breathing problems in Canada.

Dr. Kryger graduated from the McGill University Medical School in 1971. He completed an internal medicine internship at Michael Reese Hospital in Chicago and a residency at the Royal Victoria Hospital of McGill University in Montreal. He trained in pulmonary medicine at the University of Colorado, followed by two years of research training. He is board-certified in Internal Medicine, Pulmonary Medicine and Sleep Medicine; a Fellow of the Royal College of Physicians of Canada; and a Diplomate of the American Board of Sleep Medicine (ABSM).

Dr. Kryger was President of the American Academy of Sleep Medicine from 1993-94 and has been President of the Canadian Sleep Society. He is on the Board of Directors of the National Sleep Foundation (NSF) and served as Board Chair from 2007-09. He has received the William C. Dement Academic Achievement Award and the Excellence in Education Award from the AASM, as well as a Distinguished Scientist Award from the Canadian Sleep Society.

Dr. Kryger is the Chief Editor of the most widely used textbook in sleep medicine, The Principles and Practice of Sleep Medicine, which is currently in its fifth edition. Other recent books include the Atlas of Clinical Sleep Medicine and Kryger’s Sleep Medicine Review. He also has written books for the public including A Woman’s Guide to Sleep Disorders and most recently the eBook The iGuide to Sleep.

Dr. Kryger’s research was the first to show the feasibility of using noninvasive techniques to ventilate people with post-polio syndrome in their homes. His laboratory elucidated the interaction between heart failure and sleep respiration and published the...
first systematic study of oxygen in this condition. He reported the first use of computers in analyzing sleep breathing patterns and validated techniques of monitoring in which diagnostic and therapeutic data on continuous positive airway pressure (CPAP) are obtained during the same night. His lab also was among the first to report on increased healthcare utilization and mortality in sleep apnea.

2013 Mary A. Carskadon Outstanding Educator Award

Award Description: Established in 2005, the Mary A. Carskadon Outstanding Educator Award is presented to honor excellence in the field of education related to sleep medicine and sleep research.

Recipient: Robert W. McCarley, MD, was President of the Sleep Research Society (SRS) from 1991-92, and he is Professor and Chair of the Harvard Medical School Department of Psychiatry and Director of the Laboratory of Neuroscience at the VA Boston Healthcare System, where he is also Associate Director of the Mental Health Service.

Dr. McCarley graduated summa cum laude from Harvard College, where he was a National Scholar and was elected to the Alpha Chapter of Phi Beta Kappa. He was a Fulbright Scholar in Psychology at Johannes Gutenberg-Universität in Germany, and he completed his medical degree at Harvard Medical School. He interned in medicine at Brigham and Women’s Hospital in Boston and began his research career while still a resident in psychiatry at the Massachusetts Mental Health Center. He is board-certified in psychiatry and established the Laboratory of Neuroscience at the VA in 1985.

Dr. McCarley has received the SRS Distinguished Scientist Award, AASM William C. Dement Academic Achievement Award, Middleton Award from the Veterans Affairs Medical Research Service, and an award from the American Psychiatric Association. He also has received consistent peer reviewed funding from the National Institutes of Health (NIH) and VA Medical Research Service, from which he holds two Merit awards.

One of his greatest joys has been mentoring young sleep scientists. He is especially pleased to give back to his trainees many of the lessons he learned from mentors and collaborators such as Allan Hobson and Mircea Steriade.

Dr. McCarley’s work and that of his collaborators and trainees encompasses brain control of both rapid eye movement (REM) and non-REM sleep. An important line of current work has been the investigation of the homeostatic sleep factor, adenosine, including: its modulation of in vitro and in vivo cellular activity, its relationship to behavioral sleepiness, the intracellular signaling mechanisms of its A1 receptor, and its role in chronic sleep restriction. Other investigations have documented nitric oxide as a homeostatic factor, adenosine triphosphate (ATP) and brain energy levels as modulated by sleep and wake, and, most recently, optogenetic studies of the basal forebrain.

The entire field of sleep research, including his lab’s work, was recently described in “Control of sleep and wakefulness,” a comprehensive article in *Physiological Reviews* with 1,479 references (July 2012. 92: 1087-1187).

2013 Outstanding Scientific Achievement Award

Award Description: Established in 2006, the Outstanding Scientific Achievement Award is presented to individuals based on novel and seminal discoveries of a basic, clinical or theoretical nature that have made a significant impact on the sleep field.

Recipient: Amita Sehgal, PhD, is the John Herr Musser Professor of Neuroscience and an Investigator of the Howard Hughes Medical Institute in the Perelman School of Medicine at the University of Pennsylvania. She received her PhD from the Graduate School of Medical Sciences at Cornell University, where she worked with Dr. Moses Chao on the cloning and molecular characterization of the neurotrophin receptor, p75.

Dr. Sehgal is a member of the advisory council at the National Institute of Neurological Disorders and Stroke (NINDS), and she has performed editorial services for several journals, including the *Journal of Neuroscience* and the *Journal of Clinical Investigation* (JCI). She has received the Michael Brown Junior Faculty Research award and the Stanley Cohen Senior Faculty Research award at the University of Pennsylvania, and she was selected as an Assistant Investigator of the Howard Hughes Medical Institute and remains an HHMI Investigator. She also was elected to the Institute of Medicine (IOM) in 2009 and the American Academy of Arts and Sciences (AAAS) in 2011.

Dr. Sehgal conducted her postdoctoral work with Dr. Michael Young at Rockefeller University, where she conducted a genetic screen in Drosophila for mutants that have aberrant circadian (~24 hour) rhythms. She and her collaborators discovered the circadian clock mutant, *timeless* (*tim*), which was only the second Drosophila clock mutant to be identified, the first being *period* (*per*), discovered more than 20 years earlier. Dr. Sehgal and her collaborators cloned *tim*, demonstrated its interaction with *per*, and showed that the core clock comprises two co-regulated genes. Among other recent advances, her laboratory determined how the fly clock synchronizes to light (a first for animal clocks), identified dephosphorylating enzymes as components of the clock, and discovered other molecules required to transmit circadian time-keeping signals. Dr. Sehgal and her colleagues also described clocks in several tissues outside the brain, thereby establishing connections between clocks and various aspects of body physiology.

Continued on the following page →
Circadian clock mechanisms have turned out to be conserved from Drosophila to humans. Driven by this finding and the recognition that the study of sleep homeostasis could benefit from the use of a simple, genetic model, Dr. Sehgal and her collaborators developed a Drosophila model to study the homeostatic regulation of sleep. This model has been adopted worldwide and is rapidly leading to advances in sleep research.

**Young Investigator Award Recipients**

**Recipient:** Josianne Broussard, PhD  
Cedars-Sinai Medical Center, Los Angeles  
*Title of Paper:* Impaired Insulin Signaling in Human Adipocytes after Experimental Sleep Restriction

**Recipient:** John Lesku, PhD  
University of Western Australia, Perth, Australia  
*Title of Paper:* Adaptive Sleep Loss in Polygynous Pectoral Sandpipers

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**EDITOR’S INTERVIEW WITH DR. AMITA SEHGAL, WINNER OF THE SRS AWARD FOR OUTSTANDING SCIENTIFIC ACHIEVEMENT AT SLEEP 2013**

**What does winning this award mean to you?**

It means a lot, more so than many other awards, because it is the ultimate sign of acceptance of our work by the sleep field. When we first developed a fly model for sleep, some of the senior sleep researchers appeared to be skeptical. However, I believe most have now come to appreciate this model. This award attests to that.

**What achievements in your career stand out to you?**

As a graduate student, I was part of the team that cloned the first neuronal growth factor receptor, p75. That was the first big scientific achievement. Following that, it would be the identification of the timeless circadian clock gene (initiated when I was a postdoctoral fellow), the discovery that the timeless protein gets degraded by light and thereby mediates photic entrainment of the Drosophila circadian clock, the development of the Drosophila model for sleep and our recent identification of sleep mutants.

**Who were some of the researchers you admired in your early career?**

As a female scientist and geneticist, I was inspired by the work of Barbara McClintock. I also greatly admired Seymour Benzer for starting the field of behavioral genetics, and Ron Konopka for the ingenuity he showed in searching for circadian mutants.

**Who in particular influenced your career?**

My graduate advisor, Moses Chao, generated in me a love of science. I believe my fondness for ambitious, high-risk projects can also be traced to his influence. Another major influence in my life was my father, who keenly followed my career and drove me to do my best.

**What scientific questions have interested you more recently and what projects will you focusing on in the future?**

I have become interested in the effects of sleep or sleep loss on peripheral function. I’d like to incorporate studies of this in the future, along with our ongoing work on the genetics of sleep and circadian rhythms.

**What do you think should be some of the research priorities for sleep and circadian researchers over the next 5-10 years?**

I think we have a lot to learn about the basic cellular and molecular mechanisms underlying sleep, so there should be considerable more emphasis on that. On the other hand, the circadian field could benefit from some of the more translational/clinical approaches that dominate the sleep field.

**What guidance can you give to more junior sleep and circadian scientists? What are the keys to a successful research career?**

I think junior people should think big and stay focused on the science (there will be time later for service on committees etc.). As a test of whether they are asking important questions, they should periodically step away from their work and determine how significant their work sounds to a non-scientist. Sometimes even running it by a scientist who is not in the field can serve this purpose.

**What is the hardest part of your job?**

Ensuring that all those I train end up with careers of their choice. I tend to view that as my responsibility, and it can sometimes get quite stressful.

**What/when was the ‘aha moment’ when you knew you were successfully contributing to society?**

Am I successfully contributing to society? Seriously, I think it was when I was first contacted by a high school student who had read my papers and was interested in working in my area. The fact that we were reaching such young people, and inspiring them to do research, told me that I was making a difference.

**How do you keep striving for that in the midst of competing distractions?**

No matter how busy I might be, I make time for young students interested in science, even high school students with questions about their science projects.
This symposium was co-chaired by Dr. Reto Huber and Dr. Yuval Nir and included additional presentations by Dr. Vladyslav Vyazovskiy and Dr. Hans Van Dongen. The objectives were to (1) discuss local aspects of sleep on multiple levels from multi-unit recordings to high-density EEG, (2) recognize how basic knowledge of sleep regulatory mechanisms can be translated to clinical populations, and (3) explore the effects of sleep deprivation on neuronal firing patterns and cognition.

In the first presentation, Dr. Vyazovskiy talked about “Mechanisms and Function of Local Sleep: Implications for Sleep Homeostasis”. Local sleep was discussed in the context of the function of sleep and it was stressed that it is essential to understand how processes at different levels (individual neurons, oscillations in large populations, whole brain) contribute and interact. For example, the global homeostatic process of NREM sleep regulation is manifested as slow wave activity (SWA, oscillations slower than 4 Hz) at the level of the global EEG and also as slow oscillations in the membrane potentials of individual neurons, alternating between active (up/ON) and inactive (down/OFF) states. It was proposed that the purpose of down states could be to provide restoration with respect to energy homeostasis, cellular maintenance/repair or synthetic processes. The idea that sleep plays such a restorative role is supported by the local regulation of SWA. In turn, up states could provide an opportunity for selective interactions between functionally interconnected neurons, facilitating information transfer in the service of plasticity and memory. The ultimate purpose of the recovery processes during NREM sleep is to enable optimal functioning of the brain during subsequent waking behaviors.

The central tenet of the hypothesis presented in this talk was that OFF periods during sleep provide neurons with an opportunity to rest and repair minor cellular damage before it becomes irreversible. Accordingly, prolonged wakefulness or sleep deprivation activates the unfolded-protein response (UPR), as indicated by increased expression of mRNA for heat shock proteins and endoplasmic reticulum chaperones. Furthermore, high synaptic activity during waking likely results in a growing need for biosynthesis of new vesicle components (both proteins and lipids) to replace damaged elements. Thus, homeostasis can only be restored by a temporary reduction in synaptic activity, allowing the cell to clear its backlog of biosynthetic work, which is made easier without intense respiration and the associated oxidative stress. Two questions were further addressed. First, why do maintenance and plasticity-related functions at the level of single-cells or neuronal networks require behavioural sleep and its associated synchronous high-amplitude slow-wave activity? It was argued that although most cells in the body can presumably undergo rest relatively independently, neurons must rest together owing to their extensive interconnectivity. Restorative maintenance can only be achieved by a coordinated reduction in the spiking of all neurons in a functionally interconnected network. Furthermore, efficient and safe neuronal restoration requires sensory disconnection from the environment and behavioural immobility. Thus, to allow individual neurons to obtain sufficient rest, the organism must be globally and behaviourally asleep. Slow waves arise from such synchronized alternation between on and off periods across large cortical neuronal populations and reflect the conditions necessary for individual neurons to achieving rest. Second, what could be the role of REM sleep according to this framework? The regulation and adaptive role of REM sleep remains mysterious. It was proposed that REM sleep provides a sensing mechanism to monitor the progress of recovery provided by NREM sleep and assess the readiness of brain networks for waking activities while remaining offline. Instead of waking up at regular intervals and initiating typical behaviors, a more economical and safe solution would be to emulate wake-like conditions offline during REM sleep. This way, the functional state of brain circuits can be assessed with respect to their readiness for optimal functioning during subsequent wakefulness. In summary, a novel framework was presented, according to which the alternation of NREM and REM sleep episodes occurs throughout the night, until all cortical networks have obtained the needed “recovery” from preceding wakefulness (function of NREM sleep), and proven ready for optimal functioning during subsequent wakefulness (function of REM sleep).

In the second presentation, Dr. Nir talked about “Local Brain Oscillations of Sleep and Sleepiness: Human Intracranial Recordings”. Working with a unique group of neurosurgical patients, sleep studies were performed in which full-night continuous polysomnography (scalp EEG, EOG, EMG, video) was obtained along with recordings from intracranial electrodes – providing depth EEG and spiking activity from hundreds of neurons.

The first part of the talk examined brain oscillations during NREM sleep. First, analysis of the activity in each brain region confirmed that human sleep slow waves reflect alternations between active and inactive periods - as observed in animal studies. Next, by recording in multiple (8-12) distant brain regions in each participant, it was possible to examine how sleep waves in different brain regions relate to each other. The main result was that most sleep slow waves (and the underlying active and inactive neuronal states) occur locally. Local slow waves often occurred with only a faint trace in the scalp EEG. Thus, especially in late sleep, some regions can be active while others are silent. It was also found that slow waves can propagate, usually from medial prefrontal cortex to the medial temporal lobe and hippocampus.

Sleep spindles, the other hallmark of NREM sleep EEG, were also detected and examined. As was the case for slow waves, sleep spindles were predominantly local. In addition, spindle frequency was topographically organized with a sharp transition...
between fast (13-15 Hz) centroparietal spindles and slow (9-12 Hz) frontal spindles occurring later in time. In addition, deeper NREM sleep was associated with a reduction in spindle occurrence and spindle frequency.

Importantly, it was confirmed that sleep measures in the epilepsy patients were in agreement with typical findings in healthy individuals, allowing the generalizability of the findings to the general population. Moreover, the findings of local sleep oscillations were replicated in rodents without epilepsy. Overall, it was shown that sleep oscillations have greater local heterogeneity than initially assumed. Intracerebral communication during sleep may be constrained as slow and spindle oscillations often occur out of phase in different brain regions.

In the second part of the talk, it was investigated whether “local sleep” may occur during human sleep deprivation and lead to negative behavioral consequences. The same group of presurgical epilepsy patients participated in a modified version of the well-validated Psychomotor Vigilance Task (PVT) while images of faces and places were presented infrequently. Some sessions were conducted before/after full-night sleep deprivation conducted for clinical purposes and before/after normal sleep (to control for circadian and learning effects). Lapses, indicated by slow reaction times, were a robust and selective behavioral index of sleepiness. Accordingly, the extent of lapses could be predicted by time spent awake before each session and increased more than two-fold upon sleep deprivation. Next, visually-evoked neuronal activity during the PVT was examined and compared during lapses versus successful trials. During lapses, responses of individual medial temporal lobe neurons to the very same picture showed a significant suppression. Such suppression was local and was accompanied by increases in low-frequency oscillations, reminiscent of slow activity. The findings suggest that in humans, as recently described in rats, “local sleep” during wakefulness may underlie the cognitive effects of sleepiness.

In the third presentation, Dr. Huber talked about “Mapping Slow Wave Activity in Healthy Children and Children with Psychiatric Disorders”. This talk focused on the major characteristic of NREM sleep - slow waves. In the last years we have learned much about the regulation of slow waves on the cellular level. We know that when sleep pressure and slow wave activity (SWA, EEG power between 1-4.5 Hz) is high, network connectivity is increased, the synchronization of the cellular slow oscillations is increased, and this is reflected on the surface EEG by an increased slope of slow waves. On the other hand, when sleep pressure and SWA is decreasing, network connectivity is decreased and the synchronization of cellular slow oscillations decreases, which in turn is reflected in a decrease in the slope of slow waves. In summary, there seems to be a close link between sleep SWA and cortical connectivity. The most extreme changes in connectivity take place during the first two decades of life. This is nicely illustrated by the time course of synapse density displayed next to the change in amplitude of sleep slow-waves. Both seem to peak shortly before puberty and to decrease during adolescence.

As we know that cortical maturation is not taking place globally, starting in posterior brain regions over visual cortices and gradually progressing to anterior regions during childhood and adolescence, it was of interest to examine whether a similar pattern would emerge when mapping SWA. Using high-density EEG to map SWA during the first two decades of life, it was found that the maximum of SWA in the youngest children was over the occipital cortex but then with increasing age moved over the center to the frontal predominance found in adults. This time course of the topographical distribution of SWA not only paralleled the time course of gray matter maturation as assessed by magnetic resonance imaging but also fits well with anatomical and behavioral measures of maturation.

As there is growing evidence for a maturational delay in ADHD children, one must wonder whether the mapping of SWA would yield a similar result in children with ADHD. Indeed, the topography of SWA in the EEG showed a delayed pattern in ADHD compared to control children. Thus, sleep SWA seems to be a promising electrophysiological marker of cortical maturation. Of course, one key remaining question is whether sleep slow waves not only reflect cortical plasticity but also contribute to such plastic changes. In other words, does SWA play an active role in plastic changes for example during development?

An example from a clinical population may point in the same direction. Children with continuous spike-wave epilepsy (CSWS) during slow wave sleep can show quite dramatic neurodevelopmental deficits. This is a very extreme form of manipulation of slow waves, since a massive spike-wave activation invades slow wave sleep every night. In this study, the changes in synchronization were examined across the night. A good marker is the slope of slow waves since it is established that the slope of slow waves decreases across sleep in healthy control subjects. By contrast in the children with CSWS, the slow wave slope did not show such a decrease, as if the recovery function of sleep is impaired. Certainly more work is needed to prove such a relationship more conclusively. Nevertheless, this study joins growing evidence that sleep can be a powerful tool to understand brain function and plasticity in health and disease.

In the final presentation, Dr. Van Dongen talked about “Outstanding Issues in Human Sleep Loss and Cognitive Functioning: The Role of Local, Use-dependent Sleep”. In view of growing evidence in support of local sleep theory, Van Dongen collaborated with Dr. James Krueger – who first proposed the theory – to explore the role of local, use-dependent sleep in sleep loss-induced cognitive impairment. Direct evidence of a connection between local sleep and cognitive impairment came from rat experiments conducted by Rector and colleagues, who examined evoked potentials (EPs) in the barrel cortex in response to whisker twitches. When animals were awake, sometimes a local, sleep-like EP occurred in response to a whisker twitch, and this occurred more often when the whisker was twitched more frequently (use-dependence) or had previously shown a wake-like EP over a longer period of time (homeostatic regulation). Further, after repeated twitching of either whisker not only was a sleep-like EP observed, but the rat was then also more likely to make a performance error. This suggested a causal relationship between the state of a neuronal assembly involved in information processing for a cognitive task and performance outcomes on that task.

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Van Dongen and Krueger posited that a causal relationship between local neuronal assembly sleep state and cognitive performance impairment generalizes to other parts of the brain. Building on the homeostatic (i.e., prior wakefulness-dependent) and use-dependent (i.e., task-dependent) properties of local sleep, they set out to develop a unified theoretical framework to address a number of outstanding questions regarding human cognition functioning under conditions of sleep loss. The inset figure illustrates the proposed core mechanism producing local sleep and resulting in cognitive impairment, which is posited to take place at the level of cortical columns and other neuronal assemblies. Synaptic transmission for the processing of task-related information (e.g., sensory input) and background sleep/wake state-dependent neuronal activity result in (co)release of adenosine triphosphate (ATP) into the extracellular space. ATP is rapidly hydrolized to adenosine, which binds to the adenosine A1 receptor. This puts the neuronal assembly in the sleep-like state, giving it a brief opportunity to restore energy balance and otherwise recuperate from prior use. While in the sleep-like state, the neuronal assembly no longer processes information meaningfully, resulting in degradation of cognitive processing and causing performance instability.

The mechanism for cognitive impairment illustrated in the figure is fundamentally local and use-dependent and thus task-dependent. This may help to explain why performance on “simple” tasks such as the PVT is particularly sensitive to sleep loss. Since such tasks use the same cognitive pathway – and therefore to some extent the same neuronal pathway – intensively, they expose the performance instability caused by local sleep very effectively. Use-dependence may also explain why distinct components of cognition appear to be differentially affected by sleep deprivation and why inter-individual differences in vulnerability to sleep loss, despite being highly trait-like, appear to be task-specific. The explanation may lie in the processing capacity, or the redundancy of neuronal assemblies available for information processing, of different neuronal pathways. Processing capacity may vary across different components of cognition and across different individuals, and the greater the processing capacity, the less overall degradation of information processing is caused by a single neuronal assembly entering the local sleep state.

Some extracellular ATP binds to the purine type 2 receptor, P2X7, instead of being hydrolyzed. P2X7 activation leads to the release of sleep regulatory substances (SRSs) such as cytokines and neurotrophins. This has important consequences for cognition on both short (minutes to hours) and long (days to weeks) time scales. Over the short term, SRSs activate GABA-containing neurons, which in turn inhibit glutamate-containing neurons relaying cortical activation from the reticular activating system. This increases the probability of the sleep-like state with continued task-dependent use of the neuronal assembly, giving rise to the time-on-task effect. Taking a break or switching to another task that does not critically rely on the same cognitive pathway resets the time-on-task effect. A testable hypothesis following from the theory is that when performance tasks are not sufficiently cognitively distinct from each other to reset the time-on-task effect, they are critically dependent on the same neuronal pathway, and should therefore also display similar inter-individual differences in vulnerability to sleep loss.

Over the long term, SRSs such as tumor necrosis factor (TNF) and interleukin 1 (IL-1) modulate receptor trafficking and synaptic plasticity-associated processes such as synaptic scaling, thereby altering neuronal assembly activation. TNF and IL-1 also activate nuclear factor κB (NF-κB), which in turn upregulates several receptor types including the adenosine A1 receptors. Greater adenosine A1 receptor density increases the likelihood of the neuronal assembly entering the sleep-like state (synergistically with the time-on-task effect) and is the putative reason for the build-up of cognitive deficits across days of chronic sleep restriction. It is hypothesized that adenosine receptor upregulation due to chronic sleep restriction is an allostatic response aiming to preserve sleep/wake homeostasis.

Local sleep can interfere unpredictably with cognitive functioning and may jeopardize survival (e.g., as seen in sleep-deprived humans performing safety-sensitive tasks such as automobile driving). Perhaps for that reason, subcortical circuits involved in global sleep/wake regulation appear to monitor the homeostatic and use-dependent collective states of neuronal assemblies. These subcortical circuits, which include the suprachiasmatic nuclei (SCN) keeping circadian time, orchestrate local sleep states to occur preferably in whole-brain, consolidated, niche-appropriate sleep bouts. Globally orchestrated sleep bouts prevent local sleep-related deficits that could otherwise occur while interacting with the environment, thereby increasing the probability of survival and thus offering an evolutionary advantage.

Dr. Reto Huber  
University Children’s Hospital, Zurich, Switzerland

Dr. Yuval Nir  
University of Wisconsin, Madison, WI

Dr. Vladyslav Vyazovskiy  
University of Surrey, Guildford, Surrey, United Kingdom

Dr. Hans Van Dongen  
Washington State University, Spokane, WA
This year, the Sleep Research Society’s Trainee Education Advisory Committee (TEAC) held the 18th Annual Trainee Symposia Series as a part of SLEEP 2013 meeting in Baltimore. The Trainee Symposia Series remains one of the SRS’s most unique opportunities for trainees to interface with not only their peers, but also the leaders of our field. This year, 236 trainees ranging from undergraduate to graduate students, post-docs, interns, residents, and beyond, attended the sessions.

The Trainee Symposia Series spanned two days, beginning Saturday afternoon with the return of the very popular Grant Writing workshop. We were fortunate to have six NIH program officers in attendance at the grant workshop. The 50 trainees who registered for this workshop were met with the rare opportunity to work one-on-one or in small groups with members of the NIH. For four hours on Saturday afternoon, these trainees were presented with an overview of the writing and competing for NIH grants before breaking into small focus groups to review each trainee’s Specific Aims with the NIH officers in attendance. As in past years, Dr. David Dinges finished the day with his popular capstone lecture on grant writing success. We would like to extend a great gratitude to all the faculty and NIH officers that made this grant writing workshop an incredibly successful and enriching experience for the trainees.

Following the grant writing workshop, the symposia series began in full with the opening keynote and trainee datablitz. We were distinctly fortunate to have with us Dr. Eric Green, Director of the National Human Genome Research Institute. Dr. Green attested at the beginning of his lecture that he “never turns down an invitation from trainees”. In his lecture, Dr. Green’s passion for speaking to trainees was clear, giving a captivating summary of genomics research today, including personal anecdotes of the human genome project. The emphasis on emerging big data solutions and genomic approaches would be a repeating refrain throughout the remainder of the weekend, setting a perfect stage for the workshops that followed Sunday morning. Following Dr. Green’s lecture, the second annual datablitz was held and included presentations by the trainees with four of the top-ranked trainee abstracts in a shortened datablitz format. We would like to congratulate Karinna Fyfe, Eunyeon Joo, Crista Van Dort, and Leanna Garb for being invited to share their exciting work in the datablitz. Following the datablitz, TEAC held its traditional career development sessions, these omics-focused workshops positioned attendees to absorb cutting-edge science and methods to answer the most critical of today’s sleep-research questions. In a record 148 turned-in evaluations, the Trainees expressed their overwhelming approval of this year’s program, hailing both the quality of the speakers, as well as the numerous networking opportunities provided.

As always, an event such as the Trainee Symposia Series would not be possible without the efforts and support of numerous people. TEAC would like to thank the Board of Directors, as well as the President of the SRS for continuing to generously support trainee events year after year. To organize and execute an event such as this, months of preparation is required from many different individuals. The Trainee Symposia Series is truly an event for the trainees, by the trainees. Each year a subcommittee is formed, consisting of trainees from various disciplines, regions and levels of training. These trainees formulate the topics each year, and pinpoint potential speakers, invite speakers, organize the career fair, while also assisting at the event itself. They are integral to making this event a success each year. We would like to thank the members of this year’s subcommittee: Teresa Arora, Josiane Brousseau, Annette Fedson, Devon Grant, Iuliana Hastescu, Amanda Hayes, Jaime M. Hughes, Monica Kelly, Laura Kurzdzel, Elizabeth McDevitt, Andrew JK Philips, Kristina Puzino, Sarah Horsey Simpson, Ari Shecter, Rodolfo Soca, Laura Straus, Ivan Vargas, Andrew Westwood, and Allison Wilkerson. Participation in the subcommittee is open to all trainee members of the SRS and is a great opportunity to become involved in the society, meet your peers and faculty, help shape future trainee events. Students who are interested in joining the subcommittee should look out for announcements in future SRS email newsletters.

Over the course of the past year, the faculty on TEAC have dedicated themselves to putting forth the best possible symposia series possible. These faculty members represent a true dedication to trainees and training by the SRS. We would like to acknowledge these members of TEAC that made this year’s symposia possible: Philip Gehrman PhD (TEAC Chair), Lisa Meltzer PhD (TEAC Vice-Chair), Sonia Ancoli-Israel PhD, Kelly Baron PhD, Xiang Gao PhD, Monique LeBourgeois PhD, Rachel Manber PhD, Allan Pack MBChB PhD (SRS Board Liaison), Eva Szentirmai MD, Giancarlo Vanini MD, Ronald Szymbuski PhD (SRS President), Megan Ruiter PhD (Trainee Member-at-Large), Jared Saletin (Trainee Member-at-Large Elect), as well as Nicholas Cekosh, John Noel, Annie Walker-Bright, and Brittany Manning from the Sleep Research Society.

Finally we would like to thank all the speakers who attended this year’s event, often coming early to the meeting just to speak to trainees. Without these generous speakers, this event quite literally would not be possible. Thank you for your dedication to the SRS and to its trainees. This year’s speakers included: Eric Green MD PhD, David Dinges PhD, Allan Pack MBChB PhD, Namni Goel Continued on the following page →
Philip Gehrman, PhD, CBSM for the Trainee Education Advisory Committee
Janet Mullington, PhD
SRS President

Janet Mullington, PhD, is an Associate Professor of Neurology at Harvard Medical School and Beth Israel Deaconess Medical Center (BIDMC). She received her PhD from the University of Ottawa in Canada and did postdoctoral work in Germany at the Max Planck institute of Psychiatry and at the University of Pennsylvania in Philadelphia. Her primary research interest is in the area of inflammatory systems and sleep loss. Dr. Mullington is Program Director of the Harvard Clinical and Translational Science Center at BIDMC. In addition, she is the Research Fellow for the Oliver Wendell Holmes Society of Harvard Medical School, a role dedicated to serving the research training mission of the school, and she has served on the medical school’s Diversity and Multicultural Committee since 2008.

Over the last 16 years, Dr. Mullington has worked for the Sleep Research Society in many ways. She began service as a grant reviewer in 1997, volunteered as a member of the Educational Programs Committee from 2002-2005, and stemming from that experience, she was instrumental in putting together Career Development courses held at the APSS. Dr. Mullington was a contributing author for the second edition of the Basics of Sleep Guide (2009), and she is an Associate Editor for Sleep. Dr. Mullington is a member of the joint SRS-AASM Task Force, the SRS Government Relations Committee, has participated in the NIH Liaison annual meetings with NIH Directors and staff and also served as an SRS liaison to the Academic Sleep Centers Task Force of the AASM. Dr. Mullington was elected to the SRS Board of Directors in 2008 and served as board liaison for the Trainee Education Advisory Committee (TEAC) from 2008-2011, and as SRS Secretary-Treasurer 2011-2012.

Allan I. Pack, MBChB, PhD
SRS President-Elect

Dr. Allan I. Pack is the John L. Miclot Professor of Medicine, Director of the Center for Sleep and Circadian Neurobiology and Chief of the Division of Sleep Medicine at the University of Pennsylvania (Penn). He has been on the faculty at the University of Pennsylvania since 1976. He directed an NIH-funded Specialized Center of Research on sleep apnea from 1988 to 2008. He has directed two Program Project Grants—The Mechanisms of Alterations in Sleep with Age (AG17628) and currently Endophenotypes of Sleep Apnea and Role of Obesity (HL094307). He is the Founding Director of the Center for Sleep and Circadian Neurobiology and the Division of Sleep Medicine. These are the first multidisciplinary independent sleep research and clinical sleep medicine programs to be established at any medical school in the United States. Dr. Pack’s current main area of focus is on functional genomic approaches to sleep and its disorders. He uses both *Drosophila* and mouse models in his work and translates findings to humans. He is very committed to research training and currently directs two T32 grants from the NHLBI (one for postdoctoral fellows and one for graduate students) and is Principal Investigator of a K12 award. Many individuals trained in the Penn program have gone on to develop their own independent research careers at Penn, in other institutions in the United States, and in Canada. Research training is a major commitment of Dr. Pack. He has received a number of awards for his activities including the Nathaniel Kleitman Award and the William C. Dement Academic Achievement Award from the American Academy of Sleep Medicine as well and the Lifetime Achievement Award from the National Sleep Foundation.

David Gozal, MD, FAASM
Director

Dr. Gozal is currently the Herbert T. Abelson Professor and Chairman of the Department of Pediatrics at the University of Chicago and Physician in Chief of the Comer Children’s Hospital in Chicago. He received his MD from the Hebrew University of Jerusalem, completed his pediatric residency at the Haifa Medical Center in Israel, and then spent 2 years in Cameroon, West Africa, developing rural healthcare networks, for which he received the title of “Knight of the Order of Merit”. He then completed his pediatric pulmonology and sleep medicine training at Children’s Hospital Los Angeles in 1993, and joined the faculty at the University of Southern California and UCLA. In 1994, he moved to Tulane University, where he rose through the ranks and was appointed tenured Professor and Constance Kaufman Endowed Chair in Pediatric Pulmonology Research. From 1999 till 2009, Dr. Gozal was at the University of Louisville as the Children’s Hospital Foundation Chair for Pediatric Research, Distinguished University Scholar, Vice-Chair for Research, Director of the Kosair Children’s Research Institute, and Chief of the Division of Pediatric Sleep Medicine and the Sleep Medicine Fellowship Program, both of which were recognized as programs of distinction by the American Academy of Sleep Medicine.

Dr. Gozal’s research interests emphasize bench to bedside approaches to pediatric sleep disorders, with projects encompassing a wide range of interests, such as gene and cellular regulation in hypoxia and sleep disruption, murine models of sleep disorders, and genomic and proteomic approaches to clinical and epidemiological aspects of sleep in children, including collaborative work in several countries around the world.

In addition to his membership and previous committee service in the SRS, he is deputy editor for the journals *Sleep* and *Frontiers in Neurology*, associate editor of the *American Journal of Respiratory and Critical Care Medicine*, and a regular member of the NNRS study section at NIH, as well as serving on the editorial boards of several journals in the field. His research work

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is supported by several grants from the NIH, he has published over 450 peer-reviewed articles, >100 book chapters and reviews, 3 books, and > 700 scientific abstracts, and has extensively lectured all over the world.

**Jared Saletin**  
*Trainee Member-at-Large*

I am thrilled to serve as the Trainee Member-at-Large for the 2013-2014 year. I have been a trainee member of the Sleep Research Society (SRS) for the past seven years, beginning with the SLEEP2006 conference in Salt Lake City, while I was still an undergraduate. Since that time, the SRS has been continued to greatly influence my training. I have served this past year as Trainee Member-at-Large Elect and look forward to giving back to the SRS by assisting in continuing the stellar opportunities the society offers trainees.

I have had outstanding mentors during my training in sleep research. I entered sleep while pursuing my undergraduate degree in Psychological and Brain Sciences at Johns Hopkins University. After my first year of college, I became a 2006 William C. Dement Fellow under supervision of Dr. Mary A. Carskadon at Brown University—introducing me to the field of sleep, from which I’ve never left. I was struck not only by the fascinating scientific field of sleep but also by the rich environment the society created for its trainees. During my remaining undergraduate years I was a research assistant under the mentorship by Dr. Michael T. Smith at Johns Hopkins Hospital, performing my undergraduate thesis on both the intersection of sleep and pain, as well as examining larger demographic differences in sleep architecture. I then continued in 2008 to enter graduate school at the University of California, Berkeley, where I have had the pleasure of being mentored by Dr. Matthew P. Walker. At Berkeley my work has focused on the cognitive neuroscience of sleep, particularly the role of sleep in processes of learning, memory and brain plasticity. My research has combined cognitive behavioral metrics with structural and functional neuroimaging and quantitative EEG approaches to examine associations between sleep physiology, brain structural anatomy and behavioral outcomes—mainly sleep-dependent memory consolidation and memory encoding.

My work has been funded by a graduate fellowship from the National Science Foundation, has been presented at SLEEP and cognitive neuroscience meetings, and has been published in journals such as *Cerebral Cortex*, and *NeuroImage*. My future research directions aim at examining sleep-dependent brain and cognitive development early in life, particularly during the transition period of adolescence. My long-term professional goal is to remain an active sleep researcher and transition ultimately to an academic position as director of an independent sleep and developmental cognitive neuroscience laboratory.
Sleep researchers worked this summer to advocate for sleep and circadian research and awareness among members of Congress. The Senate Appropriations Committee has acknowledged these efforts by considering and passing its fiscal year (FY) 2014 Labor-Health and Human Services-Education (L-HHS) Appropriations bill and including funding increases and specific committee recommendations that support sleep research activities at the National Center on Sleep Disorders Research (NCSDR) within the National Heart, Lung, and Blood Institute (NHLBI). Please see the following summary for details:

- $3.08 billion for the National Heart, Lung, and Blood Institute (which houses the National Center on Sleep Disorders Research), an increase of $5.1 million over FY 2013 and $176.62 million more than the current operating budget under sequestration.
- **Sleep Disorders** — The Committee continues to support the implementation of the National Sleep Disorders Research Plan, including the emphasis on cross-Institute collaboration.
- **Sleep Disorders** — The Committee urges NIH to initiate new training programs in sleep and circadian sciences in all relevant Institutes and Centers, consistent with the NIH Sleep Disorders Research Plan.
- $1.26 billion for the CDC’s National Center for Chronic Disease Prevention and Health Promotion where sleep surveillance and awareness activities are supported. This is an increase of $94.4 million over the FY 2013 funding level and $287.83 million more than the current operating budget under sequestration.

In addition to the appropriations activity, SRS had a full agenda of summer advocacy events. SLEEP 2013 took place in Baltimore, Maryland. Several SRS leaders added a day of advocacy in Washington DC and visited members of the House and Senate Appropriations committees. This increased interest in sleep prompted Congressman Hank Johnson (D-GA-4) to co-sponsor a briefing to inform his colleagues about the advancements and opportunities in sleep health alongside SRS, NHLBI, the American Thoracic Society, the American Sleep Apnea Association, and the American Academy of Sleep Medicine (AASM).

The briefing showcased the newly designed NIH sleep info-graphic and was well attended by congressional staff members who asked about the prevalence of sleep disorders among youth and the impact of sleep deprivation on daily activities. Shortly after, the Friends of VA Medical Care and Health Research (FOVA) held a briefing to inform members of Congress and their staff about the progress that the Department of Veterans Affairs (VA) Medical and Prosthetic Research program has made in improving Veterans’ lives in the areas of traumatic brain injury, post-traumatic stress disorder, and sleep apnea.

The sleep research agenda has also gained traction in the Food and Drug Administration (FDA) as their efforts to advance the nomination of sleep disorders for the new Patient-Focused Drug Development Initiative resulted in narcolepsy being chosen as a condition for individual review. Sleep has now become such a contemporary issue that recommendations on treating insomnia, expanding the peer reviewed medical research program to include sleep-related illnesses, and continuing the Sleep, Activity, and Nutrition [SAN] Program as part of the Healthy Base Initiative, were included in the Senate Appropriations Committee FY 2014 Defense bill.

While the summer was filled with sleep briefings on Capitol Hill, additional activities await us this upcoming fall as Congress finalizes the FY 2014 L-HHS Appropriations bill and the Joint Task Force between SRS and AASM issues their white paper on the NIH Sleep Disorders Research Plan. SRS members will work with legislators to advance funding research opportunities for the NCSDR in addition to crafting a sleep research and public health awareness bill that the sleep research community can get behind.

_Dale Dirks and Priyanka Surio_

*Health and Medicine Counsel of Washington*
NIOSH Efforts to Prevent Hazards in the Workplace Linked to Inadequate Sleep

INTRODUCTION

Almost 15 million Americans (or 15% of full-time workers) have shift work schedules that are outside the daytime hours of 7:00 A.M. to 6:00 P.M. (www.bls.gov/news.release/flex.nr0.htm). Working at night, or at irregular hours, goes against human physiology that is hard-wired to sleep during the night and to be active during the daytime. Shift work is often dictated by society’s need for vital around the clock services in public safety, healthcare, utilities, food services, manufacturing, and transportation. In addition to shift work, the National Health Interview Survey (NHIS) from 2010 indicates that 19% of Americans worked 48 hours or more per week.¹ Long work hours often do not allow enough time for sleep.

National data show an increasing percent of workers are not getting enough sleep. NHIS data between 2004 and 2007 show that almost 30% of full-time American workers reported short sleep duration (≤ 6 hours per day).² This is an increase from 24% reported during 1985 and 1990. Some types of workers show higher rates of short sleep duration: 34% of workers in manufacturing and 44% of those who usually worked at night. Much higher rates have been reported for night shift workers in transportation and warehousing (69.7%) and health-care and social assistance (52.3%).³

Shift work and long working hours are well recognized occupational hazards. Insufficient sleep is linked to errors at work and errors while driving on the commute that endanger the worker and also other people around them. Poor sleep, shift work, and long working hours are linked to a wide range of acute and chronic diseases. The National Institute for Occupational Safety and Health (NIOSH) has a long standing commitment to preventing hazards from these demanding work hours through research, guidance and authoritative recommendations, and dissemination of information to protect workers, workers’ families, employers, and the community (see www.cdc.gov/niosh/topics/workschedules).

National Occupational Research Agenda and NIOSH Program Portfolio

Sleep-related research fits many strategic, intermediate, and activity/output goals in the National Occupational Research Agenda (NORA; www.cdc.gov/niosh/nora/default.html). The NIOSH Program Portfolio broadly guides NORA activities. The Program Portfolio is organized into 10 NORA Sector Programs that represent industrial sectors, and 24 cross-sector programs organized around adverse health outcomes, statutory programs, and global efforts (www.cdc.gov/niosh/programs).

Several sector goals directly address fatigue or sleep deprivation including (but not limited to): creating and using data collection systems for evaluation of sleep deprivation and fatigue as factors in injuries and fatalities, developing effective guidelines to reduce worker fatigue, fostering dissemination and implementation of effective teaching tools and interventions to reduce risks from demanding work schedules, and investigating workplace factors or workplace factors that may be associated with cardiovascular disease, cancer, or adverse reproductive outcomes, and determining ways to minimize exposure to such agents.

NIOSH currently funds four extramural Healthier Workforce Centers of Excellence (www.cdc.gov/niosh/TWH/centers.html). The Centers’ research examines the integration and cross-promotion of worker protection, worksite enhancement, and worker health promotion interventions. The effort strives to recognize the synergy in combining efforts to reduce personal health risk factors with traditional safety and psychosocial stress hazard reduction approaches in the workplace. Centers are located at the University of Massachusetts Lowell/University of Connecticut, Harvard University, University of Iowa, and Oregon Health and Science University.

National Data Collection

NIOSH funds the collection of occupational safety and health data for the Quality of Worklife Questionnaire (QWL) and a supplement for the NHIS.

The QWL is a module in the General Social Survey (GSS). GSS is a biannual, nationally representative, personal interview survey of U.S. households conducted by the National Opinion Research Center (NORC; www.cdc.gov/niosh/topics/stress/qwlquest.html). The 76 items in QWL include work hours, workload, worker autonomy, layoffs and job security, job satisfaction, stress, worker

Work-related sleep and work schedule issues closely fit NIOSH’s Total Worker Health™ (TWH) Cross-Sector Program (www.cdc.gov/niosh/TWH). TWH integrates occupational safety and health protection with health promotion to prevent worker injury and illness and to advance health and well-being. This more comprehensive approach addresses the systems in the workplace for organizing work schedules and insufficient sleep from personal factors such as sleep disorders and inadequate sleep practices and behaviors.

Research to Practice (r2p)

The NIOSH Research to Practice (r2p) initiative (www.cdc.gov/niosh/r2p) focuses on reducing or eliminating occupational illness and injury by increasing the transfer and translation of knowledge, interventions, and technologies into highly effective prevention practices and products into the workplace. This interactive process engages the occupational safety and health community — including researchers, communicators, decision-makers, and employer/employee groups — to work collaboratively to:

- Identify research needs;
- Design, plan, and conduct studies;
- Translate and disseminate existing knowledge, interventions, and technologies to relevant users for implementation in the workplace; and to
- Evaluate results to determine the impact on occupational safety and health.

SELECTED RECENT EFFORTS

Healthier Workforce Centers of Excellence

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well-being, and health outcomes including sleep problems. Data are available to interested researchers through NORC (www.norc.org/Research/Projects/Pages/general-social-survey.aspx).

NIOSH funded a 2010 Occupational Health Supplement for the NHIS. The data are available through NHIS (www.cdc.gov/nchs/nhis/nhis_2010_data_release.htm). The NIOSH Supplement included items about common workplace exposures and common work-related health conditions including a question about usual shift worked. The core NHIS includes a question about total hours worked per week and several questions about sleep. The next occupational health supplement is planned for 2015. To provide comments on the content being proposed for the 2015 NHIS occupational health supplement or to read more about the supplement, please visit the NIOSH Science Blog (blogs.cdc.gov/niOSH-science-blog/2013/06/24/nhis).

Medical Residents
Medical residents historically have worked frequent 24-hour or longer shifts in combination with high numbers of work hours per week. NIOSH-funded studies are examining their work hours and the link to adverse outcomes. Residents in the field were studied over the course of 3 weeks while working 24- to 30-hour shifts every third day that alternated with 8-hour shifts. During extended work shifts, residents slept an average of 2.3 hours and had no sleep during 30% of these shifts. Their response times deteriorated during each long shift and further deteriorated over the course of the subsequent shifts. A prospective nationwide survey found residents reported more than double the odds for a vehicle crash on the way home after an extended work shift. The odds for a percutaneous injury with a sharp instrument or needle stick doubled during night shift and increased by 60% during post-call days that followed an extended work shift. The odds for making a fatigue-related medical error increased by 3.5 for months with one to four extended work shifts and increased by 7.5 for months with five or more extended shifts.

Nurses/Reproductive Health/Shift Work
NIOSH studies are examining shift work and physical demands with respect to adverse reproductive outcomes among nurses, specifically the association between work schedule and risk of spontaneous abortion, preterm birth, and menstrual function. NIOSH researchers are collaborating with the Harvard Nurses’ Health Studies II and III, which are both large, ongoing prospective studies of nurses (www.nhs3.org). Results from the Nurses’ Health Study II have shown that a significant increased risk of several reproductive outcomes, including spontaneous abortion, early preterm birth, and menstrual cycle irregularities are related to shift work, particularly working the night shift. In addition, results show independent effects on reproductive outcomes from long working hours. As the Nurses’ Health Study III progresses, there will be increased opportunity to study the impact of occupational exposures on a wide variety of chronic disease outcomes, including cancers and heart disease. Access to this large dataset involves establishing collaboration with a Harvard investigator connected with the studies, submitting a study proposal, and providing Harvard Nurses’ Health Studies with funds to carry out the work.

Police/Sleep/Shiftwork/Stress
A series of studies are being carried out to understand the connection between exposures to occupational stressors and health outcomes in police officers in Buffalo, New York. Results from a few studies have already indicated that there are significant adverse health outcomes associated with sleep and shift work. One study found that the majority of officers reported feeling tired upon awakening (89.9%) and snoring (83.3%). The prevalence of snoring was 26% higher in night shift workers compared to workers on other shifts. Higher levels of perceived stress in officers were associated with shorter sleep duration and poorer sleep quality. Officers with poor sleep quality had significantly more depressive symptoms than officers with good sleep quality and both short and long sleep durations were associated with higher levels of leptin, a protein that may have implications for obesity-related conditions. Another study found that officers who worked nights and either had less than 6 hours of sleep or worked more overtime had a higher prevalence of metabolic syndrome compared to officers working the day shift. Night shift work was associated with several other problems: a higher rate of work absence, an increased risk of injury, and decreased cortisol levels on awakening indicating dysregulation of the hypothalamic-pituitary-adrenal axis.

Trucking
Information on driver fatigue and the quality, location, and length of sleep has been collected in a large national survey of over 1200 long-haul truck drivers. The survey interview included 17 questions about sleep including hours of sleep, drowsy driving, use of medication or alcohol to fall asleep, sleep quality, Epworth Sleepiness Scale, Berlin Questionnaire, multivariate apnea prediction index, and a fatigue symptom inventory. Drivers also completed a 48-hour retrospective sleep and activity diary. Results from this study are being analyzed to determine the extent of sleep disorders (including sleep apnea), drivers’ experience, and their relationship to health conditions and crashes.

Tailored Training Programs
NIOSH scientists are developing and evaluating tailored training programs for managers and workers in aviation, manufacturing, mining, nursing, retail, and trucking. These are designed to inform them of the importance of sleep and the risks linked to insufficient sleep, shift work, and long work hours and strategies to prevent these risks. NIOSH is developing a comprehensive online training program for nurses. The training program was pilot tested with undergraduate and graduate nursing students. An extramural project is currently testing the program with staff nurses. For the trucking industry, NIOSH is developing public service announcements, a brochure, and a postcard to raise awareness of the importance of sleep and the risks linked to insufficient sleep, shift work, and long work hours and strategies to prevent these risks. A website for trucking is also under development. For the mining industry, NIOSH is developing presentations for mining trainers to give miners. A helmet sticker has also been developed to help further raise awareness of this issue. NIOSH is developing a

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series of short webinars to educate workers and managers in manufacturing and retail about sleep. A training program for pilots of small aircrafts is under development.

**EXTRAMURAL RESEARCH FUNDING OPPORTUNITIES**

NIOSH supports relevant, high quality extramural research that demonstrates impact in reducing occupational disease and injury (www.cdc.gov/niosh/oep/funding.html). Current announcements with multiple receipt dates are listed in Table 1. Emphasis is placed on research that addresses needs outlined in NORA and the NIOSH Program Portfolio.

All applications for extramural research funding must (1) state which industry sectors and cross-sectors are being addressed and (2) explain how the project will contribute to the specified priority area, (3) provide information on how the project addresses r2p, and (4) provide information about expected outputs and outcomes. Interested researchers are strongly encouraged to contact the NIOSH Scientific Program Official (SPO) listed in each funding opportunity announcement for additional information.

**References**


**Disclaimer**

The findings and conclusions in this paper have not been formally disseminated by the National Institute for Occupational Safety and Health and should not be construed to represent any agency determination or policy.

Claire C. Caruso, William A. Robison, and Luenda E. Charles
On Sunday morning June 9, 2013, a legend and one of the greatest scientists of the 20th and 21st centuries left us. His close associates also lost a very dear friend and a mentor who never ceased to inspire. Franz Halberg’s passing shy of his 94th birthday leaves a void that cannot be filled.

Franz Halberg will be remembered for founding the fields of chronobiology,1 chronomics,2 and chronobioethics.3 These new transdisciplinary scientific disciplines could not have flourished without Franz Halberg’s unveiling of lawful variations as a function of TIME within the physiological range and his vision that they had far-reaching implications. Toward this goal, he not only gathered a critical mass of data himself, but with a steadily increasing network of colleagues worldwide, he also developed inferential statistical methods for their analysis and interpretation.

By adding TIME to the existing body of knowledge in all of biology and medicine, and by recognizing the crucial role this new element plays in all matters of life, Franz Halberg developed the new science of chronobiology. By insisting on an inferential statistical foundation, details of a rich time structure were revealed akin to the finer spatial resolution obtained with a microscope. His methodical scrutiny of periodicities shared between biological systems and their broad environment, seen (photic) and unseen (non-photic) influences from the sun and the cosmos led to chronomics in a way reminiscent of discoveries enabled by the advent of the telescope.

Born on July 5, 1919 in Romania, Franz studied the adrenal as a physician scientist in post-World War II Innsbruck, Austria. He continued this work at Harvard Medical School, where he held a World Health Organization fellowship in clinical endocrinology in 1948. In 1949, he moved to the University of Minnesota, which saw his breakthrough experiments that led to the important discovery that circadian rhythms are partly endogenous and can be manipulated by environmental synchronizers, notably the lighting and feeding schedules.4,5 Franz coined the term circadian, after documenting that biologic rhythms tip the scale between health and disease and even between life and death.6 His results were widely published, including a 1969 citation classic.1 By 1958, Franz had recognized the important role played by the cell’s RNA and DNA cycles, which he was first to demonstrate as complementing the hypothalamic-pituitary-adrenal system as mediator of photic inputs.7 He subsequently added pineal feed-sideways and the understanding that there are endogenous physiologic networks that respond to the cosmos.3,8

Beyond circadian, Franz demonstrated that many other built-in cycles resonate in part with their counterparts in our broad environment. His recent work focused on building a growing edifice of shared periodicities with bridges across disciplines,9,10 addressing wide-ranging applications from the optimization of individualized health care to concerns for the health of societies. He strived to understand how to enhance positive thoughts and emotions as a scaffold for tolerance and love by seeking optimal configurations of the time structured realm of the mind, what he called the chronosphere.11 He was a scholar in the true sense of the word, combining science, philosophy, poetry, and spirituality, laying the foundation of chronobioethics.

With applications in all fields of medicine and biology, Franz’s legacy is far-reaching. He will be remembered for his work in cancer chronotherapy. He showed that timing cancer treatment according to marker rhythms improves outcomes both in terms of heightened efficacy and lesser undesired side effects.12 Franz showed that a calorie is different whether it is consumed at breakfast or dinner.5,13 His principle of Primum nil nocere (above all, do no harm) prompted Franz to advocate the individualization of treatment, guided by marker rhythms, with important applications in preventive cardiology. By screening for abnormal patterns of blood pressure variability,10 appropriate circadian timed treatment more than halved the risk of stroke and other adverse cardiovascular events.

The physiological importance of the circadian system, which Franz showed had an endogenous origin, also stemmed from the ubiquitous presence of circadian rhythms in almost every variable investigated, including those related to sleep and the electroencephalogram. By 1961, Franz had documented that the total “cerebral electrical output” (EEG) was circadian periodic, peaking in early morning in healthy men, with similar variation in the alpha, delta and theta EEG frequency ranges.14 He later showed that the periodicity persists in sleep deprived subjects.15 Data were analyzed by his cosinor method and by conventional variance spectra.16 He described circadian rhythms of the total Berger-region amplitude and circadian amplitude modulations in the discrete delta, theta, alpha, sigma and beta frequency bands of EEG recordings in Macaca mulatta monkeys,17,18 concluding from the sequence of acrophases in relation to the lighting schedule that “sleep is not unitary, consisting rather of related but separately controlled rhythmic functions”.19 In monkeys, ultradian rhythms characterizing several EEG frequency bands, eye movement, muscle tone and brain temperature were shown to frequency multiply from waking to sleeping.20 About 1.7-hour ultradian rhythms modulate the alternation of waking, fast and slow sleep in the EEG of patients with narcolepsy.21,22 Under usual living conditions, Franz showed that lying-down time, sleep duration and sleep efficiency in health are characterized by a weekly (circaseptan) rhythm.23

As a gauge of complexity and nonlinearity, studies in Franz’s laboratory associated a reduced correlation dimension of the EEG with an increased regularity of breathing in deep sleep (stage IV) and an increased correlation dimension of respiratory movement during REM with an increased complexity of

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the signals. The change in complexity described by the approximate entropy of respiration was shown to depend in part on that of the EEG, suggesting the involvement of nonlinear dynamic processes in the coordination between brain and lungs. The approximate entropy of EEG being lower during stage IV and higher during wake and REM sleep, it may be useful to estimate sleep stages and the complexity in brain activity. Nonlinearity being detected in EEG signals from epileptic patients during seizures but not during seizure-free intervals or in EEG signals in health, the possibility was raised to use the approximate entropy of EEG signals estimated over consecutive intervals to determine pathological brain activity such as that occurring during absence epilepsy.

With his student Eugene Aserinsky, Nathaniel Kleitman, recognized as the father of modern sleep research, demonstrated that REM sleep was correlated with dreaming and brain activity. When sleepers were hooked to an early version of an electroencephalogram machine, they went through episodes when their eyes darted wildly back and forth several times each night. Kleitman insisted that the experiment be repeated once more, this time on his daughter Esther. In this respect, he and his daughter shared with Franz Halberg the value of self-experimentation. Esther was to contribute a series of self-measurements, notably of heart rate, which she sent to Franz. In another study and another context, her data proved invaluable in demonstrating that a biologic week resonates with circaseptan rhythms in the sun.


References

Teresa Arora, PhD
Dr. Arora’s interest in sleep was first sparked during her undergraduate Psychology degree where she discovered that no theory fully explained why all known species sleep. Her fascination with obesity was paired with sleep and she carefully sourced a PhD program in Medicine allowing her to combine research in both areas. She graduated with her PhD from the University of Birmingham, United Kingdom and investigated sleep across the lifespan in relation to metabolic health. Her thesis work demonstrated diversity by incorporating 1) published findings relating to a large (n=30,000) older Chinese cohort (Guangzhou Biobank Cohort Study) demonstrating that long sleep duration was significantly associated with the metabolic syndrome and its components; 2) published findings demonstrating that sleep duration mediated the relationship between various technology types and obesity in a large (n=750) sample of UK adolescents; and 3) findings from an experimental sleep extension/restriction study revealing alterations in metabolic hormones.

Dr. Arora has also been involved with, and has experience of, a number of clinical studies and randomized controlled trials. She regularly contributes to the Primary Care Research Into Diabetes Evolution (PRIDE), a prospective study of the long-term outcomes of diabetes and its complications, funded by the National Institute of Health Research (NIHR). Furthermore, Dr. Arora’s analyses of clinical data from Dr. Taheri’s Weight Management Clinics have shown ethnic differences in the prevalence and severity of obstructive sleep apnea in severely obese individuals, which was recently published in the Journal of Clinical Sleep Medicine.

Dr. Arora was awarded the first ever UK sleep-obesity grant from the children’s charity Action Medical Research, which allowed her to pursue her interest in adolescent sleep at a post-doctoral level. She now conducts the UK’s largest adolescent sleep study (Midlands Adolescent Schools Sleep Education Study - MASSES) investigating the longitudinal relationships and interactions between objectively measured sleep and academic performance, metabolic health and electronic device usage in a large cohort of adolescents (Mentor Shahrad Taheri, MB BS, PhD, FRCP). The findings from this study allowed Dr. Arora to receive the award of Student Researcher of the Year (2010) by the Association for the Study of Obesity (ASO), multiple publications in reputable journals, as well as an invitation to speak at the British Federation of Women Graduates (BFWG) in London, UK. Currently, her research interests are focused in the area of adolescent sleep and metabolic as well as cardiovascular health.

Dr. Arora’s proactive, motivation and commitment to sleep research was demonstrated as she successfully served on the Sleep Research Society (SRS) 2012-2013 Trainee Symposia Series Subcommittee. She is now in post as an SRS Committee member, serving on the Memberships and Communications Committee, a position she will hold for at least three years.

Aric A. Prather, PhD
Dr. Prather earned his PhD in Clinical and Biological & Health Psychology from the University of Pittsburgh where he specialized in psychoneuroimmunology under the direction of Dr. Anna Marsland. His early research focused on the effects of negative emotions and psychological stress on the immune system. As his research progressed, however, he became enamored with sleep and the extent to which the immune effects of sleep disturbance often mirrored those observed in response to psychological stress. Dr. Prather completed his clinical psychological internship at Duke University Medical Center where worked under the supervision Dr. Jack Edinger. He then went on to complete a two-year postdoctoral fellowship in the Robert Wood Johnson Foundation Health & Society Scholars program jointly housed at the University of California, San Francisco & UC Berkeley.

Dr. Prather is currently an Assistant Professor in the Department of Psychiatry and the Center for Health and Community at the University of California, San Francisco where he is supported by a career development award (K08) from the National Heart, Lung & Blood Institute (NHLBI). The primary goal of this project is to determine whether sleep serves as an important explanatory pathway in the link between chronic psychological stress and levels of systemic inflammation. His mentorship committee is comprised of Dr. Nancy Adler (primary mentor), Drs. Tom Neylan and Elissa Epel (secondary mentors), and specialized advisors Dr. Allison Harvey (sleep), Dr. Mary Whooley (cardiovascular), Dr. Firdaus Dhabhar (immunology) and Dr. Steve Cole (social genomics). This project is embedded in the Stress Aging and Emotion (SAGE) study- a longitudinal study of high stress mothers of children with Autism Spectrum Disorder and low stress mothers of typically developing children. Dr. Prather also directs the Mr. SAGE study, which recruits the male partners of the women enrolled in SAGE with the goal of examining the bidirectional and dynamic associations between sleep and daily stress/emotion regulation processes within high and low stress couples.

Finally, Dr. Prather is very interested in the social determinants that lead to disparities in sleep and health. In this regard, he was recently awarded a 2013 J. Christian Gillin, MD Research Grant from the SRS Foundation to investigate the role of race-based social stress on sleep and nocturnal autonomic functioning. These studies reflect Dr. Prather’s overall research program that seeks to elucidate the complex, interactive effects of sleep and psychological processes on age-related disease risk, particularly cardiovascular disease and infectious illness. A more complete understanding of how sleep may directly or indirectly modulate biological mechanisms (e.g., inflammation, cellular aging) implicated in disease pathogenesis will undoubtedly yield improved intervention strategies for at risk individuals.
SRSF/JAZZ Pharmaceuticals Early Career Development Research Award Update
The review process for the recent Jazz Pharmaceuticals grant is nearing completion. The Sleep Research Society Foundation commends all 27 applicants for their outstanding proposals. A review committee, consisting of 23 sleep research professionals, reviewed the applications, assessing for quality, innovation, and investigator potential. The Research Committee, which conducted the reviews, has passed their recommendations for successful candidates on to the Executive Committee, who will review the potential awardees at their meeting mid-August.

The Foundation works consistently to offer these and other research awards each year, and relies greatly on the generosity of both corporate sponsors and general membership. Make your contribution to the Sleep Research Society Foundation today online [www.sleepresearchsociety.org/donate.aspx] or by phone at (630) 737-9702. The Foundation thanks you for your support.

Call for Volunteer Submissions – Trainee Educational Activity Committee (TEAC) Trainee Subcommittee
The Sleep Research Society is now forming a subcommittee of trainees to help plan the Trainee Symposia Series and other trainee-related activities at SLEEP 2014, the 28th Annual Meeting of the Associated Professional Sleep Societies (APSS) in Minneapolis, Minnesota. This is a unique opportunity to become more actively involved in the SRS and in your field!

The planning and organization of the SRS trainee-related activities are overseen by the SRS’s Trainee Education Advisory Committee (TEAC), and its Chair, Philip Gehrman, PhD, with significant input and involvement of this subcommittee of trainees. We are looking for trainees to volunteer to be part of the planning process. Preference will be given to trainee members of the SRS that have attended at least one SRS Trainee Symposia Series. We also require that members of the committee attend the SLEEP 2014 meeting and assist the SRS staff during the Trainee Symposia Series program. We hope to assemble a committee of trainees with diverse research interests from broad geographic areas that represent the trainees within the society.

If you have any questions, or if you are interested in participating on this committee, please contact Jared Saletin, Trainee Member-at-Large, via e-mail at jsaletin@gmail.com.

Please include the following information in your e-mail:
• Name
• University
• Primary Mentor
• Degree Seeking or Obtained
• Your SRS Section (Circadian Rhythms, Basic Sleep, Sleep Disorders, or Sleep and Behavior)
• Brief Description of Research Interests
• SRS Trainee Symposia Series Events Attended and Years (i.e. Boston 2012, Baltimore 2013, etc)
• Have you previously participated on this or any other SRS committee?
• Do you plan to attend SLEEP 2014 in Minneapolis, Minnesota?

Call for Nominations – SRS Sleep Professional Awards
Each year, the Sleep Research Society honors excellence in sleep research by providing funding and recognition. Each of the SRS awards is presented at SLEEP, the annual meeting of the Associated Professional Sleep Societies. Members can nominate their colleagues for each of the awards.

In the coming weeks, the Sleep Research Society will host a call for nominations for the following awards:
• Distinguished Scientist Award – The SRS’s highest award for scientific advances in the field of sleep research is presented to a single individual for research contributions over their entire career.
• Outstanding Scientific Achievement Award – The award honors the authors of a single research contribution, based upon novel and seminal discovers of a basic, clinical, or theoretical nature.
• Mary A. Carskadon Outstanding Educator Award – The highest honor for sleep educators is awarded to individuals for an outstanding effort in disseminating basic or clinical sleep and circadian research as a mentor, teacher, or through public education.

The SRS encourages members to monitor both their email inboxes, and the SRS website for the official nomination submission dates for these and other awards. For more information on the Sleep Research Society’s offered grants and awards, please visit the SRS website.

SRS Club Hypnos at SFN Neuroscience 2013 in San Diego
The Sleep Research Society is partnering with the Society for Neuroscience to offer a professional networking event for Neuroscience 2013 attendees. The networking event will take place at the Hilton San Diego Bayfront on the evening of Monday November 11, 2013.

Club Hypnos provides an opportunity for colleagues to share interests in sleep and circadian research. As part of the Sleep Research Society’s continuing efforts to advance the field of sleep and circadian research, all interested neuroscientists are welcome to attend the reception. This is a social event that provides a networking opportunity for professionals interested in sleep & circadian research and sleep medicine. It offers meeting attendees the opportunity to interact and learn about the activities of the Sleep Research Society and the benefits of becoming a member. Refreshments and hors d’oeuvres will be served.

For more information and to register for Neuroscience 2013, please visit the Society for Neuroscience website.

Join the NIH Sleep RFA-L ListServ
To receive to-the-minute updates on new funding opportunity announcements and other important developments in the field of sleep research, we encourage you to join the NIH Sleep RFA-L listserv. To register for the listserv and view archives, please visit go.usa.gov/XHX.
of rats, aside from the wide variability in tolerance and expected deprivation of any basic biological need are comparable to those borne out in humans, whose overall physiological responses to oxidative stress. We expect that the fundamental outcomes will be areas of metabolism, immune regulation, hormone regulation, and animal studies and involve unique changes in the interdependent individuals. Characteristic findings have emerged from these studying acute and short-term sleep loss in previously well-rested ingful physical changes are not expected to become evident by ders due to deficiency to develop and become observable—mean survival times. The research initiatives fulfill goals of the NIH Sleep Disorders Research Plan in advancing our understanding of sleep functions in both the brain and the body across the lifespan.

Current Research

- **Chronic sleep loss as a state of cell injury**
  Despite morbidity and mortality in sleep-deprived and chronically sleep-restricted rats, structural damage or necrosis in major organs has not been found by light microscopy. A lack of localized effects, but a condition that is both highly reversible (with subsequent sleep) and potentially lethal, suggests that sleep loss produces widespread biochemical changes and interference with cellular functions. We have reported that the nature of these biochemical changes is related, in part, to oxidant and antioxidant imbalance, indicative of uncompensated oxidative stress. Uncompensated oxidative stress is known to result in injured cells which then are either repaired or die. We therefore interpret several signs of prolonged sleep loss, such as increased circulating cytokines and aminotransferases, as indications of bodily injury. The analyses of cell death, cell repair, and cell renewal therefore are considered crucial steps in visualizing sleep loss effects and identifying specific properties that account for sleep’s restorative nature.

  The approach is to produce a stringent state of sleep loss in rats while avoiding advanced morbidity, and thereby discover the characteristics of the cell injury that may eventuate in morbidity. This condition is met by 10 days of total or partial sleep deprivation in rats—a duration known to be sufficient for metabolic changes and mild neutrophilia to become manifest, but short enough to preclude the advanced morbidity that typically occurs by 18–26 days. We have employed fingerprinting techniques to identify the target of cell injury, whether lipid, protein, or DNA, and measured the amount of apoptosis in several vital organs. The results indicate that sleep loss produces an overall increase in oxidative DNA damage with organ-specific effects in the liver, lung, and small intestine. In the intestine, DNA damage is associated with accelerated cell turnover—considered significant given the already high metabolic rate of this tissue and its role in nutrient absorption and host defense. Sleep recovery reverses the effects of sleep loss by down-regulation of end-point damage to lipids and DNA in association with an absence of any increases in cell burdens, such as the production of new cells or imbalanced DNA damage and repair. These properties of sleep recovery are considered restorative. This evidence provides a framework for further elucidation of mechanisms and processes.

  Co-investigators include Neil Hogg, PhD (Department of Biophysics) and Aniko Szabo (Department of Population Health) at The Medical College of Wisconsin.

- **The causes and consequences of metabolic burdens in chronically sleep-restricted rats**
  We employ cycles of sleep restriction to investigate adaptive

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changes in bodily tissues in response to chronically inadequate sleep. This approach resembles the study of cyclical weight gain in which multiple cycles of starvation–refeeding in rats result in both adaptive responses (e.g., increased lipogenesis) as well as medical implications (e.g., obesity risk). Repeated cycles of disrupted sleep in rats result in a latent period of relatively stable food and water intake without weight gain. This latent period belies what follows, which is a dynamic phase marked by enormous increases in food and water intake and progressive loss of body weight, without malabsorption of calories. Despite repeated, yet limited, opportunities for rebound sleep (“weekend sleep”), the deficits accrue and pathology ensues. Analyses of tissue composition in chronically sleep-restricted rats indicate that protein and lipid amounts in internal organs are largely spared, while adipose tissues depots appeared depleted. This is counter to an expected response to calorie deficiency, which is a dramatic decrease in the masses of vital organs, such as the liver and the intestine. These findings suggest that the metabolic demands of organs may be drivers, in part, of the abnormally high rate of energy expenditure during chronic sleep loss. The hormones corticosterone (the hallmark of a behavioral distress response) and leptin (which acts to diminish food intake) are both decreased by chronic and intermittent sleep restriction, and appear to reflect low substrate availability and diminished adiposity.

After nearly 4 months of recuperation with unlimited sleep opportunities, sleep-restricted rats still consume 20% more food and 35% more water than do comparison control rats, despite normal weight, normal adipocyte morphometrics, and elevated plasma leptin concentrations (which should decreased appetite). This chronic increase in nutrients and water, along with altered negative feedback regulation and substrate use, indicate that there may be lifetime metabolic and endocrine burdens from repeated long periods of insufficient sleep earlier in life.

Ongoing studies are focused on causes and functional implications of the energy demand and the route of calorie disposal at the levels of cells and whole-body systems.

Co-investigators include Philip Clifford, PhD (Anesthesiology and Physiology), Martin Bienengraeber, PhD (Anesthesiology and Pharmacology & Toxicology), Hershel Raff, PhD (Medicine, Endocrinology) and Aniko Szabo (Population Health).

- **Pathology in connective tissues as a consequence of chronically abnormal sleep**
  The connective tissues tend to have the lowest metabolic rates among organs, yet appear to show some of the most dramatic changes during acutely prolonged and/or chronically intermittent sleep loss. The affected connective tissues are the skin, bone, and adipose tissue. The adipose tissue is remodeled from a unilocular phenotype to a multilocular phenotype which others have shown to be associated with abundant mitochondria and a state of high energy demand. With regard to bone, its formation is arrested during chronically insufficient sleep, while the processes of bone resorption continue unabated, resulting in bone that is osteopenic and osteoporotic. The bone marrow is also abnormal—megakaryocyte production is increased and red marrow adipocytes are decreased, consistent with increased demands for blood cell production and risk of thrombocytosis. Current studies are focused on determining causes and mediation of these effects.

Co-investigators include Horatiu Olteanu, MD (Hematopathology), Aleksandra Glavaski-Joksimovic, PhD (Neurosurgery), and Jeffrey Toth, PhD (Orthopaedic Surgery).

**Methodology and Techniques**
We produce acutely prolonged and chronically restricted sleep in rats by the Bergmann-Rechtschaffen paradigm and by our validated modification of this paradigm to prolong partial sleep deprivation. We employ numerous techniques for physiological measurements in live animal experiments, including indwelling catheters and pumps. Analytical tools include biochemical and antibody-based assays and microscopy.

**Training Opportunities**
Training opportunities depend on the status of extramural funding in the laboratory and a critical mass of experienced individuals. Most training opportunities have been undergraduate and graduate student internships and medical student summer research fellowships.

**Recent Publications**


Main Research Topics
At present, two major lines of work have been pursued in our research work: a) insomnia and b) the issue of sleep and memory. In the insomnia field, studies on several aspects of insomnia have been performed including diagnoses, differential diagnoses, epidemiology, psychoneuroendocrinology, autonomous nervous system, imaging and therapeutics. With respect to sleep and memory research, we not only focused on healthy young sleepers, but also included clinical samples (e.g., patients with insomnia and sleep apnea).

Ongoing Studies

- **Imaging in insomnia**
  Neuroimaging methods are currently used in a number of different studies to investigate the pathophysiology of primary insomnia. These methods include combined EEG and functional magnetic resonance imaging (fMRI) recordings, structural magnetic resonance imaging, diffusion tensor imaging, and GABA spectroscopy. Through a collaboration with the Department of Radiology, University of Freiburg Medical Centre, the Center for Sleep Research and Sleep Medicine has access to a 3 Tesla MRI scanner and an MRI-compatible EEG-recording device.

- **Perception of sleep**
  An ongoing federally funded research project will investigate the issue how sleep is perceived by healthy good sleepers and patients with insomnia. Both groups will sleep for four nights in the sleep lab and will have experimental awakenings during nights 3 and 4 from sleep stages 2 and REM – dependent variables will be awakening thresholds and the quality of sleep mentation in both groups and its relationship to the pre-awakening characteristics of the sleep EEG.

- **Meta-analysis of sleep in insomnia and mental disorders**
  A first step in this direction was a meta-analysis of the longitudinal relationships between insomnia and depression, revealing that insomniac symptoms increase the risk of becoming depressed later on by a factor of two. Ongoing analyses investigate the role of polysomnographic sleep variables in insomnia and various psychiatric disorders. Furthermore, we are looking at the effects of complimentary-alternative treatments for insomnia.

- **Freiburg sleep school**
  Within the framework of a larger initiative to establish a preventive center for mental problems/disorders at the work place and in schools/universities, our group has established a project that aims at creating the “Freiburg sleep school”. This type of school will consist of teaching modules aiming at different target populations (e.g., physicians, nurses, teachers, students and pupils) in order to inform about sleep, stress the importance of sleep and teach about maladaptive and adaptive behaviors concerning the quality of sleep.

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**Background**

The Center for Sleep Research and Sleep Medicine was established in 1990 at the Department of Psychiatry and Psychotherapy, University Medical Center Freiburg. At that time, Freiburg already had a long tradition in sleep medicine and sleep research. For example, the pathophysiologic mechanism of sleep apnea was discovered in the work group of Prof. Jung in the Department of Neurology in the 1960’s and one of the first tracheostomies to treat sleep apnea was performed in that sleep lab.

The establishment of the sleep center was mentored by the director of the Department of Psychiatry and Psychotherapy, Prof. Mathias Berger. In 1993, the still active director of the lab, Prof. Riemann, came to Freiburg in order to head the sleep center and develop a research agenda. At present, the sleep lab has a strong clinical branch, with an outpatient clinic and an inpatient facility to diagnose and treat patients with various sleep disorders. Naturally, the focus is on neuropsychiatric sleep disorders including restless legs syndrome, various parasomnias, narcolepsy, and all kinds of insomnias. In the field of research, the group has worked intensively in the field of sleep and depression including sleep deprivation work, studies in several psychiatric disorders including schizophrenia, borderline personality disorder, ADHD, alcoholism, nicotine abuse, etc. For the last decade, a major research focus has been on the topics of sleep and memory and insomnia.
Learning and memory
In line with numerous other studies, work from our lab shows that healthy sleep facilitates the consolidation of newly acquired memories. Our lab provided the first evidence that sleep-related memory consolidation is impaired in patients with insomnia and sleep apnea. Other studies have centered on potential neural mechanisms showing that EEG activity patterns that have been related to synaptic downscaling (EEG delta power) and synaptic strengthening (EEG power in the frequency range of sleep spindles) correlate with overnight memory consolidation. In adolescents, we demonstrated that the timing of learning prior to nighttime sleep is critical for the retention of new memories. Current studies include i) investigations on the impact of sleep versus reduced stimulus interference during restful waking on memory, ii) studies on the effect of sleep on the qualitative reorganization of memories, and iii) the modulation of sleep and memory through paradigms of non-invasive brain stimulation (transcranial direct current stimulation, tDCS). Sleep related interventions might bear the potential to strengthen or weaken memory traces – an approach that might be of relevance for the treatment of mental disorders, such as PTSD or exposure therapy in anxiety disorders.

Teaching/Students
With respect to teaching, our group is active in the Faculties of Medicine and Psychology. We have a constant group of 5-10 students working either on a master thesis, MD thesis, or PhD thesis in our laboratory.

Representative Recent Publications


The Sleep Research Society welcomes members who recently joined the organization. Our membership continues to grow — help us strengthen the impact of the profession by encouraging your colleagues to join. Information regarding membership can be found on the Society website (www.sleepresearchsociety.org).

FULL MEMBERS

- Charles N. Allen, PhD  Portland, OR
- Kathleen R. Ashton, PhD  Cleveland, OH
- Ashura Buckley, MD  Bethesda, MD
- Meredith E. Coles, PhD  Binghamton, NY
- Laura Creti, PhD  Montreal, QC Canada
- Kimberly M. Fenn, PhD  East Lansing, MI
- Joseph Finkelstein, MD, PhD  Baltimore, MD
- John A. Groeger, PhD  Hull, United Kingdom
- Chantelle N. Hart, PhD  Providence, RI
- Amy L. Heaton, PhD  Salt Lake City, UT
- Ilia N. Karatsoreos, PhD  Pullman, WA
- Laura Palagini, MD, PhD  Pisa, PI Italy
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- Louis Licamele, PhD  Washington, DC
- Diane C. Lim, MD  Philadelphia, PA
- Michael R. Nadorff, PhD  Mississippi State, MS
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- Laura Palagini, MD, PhD  Pisa, PI Italy
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POSTDOCTORAL FELLOW

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