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A Publication of the Sleep Research Society, USA
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PRESIDENT'S MESSAGE

It is an honor to serve as SRS president during the 50th anniversary year of the discovery of REM sleep. Not only will we, members of the SRS and dedicated sleep researchers, reflect back on the accomplishments of the last half-century, but it is also a time to look to the future of sleep research with renewed enthusiasm.

This is a time of great opportunity in our field. The rapid growth of scientific knowledge and technology has allowed sleep research to expand in many new directions and involve increasing numbers of scientists from a variety of fields. The National Center on Sleep Disorders Research is on the verge of finalizing a National Sleep Disorders Research Plan Revision. It will establish priorities for the conduct and support of research, training and health information dissemination related to sleep and sleep disorders by the NCSDR, NIH and other Federal agencies for the next five years.

From a clinical perspective, the field of sleep medicine is making considerable progress towards its goal of achieving recognition of its fellowship training programs from the Accreditation Council on Graduate Medical Education (ACGME). In addition the American Board of Sleep Medicine is moving to gain recognition of sleep medicine by the American Board of Medical Specialties (ABMS). These developments increase the potential for the establishment of academic sleep centers or even departments in universities and medical schools, which will translate to even greater opportunities for, sleep researchers.

Although we have made significant progress, there is still much for the SRS to do to ensure the future of sleep research. In 1998, the SRS produced the Vision 2020 Task Force report. As a result of this process and the hard work of SRS volunteers, the SRS has increased member services through our new web site, addressed training and job development through support of trainee programs, and established the J. Christian Gillin Endowment for Junior Faculty. In addition, we strengthened our relationship with the American Academy of Sleep Medicine in a number of areas but particularly by cosponsoring two workshops in the past year, BioInformatics in Sleep Research and Sleep and Fatigue in Medical Training. At this point, the SRS is in the initial process of creating a strategy for the coming years to renew our mission, foster scientific investigation, promote training and education, and provide forums for exchange of knowledge.

Over the next year, the SRS Board will engage in a strategic planning process that will provide a framework for our activities for the next several years, with particular emphasis on the following areas:

1. Training: The future of sleep research depends on a constant supply of young scientists entering the field. We continue to face the handicap of having few universities with the critical mass of sleep scientists needed to provide comprehensive training. We thus need to rely more heavily on specialized

trainee meetings or programs attached to other national or international meetings. The SRS needs to assess the training needs of our future scientists and determine what types of programs or support will be most helpful to meet them.

2. Research: Although the NCSDR Plan is an important step in promoting sleep research, the SRS must consider what our responsibility should be in stimulating sleep research activities, including the submission of proposals in response to the new plan and even possible direct support of research studies.
3. Job development: The first junior faculty development grant, awarded this past year, was a major milestone for the SRS. It should be only the beginning. As sleep medicine gains stature as a medical specialty, there should be growing opportunities to establish faculty positions for sleep scientists. The SRS will need to establish goals for future awards and mechanisms for the fundraising that will support our missions.
4. Relationships with AASM: We have developed a stronger alliance with the AASM while maintaining the separate identity and governance of the SRS. Our joint ventures, the journal SLEEP and the APSS Annual Meeting, have both flourished in recent years. There are clearly other areas where we will need to work together in a coordinated fashion.



A number of developments over the last few months are already having a dramatic impact on SRS activities. First, the move of the SRS administrative offices, along with the AASM, APSS, Journal SLEEP, ABSM, ADSM and WFSRS to the new corporate headquarters outside of Chicago provides significantly more space, a more convenient location, and the ability to maintain ease of communication amongst the organizations. The SRS now has a new Society Coordinator, Ms. Judy Yore, who has quickly become indispensable. Ms. Yore previously worked for the Society of Actuaries, where she served as Manager of Academic Relations and Business Manager for the Actuarial Education and Research Fund. She has extensive experience working with members in both academic and nonacademic positions. Mr. Jerome Barrett, Executive Director of AASM, deserves our thanks for recruiting Ms. Yore and overseeing a smooth transition to the new headquarters.

Several important changes have also occurred within the Associated Professional Sleep Societies. First, Dr. David White has been selected to serve as the next editor of the Journal

SLEEP, and will begin in his new role on January 1, 2003. Under the stewardship of Dr. Thomas Roth, SLEEP became the leading scientific journal in the field, increasing its impact factor from 1.8 to 4.25. The Joint Operations Committee has also selected Dr. Ronald Szymusiak as the Chair of the APSS Program. On behalf of the SRS, I would like to congratulate Drs. White and Szymusiak and encourage SRS members to provide them with your full support.

Finally, preparations are well under way for the 2003 APSS meeting in Chicago, which will celebrate the 50th anniversary of the discovery of REM sleep. This will be a joint meeting of the WFSRS. The Program Committee is already hard at work planning a number of special events that should make this a truly extraordinary occasion. Announcements will be sent out in early September and the abstract deadline is December 16, 2002. We hope to see all of you in Chicago.

The great strength of the SRS has been its volunteers, and the SRS Board will be working on ways to involve more members in projects and committees. Please contact Judy Yore (phone: 708-492-1093, e-mail: jyore@aasmnet.org) or me if you would like to volunteer your time or expertise. I greatly look forward to working with you during the coming year, and would be happy to hear from you regarding any questions, comments or concerns.

Don't forget the APSS/WFSRS 2003 abstract deadline is December 16, 2002.



Ruth M. Benca, MD, PhD
SRS President

EDITORIAL COLUMN

by Larry D. Sanford, PhD

We can safely say that our recent meeting in Seattle was a resounding success. In addition to the excellent science, there were other important events at the meeting. The SRS Board met and welcomed new members, and the highest Society awards were presented. For those not fortunate enough to attend the meeting, we have provided a short profile of Board members and congratulated the award recipients, highlighting their accomplishments. In addition, Jennifer Martin, Ph.D., the new trainee member at large and assistant editor of the Bulletin, provides her insights and reflections about trainee day activities.

With the Seattle meeting behind us we are already looking ahead to 2003. Be sure to note the changes to the abstract submission guidelines for the 2003 APSS annual meeting. We've also provided information about next year's award programs.

The quality of submissions to this year's high school essay contest reflects well on the success of the contest as a vehicle for raising awareness about issues in sleep. The five winning essays are included in this issue.

We also present the final two invited lectures given at the symposium, "Stressor-induced alterations in sleep" held in October 18-19, 2001, in Sao Paulo, Brazil. This symposium was funded by the American Physiology Society as part of its Latin American Initiative.

I want to thank Judy Yore who has done a tremendous job in helping put this issue together.

As always, your contributions and suggestions are welcome.

Announcements

We've Moved!

On July 1, the Sleep Research Society, along with the American Academy of Sleep Medicine, moved from Rochester, Minnesota to Westchester, Illinois. This move brings the association closer to other medical societies and governmental and regulatory agencies.

Please update your records. Future communications should be directed to the Sleep Research Society at its new address:

Sleep Research Society
One Westbrook Corporate Center, Suite 920
Westchester, IL 60154
Phone: 708-492-1093
FAX: 708-429-0943
e-mail: jyore@aasmnet.org

APSS News

The Joint Operations Committee is pleased to announce that David White, MD has been appointed the next editor of the journal *SLEEP*. He will be replacing Tom Roth, PhD who will complete his five-year term as editor in December. As editor, Dr. Roth made substantial contributions, implementing changes that improved the publication and increased its impact on the profession. Dr. White intends to build on these enhancements during his term.

Dr. White's acceptance of the editorship of *SLEEP* created an opening as the Chair of the APSS Program Committee. The JOC appointed Ronald Szymusiak, PhD to fill this position as the APSS prepares for the 2003 meeting in Chicago. This year's meeting, jointly sponsored by the World Federation of Sleep Research Societies, will give special recognition to the 50th anniversary of the discovery of REM sleep.

APSS

16TH ANNUAL MEETING

SLEEP RESEARCH IN SEATTLE

The 16th Annual Meeting of the Associated Professional Sleep Societies was held June 8-13 in Seattle, Washington. Following are some interesting facts and figures from this meeting:

- 4,122 meeting participants, the largest number of APSS meeting attendees to date
- 700+ abstracts presented
- 15 postgraduate courses offered
- 21 symposia sessions scheduled
- 54 opportunities to "Meet the Professor"
- 110 exhibitors displayed products and services
- First time APSS was held in a convention center
- 0 rain in Seattle during meeting!
- 708-402-0930 phone number to contact APSS to request evaluation form
- David Dinges, SRS President, gave outstanding keynote address on "Manifestations of Sleepiness: What Does it Mean to be Awake?"
- CD ROM of invited lectures available for purchase at www.APSS.org
- David White, MD concluded his term as Chair of APSS Program Committee



Attendees listen attentively to David Dinges, PhD present the Keynote Address at the 2002 APSS Annual Meeting on the topic of "Manifestations of Sleepiness: What Does it Mean to Be Awake?"



Michigan Avenue
Photo courtesy of the Chicago Convention and
Tourism Bureau

CHICAGO: OUR KIND OF TOWN

The APSS 17th Annual Meeting is June 3-8, 2003 at the Hyatt Regency in downtown Chicago. The 2003 meeting marks the 50th anniversary of the discovery of REM sleep at the University of Chicago. The Annual Meeting and this anniversary celebration will be further enhanced by the World Federation of Sleep Research Society meeting jointly with us in Chicago.

The call for Scientific Sessions and Abstracts will be mailed soon and is available for viewing on the meeting web site (www.APSS.org). All important dates are highlighted in the Call.

- The Chicago Board of Trade, the world's oldest and largest futures options exchange, was founded in 1848
- 1871 was the year of the great Chicago fire
- Navy Pier's most visible attraction is a 150 foot high Ferris wheel
- Sears Tower has 110 floors
- Spring is a great time to visit Chicago. In June the average high temperature is 80 and the average low temperature is 57!

Meet The Board

President

Ruth M. Benca, MD, PhD, earned both her medical degree and PhD at the University of Chicago. She is board certified in Psychiatry and Sleep Medicine.

Dr. Benca currently is Professor in the Departments of Psychiatry, Psychology, and Committee on Neuroscience at the University of Wisconsin-Madison, Wisconsin. She has served on the Board of Directors of both the Sleep Research Society and the American Academy of Sleep Medicine. Dr. Benca has also been actively involved with the Associated Professional Sleep Societies, serving as Program Chair and past Chair of the Joint Operations Committee.

President-Elect

Emmanuel Mignot, MD, PhD, earned his medical degree and PhD in pharmacology in Paris, France. He is board certified in sleep disorders medicine.

Dr. Mignot is Professor in the Department of Psychiatry and Behavioral Sciences at Stanford University. He has chaired the National Sleep Disorder Research Advisory Board of the National Institutes of Health and the American Academy of Sleep Medicine Research Committee. He is currently associate editor of the journal SLEEP and a board member of the National Sleep Foundation and of the National Institute of Medicine. His research is dedicated to the study of normal and abnormal sleep, with a primary genetic approach. His major focus is on narcolepsy and the hypocretin system.

Past President

David F. Dinges, PhD, is Professor of Psychology in Psychiatry, Chief of the Division of Sleep and Chronobiology in the Department of Psychiatry, and Associate Director of the Center for Sleep and Respiratory Neurobiology in the University of Pennsylvania School of Medicine.

Dr. Dinges earned his Master of Science and PhD from Saint Louis University in Experimental Physiological Psychology.

He has served on the Board of Directors of the American Academy of Sleep Medicine and the National Sleep Foundation. His research is dedicated to characterizing the physiological, cognitive and functional changes resulting from sleep loss, and evaluating behavioral, pharmacological and technological countermeasures to sleepiness and fatigue.

Secretary/Treasurer

Mark R. Opp, PhD, is Associate Professor in Anesthesiology and Physiology in the School of Medicine and a member of the Neuroscience Program at the University of Michigan. Dr. Opp earned a Master of Science degree in Biology from Walla Walla College, Walla Walla, Washington and PhD in Zoology from Washington State University.

Dr. Opp has served as the editor of the SRS Bulletin, and held

committee memberships on the Committee on Animal Research Ethics, the Trainee and Education Advisory Committee, and the Education and Scientific Review Committee. His research focuses on stressor-induced alterations in sleep.

Section Head - Circadian Rhythms

Michael V. Vitiello, PhD, is Professor and Senior Scientist, Sleep Research Group, Department of Psychiatry and Behavioral Sciences, Adjunct Professor of Psychology and Adjunct Professor of Biobehavioral Nursing and Health Systems at the University of Washington in Seattle.

He received his BA in Psychology from Columbia University in 1973 and his PhD in Physiological Psychology from the University of Washington in 1980.

Dr. Vitiello's research focuses on the causes, consequences and treatments of sleep disturbance in the elderly. He is Editor-in-Chief (for the Americas) of Sleep Medicine Reviews, a Fellow of the Gerontological Society of America and holds an Independent Scientist (K02) Research Career Award from the National Institute of Mental Health.

Section Head - Basic Sleep

Barbara E. Jones, PhD, is Professor in the Department of Neurology and Neurosurgery at McGill University and the Montreal Neurological Institute. She earned her PhD from the University of Delaware in Physiological Psychology after doing a portion of her graduate training at the Faculte de Medecine in Lyon, France.

Her original training in Sleep Research was obtained under Michel Jouvet in Lyon, followed by postdoctoral studies in biochemical pharmacology with Jacques Glowinski in Paris, and then in chemical neuroanatomy with Robert Moore in Chicago. Her research has concentrated upon the physiological and anatomical elucidation of chemically specific neuronal systems that are responsible for the generation of the sleep-wake states.

Section Head - Normal and Pathological Excessive Daytime Sleepiness

Joyce A. Walsleben, RN, PhD, is the director of the New York University School of Medicine's Sleep Disorders Center and Research Associate Professor in the School of Medicine. Dr. Walsleben earned her doctorate in biopsychology from the State University of New York at Stony Brook in 1987 and became board-certified in sleep disorders medicine in 1988. She is a Diplomate, American Board of Sleep Medicine.

As a clinician and researcher she specializes in the diagnosis and treatment of a variety of sleep disorders, including sleep apnea and narcolepsy. In addition, she is a co-PI in the Sleep Heart Health study, a multi-center NIH grant to examine the role of sleep apnea in cardiovascular disease. She served on the New York State Task Force on the Impact of Fatigue and Driving between 1993-1999. She is a member of several professional organizations and serves on the scientific councils of the National Sleep Foundation, the Narcolepsy Network, and the Society for Woman's Health Research and is a member of the Bioterrorism

Task Force, American Thoracic Society. She is a co-author of *A Woman's Guide to Sleep* published October 2000.

Section Head - Sleep and Behavior

Roseanne Armitage, PhD, is Professor of Psychiatry and Psychology at the University of Texas Southwestern Medical Center at Dallas, Texas and is the Director of the Sleep Study Unit. Dr. Armitage completed her Masters and PhD in sleep research at Carleton University in Ottawa, Canada and post-doctoral research at the University of Ottawa. Her research focuses on sex differences in quantitative sleep EEG, the neurobiology of major depressive disorders, risk factors for psychiatric illness across the life cycle, and sleep regulation.

She has been an active participant in APSS since 1981, recently serving on the Program Committee, the Training Education and Advisory Committee and the Nominating Committee for the Sleep Research Society. She has also served on numerous committees and task forces dedicated to education, clinical and basic research, and practice standards in sleep EEG.

Publications Chair

Christine Acebo, PhD, is Assistant Professor (Research) in the Department of Psychiatry and Human Behavior at Brown University Medical School and Assistant Director of the E. P. Bradley Hospital Chronobiology and Sleep Research Laboratory in Providence, RI. Dr. Acebo earned her PhD in Biobehavioral Sciences from the University of Connecticut. The research problems that most engage her include the development of tools to assess sleep/wake patterns in naturalistic settings and the development of analytic procedures for complex data sets.

Membership Chair

Jodi A. Mindell, PhD, is Associate Professor of Psychology and Director of Graduate Studies at St. Joseph's University and Associate Director of the Sleep Disorders Center at the Children's Hospital of Philadelphia. Dr. Mindell earned her MS and PhD from University at Albany, State University of New York in Clinical Psychology, completing her internship at Brown University.

Dr. Mindell also serves on the Board of Directors of the National Sleep Foundation. Her research is dedicated to the assessment and treatment of pediatric sleep disorders, as well as its impact on families.

Program Chair for Trainees

Eric A. Nofzinger, MD, is an Associate Professor of Psychiatry at the University of Pittsburgh School of Medicine where he is currently the director of the Sleep Imaging Research Program. He has been there since 1987, completing residency training in Psychiatry and in Sleep Disorders Medicine and an NIMH research fellowship with emphasis in sleep research.

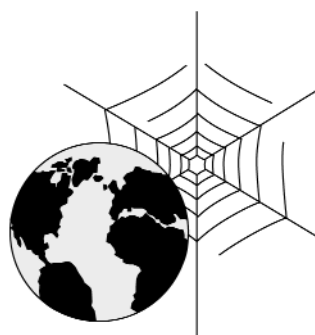
Dr. Nofzinger has provided service to the sleep research community and to training in a variety of capacities over the years, including: a member of the DSM-IV work group on sleep disorders; a representative, Department of Veteran's Affairs to the Advisory Committee for the National Center for Sleep Disorders

Research; a member of the Research Committee, American Academy of Sleep Medicine (AASM); an AASM Representative at an NIMH Research Roundtable; a member of the AASM Foundation Grant Review Board; a Member of the General Clinical Research Centers Review Committee for the National Center for Research Resources (NCRR) at NIH; and a member of the Sleep Research Society Trainee Committee.

Dr. Nofzinger's areas of research interest include human functional neuroimaging studies of sleep in health and in disease where he has received funding to study depression, insomnia, aging, schizophrenia, alcoholism and obstructive sleep apnea syndrome.

Trainee Member at Large

Jennifer L. Martin, PhD, earned her doctorate in clinical psychology at the San Diego State University / University of California, San Diego Joint Doctoral Program in Clinical Psychology. She completed her clinical internship in behavioral medicine at Brown University. Dr. Martin is currently a post-graduate researcher at the University of California, Los Angeles where she studies non-pharmacologic interventions for sleep disruption in aging and dementia.



New Website

www.sleepresearchsociety.org

A new Sleep Research Society web site was introduced in April 2002. This Bulletin is posted on the site. We hope that you take time to visit and explore the many areas of interest on the site.

Site features include:

- Online access to Society information, opportunities and resources
- Sleep related announcements and a calendar of events
- Membership directory accessible to members
- Personal and laboratory web pages for posting individual, center and research laboratory information.
- Downloadable classic research papers
- Links to other sites that pertain to sleep and sleep disorders

The site was designed to be a resource for members and to educate and inform the public. We suggest you visit us at www.sleepresearchsociety.org often to see the latest updates. Your suggestions and comments are welcome.

Award Presentations

The Sleep Research Society (SRS) presented several awards at the 2002 Associated Professional Sleep Societies (APSS) meeting in Seattle, Washington this June. The Distinguished Scientist Award is the highest award presented by the SRS. Awarded annually since 1989, it is given for significant original and sustained contributions of a basic, clinical or theoretical nature. This year, Adrian Morrison, DVM, PhD and Jerome Siegel, PhD were recognized.

As noted in the award announcement distributed at the meeting, Dr. Morrison trained in veterinary medicine at Cornell University and received his PhD in anatomy from the University of Pennsylvania. He entered the field of sleep research during his postdoctoral studies at the Institute of Physiology of the University of Pisa in Italy. Dr. Morrison is currently Professor of Behavior Neuroscience at the University of Pennsylvania. He has been an active volunteer in the profession, sitting on the Boards of various organizations, chairing committees and serving as editor of several journals.

Over the years, Dr. Morrison has made substantial contributions to sleep related research. His early work focused on the control of motor reflexes and the role of the vestibular nuclei in the production of rapid eye movements. He then turned his attention to the phenomenon of REM sleep without atonia, work that aided in the recognition and understanding of REM Sleep Behavior Disorder. Dr. Morrison and his students then demonstrated that pontogeniculo-occipital (PGO) waves could be elicited by external stimuli, which led them to the appreciation of these waves as a sign of alerting within the brain. His work on atonia and PGO waves led to his proposal that atonia is secondary to reticular activation and that REM sleep is one variant of this relationship, orienting being another and narcolepsy, a pathological variant. Dr. Morrison's current research focuses on the study of the amygdala's role in modulating sleep.

Dr. Siegel received his undergraduate degree from the City College of the City University of New York, and his doctorate degree in Physiological Psychology from the University of Rochester, New York. He was appointed Research Psychologist at Sepulveda VAMC in Los Angeles in 1976. He remains at this institution today, having been appointed Chief of Neurobiology Research in 1980 and, more recently, Senior Research Career Scientist.

Dr. Siegel has served on several committees and the Boards of several organizations in his career, including serving as Chairman of the Neurobiology Review Board for the Veterans Administration, and President of the Sleep Research Society. He also served as Chair on the Association of Professional Sleep Societies' Scientific Program Committee, Program co-chair of the World Federation of Sleep Research Societies, and associate editor of the Journal SLEEP. He is currently serving on the Narcolepsy Network Medical Advisory Board and on the Editorial Board of the Archives of Italian Biology.

Dr. Siegel has received numerous awards and grants for his

research. He was given the M.E.R.I.T. Award by the National Institutes of Health Heart, Lung, and Blood Institute for funding of research on the control of muscle tone by the nucleus magnocellularis. He also received the Javits Award from the National Institute of Neurological Disorders and Stroke for his research on immunologic factors in narcolepsy. Dr. Siegel won the Narcolepsy Network Scientist/Clinician Award in 2000. The SRS also presented him with a special award in 2001 recognizing his research leading to the discovery of the loss of hypocretin cells as the cause of human narcolepsy.

The discoveries Dr. Siegel has made in the area of REM sleep will have a lasting impact in the sleep field and beyond. He has made important contributions to the understanding of the brainstem neuronal mechanisms controlling REM sleep and has made significant findings relating to the study of human narcolepsy.

The Young Investigator Award recognizes an outstanding research effort by a new investigator in the field of sleep research. The basis for evaluation of candidates is a single publication in a refereed journal. The Sleep Research Society presented this award to Marcos G. Frank, PhD for his article "Sleep Enhances Plasticity in the Developing Visual Cortex," published in the journal *Neuron*, volume 30, April 2001. Dr. Frank's research tested the hypothesis that sleep is essential for synaptic plasticity. It is the first study that definitively implicated sleep in the mechanisms of experience dependent cortical plasticity. This study should stimulate additional research on the role of sleep in learning and memory and in brain development.

Dr. Frank earned his PhD at Stanford University and did postdoctoral work at the University of California, San Francisco. He is currently on the faculty in the Department of Neuroscience at the University of Pennsylvania in Philadelphia.

We congratulate the SRS award recipients for 2002.



Adrian Morrison, DVM, PhD and Jerome Siegel, PhD were the 2002 Distinguished Scientist Award winners.

SLEEP RESEARCH SOCIETY AWARDS

The SRS Awards Committee is currently soliciting nominations for the Distinguished Scientist Award and applications for the Young Investigator Award.

SRS Distinguished Scientist Award

This is the Society's highest award for scientific advances in the field of sleep research. The award is given for significant, original and sustained contributions of a basic, clinical or theoretical nature. Members of the Sleep Research Society are invited to submit nominations to the Awards Committee. A letter outlining the scientific contributions made by the nominee and the reasons why the individual should be honored should accompany the nomination. Candidates need not be current members of the Sleep Research Society.

Nominations will be reviewed, and the SRS Awards Committee, which may also offer nominations of its own, will present the award. The deadline for SRS Distinguished Scientist nominations is Monday, December 16, 2002.

SRS Young Investigator Award

This award recognizes an outstanding research effort by a new investigator in the field of sleep research. The basis for evaluation of candidates is a single publication in a refereed journal. The candidate should be the first author; and the article must be published or officially accepted for publication by the application deadline. On the application deadline, the candidate must be 35 years old or younger or within 5 years of obtaining a terminal degree. Exceptions to the age rule will be considered for those applicants who feel that extenuating circumstances warrant such consideration. A letter detailing these considerations must be included with the application.

The award consists of a plaque and a travel honorarium that may be applied toward travel to the 2003 Annual APSS Meeting. The plaque will be presented at a ceremony at the Annual APSS Meeting. To apply, candidates must submit 5 copies of the paper, a single CV, documentation of age (a copy of a driver's license, birth certificate or passport) and, if appropriate, a letter outlining extenuating circumstances regarding the age criterion. If a paper is in press at the time of application, a copy of the written notification of the paper's acceptance for publication must also be included. Applicants should provide the name of a senior investigator who will provide a letter of recommendation. The senior investigator does not need to be an author on the paper or abstract, but should be familiar with the candidate's role on the research project. The candidate is responsible for ensuring that the letter of recommendation from the senior investigator arrives by the application deadline. In addition, a candidate must be a member in good standing of the SRS or must include a completed application for membership and fee with the award application. Repeat applications from unsuccessful applicants from previous years are encouraged.

Candidates are welcome to apply for both the Young Investigator

Award and the Trainee Travel Fellowship, but in the event the candidate receives the Young Investigator Award, she/he will receive only this award. Multiple awards may be recognized dependent on the quality of the applications.

The deadline for receipt of applications is Monday, March 3, 2003.

Nominations and applications for SRS awards should be sent to:

Judy Yore
Sleep Research Society
One Westbrook Corporate Center, Suite 920, Westchester, IL 60154
Phone: 708-492-1093; Fax: 708-492-0943
e-mail: jyore@aasmnet.org

AMERICAN ACADEMY OF SLEEP MEDICINE AWARD

AASM Young Investigator Award

All students and postdoctoral residents and fellows who are under 40 years of age on December 1, 2002 are eligible for consideration in the AASM Young Investigator Award. Selection is based on scientific merit, innovation, logic, and evaluation by the APSS Program Committee's designated reviewers and the AASM Research Committee.

After reviewing submissions, the AASM Research Committee contacts semi-finalists for their respective Senior Investigator information associated with the work. Final candidates will be selected by the Committee upon confirmation that the nominated project was an original idea produced by the young investigator in an independent manner and that the candidate is under 40 years of age.

The top finalist and four honorable mentions will be recognized at the Annual Meeting and will receive honoraria.

Look for additional information, including submission deadlines on the meeting web site (www.aasmnet.org). Please address questions relating to this award to the AASM Research Committee at 708-492-0930.

ASSOCIATED PROFESSIONAL SLEEP SOCIETIES TRAVEL AWARDS

The Sleep Research Society offers a limited number of travel awards to help trainees attend the APSS Annual Meeting. There are two types of travel awards: awards that are issued on the basis of scientific merit and awards for trainees who have never attended an APSS Annual Meeting. Trainees may apply for only one type of travel award. All eligible applicants must be members of the SRS and currently engaged in sleep research.

Travel Awards are primarily intended to further the career development of students who are actively pursuing an academic degree, or who are in the early stages of post-graduate training in the areas of sleep. Eligible trainees include undergraduate stu-

dents, graduate students, postdoctoral fellows or medical interns/residents who are within four years of receiving the doctoral degree or completion of medical internship. Trainees who have been accepted but who are not yet enrolled in a college or university degree program are also eligible. The dollar amount of the award to each recipient (not to exceed \$500) depends on the type of the award.

Historically, there are many more Travel Awards based on Scientific Merit issued than First Time Trainee Travel Awards. Award winners will be notified by e-mail (or fax if e-mail address is not provided) prior to the advance registration deadline for the APSS Annual Meeting.

Trainee Award Based on Scientific Merit

To apply for a Trainee Travel Award Based on Scientific Merit, you must be the first author of an abstract. Although the same first author may submit up to two abstracts only one of the abstracts may be submitted for consideration of this award. To apply, please check the appropriate box and complete all of the required information on the APSS Annual Meeting abstract submission form and submit by the designated deadline.

Trainee abstracts will be ranked based on scientific merit regardless of presentation type. The scientific merit of abstracts is recognized at three levels:

- Trainee Research Excellence Awards - top 10 trainee abstracts
- Trainee Research Merit Awards - meritorious trainee abstracts
- Trainee Travel Award - good abstracts that were not ranked high enough to receive a merit or excellence award

First Time Trainee Travel Award

Trainees who have never attended an APSS Annual Meeting may apply for a limited number of First Time Trainee Travel Awards. Applicants for this award will not be considered for a Merit-Based Trainee Travel Award. To apply for this award you must submit the following:

- CV of applicant - should include current mailing address, and email address (preferable) or fax number for the most efficient method of correspondence.
- A letter from the applicant certifying that she/he has never before attended an APSS Annual Meeting and a statement expressing how this meeting will be beneficial to her/his career development.
- A letter from the trainee's mentor or departmental advisor describing the trainee's research involvement, and certification that the applicant is an eligible trainee.

Deadline for receipt of the First Time Trainee Travel Award Application: February 3, 2003
Send complete application to:
SRS First Time Trainee Travel Award
One Westbrook Corporate Center, Suite 920
Westchester, IL 60154



Chicago River
Photo courtesy of the Chicago Convention and Tourism Bureau



NEWS FLASH

NEWS FLASH ABSTRACT SUBMISSION GUIDELINES APSS 17TH ANNUAL MEETING

Please note that the APSS Program Committee has made several changes relating to abstract submissions for the APSS 17th Annual Meeting. Please note the following:

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The Role of Cytokine-Neurotransmitter Interactions in Immune Stressor-induced Alterations in Sleep

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Introduction

Vertebrate animals subjected to immune stressors in the form of acute bacterial or viral infection undergo specific alterations in behavior (Toth, 1999). These changes in behavior, often termed sickness behavior, include social isolation, significant alterations in sleep patterns, decreased appetite, and lethargy. How immune responses to infection induce these changes, and the complex regulatory mechanisms in brain responsible for these alterations in behavior, is not well understood. It is also not known whether the changes in behavior that occur during infection promote host defense and ultimately improve survival. Phylogenetic data and conventional wisdom suggest that sickness behavior during acute infection promotes survival by protecting a vulnerable animal and by altering the host environment such that pathogen growth is inhibited [see (Opp, 1999)]. We present evidence in this review that interactions in brain between neurotransmitters and immunomodulators are of functional relevance to the alterations of sleep in response to immune stressors. We focus on corticotropin-releasing hormone (CRH) and the cytokine interleukin (IL)-1 as prototypical representatives of neurotransmitters and immunomodulators, respectively. We also suggest that the specific changes in sleep in response to immune stressors serve to support the efforts of the immune system to combat the pathogen by facilitating the production of fever.

Immune Stressor-induced Alterations in Sleep

Although the precise quantitative and temporal changes vary substantially depending on the infecting microorganism and the route of infection, infection-induced alterations in sleep are characterized by an initial enhancement of slow-wave sleep (SWS), which is subsequently followed by SWS suppression (Toth and Opp, 2001b). Although during the early stages of infection there is an increase in the amount of time spent in SWS, the SWS that occurs during this period is extremely fragmented. The fragmented nature of SWS in response to infection is manifest by many more transitions between SWS and waking, with the result that individual SWS epochs are much shorter than normal. REM sleep is generally inhibited for the duration of the infection. These biphasic responses of SWS to true infection (ie., in which there is replication of the pathogen) may also be observed when immune activation is induced by the injection of immunomodulators such as IL-1 [eg., (Opp and Imeri, 2001)], or the bacterial cell wall product lipopolysaccharide (LPS). As such, certain facets of infection-induced alterations in sleep may be mimicked by administration of immunomodulators into otherwise normal animals. Although there are many other aspects of infection-induced alterations in sleep that could be discussed, these are beyond the scope of this paper and the interested reader is referred to a paper by

Toth and Opp (2001b). The salient features of infection-induced alterations in sleep for the purposes of this discussion are the biphasic temporal alteration and fragmentation of SWS and REM sleep suppression.

CRH and IL-1 Interactions

CRH is well-known as a mediator of many of the complex behavioral, physiological, and autonomic responses to stressors [reviewed (Dunn, 1990; Owens and Nemeroff, 1991)]. CRH, and other peptides and hormones that comprise the hypothalamic-pituitary-adrenal (HPA) axis such as adrenocorticotrophic hormone (ACTH) and corticosterone (CORT), generally induce waking when administered into experimental animals or humans [reviewed (Opp, 1995)]. In contrast, cytokines such as IL-1 increase SWS [reviewed (Opp et al., 1992)]. Cytokines are also potent stimulators of HPA axis activity [reviewed (Turnbull and Rivier, 1999)]. Indeed, the HPA axis constitutes the major negative feedback mechanism for cytokine actions in brain. For these and other reasons we hypothesize that the complex bi-phasic alterations in sleep of animals subjected to immune stressors are due, in part to interactions between cytokines and the HPA axis.

According to our working hypothesis, the initial enhancement of SWS after infection is mediated by cytokine actions in brain. It is now well-known that many cytokines and their receptors are in brain, where they respond to activation of the peripheral immune system. IL-1, tumor necrosis factor (TNF), and perhaps other cytokines [e.g., IL-6] are synthesized and released in brain in response to infection, and act on sleep regulatory centers in the hypothalamus and brain stem to increase SWS and suppress REMS. There are several mechanisms by which the peripheral immune system and the brain engage in bi-directional communication. A review of these mechanisms is beyond the scope of this paper; the interested reader is referred to a paper by Dantzer (1994). Although there is ample evidence to suggest that the initial enhancement of SWS during the early stages of infection is mediated by cytokine actions in brain, the definitive experiments to test this hypothesis remain to be conducted. To the best of our knowledge there have been no studies of the effects of antagonizing cytokine actions in brain on alterations in sleep during infection. However, the disruption of normal cytokine networks in brain impacts responses to infection, as mice lacking a functional IL-10 gene exhibit different sleep responses to influenza challenge than background strain control animals (Toth and Opp, 2001a). IL-10 is a key cytokine for the regulation of IL-1 and TNF actions, and as such these data provide additional evidence that cytokines do play a role in infection-induced alterations of SWS.

CRH and the HPA axis comprise the major negative feedback mechanism for cytokine actions in brain (Turnbull and Rivier, 1999). The ultimate means by which the HPA axis modulates cytokine actions in brain is by the inhibition of cytokine synthesis by glucocorticoids (Pezeshki et al., 1996). Immune challenge by infection increases first cytokines (ie., the immune response), which is followed later by increases in CRH and HPA axis activity. The second phase of the bi-phasic SWS response to infection, the period when sleep is suppressed, may thus be due to either an increase in substances known to enhance / modulate waking (CRH / HPA axis), and / or due to the glucocorticoid-mediated reduction in the synthesis of substances that enhance SWS (eg., cytokines). Evidence supporting this hypothesis is derived from studies of rat strains that differ in HPA axis activity induced by immune challenge (Opp and Imeri, 2001). Fischer 344 (F344) rats and Lewis rats are inbred rat strains derived from the outbred Sprague-Dawley. Lewis rats have a genetic defect rendering them less sensitive than Sprague-Dawley rats to immune challenge with respect to HPA axis activation. F344 rats have enlarged corticotrophs, and secrete more CRH in response to immune challenge than either Lewis or Sprague-Dawley rats. Therefore, these rat strains diverge with respect to HPA axis activation by immune challenge, with one strain (Lewis) being hyporesponsive and another (F344) being hyperresponsive.

We exploited these genetic differences by administering IL-1 ICV into rats of each strain and determining effects on sleep-wake behavior (Opp and Imeri, 2001). Sprague-Dawley rats responded to IL-1 administration as previously reported. SWS was increased in the first postinjection hour, after which there was a 2-h period when SWS did not differ appreciably from controls. SWS began to increase in postinjection hour 4, reaching the peak magnitude of effect in postinjection hour 5. Lewis rats, with reduced HPA axis responsiveness to immune challenge, exhibited increased SWS earlier, with the peak effect observed in postinjection hour 4. The SWS response of these animals to IL-1 was apparent in postinjection hour 3. In contrast, F344 rats exhibited a profound suppression of SWS in postinjection hour 2, which we attributed to an IL-1-induced surge in CRH / HPA axis activity (this rat strain has a hyperresponsive HPA axis to immune challenge). We demonstrated that this is indeed the case because blocking CRH actions with a specific receptor antagonist before administration of IL-1 completely eliminated this IL-1-induced suppression of SWS. As such, aspects of IL-1-induced changes in SWS of these rat strains are attributed to actions of CRH.

Functional Considerations for Immune Stressor-induced Alterations in Sleep

The functions of sleep present a great mystery to neuroscientists. It is not the purpose of this review to posit functional explanations for this fundamental CNS process. We will however, suggest that the precise manner in which sleep architecture is altered by infectious challenge is ideally suited to facilitate the production of fever. Given the critical role fever plays in host defense, infection-induced alterations in sleep may be viewed as an adaptive response that improves the probability the host will survive the infectious challenge. We have previously outlined in greater detail the evidence supporting this hypothesis, and the interested reader is referred to this earlier work (Opp, 1999). We provide

here a very brief synopsis of the evidence supporting our hypothesis.

1) Sleep and thermoregulation are closely linked, yet separable physiological processes. In healthy animals, there is a regulated decrease in brain / body temperature upon entry into SWS. [Although sleep-wake state-dependent changes in body temperature are apparent (Obál et al., 1985), we focus in this discussion on brain temperature]. Upon entry into SWS, brain temperature decreases to the regulated asymptotic values that is species appropriate, and is due to changes in peripheral vasomotor tone. Generally speaking, the longer and more consolidated the SWS episode, the greater the decrease in brain temperature, until the regulated asymptote is reached. Sleep-wake dependent changes in brain temperature are superimposed on the normal circadian brain temperature fluctuations.

2) The early studies of Parmeggiani and colleagues (Parmeggiani et al., 1977; Parmeggiani, 1988; Parmeggiani and Rabini, 1967), and of Heller and his group (Heller, 1977; Heller et al., 1983; Heller et al., 1988), convincingly demonstrated that during REM sleep the major thermoregulatory effector mechanisms of shivering, panting or sweating are impaired, if not completely absent. Therefore, in a functional sense REM sleep renders normally homeothermic vertebrates poikilothermic.

3) There is empirical evidence that fever is beneficial to the host. Studies of ectotherms, in which fever may be manipulated by altering environmental temperature, indicate that fever imparts survival value. Studies of this nature [eg., (Kluger et al., 1975)] indicate the probability of dying increases if the host is not permitted to fever in response to pathogen challenge. [The use of antipyretics during infection is an interesting topic that must wait for another discussion.] There are thought to be two means by which fever improves host survival; immune responses of vertebrates are more efficient at temperatures slightly greater than normal body temperature, and many pathogens (particularly bacteria) replicate more slowly at temperatures slightly above normal body temperature of the host.

4) Fever, although beneficial to the host, is costly in terms of energetic expenditure. It is estimated that overall metabolism must increase by about 13% per 1 °C fever developed, and that through the duration of an infectious challenge, overall metabolism may increase 30 to 50% (Kluger, 1979). Although many factors influence the metabolic costs of producing a fever, and the numbers stated above are likely to differ depending on the specific host and pathogen interaction, it is clear that fever presents a major metabolic burden to the host.

We now come to the question of why an animal should sleep differently during the early stages of acute infection. We suggested that the alterations in sleep that occur during the course of an acute infection serve to support the generation of fever. Because there is empirical evidence that fever increases survival of the host, anything the animal can do to support the generation of fever increases survivability. Fever is an energetically costly undertaking. Sleep conserves energy, and by sleeping instead of being active, the animal makes available additional energy reserves for fever production. In addition, the manner in which sleep archi-

ture is altered during the early stages of infection also supports the generation of fever. The suppression of REM sleep is critical because shivering is the most effective means of generating a fever. Because this stage of sleep, during which time shivering can not occur is abolished, the period when shivering can occur is effectively increased. In addition to suppression of REM sleep, NREM sleep becomes fragmented. During NREM sleep episodes, heat dissipation occurs exponentially until reaching a regulated asymptote. Fragmenting NREM sleep reduces heat dissipation because the NREM episodes are of such short duration. Therefore, the combined effect of REM sleep suppression and NREM sleep fragmentation during the chill phase of fever is to increase heat production and reduce heat loss, i.e. facilitate the production of fever. Of what functional significance is a biphasic sleep response to acute infection? During the production of fever, energy reserves have been depleted, and these stores need replenished. Later in the infectious challenge, sleep is suppressed, which provides additional time for the animal to forage / feed in an effort to restore energy reserves.

Conclusions

There are profound changes in the behavior of sick animals that may serve an adaptive function in combating the pathogen. The mechanisms that mediate responses to immune stressor-induced alterations in sleep are likely to include interactions between immunomodulators, such as the cytokine IL-1, and neurotransmitters, such as CRH. Although we have touched only briefly on this topic in this review, there is ample evidence to support the hypotheses that IL-1 and CRH are regulators / modulators of SWS and waking, respectively and that their interactions may be important determinants of the alterations in behavior that occur in response to immune stressors. Whether or not the precise changes in sleep that occur in response to infectious challenge facilitate recovery remains to be determined, yet evidence provided herein suggests that by supporting the generation of fever, immune stressor-induced alterations in sleep may indeed serve this function.

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Alterations in Sleep During Infectious Disease: Interactive Influences of Stress and Host Defense

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The generation of an immune response, activation of the hypothalamic-pituitary-adrenal (HPA) axis, and alterations in normal sleep patterns are all components of the host reaction to infectious challenge. An effective immune response is essential to achieving the optimal outcome of elimination of the pathogen, recuperation, and survival. In contrast, an inadequate immune response can permit excessive microbial proliferation and damage and perhaps result in death, whereas an excessive or unregulated host response can result in unnecessary inflammatory damage, as well as potential disability or death. However, the complex processes of the immune response, the HPA response, and patterns of sleep are highly interrelated, and all three are likely to influence the clinical outcome of infection.

The development of increased sleep propensity during microbial infections and a resultant increase in time spent asleep may confer survival advantages during infectious disease. One likely advantage is reduced energy utilization due to inactivity and a lower metabolic rate (Zepelin, 2000). Such energy conservation may help the host to maintain metabolic homeostasis despite the metabolically costly generation of fever during periods of infection-related anorexia. Because animals typically seek a protected location for sleep, increased somnolence may also promote survival by causing the animal to remain in a relatively safe location during periods of disease-related debilitation (Hart, 1988; Sapolsky et al., 2000). Sleep is also postulated to potentiate immune responses and could perhaps in that way confer an additional host benefit during infectious disease. Indeed, animals that show a robust enhancement of sleep after microbial challenge are more likely to survive than are animals that show little or no enhanced sleep (Toth et al., 1993). However, sleep during microbial infections may also engender some disadvantages for the host. Prolonged somnolence is likely to reduce opportunities for food and water consumption, thereby exacerbating energy deficits that accrue due to fever and anorexia. In addition, excessive sleep or sleepiness is associated with reduced arousal and inattention to the environment, which could have detrimental consequences if prolonged unnecessarily. Thus, an appropriate balance between sleep and arousal during the infectious state would perhaps achieve the optimal survival advantage.

In studies of rabbits, the alterations in sleep that develop over the course of an infectious disease are temporally bimodal, with an initial phase of increased sleep and a subsequent period in which both the amount and the depth of sleep are reduced (Toth & Krueger, 1988; Toth & Krueger, 1989; Toth & Krueger, 1990; Toth et al., 1994). These alterations in sleep can be dissociated from fever, vary depending on the specific challenge organism and the route of inoculation, and are modified by changes in host immune competence or in the environment (Toth, 1999). The

phase of reduced sleep is not dependent on a prior phase of sleep enhancement (Toth et al., 1995a) and therefore appears to be actively induced, rather than a simple rebound effect related to a previous period of hypersomnolence. Consistent with this idea, studies with mice indicate that infection-related sleep suppression and enhancement are controlled by different genetic mechanisms (Toth & Williams, 1999). Thus, microbial infection seems to induce a balance between or temporal progression from sleep-promoting to sleep-reducing mechanisms.

Like patterns of sleep, immune and inflammatory responses during infectious disease are also subject to both potentiating and suppressive influences. Indeed, many of the same substances that regulate the immune response also influence sleep in consistent ways. Thus, pro-inflammatory cytokines such as interleukin 1, tumor necrosis factor, and interferon not only promote the immune response but also increase slow-wave sleep. In contrast, immunosuppressive and anti-inflammatory substances like corticotropin-releasing hormone, adrenocorticotrophic hormone, glucocorticoids, interleukin 10, and melanocyte stimulating hormone tend to limit inflammation and the immune response and also promote arousal. Biphasic patterns of sleep during infectious disease are likely to reflect the progression from an initial immune-enhancing physiologic response to the microbial challenge into an eventual homeostatic state in which the immune response is regulated or curtailed. A temporal progression from increased somnolence to reduced or fragmented sleep during infectious disease suggests that the milieu of sleep-modulatory substances changes as the disease progresses, with somnogenic stimuli becoming gradually supplanted by substances that suppress sleep.

The HPA axis contributes to the regulation of the immune response and could potentially contribute to the generation of biphasic alterations in sleep. Sleep, the HPA axis, and the immune response are highly interrelated. Glucocorticoids are predominantly considered to be anti-inflammatory and in general dampen immune responsiveness, particularly when present in high concentrations; however, low or basal concentrations may facilitate some facets of the host response, particularly early in the course of the response (Dhabhar & McEwen, 2000; Sapolsky et al., 2000; Van Reeth et al., 2000). Infectious or inflammatory challenges can cause immune activation that precedes HPA activation and eventually acts to potentiate or trigger subsequent glucocorticoid release (Sapolsky et al., 2000). Glucocorticoids inhibit the synthesis, release, and actions of many mediators of inflammation and the immune response, and also influence normal sleep architecture (Van Reeth et al., 2000). Sleep onset is associated with the short-term inhibition of cortisol secretion, whereas awakening is associated with a surge of secretion (Van Cauter, 2000). In addition, cortisol secretion is inversely related to EEG slow-wave amplitude

(an index of the depth of slow-wave sleep) during sleep and is temporally coupled to EEG activity (a measure of central alertness) during waking (Van Reeth et al., 2000). Human subjects who are deprived of sleep lose the modulatory impact of sleep-wake transitions on cortisol regulation, resulting in a blunted amplitude of its daily rhythm, higher basal levels during quiescent periods, and elevated evening levels (Van Cauter, 2000; Van Reeth et al., 2000). Thus, increased sleep may curtail HPA activation, thereby limiting circulating concentrations of glucocorticoids and facilitating greater immune responsiveness during the early phases of the infection. Conversely, arousal or reduced sleep could promote the availability of glucocorticoids to limit inflammation later in the course of the response, thereby protecting the organism from the deleterious effects of an unconstrained inflammatory response.

A role for endogenous glucocorticoids in influencing sleep during microbial disease, either directly via the central nervous system or indirectly via immunosuppressive and anti-inflammatory actions, is supported by numerous observations that corticotropin releasing hormone and glucocorticoids, as well as numerous other endogenous and exogenous immune-modulating substances, markedly influence sleep patterns in normal animals (Opp, 1995; Van Reeth et al., 2000). In healthy rabbits, for example, the administration of an immunosuppressive dose of cortisone produces reductions in both the time spent in slow-wave sleep and in delta-wave amplitude during slow-wave sleep (Toth, 1995a; Toth et al., 1992). Glucocorticoids also produce consistent alterations in sleep in animals undergoing microbial infection. In rabbits that are infected with *Escherichia coli* or *Candida albicans*, administration of cortisone attenuates both the infection-induced increase and decrease in sleep, as well as the severity of some clinical signs, yet the infection persists (Toth, 1995a; Toth et al., 1992). Strains of mice that show robust glucocorticoid responsiveness in stress paradigms also tend to show patterns of suppressed sleep during influenza infections, whereas strains that are less stress-responsive show patterns of sleep enhancement (Eleftheriou & Bailey, 1972; Moynihan et al., 1994; Shanks et al., 1994; Toth et al., 1995b). Genetic analysis indicates that these two patterns of altered somnolence are likely to be mediated by different portions of the genome (Toth & Williams, 1999). An initial mapping study revealed that the "suppressed sleep" phenotype shows linkage to a quantitative trait locus (QTL) (*Sr1p1*, sleep response to influenza, light phase) that incorporates the gene *chrh-2* (corticotropin-releasing hormone receptor 2) (Toth & Williams, 1999). However, clearly numerous other genes are also located in the confidence interval that defines *Sr1p1*, and considerable work remains before the gene underlying this QTL is identified.

In general, studies of the impact of sleep or lack of sleep on various immunologic parameters have produced divergent conclusions (reviewed in Benca & Quintans, 1997; Everson, 1997). In some reports, sleep deprivation appears to enhance immune responses (e.g., Bergman et al., 1996; Dinges et al., 1994; Renegar et al., 1998), whereas in others, it appears to suppress immune function (e.g., Brown et al., 1989; Everson, 1993; Irwin et al., 1994) or to have little effect (e.g., Toth et al., 1995a; Toth & Rehg, 1998). A number of issues complicate evaluation of the relationship between sleep, stress and host defense (Benca &

Quintans, 1997; Horne, 1978; Toth, 1995b). One important consideration is distinguishing between stress-related effects and effects attributable to sleep or lack of sleep per se. Many stressors adversely influence immune competence and the response to microbial challenge in humans and animals (Sapolsky et al., 2000), and stress is also known to influence sleep (Hall et al., 1998; Van Reeth et al., 2000). Studies of sleep and sleep deprivation are generally designed to minimize non-specific stress and may include the measurement of physiologic parameters that are thought to reflect the degree of stress. However, many human situations that are commonly associated with either poor quality or inadequate amounts of sleep are also associated with stress (e.g., preparation for examinations, shift work, depression, bereavement). Thus, from one perspective, the theoretical value of making stringent distinctions between stress- and sleep-related effects on immune competence may exceed the practical utility of such an effort. Another consideration relevant to the evaluation of sleep- and stress-induced alterations in immune competence is that even statistically significant changes in immune indices can nonetheless have little or no impact on the host in terms of likely biological, clinical, or functional significance (Benca & Quintans, 1997; Brown et al., 1992; Horne, 1978; Toth, 1995b). Relatively few studies to date have evaluated the impact of sleep or sleep deprivation on the immune responses of animals that are actually undergoing microbial infections. The type of infectious challenge and the selected outcome measures can markedly influence conclusions regarding the immune-modulatory effects of either stress or sleep (e.g., Toth & Rehg, 1998). Consideration of these issues is important to the interpretation of data concerning the relationships between stress, sleep, and the immune response.

In summary, microbial challenge elicits physiological responses that both promote and limit the generation of a host immune response. Pro-inflammatory factors are likely to also elicit sleep potentiation, whereas activation of the HPA axis and other immune-modulatory networks tend to promote arousal. An appropriate balance of immune responsiveness, HPA activation, and sleep propensity is likely to promote the ideal clinical outcome of elimination of the pathogen, recuperation, and survival.

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The Student BITS segment is an ongoing forum for issues pertaining to sleep and to the training and retention of sleep scientists, from a trainee's perspective. Undergraduate, graduate, or postdoctoral trainees interested in submitting an article for Student BITS are invited to contact Jennifer L. Martin, Assistant Editor, SRS Bulletin, Student BITS at jemartin@UCSD.edu.

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Trainee Day at APSS

As the incoming trainee member-at-large, I wanted to take this opportunity to discuss the highlights of the Trainee Day event at the 2002 APSS meeting in Seattle. I hope this will inspire trainees to attend future SRS sponsored training events and will educate others in the SRS about the importance and utility of this day dedicated to trainee-related activities.

While composing this synopsis, I began to reflect on my own experiences at Trainee Day over the past several years. I no longer recall the first Trainee Day I attended (is this a sign of aging?); however, I do recall the aspects of Trainee Day that have been helpful to me as a student in the field of sleep research. Some of what has been helpful to me is related to career development. A few years ago, I attended a workshop by Dale Edgar, PhD and Michael Perlis, PhD on writing a CV - an important skill often neglected in graduate training. Each student brought his or her CV to the workshop for review and feedback to improve this "marketing tool."

What I recall as most helpful, however, are the very personal and friendly interactions I have had with senior faculty in the field of sleep research. In fact, this year, forty percent of the student evaluations from Trainee Day indicated that networking with faculty was the single most helpful aspect of the day. A few years ago, I attended a "meet the professor lunch" with Richard Bootzin, Ph.D. and Mary Carskadon, PhD on "how to teach a sleep course." Following the lunch, Dr. Carskadon and I had a wonderful discussion about the topic. At that time I could not have predicted that I would spend the past year as a clinical psychology intern at Brown University in 2001-2002, and would contact Dr. Carskadon to discuss spending my allocated research time in her

lab. This is but one example of the positive interactions I have had with faculty at Trainee Day.

Finally, I would like to highlight the interactions I have had with fellow trainees at Trainee Day events. In many ways, it was these interactions which inspired me to participate on the Trainee Day committee for the past two years, and to run for the trainee member-at-large position within the SRS. The peers and future colleagues I have met have inspired me to be involved in these activities and to represent the interests of trainees to the SRS Board.

In 2002 over 130 trainees participated in a one-day event focused on sleep research. The day began with the SRS program Chair for Trainees, Ron Szymusiak, PhD, highlighting the successes of Trainee Day events over the past several years. Specifically, Dr. Szymusiak noted that a considerable number of trainees go on to attend future SRS sponsored trainee events and to submit abstracts for presentation at the annual APSS meetings.

Following Dr. Szymusiak's remarks, outgoing trainee member-at-large Scott Doran, PhD welcomed the trainees and reviewed the schedule for the day. Trainees then attended workshops and meet-the-mentor lunches, and interacted with one another during breaks throughout the day. The remainder of the morning was spent in one-hour workshops led by invited faculty who generously donated their time to participate. A broad array of topics from genetic models of sleep and circadian rhythms to REM and post-traumatic stress disorder were presented.

Following the third workshop, a career fair was held in which faculty, programs, labs, clinics and companies were invited to advertise open positions. For trainees looking for positions, this was described as a helpful part of the day. The career fair led directly into the evening trainee reception, which was well attended by both students and faculty. During the reception, trainees had the opportunity to interact with faculty and fellow trainees. Ninety-one percent of Trainee Day participants rated the overall quality of the day as very good to excellent. On behalf of the Trainee Day committee and the participants in the 2002 Trainee Day, I would like to extend a special thank you to the faculty who participated in the workshops and meet-the-mentor lunches. These individuals are listed in the box below.

Ted Abel
Richard Allen
Gary Aston-Jones
John Caldwell
Mary Carskadon
Charles Czesler
William Dement
Marcos Frank
Robert Greene
Alfred Lewy

Mark Mahowald
Andrew Monjan
Charles Morin
Mark Opp
Allan Pack
Michael Perlis
Gina Poe
Dieter Riemann
Richard Ross
Thomas Roth

Stephen Sheldon
Peter Shiromani
Peter Soja
Robert Stickgold
Sigrid Veasey
Hans VanDongen
Masashi Yanagisawa
Phyllis Zee

High School Essay Contest Winners

The Sleep Research Society (SRS) 2002 Essay Awards Program received entries from across the country. Essays, limited to one thousand words in length, were accepted on any topic related to sleep. Specific topics included sleep and performance, sleep and society, the brain in sleep, dreams, and the functions of sleep.

2002 marks the eleventh year that the SRS has sponsored this competition. A panel of experts from the organization selected the five winning essays on the basis of content, scientific application and originality. In recognition of their accomplishments, winners were awarded \$250 each and a certificate of merit.

Waking Before the Sun

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Researchers have shown that most people in today's society are not receiving enough sleep, especially adolescents who need more sleep now than at any other time of their lives (NSF, 2000). The National Sleep Foundation (NSF) (2000) recommends adolescents receive 8.5 to 9.25 hours of sleep per night. Their growing bodies require sleep in order to develop, especially because of the brain maturation that takes place during sleep (Dahl, 1999). Many adolescents are not getting enough sleep, especially during the week (NSF, 2000). A study performed by Wolfson and Carskadon (1998) showed that students are continually decreasing in the amount of sleep they receive during a typical school night, from 7 hours, 42 minutes in 13 year olds to 7 hours, 4 minutes in 19 year olds. These averages are much less than the amount of sleep advised by the NSF and it reports (2000) that only 15% of adolescents sleep the minimum 8.5 hours on a school night, while 26% are sleeping less than 6.5 hours. Insufficient sleep in adolescents has serious detrimental effects on their mind and body (NSF, 2000). Early starting schools can be restricting the amount that students sleep and thereby impacting the mental and physical health of the students.

The lack of sufficient sleep in adolescents can have negative impacts on the mind, body, and mood of children. The lack of sleep can lead to the weakening of the immune system which makes a person more susceptible to diseases, viruses and bacteria (Myers, 2001). It may also lead to problems with cognitive performance such as an increase in time it takes to respond to a stimulus and a decrease in accuracy (Mikulincer et al., 1989). A reduction in a person's average amount of sleep can also result in the difficulty to control emotions and increased behavioral problems (Holes et al., 1976). Signs of sleepiness including inability to focus and difficulty in "staying still" resemble behaviors common in adolescents with Attention Deficit Hyperactivity Disorder (Dahl, 1999). In addition, the NSF (2000) found that over half (55%) of all 'fall-asleep car crashes' are caused by people aged under 25.

Sleep not only has a relationship with behavior and mood but also

with academic performance. Wolfson and Carskadon (1998) found a significant positive correlation between grades and sleep among high school students. Students with higher grades (A to A-averages) slept for an average of seven hours and twenty-two minutes, while students with low grades (D to F averages) received only six hours and forty-eight minutes of sleep per night (Wolfson and Carskadon, 1998). Students who reported themselves as long sleepers, receiving nine or more hours of sleep per night, reported significantly higher grade point averages than those of short sleepers, who receive less than six hours of sleep per night (Kelly et al., 2001).

Differences in circadian rhythms may contribute to varying degrees of academic performance among students. Thinking is sharpest and memory most accurate when people are at their daily peaks in circadian arousal. (Myers, 2001) Some individuals experience their 'peak activation time' in the morning, while others have it during the afternoon or evening. Students that are most alert in the morning have a significantly higher grade point average than students reporting maximum alertness at other periods of the day (Biggers, 1980). Beaulieu (1991) also found that students reporting early morning peak activation had higher GPA's than those who were more alert in the afternoon or evening. However, there are a significantly smaller number of morning alert students than those who are more alert in the afternoon or evening (Beaulieu, 1991). Secondary school primarily takes place during the early morning, a time when some of the students may not be at their most alert. Students, who are more alert in the evening naturally go to sleep and wake up later. When these evening alert people are forced to wake up earlier than normal, they have difficulty waking up and concentrating (Gofer et al., 1999).

School starting times prohibit adolescents from waking up at later times and therefore receiving more sleep. Recently, chronobiologists have found that adolescence causes a change to the circadian rhythm, pushing back the onset of sleepiness to a later hour of the night (Lindsley, 1996). According to this change, the amount of time that a young person sleeps is directly related to what time they need to wake up. Students, age 10 to 14 years old, have been shown to sleep more on weekends and vacations as a result of waking up later (Szymczak et al., 1993). Also, high school students who start school at 7:30 AM or earlier obtain less total sleep on school nights due to earlier wakeup times (Allen, 1991).

Starting secondary schools later is an option that administrators can consider when taking into consideration their students' sleep patterns. The solution to adolescent sleep is easy if schools were able to just push back their start times indefinitely. There are many other factors that school districts must contend with when considering shifting their high school's schedule to later in the day. If schedules change, there may be a need for more buses to transport people to different places, which would be accompanied by an increase in the amount of money that is needed for transportation and the traffic conditions during the different times of bus routes may impact the number of buses needed. Many extracurricular activities and athletic activities will be pushed back as well. Some sports require light outside and ending school later may inhibit the ability of schools to participate in certain outdoor activities. Many schools provide their facilities to community activities, such as

adult education, which would need to change its schedule as a result of the high school changes. These are only a few of the factors schools need to consider when wanting to change school starting times, however districts should begin to reconsider having their oldest students going to school the earliest.

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The Causes and Effects of Sleep Deprivation in Adolescents

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Waking up early, going to school, doing all sorts of after school activities, then going home after a long day to do homework until late into the night; this is the typical day for many young adults.

The teenagers of today get far less sleep than what is recommended. This sleep deprivation can have dramatic effects.

Teenagers are driven to sleep longer than adults, not because of the stereotypical lazy teenager image, but because of biological forces. Such biological forces include changes in sleep patterns. One of these biological forces is the secretion of the sleep related hormone melatonin. If this hormone is secreted at a different time than it normally is, it can change the circadian rhythms that guide a sleep-wake cycle. This is one reason why adolescents take much more time to "wind down" than adults. At 7:30, a teenager is still fully awake and alert, whereas an adult is beginning to feel sleepier. By 10 o'clock the adult is ready to go to bed, but the teenager may not be ("Adolescent Sleep").

Sleep deprivation effects show up in school. Recent research has revealed an association between sleep deprivation and poorer grades. Many studies support this thought. One survey in 1998 was done on more than 3,000 high school students by Amy R. Wolfson, PhD of the College of the Holy Cross and Mary A. Carskadon, PhD, of Brown University Medical Center. They found that students with C's, D's, and F's, were getting 25 minutes less sleep and going to bed 40 minutes later than students getting A's and B's. These students also have more disciplinary problems, sleepiness in class, and poor concentration (Carpenter, 2001).

Sleep Deprivation can also lead to conditions that can be harmful to one's body, mind, and even society. Teenagers are put at risk for cognitive and emotional difficulties when experiencing a lack of sleep (Carpenter, 2001). Things that can be negatively affected by lack of sleep include mood, motivation, awareness, coordination, and even memory (Caldwell, 1997). Society can be affected with such things as traffic accidents. For example, the National Highway Traffic Safety Administration says that fatigue and drowsiness cause more than 100,000 traffic accidents a year and that young drivers are driving in more than half of these crashes (Carpenter, 2001).

Teenagers often do not realize that they need to be getting more sleep. In many ways, culture teaches people that younger children need more sleep than adolescents. However, recent studies have shown that young adults need more sleep than adults or children. The changes that adolescents go through require them to have about nine hours of sleep a night (Maas, 1998). However, a recent survey done by Mary Carskadon and Amy Wolfson of 3,120 high school students found the average hours of sleep per night to be 7 hours and 20 minutes (Corm, 1996). Similarly, in a 1998 study that these two women conducted, they found that 26% of high school students routinely sleep less than 6.5 hours on school nights, and only 15% sleep 8.5 hours or more (Carpenter, 2001).

Another reason that such sleep deprivation occurs is the large differences between the weekend and school night sleeping schedules in many teenagers. Surveys have shown that these irregular sleeping habits have been related to academic difficulty, daytime sleepiness, depression, and sleep/wake behavior problems. This survey has also related sleep schedules to the occurrence of sick days away from school in girls and the occurrence of injuries requiring treatment in boys. Other surveys have related irregular sleep/wake schedules with high levels of daytime sleepiness

(Acebo).

There is also the issue of how sleep deprivation affects adolescents' emotions and behaviors. Many sleep-deprived teenagers are more susceptible to psychopathologies such as depression and ADHD. These teenagers have also shown difficulty in controlling their emotions and impulses. The lack of sleep leads to repeated fatigue and sleepiness, unstable emotions, poor decision-making, and risky behavior (Carpenter, 2001).

Sleep deprivation in adolescents is a major problem that needs to be dealt with. There are many things that teenagers can do to somewhat stifle the growing trend toward sleep deprivation. First of all, they can stay away from caffeine and nicotine, both of which are stimulants, after noon. Second, they can avoid heavy studying or computer games before bed. These can be arousing. Third, they can avoid bright light in the evening, but open blinds or turn on lights as soon as the morning alarm goes off to help with waking up. Lastly, they can avoid sleeping more than 2 or 3 hours past their normal awakening time in order to not disrupt their body clock ("Adolescent Sleep").

Sleep deprivation in teenagers is very prevalent in society. This phenomenon has many biological as well as environmental causes. The effects of this sleep deprivation can be very dramatic ranging from a drop in grades to a fatal automobile accident. Sleep deprivation in adolescents is a problem that needs attention.

Lucid Dreaming

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"I realized I was dreaming. I raised my arms and began to rise (actually, I was being lifted). I rose through black sky that b/ended to indigo, to deep purple; to lavender, to white, then to very bright light. All the time I was being lifted there was the most beautiful music I have ever heard. It seemed like voices rather than instruments There are no words to describe the JOY I felt. I was very gently lowered back to earth, I had the feeling that I had come to a turning point in my life and I had chosen the right path, The dream, the joy I experienced, was kind of a reward, or so I felt. It was a long, slow slide back to wakefulness with the music echoing in my ears. The euphoria lasted several days; the memory, forever (LaBerge, 3)."

Lucid dreaming is defined simply as a state where one is consciously aware of being in a dream. Yet this straightforward definition gives no indication of the complexity and elaborateness of the term and the theory behind it. As demonstrated by the above quote, lucid dreaming is an exciting adventure for the elite few who have a natural penchant towards it. For the dreamer in the quote, lucid dreaming gave rise to a feeling of utmost euphoria and joy. This dreamer felt spiritually lifted, like the burdens of everyday life had been cast aside. Many lucid dreamers experience similar responses, however, lucidity can also be associated with feelings of power, wholeness, death, or awakening. Some of

the characteristics often used to describe lucid dreams are heightened quality and clarity of visual images, and an overwhelming sense of reality. In fact, the majority of people who experience this phenomenon find it difficult to distinguish between their dream and waking reality (Godwin, 10). Many a lucid dreamer has described his or her own lucid dreams as "real, true, ecstatic, blissful, revealing, wondrous, full of gratitude, magical and divine"(Godwin, 80).

One biological approach to the explanation of lucid dreaming is a theory that involves the immediate awakening of specific neurons, called aminergic neurons, located in the brain stem. Aminergic neurons usually relate to learning, but can also relate to critical attention tasks. These neurons generally decrease their activity during the normal sleep cycle (causing the dreamer not to question the strange events taking place in their dreams, assuming that they are part of reality, and not, in fact, part of a dream). However, in lucid dreamers, these neurons often spontaneously activate and "awaken" during the sleep cycle. The sudden "awakening" of these neurons would explain the dreamer's abrupt realization that what they are experiencing is actually a dream, and their ability to instantly be attentive to every last detail of their once blurry, unfocused dream (Godwin, 73).

In 1991, Lynne Levitan and Stephan LaBerge performed and analyzed a laboratory study containing 107 lucid dreams from 14 people. The 14 subjects were each taught a specific series of eye movements to signify that they were aware that they were dreaming. This technique was used for the reason that lucid dreams occur during REM (rapid eye movement) sleep, and eye motion: would be monitored closely. To perform the experiment, the subject's brain waves, chin muscle activity and eye movements were closely observed. A measurement was taken of the time that passed between the subject falling into sleep and the occurrence of the specific pattern of eye movements recognized clearly by a person not involved in the experiment. This experiment gave concrete evidence that people could be aware of the fact that they were dreaming, and that lucidity really does exist (The Lucidity Institute).

Another dreamer notes:

"I became lucid, while being chased by a tiger, and wanted to flee. I then pulled myself back together, stood my ground, and asked, 'Who are you?' The tiger was taken aback but transformed into my father and answered, 'I am you father and will now tell you what you are to do'... I rejected his threats and insults ... (LaBerge, 194)"

This quote explains, without question, the reason behind the fixation with lucid dreaming. From a scientist's point of view, lucidity is another fascinating, missing piece of the elaborate puzzle that is the human brain. By answering the questions brought up by lucid dreaming, such as, "What happens in the brain that makes a dreamer suddenly lucid?" scientists will come closer to a better understanding of the complex human brain. From a dreamer's point of view, lucidity, for so many people, is a way to face one's fears or to satisfy a yearning for something unattainable in the real world. For instance, in the above quote, it is apparent that the dreamer has trouble standing up to authority figures, in this case

his father, but finally in this dream; he is able to do so, and thus gains satisfaction and confidence in himself. Due to the solutions that lucid dreaming seems to provide, people are drawn to its possibilities and are even willing to spend years of their lives dedicated to learning the techniques behind finding these solutions.

According to Antonio et al. (1992) lucid dreaming can be influenced/induced by scientists. In an experiment performed in 1983, results showed that not only did the technique work, but the induced lucid dreams from normally non-lucid dreamers were more vivid and more positive than the induced dreams of the normally lucid dreamers. This finding gives inspiration to all of the people in the world, searching for a way to use lucid dreaming as a way to solve their problems such as jealousy anger, feelings of worthlessness, uncontrollable fears, etc.

In conclusion, lucid dreaming, although current scientific explanations for it are unstable at best, seems to provide many people with a solution to many different troubles. Many people follow lucid dreaming research for the simple reason that lucid dreaming provides an easy way to conquer varying problems that can seem overwhelming.

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Familial Advanced Sleep Phase Syndrome: The Case of the Morning Larks

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Morning larks vs. night owls. Humans run on internal biological clocks that allow their bodies to keep track of day and night. These rhythms, known as circadian rhythms, cycle every 24 hours and help control endocrine activity, metabolic function, and body temperature. However, the most crucial function of circadian rhythms is its role in the human sleep-wake cycle. Regulated by a central circadian clock in the superchiasmatic nucleus of the hypothalamus, sleep propensity is controlled through melatonin secretions in the body (Reid et al., 2001; Jones et al., 1999).

Without it, there would be chaos.

A number of sleeping disorders have originated from alterations in the circadian sleep-wake cycle; among these are advanced sleep phase syndrome (ASPS) and delayed sleep phase syndrome (DSPS). Advanced sleep phase syndrome is characterized by an onset of early evening sleep followed by an offset of sleep by early morning awakening, the "morning larks." Delayed sleep phase syndrome is just the opposite, where patients experience an onset of extremely late evening sleep also followed by a late morning awakening, the "night owls" (Reid et al., 2001).

Those affected with advanced sleep phase syndrome and labeled as "morning larks," function with a 4-hour advance in sleep, body temperature, and melatonin secretions. While some individuals can cope with the advancements in sleep-wake cycles, others find it a disabling disorder. Individuals with FASPS generally experience excessive sleepiness around 7:30 pm, the peak time when people are socializing. This is followed by a normal duration of sleep and a spontaneous awakening around 4:30am, the time when regular individuals are in their heaviest sleep time of the 24-hour period. Thus, patients are unable to take part in normal social schedules and activities throughout their lives (Toh et al., 2001).

Familial cases of ASPS (FASPS) have been discovered in recent years, suggesting that disorders associated with circadian rhythms are, in fact, hereditary and regulated by genes. Other sleep disorders such as delayed phase sleep syndrome, restless leg syndrome, and narcolepsy have also been associated with familial connections. A familial identification of a disorder provides scientists with the unique opportunity to identify the specific gene responsible for the alterations in human circadian rhythms through the studies of pedigree diagrams and genomic linkage analysis (Reid et al., 2001).

Three families have thus far been identified to carry the gene responsible for FASPS. With the consent of family members, scientists have conducted research utilizing physician interviews, the Home-Ostberg questionnaire, a tool used to evaluate patients of "morning lark" or "night owl" tendencies, and family histories of sleep. Dim light melatonin onsets (DLMO) were also collected to monitor the inner biological sleep cycles and affected individuals had a mean DLMO at least 3 1/2 hours earlier than that of normal individuals. Affected individuals varied from ages as early as eight years old to early adulthood. Using the data collected, scientists constructed a family pedigree, mapping the family of affected and unaffected individuals. The pattern of affected individuals showed at least one family member in each generation experiencing FASPS, following the genetic model of segregation that occurs from an autosomal dominant gene (Reid et al., 2001).

As identified by researchers Christopher Jones and Louis Ptacek of the University of Utah (Howard Hughes Medical Institute, Salt Lake City, UT), the autosomal dominant gene *hPer2* is theorized to be a circadian rhythm variant. Short and long *t* autosomal semi-dominant circadian rhythm mutants were discovered in fungi, plants, and animals after mutagenesis screening and spontaneous mutation recognition procedures. Long-period mutants were seen to be phase delayed while short-period mutants were phase-advanced. These abnormal circadian phenotypes were then genet-

ically analyzed to identify and characterize the genes responsible for circadian rhythms. In order to find the genetic basis of FASPS, Jones and Ptacek also performed linkage pedigree analysis studies in a large family carrying the mutated gene. They found that the human *Per2* gene is comprised of 23 exons, in which a mutation in the form of base pair deletions and point substitutions on specific sites of nucleotide base-pairs. This causes a deficiency in phosphorylation of *hPer2* which could degrade the circadian cycle and/or accelerate nuclear entry, causing phase-advance rhythm and sleep-wake rhythm as observed in FASPS (Toh et al., 2001).

The identification of genes responsible for circadian rhythms proves that studies on the human internal biological clock can be researched further in the future. Other families, which carry mutations on the FASPS allele, can provide information and opportunities to identify possible mutations on other genes that alter or affect human circadian rhythms. The knowledge and discoveries gained from these studies can help scientists more fully understand the workings of the human inner clock and create treatments to aid those affected with FASPS and other circadian sleep-wake cycle disorders. The familial identification of sleep disorders can play an important role in identifying genes responsible for the regulation of human sleep and circadian rhythms.

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Sleep Apnea

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Obstructive sleep apnea (OSA) is the cessation of breathing due to mechanical blockage of the airway. This occurs when the soft tissue in the back of the throat collapses and closes during sleep. It may occur hundreds of times during the night and each episode may last for a minute or even longer. Some symptoms of sleep apnea include restless sleep, loud, heavy snoring, loss of energy, headaches, trouble concentrating, forgetfulness, obesity, excessive daytime sleepiness, and significant social and emotional problems (Hargrett, 2001). Not all people with sleep apnea experience; all of these symptoms, and not everyone who has these

symptoms has sleep apnea. If it remains untreated, sleep apnea may cause high blood pressure and other cardiovascular diseases, memory problems, impotency, and even death. Sleep apnea may occur to anyone; it has been shown that it is more common in men (4%) than it is in women (2%). Also, it seems to run in the family, suggesting that it maybe genetic (Hargrett, 2001). Some risk factors include being male, overweight, and over forty years of age, but sleep apnea may occur to anybody. The first major step to reduction of sleep apnea is weight loss. Studies by Harman (1982) and Bearpark (1995) certainly support this idea.

Harman (1982) looked into the effect of weight loss on sleep-disordered breathing and oxygen desaturation in abnormally obese men. He observed four obese men who had significant sleep disordered breathing and oxygen desaturation after they had lost an average of 108 kg. All of the men showed a correlation between their weight loss and the significant reduction in the number of occurrences per hour of sleep disordered breathing events. Three of the men had improvement in the severity of desaturation accompanying the abnormal breathing. The two men with daytime drowsiness showed the most dramatic improvement in desaturation prior to weight loss. Harman's study suggests that obesity is a cause, not an effect, of the sleep apnea syndrome. (Harman, 1982).

In a study by Bearpark, 294 men aged 40 to 65 were monitored to measure snoring and sleep apnea. He observed that smokers snored for a greater percentage of the night than nonsmokers, and he concluded that alcohol consumption had no relation to the sleep disordered breathing. In this study, Bearpark concluded that sleep apnea is extremely common in middle-aged men, and in this age range, it is associated with obesity and not with age. (Bearpark, 1995).

Many other case studies support the belief that obesity has a factor in sleep apnea. Mortimore (1998) conducted a study on local fat deposition. He measured the general obesity, BMI, neck circumference, and percentage total body fat with neck fat deposition using an MRI in three matched subject groups. Nine non-obese, non-snoring control subjects, nine non-obese patients with OSA, and nine obese patients with OSA were matched to the other groups in terms of age. Percentage total body fat was 30 and 44% greater in non-obese and obese patients with OSA, respectively, than in the control subjects. Neck tissue volume was 10% greater in non-obese and 28% greater in obese patients with OSA than in control subjects. The percentage of neck tissue volume attributed to fat was 27% greater in non-obese and 67% greater in obese patients with OSA than in control subjects. There were no significant differences between non-obese patients with OSA and control subjects with respect to fat located in other areas of the neck; obese patients with OSA had 42% more fat than control subjects ($p < 0.05$). Mortimore concluded that even relatively non-obese patients with OSA have excess fat deposition, especially anterolateral to the upper airway when compared with control subjects with the same level of obesity assessed using BMI and NC. This excess fat probably contributes to their susceptibility to OSA. (Mortimore, 1998).

Finally, Rajala (1991) examined tests on twenty-seven extremely obese patients (13 men and 14 women) whose body mass index



LETTER

FROM THE SECTION HEAD OF SLEEP AND BEHAVIOR, SRS

Dear Sleep and Behavior Members,

It is with pleasure that I begin my term as Section Head of Sleep and Behavior. I appreciate your confidence in me and will do my best to represent and meet the needs of the membership. There are several things that I would like to accomplish for Sleep and Behavior over the next three years for which I need your input and help.

Foremost is to ensure that the research priorities and interests of the members of Sleep and Behavior are represented in the NIH Sleep Disorders Strategic Research Plan that is currently being updated by the National Center on Sleep Disorders Research (NCSDR) and the Sleep Disorders Research Advisory Board. This plan will have a big impact on federal funding priorities and program announcements and the direction of sleep research in the next 5 years. If you have not already read a draft this document, please do so. You will be able to obtain a copy from either the SRS website, AASM central office or NIH/ NCSDR websites shortly. I will be sending my comments to the working group and would be pleased to include yours as well.

Second, I strongly encourage you to consider submitting a proposal for an SRS-sponsored symposium or workshop on topics related to our diverse interests in sleep and behavior for inclusion at other scientific meetings. The Circadian Rhythms Section coordinated a workshop entitled "Sleep and Circadian Rhythms: Views to the Future" at the Society for Research on Biological Rhythms this past year. Not only do these programs increase awareness of SRS among other societies and attract students, trainees, and new members, but they also put the spotlight on our section.

Sleep and Behavior is the largest Section within SRS, with nearly 300 members. I welcome your suggestions and comments on how we can begin to establish our identity as a section and to take a more active role in highlighting our best work. Let's start with the annual APSS meeting, June 3-8, 2003 in Chicago. Let me know what you want to see included and what proposals you are planning to submit. Each of the Section Heads serves on the Program Committee; getting a "heads up" on a proposal is very helpful.

-----Roseanne Armitage

was greater than or equal to 40 kg m⁻². They ranged from 23-51 years old, and their BMI ranged from 40.0-62.9 kg. Eleven patients had an oxygen desaturation index (ODI) of 10 h⁻¹ (10 men and one woman). They had excessive daytime sleepiness, arterial hypertension, and other symptoms of OSA. Nine patients received antihypertensive treatment, six of which had OSA. In three patients with OSA, surgery was performed and a 30-38% reduction in BMI was achieved. Eight patients with OSA were treated with an intensified dietary regimen, and the BMI reduction ranged from 2.6 to 33%. OSA was either cured or significantly improved in six (55%) patients, with a mean reduction in BMI of 27%, while in patients with persistent OSA the mean reduction in BMI was only 7%. (Rajala, 1991).

There are still many studies and much research being done today to try to reduce sleep apnea even more. Many studies have shown a correlation between weight loss and sleep apnea. Harmen showed that obesity is a cause, not an effect of OSA; Rajala gathered that reduction in BMI significantly improves, and maybe even cures OSA. Bearpark's study showed that OSA is not caused by age, but by obesity. Finally; Mortimore's studies indicated that excess fat contributes to a person's susceptibility to OSA. Although other treatments have been utilized, as can be seen in the studies mentioned above, there is a high correlation between obesity and OSA. Weight loss is a behavioral modification that could go a long way in at least to begin the end to this problem. Therefore, weight loss should be the first course of treatment for reduction of severity of OSA. Many more years of research and breakthrough are to come, and hopefully someday, sleep apnea will be eliminated forever.

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