Welcome from the CSS President
Helen Driver, PhD, RPSGT, DABSM

On behalf of the CSS executive, the scientific and local organising committees, it is with great pleasure that we welcome you Toronto and to the Canadian Sleep Society's 4th conference "Waking Up to Sleep Disorders".

Soon after our 2007 meeting in Montreal, Shelly Weiss started looking for a suitable venue for this meeting. We were joined for a few interesting hotel tours by Kimberly Cote, James MacFarlane, Carol Mously, and Michael Fitzpatrick to find a venue that would accommodate our conference size and range of requirements. We trust that you'll be comfortable during our sojourn in this newly renovated and centrally located space.

With tremendous dedication from the organized and efficient Kimberly Cote, in collaboration with Shelly Weiss, James MacFarlane and Eileen Sloan, technologist representatives Carol Mously and Natalie Morin, and student representative Stuart Fogel, as well as the various committee members and reviewers, we have developed a diverse program to meet the needs of students, technologists, clinicians and researchers. We are very honoured to have seven top-notch keynote speakers, from abroad and locally, who are giving freely of their expertise and time to form such an integral component of our program. Given the high calibre of these speakers and the over-reaching content of their presentations, there will be no concurrent sessions during the keynote addresses. The symposium submissions were varied and of excellent quality, they were selected with care to minimize content overlap and to meet requirements for CME credits and our budget! In response to feedback from technologists, this year we have introduced technical workshops that will run concurrently with the scientific symposia. We recognize the valuable expertise and contributions from our exhibitors and you will notice that we have explored new ways of extending their participation in the program.

In addition to the annual student and technologist abstract prizes, this is the second year of the four CSS and Institute for Circulatory and Respiratory Health (ICRH) Student Travel Awards, and we have three other awards. (1) The Student Outstanding Achievement Award,

is a new prize, initiated by the student representatives, based on the scientific merit of a publication by a student in the field of sleep research; the first recipient of this award is Patty Brooks, University of Toronto. (2) The Broughton Young Investigator Award goes to Dr. Jean-François Gagnon, Université de Montréal. (3) The Distinguished Scientist Award, in recognition of a scientist who has made significant contributions to the field of sleep research in Canada, goes to Dr. Carlyle Smith, Trent University.

We are grateful to Gilles Lavigne, who is receiving an honorary doctorate from the University of Zurich, for his guidance and encouragement. From the CSS office, F+A (Felicissimo, Rossie International in Montreal) - Jason Rossie, June Viau, Katherine Jolin and Sabrina Santucci have helped us organize and present this conference, we could not do this tremendous work without their professional help. A heartfelt thank you to our sponsors and exhibitors, especially in these times of financial constraint, for their contributions. A final thanks to all delegates for joining us in our ongoing learning, professional development, and social interactions.

Everything matters when you can't sleep.

Mot de la Présidente
Helen Driver, PhD, RPSGT, DABSM

Au nom de l'Exécutif de la SCS, du Comité scientifique et du Comité organisateur local, j'ai l'insigne plaisir de vous accueillir à Toronto à l'occasion du 4e Congrès de la Société canadienne du sommeil, dont le thème cette année sera "Vigilants aux troubles du sommeil".

Peu après notre Congrès de 2007 à Montréal, Shelly Weiss débutait ses recherches visant à trouver un lieu adéquat pour notre événement. Se sont joints au processus d'inspection des hôtels nos collègues Kimberly Cote, James MacFarlane, Carol Mously et Michael Fitzpat-rick, qui ont tous contribué au choix d'un établissement répondant aux besoins d'un événement de cette envergure. Nous souhaitons que l'espace que nous avons retenu soit conforme à toutes vos attentes.

Grâce au grand dévouement de Kimberly Cote, dont les talents d'organisatrice et l'efficacité nous laissent admiratifs, en collaboration avec Shelly Weiss, James MacFarlane, Eileen Sloan, les porte-parole des technologues Carol Mously et Natalie Morin, le porte-parole étudiant Stuart Fogel, ainsi que les nombreux comités et évaluateurs, nous avons relevé le défi d'élaborer un programme varié qui saura répondre aux besoins des étudiants, des technologues, des cliniciens et des chercheurs. Nous sommes honorés de la présence parmi nous de sept sommités dans notre domaine qui partagent à la fois leurs connaissances et leur temps en participant à notre programme scientifique. Eu égard à l'importance de ces conférenciers et du contenu de leurs interventions, nous ne présenterons pas de séances parallèles lors des conférences d'honneur. Par ailleurs, les propositions de symposiums retenues sont variées et de grande qualité, de manière à ne pas chevaucher des thématiques complémentaires et à satisfaire les critères d'ÉMC… et de notre budget. Pour répondre aux besoins exprimés par les technologues, nous offrirons cette année des ateliers techniques en parallèle aux symposiums scientifiques. Toujours reconnaissants à l'endroit des contributions de nos exposants, nous avons exploré de nouvelles façons de maximiser leur participation au programme.

En plus des prix annuels décernés aux étudiants et aux technologues pour le meilleur résumé scientifique, nous offrons pour la deuxième fois quatre bourses de voyage SCS/ISCR. Grâce à une initiative des porte-parole étudiants, nous accorderons pour la première fois cette année le Prix d'excellence pour étudiants, basé sur le mérite scientifique d'une publication par un étudiant dans le domaine de la recherche sur le sommeil. Nous félicitons Patty Brooks, de l'Université de Toronto, qui remporte cet honneur. Le prix Roger-Broughton pour jeune chercheur est attribué au Dr Jean-François Gagnon, de l'Université de Montréal, alors que le prix du Chercheur émérite, décerné au chercheur dont le travail a contribué de manière insigne au domaine de la recherche sur le sommeil au Canada, est présenté au Dr Carlyle Smith, de l'Université Trent.

Nous exprimons notre grande reconnaissance à Gilles Lavigne, récipiendaire d'un doctorat honoris causa de l'Université de Zurich, pour son encouragement et ses sages conseils. Nous remercions également le personnel du bureau de la SCS à Montréal, Felicissimo, Rossie et associés. En effet, Jason Rossie, June Viau, Katherine Jolin et Sabrina Santucci nous ont aidé à organiser et à concrétiser ce projet; sans eux, nous n'aurions pu abattre tout ce travail de planification. Un grand merci également à nos exposants et à nos commanditaires qui sont au rendez-vous malgré une conjoncture économique difficile. Enfin, merci à tous les participants de se joindre à nous dans notre grand projet d'éducation, de croissance professionnelle et d'interactivité sociétale.

Welcome from the CSS Vice-President (Research)
Kimberly Cote, MSc, PhD

The Scientific Program Committee has organized an exciting program for the 4th Congress of the Canadian Sleep Society in Toronto. The program includes 7 international and Canadian keynotes speakers, 11 scientific symposia, and 4 technical workshops. The conference gets started on Sunday with ½-day programs for students and technologists, as well as applied symposia. In addition, we have over 80 poster presentations in the York A/B room which will be on display Monday and Tuesday. These presentations cover a wide range of topics from basic neurophysiology to clinical sleep disorders. Other events scheduled throughout the program include workshops on CPAP mask fitting, and CPAP compliance, as well as discussion sessions on the BRPT eligibility requirements for Canadians, and new scoring rules for Francophone technologists. Industry exhibitors will be available in the Trinity Ballroom to answer your question on their latest products and services.

Be sure not to miss opening ceremonies on Monday at 8:30 A.M. in the Grand Ballroom where we'll present a number of awards including the Distinguished Scientist Award, the Roger Broughton Young Investigator Award, the new CSS Student Outstanding Achievement Award, the CSS Abstract Prizes to students and technologists, and the Institute for Circulatory and Respiratory Health / Canadian Sleep Society joint Travel Awards.

Don't miss the social events! A 'Sleep Professionals Social' Sponsored by ResMed & Medigas is taking place Sunday night at the Hard Rock Café. The 'Salsa Party' on Monday night is not to be missed! Join us for a night of dancing and sizzling Latin music with Toronto's own Cassava Latin Rhythms.

Welcome and Enjoy!

Mot de la vice-presidente (recherche)

Kimberly Cote, MSc, PhD

Le Comité scientifique a conçu une programmation stimulante dans le cadre du 4e Congrès de la Société canadienne du sommeil à Toronto. Le programme se distingue par les sept conférenciers d'honneur canadiens et internationaux, les onze symposiums scientifiques et les quatre ateliers techniques. Le Congrès débute le dimanche par des séances d'une demi-journée pour étudiants et technologues, ainsi que des symposiums appliqués. En outre, plus de 80 présentations par affiches seront exposées au Salon York AB le lundi et le mardi. Ces présentations traitent d'une vaste gamme de sujets, depuis la neurophysiologie fondamentale aux troubles cliniques du sommeil. Parmi les autres activités prévues, citons des ateliers sur l'ajustement du masque CPAP et la conformité CPAP, ainsi que des séances de tribune libre sur les critères d'admissibilité à la BRPT pour les canadiens, ainsi que les nouveaux règlements sur la notation pour technologues francophones. Les exposants seront heureux de vous accueillir au Salon Trinity afin de répondre à vos questions relatives aux nouveaux produits et services.

Ne manquez pas la cérémonie d'ouverture le lundi matin, 8h30, au Grand Salon, lors de laquelle seront décernés plusieurs prix, dont celui du Chercheur émérite, le prix Roger-Broughton pour jeune chercheur, le nouveau prix d'excellence SCS pour étudiants, les prix SCS pour les meilleurs résumés scientifiques soumis par des étudiants et des technologues, ainsi que les bourses de voyage offertes conjointement par l'Institut de la santé circulatoire et respiratoire et la Société canadienne du sommeil.

Enfin, n'oubliez pas les activités sociales. La soirée pour professionnels du sommeil, offerte par ResMed & Medigas, aura lieu le dimanche soir au Hard Rock Café. La soirée salsa du lundi soir est à inscrire dès maintenant à vos agendas, avec en perspective des rythmes latinos enveloppants et toute une soirée de danse en compagnie du groupe torontois Cassava Latin Rhythms.

Au plaisir de vous souhaiter à tous bienvenue et bon congrès.

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The Clock
By Gail Dendy

The old clock's winding down:
its parts jammed with dust and
grease,
the works an intricate metallic lace,
slowing
the years, the days, the hours.
With a trilling whirr, it breaks:
one lifetime stirring
as feather-ready as birds in flight

clapping their way across the sky,
collecting up hours and minutes and
seconds

then tipping them over, just for fun

Committees

CSS 2009 Scientific Program Committee

CHAIR:

Kimberly Cote, PhD   CSS Vice-President (Research), Brock University

MEMBERS:

Helen Driver, PhD   CSS President, Queen's University, Kingston General Hospital
Gilles Lavigne, PhD   CSS Past-President, Université de Montréal
Shelly Weiss, MD   CSS Vice-President (Clinical), University of Toronto
Charles Samuels, MD   CSS Member-at-large (Membership), University of Calgary
Penny Corkum, PhD   Dalhousie University
Marie Dumont, PhD   Université de Montréal
Richard Horner, PhD   University of Toronto
Charles Morin, PhD   Université Laval

CSS 2009 Organizing Committee

CHAIR:

Shelly Weiss, MD   CSS Vice-President (Clinical), University of Toronto

MEMBERS:

Kimberly Cote, PhD   CSS Vice-President (Research), Brock University
Helen Driver, PhD   CSS President, Queen's University, Kingston General Hospital
Gilles Lavigne, PhD   CSS Past-President, Université de Montréal
James MacFarlane, PhD   CSS Member-at-large (Newsletter), University of Toronto
Eileen Sloan, PhD, MD   CSS Secretary-treasurer, University of Toronto
Brian Murray, MD   University of Toronto
John Peever, PhD   University of Toronto

TECHNOLOGIST:

Carol Mously, BSc, RPSGT CSS Member-at-large (Technologist), Toronto Sleep Institute

STUDENTS:

Stuart Fogel, MA   CSS Member-at-large (Student), Queen's University
Patti Brooks, BSc   University of Toronto

CSS 2009 Technologist Committee

CHAIRS:

Carol Mously, BSc, RPSGT	CSS Member-at-large (Technologist), Toronto Sleep Institute
Natalie Morin, BSc, RPSGT	CSS Member-at-large (Technologist), Sleep Strategies

MEMBERS:

Helen Driver, PhD, RPSGT	CSS President, Queen's University, Kingston General Hospital
Kimberly Cote, PhD	CSS Vice-President (Research), Brock University
Lori Davis, RPSGT	West Park Healthcare Centre
Roger Goodbout, PhD, RPSGT	BRPT board, Université de Montréal
Kenan Hoelke, RPSGT	Royal Victoria Hospital, Barrie

CSS 2009 Student Committee

CHAIR:

Stuart Fogel, BSc, MA	CSS Member-at-large (Student), Queen's University

MEMBERS:

Kimberly Cote, PhD	CSS Vice-President (Research), Brock University
Helen Driver, PhD	CSS President, Queen's University, Kingston General Hospital
Patti Brooks, BSc	University of Toronto
Christian Burgess, BSc	University of Toronto
Catherine Milner, MA	Brock University
Jennifer Vriend, BSc	Dalhousie University

CSS 2009 CME Committee

This event has been held-over to April 2010 for a one-day CME in association with the Continuing Education and Professional Development Office, University of Toronto.

CHAIR:

James MacFarlane, PhD	CSS Member-at-large (Newsletter), University of Toronto

MEMBERS:

Shelly Weiss, MD	CSS Vice-President (Clinical), University of Toronto
Adam Blackman, MD	University of Toronto, Toronto Sleep Institute
Mohammed Hussain, MBBCh	Tri-Hospital Sleep Laboratory, Ontario
Elliot Lee, MD	University of Ottawa
Christopher Li, MD	University of Toronto
Len Makerewich, MD	Niagara Snoring & Sleep Centre

CSS 2009 Public Lectures

Eileen Sloan, PhD, MD	CSS Secretary-treasurer, University of Toronto
Adam Blackman, MD	Toronto Sleep Institute

CSS 2009 Fund Raising Committee

CHAIR:

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Reviewers

We would like to thank those individuals who gave their time to review and adjudicate abstracts and awards.

General Abstracts and Student and Technologist Abstract Prize Reviewers

Kimberly Cote, PhD	CSS Vice-President (Research), Brock University
Helen Driver, PhD	CSS President, Queen's University, Kingston General Hospital
Gilles Lavigne, PhD	CSS Past-President, Université de Montréal
Shelly Weiss, MD	CSS Vice-President (Clinical), University of Toronto
Charles Samuels, MD	CSS Member-at-large (Membership), University of Calgary
Penny Corkum, PhD	Dalhousie University
Marie Dumont, PhD	Université de Montréal
Richard Horner, PhD	University of Toronto
Charles Morin, PhD	Université Laval

Roger Broughton Young Investigator Award Judges

Kimberly Cote, PhD	CSS Vice-President (Research), Brock University
Helen Driver, PhD	CSS President, Queen's University, Kingston General Hospital
Gilles Lavigne, PhD	CSS Past-President, Université de Montréal

Canadian Sleep Society (CSS) / Institute for Circulatory and Respiratory Health (ICRH) Student Travel Award Judges

Penny Corkum, PhD	Dalhousie University
Marie Dumont, PhD	Université de Montréal
Richard Horner, PhD	University of Toronto

Canadian Sleep Society (CSS) Student Outstanding Achievement Award Judges

Sharon Chung, PhD	Toronto Western Hospital
Wendy Hall, RN, PhD	University of British Columbia
Tore Nielsen, PhD	Université de Montréal
Ben Rusak, PhD	Dalhousie University

Distinguished Scientist Award

The Canadian Sleep Society (CSS) is pleased to announce that the recipient of the 2009 Distinguished Scientist Award is Dr. Carlyle Smith, PhD, CPsych of Trent University. This award is in recognition of a scientist who has made significant contributions to the field of sleep research in Canada. The first recipient of the award was Dr. Jacques Montplaisir of the University of Montreal in 2007.

Carlyle Smith is Lifetime Professor Emeritus of Psychology at Trent University in Peterborough, Ontario, Canada and is also a registered clinician. He is an internationally known expert on the topic of sleep and is Director of a world class sleep research laboratory at Trent University to investigate the many different aspects of sleep. His research has been steadily increasing our understanding of how the brain, during sleep, consolidates information that was learned earlier in the waking state, using both animal and human subjects. He has recently become interested in how sleep changes with aging and how these changes affect learning.

Carlyle Smith graduated as a Biochemistry major (B.Sc.) from the University of Manitoba (1963). He earned an M.A.(1969) and Ph.D. (1971) in Physiological Psychology from the University of Waterloo. He did postdoctoral work with Prof. Michel Jouvet at Universite Claude Bernard in Lyon, France (1971-72). He then accepted a position at Trent University in the Department of Psychology and continued to do sleep research with rats and began to examine sleep in human subjects as well in 1985. Over the years he has been a sabbatical research guest at the Universite de Nice (Prof . Claude Gottesmann: 1979), Universite de Paris (Prof. Vincent Bloch : 1986-87, 1993-94), University of Ottawa, Faculty of Medicine (Dr. Roger Broughton, 1980), Cyclotron Research Institute, Liege, Belgium (Dr. Pierre Maquet: 2002).

He currently holds research grants from the National Science and Engineering Research Council (NSERC) and the Canadian Institutes of Health (CIHR). He has also recently held grants from the Alzheimer's Society of Canada and has built a state of the art sleep lab with infrastructure funds from the Canadian Foundation for Innovation (CFI) in conjunction with the University of Toronto as part of the Centre for Biological Timing and Cognition (CBTC).

He has published widely on the topic of sleep and memory and co-authored a book (Sleep and Brain Plasticity, 2003) with Dr. P. Maquet (U.of Liege, Belgium) and Dr. R. Stickgold (Harvard U., Boston). Dr. Smith was awarded the Distinguished Research Award (Trent University) in 2000. His work has been the subject of numerous T.V. documentaries (Scientific American Frontiers, W-5, 20/20, Discovery Channel, CTV Specials, CBC documentaries as well as Japan's science show, Science 2000). He has also been featured in a number of newspaper articles including the New York Times, Globe and Mail, Toronto Star and interviewed on radio talk shows such as CBC Morning, Quirks and Quarks. He is listed in the Canadian "Who's Who".

He has also made a significant contribution to the advancement of sleep and dream research in Canada as Secretary and Vice-President (Research) of the Canadian Sleep Society. The Canadian Sleep Society was originally incorporated in Peterborough, Ontario.

He has taught courses on Sleep and Sleep Disorders as well as on Dreams and Dreaming and currently has Adjunct status with both Brock (Dept. of Psychology) and Queens Universities (Department of Psychology, Department of Neuroscience). He currently supervises 6 graduate students.

List of selected publications available at: www.css-meeting.ca

Roger Broughton Young Investigator Award

The Roger Broughton Young Investigator Award honours the contributions of Dr. Roger Broughton, founding President of the Canadian Sleep Society (1986 – 1988), and one of the founding figures of Canadian sleep research. In addition to a distinguished research career and many important contributions to the sleep literature through articles and books, Dr. Broughton co-founded the first sleep medicine clinic in Canada. In addition to numerous other honours, in 1997 Dr. Broughton was awarded the William C. Dement Award by the American Sleep Disorders Association for lifetime academic achievement in the field.

The Canadian Sleep Society (CSS) is pleased to announce that the recipient of the 2009 Roger Broughton Young Investigator Award is Dr. Jean-François Gagnon of the Université de Montréal. This award is in recognition of important early career research contributions to sleep research.

Previous winners of this award were: Dr. Kimberly Cote in 2004, Dr. Penny Corkum in 2007 and Dr. John Peever in 2007.

Jean-François Gagnon is an Associate Professor in Psychiatry at the Université de Montréal, Quebec, Canada and also a Clinical Neuropsychologist. His laboratory is based at the Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Coeur de Montréal. In recent years, his research focus has been on REM sleep behaviour disorder (RBD) and its association with neurodegenerative diseases. His research has contributed to major advances in the understanding of the association between RBD and neurodegenerative diseases, particularly Parkinson's disease. Most of these studies have been published in the top 10 journals in clinical neurology (Lancet Neurology, Annals of Neurology, Neurology, Sleep). His findings indicate that RBD is much more than a sleep disorder. In fact, in several patients, it appears to be an early stage of neurodegenerative disease. Greater awareness of this issue could lead to substantial advances in understanding mechanisms, refining the diagnoses, and developing treatments for neurodegenerative diseases.

Jean-François Gagnon graduated as a Psychology Baccalaureate and Master (B.A. and M.Ps.) from Université Laval, Quebec city, Quebec (1996). He earned a PhD (2005) in Neuropsychology from Université du Québec à Montréal, Quebec. He did postdoctoral work with Dr. Jacques Montplaisir at the Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Coeur de Montréal (2005-2006), and with Dr. Julien Doyon at the Unité de Neuroimagerie Fonctionnelle, Institut universitaire de Gériatrie de Montréal.

He currently holds research grants from the Canadian Institutes of Health (CIHR) and the Fonds de la recherché en Santé du Québec. He currently supervises five graduate students.

List of selected publications available at: www.css-meeting.ca

Student Outstanding Achievement Award

This award is for the scientific merit of a single publication by a student in the field of sleep research. Funding for these awards is made through the CSS student fund – Thank you to CSS members who made contributions to the student fund with their CSS registration. The CSS is pleased to present the inaugural award to:

PATTI BROOKS of the University of Toronto, for the following publication:

Brooks, P.L. and Peever, J.H. Glycinergic and GABAA-mediated inhibition of somatic motoneurons does not mediate REM sleep motor atonia. The Journal of Neuroscience, 2008, 28(14):3535–3545. Also see commentary and reply in Sleep 2008;31:1473-97.

Student and Technologist Abstract Prize Winners

This year we have one student and one technologist abstract prize. Please note: the winners of the student abstract competition for 2008 and the winner from 2009 are giving oral presentations during the Student Program on Sunday, April 26th. Congratulations!

TECHNOLOGIST PRIZE - LAURA RAY
Systematic Optimization of Automated Sleep Spindle Detection
Ray LB.1, Peters KR.1, Fogel SM.2, Smith CT. 1
Department of Psychology, Trent University, Peterborough, Ontario, Canada; 2Neuro-science Department, Queens University, Kingston, Ontario, Canada

STUDENT PRIZE - RONA KERTESZ
Event-Related Potentials (ERPs) Reveal Failure to Inhibit Stimuli During the Pre-Sleep Waking Period for Patients with Sleep-Onset Insomnia
Kertesz R. S., Cote K. A.
Department of Psychology, Brock University, St. Catharines, ON, Canada

For more information about CSS award opportunities, as well as application and evaluation procedures, please refer to the CSS website: http://www.css.to/awards.html

Past-Presidents of the Canadian Sleep Society

Roger Broughton, MD, PhD (1986-88)

Robert D. Ogilvie, PhD (1988-90)

Meir H. Kryger, MD (1990-93)

Alistair MacLean, PhD (1993-96)

Charlie George, MD (1996-99)

Joseph De Koninck, PhD (1999-2002)

Charles Morin, PhD (2002-2005)

Gilles Lavigne, DMD, PhD (2005-2008)

CME and CEC Information and Certificates of Attendance

This educational activity has been approved for CME:
• The College of Family Physicians of Canada - 15 Mainpro-M1 credits.
• The Royal College of Physicians and Surgeons of Canada - 15 Section 1 credits.
• The Physician's Recognition Award of The American Medical Association - 15 Category 1 credits.

Continuing Education Credit (CEC) hours for attendance at this conference are recognized by the Board of Registered Polysomnographic Technologists (BRPT). The maximum number of CEC hours possible through the CSS is 16.25 hours calculated as follows:

> • Sunday, April 26 = 3.25 hours;
> • Monday, April 27 = 6.0 hours;
> • Tuesday, April 28 = 7.0 hours.

Dental Symposium, Sunday April 26th
This program is eligible for 3 credit points for Ontario and Québec dentists under the "Non-Approved Sponsor / Non-Dental Topic" category.

Please complete your evaluation form and return it to the registration desk.

General Information

Public Transportation

The Metro is the subway system. Maps are located in each station. Operating hours are from 05:30 – 01:00. City buses run frequently and provide an alternative mode of transportation.

Shopping Hours

Stores, boutiques and department stores are open from 09:00 to 18:00 daily, to 21:00 on Thursdays and Fridays, and until 17:00 on Saturdays.

Gratuities

In Canada, gratuities are not included in restaurant bills, but are left to the discretion of the client. It is customary to leave a gratuity of 15% of the total before taxes. This same policy applies to taxi fares. Porters accept $1 CDN per piece of luggage.

Child Care

Most major hotels will gladly arrange childcare upon request.

Congress Venue

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525 Bay Street, Toronto, ON M5G 2L2
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Fax: 416-597-9211
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Speaker Disclosure

Speakers have been requested to disclose to the audience any real or apparent conflict(s)
of interest that may have a direct bearing on the subject matter of this program.

Registration Hours

Sunday, April 26th 11:00 – 16:00
Monday, April 27th 07:00 – 18:00
Tuesday, April 28th 07:00 – 15:00

*Don't miss the Reception (Salsa Party) on Monday, April 27th
from 19:00 – 21:00 in the Grand Ballroom*

It's time for a fiesta!! Put on your dancing shoes and join all the excitement for a one
night extravaganza at the Reception-Salsa Party. Be seduced by the hot sounds of
Cassava Latin Rhythms from Toronto, mixing many diverse styles of Latin music.
http://www.cassavalatin.com/

Note: a $20 entrance fee applies. This includes one drink ticket and appetizers.

Acknowledgements

We graciously acknowledge the following Sponsors and Exhibitors for their participation and support:

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Covidien Ltd. (NYSE: COV, BSX: COV)   **(Booth #123, 125, 224)**

Covidien is a leading global healthcare products company that creates innovative medical solutions for better patient outcomes and delivers value through clinical leadership and excellence. Covidien manufactures, distributes and services a diverse range of industry-leading product lines, including surgical devices, energy-based devices, respiratory and monitoring solutions, pharmaceutical products, imaging solutions, patient care and safety products, and medical supplies. With 2006 revenue of nearly $10 billion, Covidien has more than 43,000 employees worldwide in 57 countries, and is products are sold in over 130 countries. www.covidien.com

Covidien est une compagnie internationale de soins de santé de premier rang qui crée des solutions médicales innovantes améliorant l'évolution de l'état de santé du patient et qui apporte de la valeur par son leadership et son excellence cliniques. Covidien fabrique, distribue et approvisionne une gamme variée de lignes de produits leaders du secteur, notamment des dispositifs chirurgicaux, des dispositifs à champ énergétique, des systèmes respiratoires et de monitorage, des produits de soins et de sécurité du patient, des solutions d'imagerie, des solutions d'imagerie, des produits pharmaceutiques et des fournitures médicales. Avec un chiffre d'affaires 2006 de près de 10 milliards de dollars, Covidien emploie plus de 43,000 collaborateurs dans 57 pays, et ses produits se vendent dans plus de 130 pays. www.covidien.com

DeVilbiss Healthcare   **(Booth #210)**

DeVilbiss Healthcare is a world leader in the design, manufacturing, and marketing of respiratory medical products that address the respiratory needs of patients in institutional and homecare settings. DeVilbiss products are manufactured in the United States, Europe and Asia and are distributed in more than 100 countries around the world.

Elsevier Canada   **(Foyer 1)**

Elsevier Canada is a team of leading publishers including Saunders, Mosby, Churchill Livingstone, Butterworth-Heinemann, Hanley & Belfus, MDConsult and FIRSTConsult dedicated to meeting the information needs of health science professionals. We publish high-quality textbooks, references, periodicals, and electronic products for medicine, nursing, dentistry, veterinary medicine, and health professions.

Embla   **(Booth #220, 222)**

Embla® is a global company with a philosophy and vision 'To be Closer to our Customers' by proactively understanding our customers' needs and providing solutions to their challenges. Our 1-year Hardware Warranty and elite technical support team, which is fully staffed 24/7, are examples of such solutions.

Innovation is also a theme among Embla's impressive product line including the Embletta® portable diagnostic recorder which boasts over a half-million sleep studies performed worldwide, the Embla Enterprise™ business management system and our renowned AASM compliant PSG software platforms, which are used in over 60 countries. Contact us at 888-662-7632 or www.embla.com

Fisher & Paykel Healthcare Inc.   **(Booth # 105, 107)**

Fisher & Paykel entered the respiratory care market in 1971 with the development of a unique respiratory humidifier system for use in critical care. We now offer a broad range of products and systems for use in respiratory care, the treatment of Obstructive Sleep Apnea, neonatal care and operating rooms.

Grass Technologies   **(Booth #113)**

Grass Technologies offers a wide range of instrumentation for PSG, EEG, LTM - from lab-based to ambulatory to WIRELESS recorders — at affordable prices. Systems feature the world-renowned accuracy, dependability and performance of Grass amplifiers, and powerful software. A full line of electrodes, transducers, etc. is also available - visit our Online Store

Home LifeCare Services and Breathing Solutions   **(Booth #212)**

Home Lifecare Services and Breathing Solutions are Canadian owned and operated providing service for the last 2 decades, specializing in CPAP, Oxygen and respiratory equipment and supplies. We have a team of highly skilled healthcare professionals capable of providing the highest level of service and care to our clients. We cover Barrie to G.T.A. Hamilton to Bowmanville.

Login Canada   **(Foyer 1A)**

Login Canada has become the premier book distributor of health sciences, scientific/ technical, and trade books and electronic products. We currently stock over 60,000 titles from more than 500 publishers with access to over 551,932 titles in medicine, nursing, pharmacy, dentistry, veterinary medicine, allied health, psychology, social sciences, computer sciences, engineering, telecommunications, music and film, and a broad range of other topics.

McArthur Medical Sales Inc.   **(Booth #216)**

Established in 1984 as distributor of high quality products from leading manufacturers. "McArthur medical Sales Inc. success is built on creating and maintaining long-term business relationships with our customers. Our continued success is the result of our customers satisfaction." Visit our exhibit to view: SenTec Digital Monitoring System for real time measurement of transcutaneous pCO2, continuous SpO2 and pulse. Compatible with most common polysomnographic systems. SleepInnov BlueNight ambulatory and wireless solution for sleep apnea screening, 6MWT and cardiac and pulmonary exercise training.

Medigas   **(Booth #117, 119)**

Medigas is a trusted name in CPAP therapy, being one of the first homecare organizations to bring CPAP technology to Canada. Over the years, we've learned how to make CPAP therapy a positive experience by developing a sleep program that places your patient in the centre of the quality services and products we provide. By choosing Medigas for your patients, you are selecting an accredited healthcare provider with a skilled team of professionals committed to providing OSA education, compliance monitoring and ongoing follow-up. In 2009, Medigas celebrates 40 years serving the healthcare market – Breathe well. Sleep well. Live well.

Medtronic of Canada   **(Booth #208)**

Medtronic is passionate about improving the health of people and communities throughout the world. Medtronic ENT is a leading developer of products to treat people with diseases of the ear, nose, and throat (ENT). Medtronic ENT's main products include powered tissue-removal systems and other microendoscopy instruments, implantable devices, nerve monitoring systems, disposable fluid-control products, and a palatal implant system for treating snoring and mild to moderate Obstructive Sleep Apnea (OSA). Medtronic is committed in providing lifelong solutions that can assist in managing patients medical conditions.

Northern Optotronics Inc.   **(Booth #109)**

For the past 11 years Northern Optotronics Inc (NOI), a leading Canadian-owned Corporation, has surpassed customer expectations for consistent, flexible and reliable products and service. NOI is your single most trusted source to meet all of your equipment requirements and your best choice for Compumedics supported products. Superior Products, Extraordinary Service, Unsurpassed Knowledge, Simply the Best.

Philips Respironics   **(Booth #128, 129)**

Philips Respironics is a leading developer, manufacturer and distributor of innovative products and programs that serve the global sleep and respiratory markets. Focusing on emerging market needs, the Company is committed to providing valued solutions to help improve outcomes for patients, clinicians and health care providers. Philips Respironics markets its products in more than 140 countries and employs more than 5000 associates worldwide.

REM-A-TEE Anti Snore Shirt   **(Booth #204)**

Featuring innovative sleep products designed to treat mild to moderate POSITIONAL sleep apnea and snoring. Using a patented three bumper system integrated into a t-shirt or Bumper Belt will ensure the wearer remains sleeping on their side throughout the night. For patients who cannot tolerate Oral Appliances or CPAP, POSITIONAL therapy can be an excellent cost effective option. Free Samples and brochures are available for your sleep center. Stop by our booth or call us to register and receive a sample kit. We look forward to working with you. 1-866-927-6739. http://www.rematee.com.

ResMed   **(Booth #200, 202)**

ResMed (ASX: RMD and NYSE: RMD) is a leading developer, manufacturer and marketer of products for the screening, treatment and long-term management of sleep-disordered breathing (SDB) and other respiratory disorders.

ResMed operates in over 65 countries via 16 direct offices and a network of distributors with extensive knowledge and experience of local markets.

The ResMed Story
• Formed in 1989
• Primary purpose was to commercialize a device for treating obstructive sleep apnea <http://www.resmed.com/en-uk/about_us/understanding-sdb-english-640x380. swf> (OSA), a major subset of sleep disordered breathing (SDB)
• Developed in 1981 by Professor Colin Sullivan and colleagues at the University of Sydney, nasal continuous positive airway pressure (CPAP) provided the first successful noninvasive treatment of OSA.

- Since 1989, ResMed has maintained its focus on SDB
- Operations have grown dramatically via introduction of a number of highly innovative product lines

RestAssure Inc.   **(Booth #218)**

RESTAssure is a recognized leader in providing respiratory healthcare products and services.Our client care services are continually expanding and we are committed to maintaining a standard of excellence. The RESTAssure team works in partnership with our clients as well as healthcare facilities, manufacturers and the government to ensure that we are providing the best products and services. We deliver the highest quality service for effective and cost-efficient home-healthcare solutions. RESTAssure focuses on improving quality of life for our clients through continuous product-quality improvement initiatives and public education. RESTAssure is your best choice for complete home respiratory care.

Shoppers Home Health Care   **(Booth #226)**

With CPAP services available at over 20 stores across Ontario, Shoppers Home Health Care® carries a complete selection of CPAP products from all major manufacturers, with Shoppers Optimum Points® available on all purchases. Our professional team of Registered Respiratory Therapists, Registered Nurses, and Registered Polysomnographic Technologists are on hand to help all of our clients find the CPAP solution that works for them. Full Oxygen service is also available at select stores

Sleep Strategies Inc.   **(Booth #100)**

Sleep Strategies Inc. is a leading provider of professional sleep scoring/analysis and consultation services for sleep disorder facilities worldwide. Sleep Strategies emphasizes patient care - timely, accurate and cost effective sleep analysis means swift diagnoses, ultimately increasing the number of patients treated. We play a key role in the successful growth of leading hospitals, private sleep disorder laboratories, university research organizations and pharmaceutical companies. Sleep Strategies achieves this through our ongoing commitment to superior quality, affordability and exceeding our client's expectations. For more information, visit www.sleepstrategies.com or call 1.800.905.0348.

SOMNOmedics GmbH   **(Foyer 2)**

SOMNOmedics GmbH is a leading Developer, Manufacturer and Distributor of innovative products for sleep diagnostics and therapy. 3 lines of devices: SOMNOscreen™ plus PSG: mobile 33-Channel miniaturized Sleep Lab. SOMNOwatch™: ambulatory monitors PLM, Activity, Body Position, Tremor Analysis and Respiratory Screening, long-term-EEG and -ECG. SOMNOscreen™ plus EEG 10-20: mobile 43-Channel EEG-/PSG-System. The devices can be upgraded at any time.

SOMNOmedics GmbH was founded in 2000 and is located in the North of Bavaria (Germany). The Company developed from a distributor of Sleep Diagnostic Devices into a leading Developer Manufacturer & Distributor of innovative products for sleep diagnostics and therapy. With the SOMNOscreen plus, SOMNOmedics offers the possibility to use one and the same device as an Ambulatory Screener or to upgrade it to a full PSG System with Digital Video. The product line has grown to include the SOMNOscreen plus EEG 10-20 and the SOMNOwatch, a small "all-rounder" for screenings, follow-up or Research.

Stellate Systems   **(Booth #121)**
Founded in 1986, Stellate is a leading global supplier of advanced solutions for Sleep Diagnostics, EEG, ICU and Long Term Monitoring. The company's Harmonie systems can be found at a wide range of institutions from small clinics to large universities and hospitals. Stellate products are backed by comprehensive 24/7 customer support delivered by a team of highly qualified EEG and PSG technologists.

Valeant Canada Ltd.   **(Booth #111)**
Valeant Canada Limited is a subsidiary of Valeant Pharmaceuticals International, a global pharmaceutical company. Valeant Pharmaceuticals International's vision is to discover, develop, acquire and commercialise innovative products for the treatment of diseases with significant unmet medical needs primarily in the areas of neurology and dermatology.

Valeant Canada limitée est une filiale de Valeant Pharmaceuticals International, société pharmaceutique intégrée. La vision de Valeant Pharmaceuticals International est de découvrir, mettre au point, acquérir et commercialiser des produits novateurs servant à traiter des maladies présentant d'importants besoins médicaux non satisfaits, surtout dans les domaines de la neurologie et de la dermatologie.

VitalAire Canada   **(Booth #228)**
VitalAire provides CPAP Therapy through our 80 offices across Canada. Our large product selection assures your patients access to the newest treatment technology and a comfortable interface fit. We are accredited and our Standard Clinical Program ensures that your patients receive the information they need to achieve effective CPAP therapy.

XLTEK   **(Booth #206)**
XLTEK, a division of Natus, and Bio-logic, a division of Natus, design, manufacture and distribute a wide range of neurology & sleep diagnostic systems and supplies.

The XLTEK & Bio-logic product lines are designed to deliver the latest innovations in EEG, Epilepsy, ICU, Ambulatory, PSG, EMG and IOM needs.

Keynote Speakers

Dr. Irene Tobler, University of Zurich
Title: "Sleep regulation in animals"
Monday, April 27, 2009

Dr. Irene Tobler, PhD, is a Professor of Zoology in the Faculty of Science at the University of Zurich, and a Senior Research Scientist at the Institute of Pharmacology and Toxicology, University of Zurich, Switzerland. Her research interests include: sleep and sleep regulation: comparative aspects; mechanisms of sleep regulation (sleep substances; adenosine system; role of GABA in sleep; gene-targeted animal models); daily torpor and its relationship to sleep; sleep and memory: contributions from rodents; phylogeny of sleep and its regulation (studies include a large diversity of species: rat, mouse, Syrian hamster, cat, dog, sheep, rabbit, Djungarian hamster, Guinea pig, pigeon, perch, cockroach, scorpion, elephant, giraffe, blind mole rat, Macaque and Ibex); and animal models for sleep and ageing.

Dr. Tobler's research is funded by the Swiss National Science Foundation; University of Zurich; EU project - Marie Curie Training Program: "The Biological and sociological effects of sleep restriction"; and EU project - Specific Targeted Research Project: "Enough Sleep".

Dr. Tobler has participated on the board of number of societies, including the European Sleep Research Society (Treasurer, 1986-1984; President, 2000-2004), World Federation of Sleep Research Societies (European Delegate, 1991-1994), American Sleep Research Society (European Delegate, 1991-1994), Swiss Society for Sleep Research, Sleep Medicine and Chronobiology (Secretary, 1994-1998, Vice President, 1998-2000¸ President, 2000-2004), and the Swiss Society for Neuroscience, (Member of the Board, 1999-2001). In addition, Dr. Tobler has acted as Associate Editor for the Journal of Sleep Research (1992-2007), and has been on the editorial board of the Archives Italiennes de Biologie since 2002. She has also been involved with the Committee for Research in Animals of the Canton of Zurich (Member 1995-2000; President 2000-2003), the Medical-Ethical committee in Physiology-Pharmacology, and the Ethical Committee for animal protection of both Universities in Zurich.

Dr. Tobler has over 160 publications in peer-reviewed journals, and was recipient of the 7th Pisa Sleep Award, University of Pisa, in June 2008.

Dr. Derk-Jan Dijk, University of Surrey
Title: "Sleep/wake regulation: Circadian, homeostatic and genetic components"
Date: Monday April 27, 2009

Dr. Derk-Jan Dijk, PhD, is Professor of Sleep and Physiology and Director of the Surrey Sleep Research Centre in the Faculty of Health and Medical Sciences at the University of Surrey in Guildford, United Kingdom. Prior to his appointment at the University of Surrey, he served as Assistant Professor of Medicine (Neuroscience) at Harvard Medical School in Boston, Massachusetts. Dr. Dijk received his BSc and MSc in Biology and his PhD in Medical Sciences from the University of Groningen in The Netherlands. He completed his postdoctoral fellowship at the Institute of Pharmacology, University of Zürich, in Switzerland. His research interests include regulation of sleep and circadian rhythms in humans; role of sleep homeostasis and circadian rhythms in the regulation of waking performance; monitoring of alertness and performance; age-related changes in sleep timing, structure, and quality; effects of hypnotics, melatonin, and light on the spectral composition of the electroencephalogram (EEG), sleep, and performance; and sleep and circadian rhythms in shift workers morning and evening types.

Dr. Dijk served as acting co-director of the Clinical Research Centre (formerly HPRU) at the University of Surrey, which is world-renowned for the research and development of CNS active drugs. Dr. Dijk is associated with several professional organizations, including the European Sleep Research Society (ESRS), the Sleep Research Society, and the Society for Research on Biological Rhythms. He served as Chair of the Scientific Committee of the ESRS and as member of the scientific committee of the World Federation of Sleep Research Societies. He currently is a deputy editor of SLEEP. Dr Dijk has published more than 100 original reports and numerous reviews on his areas of research in journals such as Sleep, The Journal of Neuroscience, American Journal of Physiology, and European Journal of Pharmacology, among others.

Dr. Mary A. Carskadon, Brown University
Title: "Sleep & Sleepiness in Adolescents"
Date: Monday April 27, 2009

Dr. Mary A. Carskadon, PhD, received a doctorate with distinction in Neuro- and Biobehavioral Sciences with a specialty in sleep research from Stanford University (1979) where she trained under the mentorship of William C. Dement, MD, Ph.D. In 1999, she was awarded an honorary doctor of sciences degree from Gettysburg College, her undergraduate alma mater (Psychology, 1969). She is currently Director of Chronobiology/Sleep Research at the E.P. Bradley Hospital and Professor of Psychiatry and Human Behavior at the Warren Alpert Medical School of Brown University in Providence, RI. Dr. Carskadon is a past president of the Sleep Research Society (SRS), a Fellow of the American Academy of Sleep Medicine (AASM), member of the Board of Scientific Counselors, National Space Biomedical Research Institute, deputy editor of Sleep, and associate editor of Behavioral Sleep Medicine. She has served on a number of working groups, task forces, and review committees on issues of sleep, sleeplessness, and sleepiness. Carskadon received the National Sleep Foundation's Lifetime Achievement Award (2003), the American Academy of Sleep Medicine Mark O. Hatfield Public Policy Award (2003), the Outstanding Educator Award of the Sleep Research Society (2005), and the Distinguished Scientist Award of the Sleep Research Society (2007). She has also been elected Fellow of the Association for Psychological Science (2007) and Fellow of the American Association for the Advancement of Science (2007).

Dr. Carskadon's early research culminated in the development and application of a standardized measure for daytime sleep tendency, the multiple sleep latency test. Dr. Carskadon's current scientific activities include research examining the interrelation between the circadian timing system and sleep/wake patterns of children and adolescents; another research theme focuses on the effects of family history of alcohol abuse/dependence on sleep and circadian rhythms in young humans. New research initiatives include assessing sleep restriction and depressed mood in college students and role of sleep in overnight learning enhancement for children with ADHD and as a function of pubertal maturation in healthy youngsters. Her research findings have raised public health issues regarding the consequences of insufficient sleep in adolescents as well as concerns about early starting times of schools.

Dr. Eus Van Someren, Netherlands Institute for Neuroscience and VU Medical Center
Title: "Melatonin and light treatment in dementia"
Date: Monday April 27, 2009

Dr. Eus J.W. Van Someren, PhD, is Head of the Sleep and Cognition research group at the Netherlands Institute for Neuroscience (NIN), an institute of the Royal Netherlands Academy of Arts and Sciences, and is Associate Professor at the Departments of Medical Psychology, Clinical Neurophysiology and Neurology at the VU University Medical Center (VUmc). Originally trained as a physicist, psychophysiologist and neuropsychologist, he now leads a group working on quantifying instability of the states of sleep and wakefulness in elderly and dementia; the role of sleep in cognitive information processing; the interaction between sleep and thermoregulation; brain activity in chronic insomnia. His position at the VUmc facilitates access to patient groups; and moreover involves a pivotal role in multimodal brain imaging (EEG combined with fMRI, MEG, TMS) which now focuses on brain changes in insomnia. He is author of about 100 peer-reviewed publications in scientific journals. Presently, he is founding a National Sleep Registry that will facilitate progress in the understanding of risk factors for insomnia.

Dr. Charles Morin, Laval University
Title: "Insomnia: From epidemiology to treatment"
Date: Tuesday April 27, 2009

Dr. Charles Morin, PhD, is Professor of Psychology and Director of the Sleep Research Center at the Université Laval in Quebec City. He received a doctorate degree (PhD) in clinical psychology from Nova University in Florida and completed a post-doctoral fellowship in sleep medicine at Virginia Commonwealth University, where he remained on the faculty as Professor of Psychiatry and Director of the Sleep Disorders Center from 1987 to 1994. He currently holds a Canada Research Chair on Sleep Disorders and is past President of the Canadian Sleep Society. He is Associate Editor for the journals Sleep and Behavioral Sleep Medicine and is on the editorial board of several other journals. He is a member of the DSM-V workgroup in charge of updating diagnostic criteria for sleep disorders. He has published four books and over 150 articles and chapters. Dr. Morin's main interests are in the epidemiology and treatment of insomnia, with a special expertise in the development and validation of cognitive-behavioural and combined psychological and pharmacological approaches for treating insomnia. His research is funded by the Canadian Institutes of Health Research and the National Institute of Mental Health.

Dr. Ruth Benca, University of Wisconsin-Madison
Title: "Sleep, seasonality and mood"
Date: Tuesday April 27, 2009

Dr. Ruth Benca, MD, PhD, is Professor in the Departments of Psychiatry and Psychology at the University of Wisconsin-Madison and the Director of the University of Wisconsin Center for Sleep Medicine and Sleep Research.

Dr. Benca received her PhD in pathology and her medical degree from the University of Chicago in Illinois before completing a residency in psychiatry and a fellowship in sleep disorders medicine, also at the University of Chicago. She is a member of several professional organizations, including the Society for Neuroscience, the Sleep Research Society (SRS), the American Academy of Sleep Medicine (AASM) and the American Psychiatric Association; she has served as President of the SRS and on the Board of Directors of the AASM. Additionally, she is a Deputy Editor of Sleep: Journal of Sleep and Sleep Disorders Research. Dr. Benca has served as principal investigator for a number of basic and clinical research studies and has presented her research findings at a variety of national meetings. She has coauthored numerous articles, reviews and book chapters, and her research has been funded by the National Institute of Mental Health and the Department of Defense. Among her awards and honors, Dr. Benca was recently selected to participate in the Hedwig van Amerigen Executive Leadership in Academic Medicine (ELAM) Program for Women (2004–2005).

Dr. Doug Bradley, University of Toronto
Title: "Sleep apnea and cardiovascular disorders"
Date: Tuesday April 27, 2009

Dr. Douglas Bradley, MD, is Professor of Medicine and Director of the Sleep Research Laboratory at the Toronto Rehabilitation Institute, and of the Centre for Sleep Medicine and Circadian Biology at the University of Toronto. His research focuses on the relationship between sleep apnea and cardiovascular diseases. He holds several peer-reviewed grants from the Canadian Institutes of Health Research and other agencies and has published over 160 papers and book chapters on sleep apnea and related topics.

Program-at-a-glance

Saturday, April 25 — Pre-conference events

Time	Event
15:00-17:00	Public Lectures (Salon A/B)
19:00	Satellite Program (Respironics) (Westin Harbour Castle Hotel)

Sunday, April 26

Registration Open — Exhibits

Time	Event
8:30-12:00	Committee Meeting - CSS Portable Monitoring for OSA (Dundas Room)
10:30-12:30	
11:00	Registration Opens
13:00-17:30	Technical Program (Salon C) — Coffee Break 14:45 - 15:15
13:00-17:00	Student Program (Salon C) — Coffee Break 14:30 - 15:00
13:00-17:00	Symposium 1 & 2 (Salon D) — Coffee Break 14:30 - 15:00
13:00-16:30	Symposium 3 A & B (Dental) (York) — Coffee Break 14:30 - 15:00
14:30-15:30	EXHIBITION (Trinity Ballroom)
19:00	Sleep Professional Social (Hard Rock Café)

Monday, April 27

Registration Open — Exhibits Open

Time	Event
7:00	Registration Opens
8:30-12:00	Sponsored Workshop I (Bay Room) Respironics
8:30-9:00	Opening Ceremonies and Awards (Grand Ballroom)
9:00-10:30	Keynote Lectures 1 & 2 (Grand Ballroom)
10:30-11:00	COFFEE BREAK & POSTER VIEWING (Trinity Ballroom & York A/B)
11:00-12:30	Symposium 4 (Salon C) / Symposium 5 (Salon D) / Technical Workshop 1 (Bay Room)
12:30-13:30	LUNCH & POSTER VIEWING (Trinity Ballroom & York A/B) / Lunch Hour Discussion I (Francophone Session) (Bay Room)
13:30-15:00	Keynote Lectures 3 & 4 (Grand Ballroom)
15:00-15:30	COFFEE BREAK & POSTER VIEWING (Trinity Ballroom & York A/B)
15:30-17:00	Symposium 6 (Salon C) / Symposium 7 (Salon D) / Technical Workshop 2 (Salon D)
17:00-18:00	Annual General Meeting (For CSS members) (Salon D)
19:00-21:00	Reception: Salsa Party (Salon A/B)

Tuesday, April 28

Registration Open — Exhibits Open

Time	Event
7:00	Registration Opens
8:30-12:00	Sponsored Workshop II (Bay Room) Fisher&Paykel
8:30-10:00	Keynote Lectures 5 & 6 (Grand Ballroom)
10:00-10:30	COFFEE BREAK & POSTER VIEWING (Trinity Ballroom & York A/B)
10:30-12:30	Symposium 8 (Salon C) / Symposium 9 (Salon D) / Technical Workshop 3 (Salon D)
12:30-13:30	LUNCH & POSTER VIEWING (Trinity Ballroom & York A/B) / Lunch Hour Discussion II (BRPT) (Bay Room)
13:30-15:00	Symposium 10 (Salon C) / Symposium 11 (Salon D) / Technical Workshop 4 (Salon A/B)
15:00-15:30	COFFEE BREAK & POSTER VIEWING (Trinity Ballroom & York A/B)
15:30-17:30	Keynote Lecture 7 (Grand Ballroom) / Poster Data Blitz (Grand Ballroom) / Evaluation & Closing Remarks (Grand Ballroom)

Sponsored Workshops and Social Events

SATURDAY, APRIL 25, 2009

19:00 (Westin Harbour Castle)
Evening lecture with Dr. David White on
"The Future of Sleep Medicine"
Hosted by Respironics at the Westin Harbour Castle Hotel,
1 Harbour Square, Toronto, Ontario M5J 1A6
From 7PM, with appetizers. Following the lecture there will be a band "Catch 22"

SUNDAY, APRIL 26, 2009

19:00 (Hard Rock Café)
"Sleep Professionals Social"
Hosted by ResMed and Praxair (Medigas)

MONDAY, APRIL 27, 2009

08:30 – 12:00 SPONSORED WORKSHOP I (Bay Room)
Respironics (Philips Home Healthcare Solutions)
First Impressions Workshop – Hands on Mask Fitting and
Advance Compliance Tools
Note: For this workshop attendance certificates will be awarded for CEC hours by the Education Provider, not the CSS. Please note that the CEC hours for attending this sponsored workshop must be deducted from hours for attending the 4th Conference of the CSS (as both events occur simultaneously).

TUESDAY, APRIL 28, 2009

08:30 – 12:00 SPONSORED WORKSHOP II (Bay Room)
Fisher & Paykel Healthcare Inc.
Using Technology to Increase Patients CPAP Compliance
Note: This educational activity has been approved for 2.5 hours through the Canadian Sleep Society. Attendance certificates will be awarded for CEC hours by the Education Provider, not the CSS. Please note that the CEC hours for attending this sponsored workshop must be deducted from hours for attending the 4th Conference of the CSS (as both events occur simultaneously).

Pre-conference event: SATURDAY, APRIL 25, 2009

15:00 – 17:00 PUBLIC LECTURES (Salon A/B)

Disturbed sleep and mental health - What happens and what to do about it?
• Dr. Jon Fleming, Department of Psychiatry, University British Columbia, Co-Director UBC Sleep Disorders Programme

Insomnia: What role do the lungs play?
• Dr. Michael Fitzpatrick, Sleep Disorders Clinic and Department of Medicine, Kingston General Hospital, Queen's University, Kingston

Chair: Dr. Eileen P. Sloan, Department of Psychiatry, Mount Sinai Hospital and University of Toronto

Co-Chair: Dr. Adam Blackman, Toronto Sleep Institute

Scientific Program

13:00 – 17:30	**TECHNICAL PROGRAM (Salon A/B)**
	Chairs: Carol Mously, BSc, RPSGT, Toronto Sleep Institute
	Natalie Morin, BSc, RPSGT, Sleep Strategies Inc.

13:00 – 13:30	Staging Sleep – From Old to New
	Natalie Morin, BSc, RPSGT, Sleep Strategies Inc.

13:30 – 14:00	Distinguishing Seizures, Parasomnias and Artifacts
	Brian Murray, MD, University of Toronto Sunnybrook Health Services Centre

14:00 – 14:45	Cardiac Physiology and Heart Disease in PSG
	Doug Scullion, BSc, RPSGT, ResMed

14:45 – 15:15	**BREAK**

15:15 – 16:00	Movement Disorders During Sleep
	Laree Fordyce, RPSGT, CCRP, REEGT, CENGT, Centre for Sleep and Human Performance, Calgary

16:00 – 17:30	**TECHNICAL WORKSHOP Cont'd (Salon A/B)**
	Hypoventilation and Non-invasive Ventilatory Support: Overview and Case Studies of BiLevel S/T Titrations
	Moderator: Helen Driver, PhD, RPSGT, DABSM, Queen's University and Kingston General Hospital

In the sleep laboratory setting, bilevel non-invasive ventilatory support (BPAP) is used primarily to treat chronic forms of hypoventilation, although it can be employed in the management of several other conditions. The purpose of bilevel therapy is to augment the patient's ventilation by boosting the tidal volume and/or respiratory rate. Patients with acute and chronic neuromuscular disease, the obesity hypoventilation syndrome, and kyphoscoliosis commonly benefit from this treatment. Some patients with central sleep apnea may also benefit from bilevel treatment. Bilevel titration involves adjustment of

inspiratory and expiratory pressures to optimize ventilation and patient comfort. Bilevel units are available in three different modes: (1) Spontaneous (S) breathing; (2) Spontaneous/ Timed (S/T) (assist/control, AC) with a back-up rate, (3) Timed (T) (control) breathing. This workshop for sleep technologists will include case studies to outline titration procedures.

Learning Objectives:
1. List the important differences between CPAP and BPAP.
2. Describe how to perform an overnight in-laboratory BPAP titration.
3. List 5 clinical conditions for which BPAP therapy is appropriate.
4. Recognize the polysomnographic (PSG) features of hypoventilation.
5. Recognize and correct BPAP titration problems (based on PSG case studies).

Note: There will be interactive discussion during the case study presentations - opinions will be solicited from the attendees.

16:00 - 16:20	Polysomnography to Document Hypoventilation and PSG Samples from Case Studies Lori Davis, BSc, RCPT(P), RPSGT, West Park Hospital, Ontario.
16:20 - 16:35	PSG Case Study: Obesity Hypoventilation Syndrome (OHS) Fern Toop, RPSGT, Kingston General Hospital, Ontario.
16:35 - 16:50	PSG Case Study: Paediatric Patrick Raymondo, RPSGT, Ottawa General Hospital.
16:50 - 17:00	A Model for Patient Access to BPAP Units - The Ventilator Equipment Pool (VEP) Ray Milton, RRT, Coordinator, VEP Ontario.
17:00 - 17:15	Indications and Troubleshooting in BPAP Titration Michael Fitzpatrick, MD, DABSM, Queen's University and Kingston General Hospital.
17:15 - 17:30	Recording Hypoventilation and BPAP Titration Algorithm: Q&A Panel: Includes the moderator, the five speakers, Kathy Lutley-Borland, BSc, RET, RPSGT, Ottawa, Debra Medin, MS, RRT, RPSGT, Pediatric Sleep Disorders Laboratory, The Hospital for Sick Children, Toronto.

13:00 – 17:30	**STUDENT PROGRAM (Salon C)**
	Chairs: Stuart Fogel, MA, Queen's University
	Vincent Moreau, Univérsité Laval
13:00 – 13:30	Teaching Sleep
	Dr. Mary A. Carskadon, PhD, Brown University
13:30 – 14:00	Cognition and Circadian Rhythms
	Dr. Lynn Hasher, PhD, University of Toronto
14:00 – 14:30	Quantitative Analysis of Sleep and EEG Data
	Dr. Derk-Jan Dijk, PhD, University of Surrey
14:30 – 15:00	**BREAK**
15:00 – 15:15	Enhanced Cholinergic Activity at the Hypoglossal Motor Nnucleus Suppresses Genioglossus Muscle Activity
	Kevin Grace, University of Toronto
15:15 - 15:30	Impact of Sleep Deprivation on Postural Control in Young and Older Subjects
	Rebecca Robillard, BSc, University of Montreal
15:30 -15:45	GABAB-mediated Inhibition Plays a Critical Role in Mediating REM Sleep Atonia
	Patti Brooks, University of Toronto
15:45 - 16:00	Event-related Potentials (ERPs) Reveal Failure to Inhibit Stimuli During the Pre-sleep Waking Period for Patients with Sleep-onset Insomnia
	Rona Kertesz, MA, Brock University
16:00 - 16:30	Academic and Industry Collaborations: Brightening the Sleep Landscape
	Dr. Chris Winrow, PhD, Merck & Co. Inc.
16:30 - 17:00	Cognitive Behavioural Therapy for Insomnia
	Dr. Charles Morin, PhD, Laval University
17:00 – 17:30	Discussion

13:00 – 14:30   **APPLIED SYMPOSIUM 1**   **(Salon D)**
      Sleep and Sleep Disorders: Measuring the Impacts on Public Health
      Chair: Louise McRae, BSc. Public Health Agency of Canada

Behavioural risk factors for chronic diseases are well-recognized. Interest in the role of sleep and sleep disorders in the development and management of chronic diseases is growing. This symposium offers an opportunity to discuss sleep and sleep disorders in the context of public health. Evidence of the role of sleep and sleep disorders in traffic collisions and in the development of diabetes, cardiovascular disease, obesity, depression and other chronic diseases will be presented. Discussion will focus on the importance of sleep and sleep disorders among the well-recognized chronic disease behavioural risk factors and issues specific to children. There are a number of existing Canadian population-based indicators relating to sleep such as duration, quality, daytime sleepiness, and use of sleeping pills, etc. The indicators most pertinent to chronic diseases surveillance will be considered.

Learning objectives:
1. Understand sleep and sleep disorders in the context of public health and chronic disease surveillance.
2. Understand the association of sleep and sleep disorders with cardiovascular disease, obesity, and other chronic diseases.
3. Understand the importance of sleep and sleep disorders among the chronic disease risk factors.
4. Contribute to the development of a list of key indicators for sleep and sleep disorders most pertinent to chronic disease surveillance.
Note: Each talk is followed by a 5-minute question period.

13:00 - 13:15   Sleep Apnea in Canada
      Louise McRae, BSc, Public Health Agency of Canada

13:20 - 13:35   Sleep, Metabolism and Obesity
      Angelo Tremblay, PhD, Laval University

13:40 - 13:55   Paediatric Sleep Disordered Breathing: Current and Future Health Concerns
      Ian MacLusky, MBBS, FRCP(C), University of Ottawa
      Children's Hospital of Eastern Ontario

14:00 - 14:15	Sleep, Sleep Disorders and Cardiovascular Disease Najib Ayas, MD, University of British Columbia.
14:20 - 14:30	Panel Discussion and Question Period
14:30 – 15:00	**BREAK**
15:00 – 17:00	**APPLIED SYMPOSIUM 2 (Salon D)** Sleep Deprivation, Circadian Factors and Human Performance: A Research Update on Measurement Techniques and Interventions. Chair: Charles Samuels MD, CCFP, DABSM. Medical Director Centre for Sleep and Human Performance, Principle Investigator, Calgary Police Service Health and Human Performance Research Initiative.

Sleep deprivation research has a well established foundation in the field of sleep research, however, lack of standard research techniques in field settings is limiting the practical application of the results of this research in various field settings. This symposium will begin to bride the gap between the laboratory and real life through presenting the work of researchers who are applying the basic principles of sleep deprivation and circadian rhythm research techniques with scientific rigor in medical, military, and para-military field settings.

Learning Objectives:
1. Review a unique psychophysical measurement technique in sleep deprivation and human performance research.
2. Discuss current military research that addresses effective countermeasures in demanding high performance field settings.
3. Discuss occupational and patient safety issues in medical residents under conditions of sustained wakefulness.
4. Review unique mathematical modeling of actigraphy data for analytical purposes in sleep deprivation and circadian rhythm research.

15:00 - 15:05	Introduction Charles Samuels, MD, University of Calgary
15:05 - 15:30	Psycho-Physiological Profiles of Sleep Deprivation in Operational Environments Chris Berka, CEO & Founder, Advanced Brain Monitoring Inc.

| 15:30 - 15:50 | Occupational Fatigue Management Programs: Assessing Sleep, Sleepiness and Sleep Disorders in the Real-World |
| | Steve Lockley, PhD, Harvard Medical School |

| 15:50 - 16:10 | Occupational and Patient Safety Consequences of Sustained Wakefulness in Residents: Observational Studies |
| | Najib Ayas, MD, University of British Columbia |

| 16:10 - 16:30 | Waiting Time Distribution of Actigraphy Measured Sleep |
| | Charles Samuels, MD, University of Calgary |

| 16:30 - 17:00 | Panel Discussion and Question Period |

13:00 – 16:30   **DENTAL SYMPOSIUM 3 A & B (York)**
Oral Appliance a First Line Treatment for OSA -- How, When, Why?
Chair: Luc Gauthier, DMD, MSc, Université de Montréal, Québec.

In the past, oral appliances (OA) have been considered to be an alternative treatment for obstructive sleep apnea (OSA). Although CPAP remains the Gold standard for treatment, recent studies and AASM recommendations support the use of oral appliances as a first line to treat mild-to-moderate OSA. This symposium will provide an up-to-date review of current research related to the use of oral appliances (e.g., how they work, when to use them, and why they are effective). In addition, recent developments will be presented on bruxism and pain in relation to sleep-related breathing disorders.

Learning Objectives:
1. Understand the role, limitations, and indications of OA for treating OSA.
2. Understand the mechanisms of how OA works.
3. Understand the role and the link between OSA, bruxism, pain, and sleep.
4. Understand the place of the OA in the future OSA treatment.

13:00 - 13:05   Introduction
Luc Gauthier, DMD, MSc, Université de Montréal

15:00 - 16:30   **DENTAL SYMPOSIUM 3A (York)**
The Dentists' Approach to Patients with Sleep Apnea
Chair: Frédéric Sériès, MD, Université Laval
Note: Each talk is followed by a 5-minute question period

13:05 - 13:20	Dental Examination for Sleep Apnea: Indications and Contraindications of OA (How, When, Why OA for OSA) Patrick Arcache, DMD, Université de Montréal

13:25 - 13:40	Cranio-facial Development in Children with OSA Catherine Ashton-McGregor, DDS, Kingston

13:45 - 14:00	Are there OA Specificities? Comparison between the Silencer and Klearway Luc Gauthier, DMD, MSc, Université de Montréal

14:05 - 14:20	Oral Appliances and Side Effects: When to Stop, How to Manage? Fernanda R. Almeida, DDS, PhD, University of British Columbia

14:25 - 14:30	Panel Discussion and Question Period

14:30 - 15:00	**BREAK**

15:00 - 16:30	**DENTAL SYMPOSIUM 3B (York)** Dental Sleep Medicine in Practice Chair: Luc Gauthier, DMD, MSc, Université de Montréal Note: Each talk is followed by a 5-minute question period

15:00 - 15:15	Bruxism and Sleep Patrick Arcache, DMD, Université de Montréal

15:20 - 15:35	Overview on Pain and Sleep Interaction Nelly Huynh, PhD, Université de Montréal

15:40 - 15:55	CPAP or Oral Appliances? I Need to Know! Frédéric Sériès, MD, Université Laval

16:00 - 16:15	Morning Headaches and Oral Appliances: Is there a Link? Laurent Franco, DMD, Université de Montréal

16:20 - 16:30	Panel Discussion and Question Period

MONDAY, APRIL 27, 2009

08:30 – 09:00   OPENING CEREMONIES AND AWARDS (Grand Ballroom)

09:00 – 09:45   **KEYNOTE 1 (Grand Ballroom)**
Chair: Helen Driver, PhD
Sleep Regulation in Animals
Dr. Irene Tobler, PhD, University of Zurich, Switzerland

09:45 – 10:30   **KEYNOTE 2 (Grand Ballroom)**
Chair: Kimberly Cote, PhD
Sleep/Wake Regulation: Circadian, Homeostatic and Genetic
Components
Dr. Derk-Jan Dijk, PhD, University of Surrey, England
Note: Each keynote lecture includes an 8 minute discussion period

10:30 – 11:00   **COFFEE BREAK (Trinity Ballroom)**
POSTER VIEWING (York A/B)

11:00 – 12:30   **SYMPOSIUM 4 (Salon C)**
From the Synapse to Sleep Homeostasis
Chair: Valérie Mongrain, PhD, Center for Integrative
Genomics, Genopode Building, University of Lausanne, Switzerland.

It is currently acknowledged that sleep is regulated by two main processes, a circadian and a homeostatic process. This symposium will highlight the important links between the physiology and dynamics of the synapse and the homeostatic process of sleep regulation through the presentation of novel and pioneering data regarding sleep regulation and its underlying molecular mechanisms. More specifically, the symposium will address the issue of how the regulatory elements present at the synapse could contribute to the recovery process of sleep and which neuronal systems in mammals show evidence of such sleep-dependent synaptic dynamics. Moreover, the symposium will serve to unravel the mechanisms by which sleep intensity depends on neuronal activity and the role of sleep for neuronal integrity and central nervous system equilibrium.

Learning objectives:

1. Establish which important structural and functional elements of the synapse are undergoing sleep-dependent changes.

2. Draw a portrait of synaptic events submitted to modification with sleep and wakefulness and their role in neuronal function.

3. Characterize the neuronal populations and structures showing sleep-dependent synaptic plasticity and depict relations with other neuronal functions.

Note: Each talk is followed by a 5-minute question period.

11:00 -11:15	Sleep and Synaptic Homeostasis Chiara Cirelli, MD, PhD, University of Wisconsin/Madison
11:20 -11:35	Activity-dependent Changes in Neurotransmitter Receptors during Waking and Sleeping Barbara E. Jones, PhD, Montreal Neurological Institute
11:40 -11:55	Synaptic Plasticity in Hypocretin Neurons: Re-setting Arousal Threshold Xiao-Bing Gao, PhD, Yale University School of Medicine
12:00 -12:15	Sleep and Stress-induced Alterations in Homeostasis Larry D. Sanford, PhD, Eastern Virginia Medical School
12:20 -12:30	Panel Discussion and Question Period
11:00 – 12:30	**SYMPOSIUM 5 (Salon D)** Narcolepsy: Recent Findings in Neurobiology, Neuropharmacology, and a Clinical Update Co-Chairs: Shelly K. Weiss, MD, University of Toronto Brian Murray, MD, University of Toronto

Narcolepsy, a chronic neurologic sleep disorder is about as prevalent as multiple sclerosis, yet it may not be readily diagnosed. This symposium will focus on basic and clinical research and a clinical update of this disorder. The clinical features of narcolepsy/cataplexy in both adult and pediatric patients, a discussion of the clinical neurophysiologic

findings used to help in differential diagnosis, and management options with case examples will be reviewed. Research findings from the mouse model of human narcolepsy (i.e. hypocretin/orexin deficient mice) will be outlined illustrating the neurobiology of sleepiness and cataplexy in this animal model. This research will illustrate the neurochemical mechanisms that trigger cataplexy as well as the development of pharmacological strategies for alleviating cataplexy and sleepiness in narcolepsy. The symposium will conclude with a review of the history of -hydroxybutyrate (GHB), its emergence both as a drug of abuse and a treatment modality for narcolepsy, and its putative mechanism of action.

Learning objectives:
1. Review the clinical features, differential diagnosis of excessive sleepiness, and current management of pediatric and adult narcolepsy.
2. Expand understanding of the neurobiology of sleepiness and cataplexy from research using a mouse model of human narcolepsy.
3. Learn about the neurobiology of y-hydroxybutyrate (GHB) and its use in narcolepsy.

11:00 - 11:05	Introduction Shelly K. Weiss, MD, University of Toronto
11:05 - 11:20	Neurobiology of Narcolepsy and Cataplexy John H. Peever, PhD, University of Toronto
11:20 - 11:35	Pediatric Narcolepsy: An Update on the Clinical Management of Pediatric Patients Shelly K. Weiss, MD, FRCPC, University of Toronto
11:35 - 11:50	Adult Narcolepsy: An Update on the Clinical Management of Adult Patients Brian Murray, MD, FRCPC, University of Toronto
11:50 - 12:05	The Neurobiology of y-hydroxybutyrate (GHB): A Club Drug with Therapeutic Applications in Narcolepsy Carter Snead MD, FRCPC, University of Toronto
12:05 - 12:30	Panel Discussion and Question Period

11:00 – 12:30   **TECHNICAL WORKSHOP I (Salon A/B)**
Pediatric Polysomnography
Chairs: Carol Mously, BSc, RPSGT for the CSS
Jacinthe Lavergne, BSc, RRT, RPSGT, Montreal Children's Hospital

Learning Objectives:
1. Review and recognize the indications for performing polysomnography on a child.
2. Understand the consequences of sleep disorders in children.
3. Describe the setup and recording procedures specific to the pediatric population, as well as the pleasures and challenges of working with different age groups.
4. Identify and understand the differences in the scoring procedures and rules for sleep and respiratory events, comparing the adult and the pediatric criteria.

11:00 - 11:35   Indications for pediatric polysomnography and difference between adult vs. pediatric PSG
Jacinthe Lavergne, BSc, RRT, RPSGT, Montreal Children's Hospital

11:35 - 12:10   Infant sleep vs. adult sleep including the recording and scoring of respiratory events
 K. Jeremy Gibbons, BSc, RPSGT, Hospital for Sick Children, Toronto

12:10 – 12:30   Question Period

12:30 – 13:30   **LUNCH (Trinity Ballroom)**
POSTER VIEWING (York A/B)

12:30 – 13:30   **Lunch Hour DISCUSSION I (Bay Room)**
Discussion on New Scoring Rules for Francophone Technologists
Hosted by Natalie Morin, RPGST, CSS member-at-large (Technologist)

13:30 – 14:15   **KEYNOTE 3 (Grand Ballroom)**
Chair: Penny Corkum, PhD, Dalhousie University
Sleep & Sleepiness in Adolescents
Dr. Mary A. Carskadon, PhD, Brown University, USA

14:15 – 15:00   **KEYNOTE 4 (Grand Ballroom)**
Chair: Marie Dumont, PhD
Melatonin and Light Treatment in Dementia
Dr. Eus Van Someren, PhD, Netherlands Institute for Neuroscience and VU Medical Center, Netherlands

15:00 – 15:30   **COFFEE BREAK (Trinity Ballroom)**
                                    POSTER VIEWING (York A/B)

15:30 – 17:00   **SYMPOSIUM 6 (Salon C)**
                                    Insomnia Comorbid with Medical and Mental Disorders:
                                    Nature, Impact, and Management
                                    Chair: Geneviève Belleville, PhD, Université du Québec à Montréal
                                    & Centre de recherche Fernand-Seguin, Hôpital Louis-H-Lafontaine.

Insomnia with a comorbid or concomitant medical or mental disorder is often conceptualized as "secondary insomnia". It is therefore assumed that attention and appropriate care for the "primary disorder" will diminish the associated sleep disturbances. Data suggesting otherwise has been accumulating. The reduction of symptoms of a primary mental or medical disorder does not always result in amelioration of associated insomnia. Further, comorbid sleep disturbances may negatively affect the course of a primary disorder, as well as its treatment. Further information about comorbid insomnia with various medical or mental disorders will allow a better understanding of the pathophysiology of sleep disturbances and the development optimal treatment strategies.

Learning Objectives:
1. Review the latest findings and new ideas related to comordid insomnia among individuals suffering from various medical and mental disorders.
2. Describe relationships between insomnia and the course of its comorbid disorder, as well as its impact on psychological distress and cognitive functioning.
3. Discuss applications of cognitive-behavioral strategies to address comorbid insomnia in populations suffering from various medical and mental disorders.
Note: Each talk is followed by a 5-minute question period.

15:30 - 15:40   Heterogeneous Insomnia Subtypes: Implications for Understanding the Arousal Construct and its Relation to Comorbidity and Spectral EEG
                              Colleen E. Carney, PhD, Ryerson University

15:45 - 15:55   Sleep and ADHD in Children: Does Fidgety Philip Fidget All Night?
                              Vincent Moreau, PhD, candidate, Université Laval

16:00 - 16:10   The Impact of CBT for Anxiety Disorders on Sleep Disturbances: A Meta-Analytic Review
                              Geneviève Belleville, PhD, Universitè du Quèbec à Montréal

16:15 - 16:25	Insomnia Comorbid with Cancer: Epidemiology and Management
	Josée Savard, PhD, Université Laval
16:30 - 16:40	Nature and Behavioral Treatments of Sleep Disturbances in Military
	Veterans with Posttraumatic Stress Disorder
	Anne Germain, PhD, University of Pittsburgh
16:45 - 17:00	Panel Discussion and Question Period

15:30 – 17:00 **SYMPOSIUM 7 (Salon D)**
Sleep Disturbances during Pregnancy and Menopause
Chair: Helen S. Driver, PhD, Queen's University and Kingston General Hospital.

Women often develop complaints of poor sleep in relation to the pivotal periods of pregnancy and menopause that are associated with changing hormone profiles, and changes in life-style and/or environment. While unrefreshing sleep and excessive daytime sleepiness may be anticipated during these periods in a woman's life, these complaints should also be considered as indicative of a primary sleep disorder including sleep-disordered breathing. During pregnancy, women have traditionally been thought to be protected from developing breathing disorders during sleep, but there is growing concern regarding increased risk for obstructive sleep apnea (OSA) in this population particularly in women with preeclampsia and with the increasing prevalence of obesity. Sleep problems occur in 50-80% of menopausal women, with vasomotor symptoms (hot flashes) reported by approximately 75% of postmenopausal women and 40% of perimenopausal women. This symposium will address important aspects regarding causes of disturbed sleep during pregnancy and menopause with a focus on therapeutic interventions.

Learning Objectives:
1. Review causes of disturbed sleep and the potential impact of sleep disruption during pregnancy, with an emphasis on sleep apnea.
2. Address areas of clinical concern, including the medical and psychiatric complications of insomnia and primary sleep disorders during pregnancy.
3. Discuss the effects of the climacterium (menopausal transition) on sleep in particular with regard to insomnia and sleep-disordered breathing.
4. Evaluate various treatment options, including hypnotic medication, hormone therapy and non-pharmacologic approaches.

15:30 - 15:45	The Impact of Sleep Disruption during Pregnancy and the Post-partum Period
	Eileen P. Sloan, PhD, MD, University of Toronto
15:45 - 16:00	Sleep-disordered Breathing during Pregnancy, including Discussion on Case Studies
	Helen S. Driver, PhD, RPSGT, DABSM, Queen's University
16:00 - 16:10	Questions – Pregnancy
16:10 - 16:30	Dealing with Menopausal Sleep Disturbances
	Päivi Polo-Kantola MD, PhD, Turku University
16:30 - 16:45	Insomnia: Therapeutic Options for Women
	Judith R. Davidson, PhD, Queens University
16:45 - 16:55	Questions – Menopause
16:55 - 17:00	Panel Discussion and Question Period
15:30 – 17:00	**TECHNICAL WORKSHOP 2 (Salon A/B)**
	Sensor Technology for Respiratory Recordings during Polysomnography
	Chairs: Kenan Hoelke, RPSGT, for the CSS
	Glenna Labelle, RRT, Sleep and Home Respiratory Product Manager International Group, (Respironics) Philips Home Healthcare Solutions

This workshop is intended to provide participants with a working knowledge of the basic operating principles of devices used in polysomnography to record indications of the adequacy of ventilation and respiration. Along with a solid understanding of how devices work, discussion of the best practices for use and care of the devices, and scoring of the recordings will be included in the workshop. Basic operating principles of devices will be described and compared with alternative measures, for example for recording respiratory effort - esophageal pressure versus intercostal EMG versus piezo belts versus Impedance versus inductive plethysmography. Comparisons between manufacturers are beyond the scope of this workshop. Where there is a significant difference in operating principles of similar devices between manufactures, this can be

pointed out but specific operational differences between two similar devices used for the same respiratory recording classification (e.g. airflow, effort or SpO2) are left for the participants to explore in depth with the manufacturers directly in the exhibit hall.

Note: This workshop will be geared more towards the Sleep Technologist, though Physicians who are interested in the sensor technology would also benefit from attending. Learning Objectives:

1. Understand basic operating principles of sensors used to record airflow and respiratory effort.
2. Recognize which technology is best for measuring a specific physiological signal.
3. Appreciate how to use the sensor to obtain the best possible signals.
4. Explore how to perform effective troubleshooting with each of these types of sensors.

15:30 – 16:10	Flow and Respiratory Sensors
	Don C. Bradley, BSc, Founder and CTO Braebon Medical Corporation
16:10 – 16:50	Recording Respiratory Effort, O2 and CO2
	Pam Minkley, RPSGT, RRT, CPFT, (Respironics) Philips Home Healthcare Solutions
16:50 - 17:00	Integration and Summary
17:00 – 18:00	**ANNUAL GENERAL MEETING (for CSS Members)(Salon D)**
19:00 – 21:00	**RECEPTION: SALSA PARTY (Salon A/B)**
	Note: you may purchase tickets for $20 at the Registration Desk.

TUESDAY, APRIL 28, 2009

08:30 – 09:15   **KEYNOTE 5 (Grand Ballroom)**
Chair: John Fleming, MD
Insomnia: From Epidemiology to Treatment
Dr. Charles Morin, PhD, Laval University, Canada

09:15 – 10:00   **KEYNOTE 6 (Grand Ballroom)**
Chair: Eileen Sloan, PhD, MD
Sleep, Seasonality and Mood
Dr. Ruth Benca, MD, PhD, University of Wisconsin-Madison, USA
Note: Each lecture includes an 8 minute question period

10:00 – 10:30   **COFFEE BREAK (Trinity Ballroom)**
POSTER VIEWING (York A/B)

10:30 – 12:30   **SYMPOSIUM 8 (Salon C)**
Fatigue in the Workplace: Contributions of Irregular Work Schedules
and Sleep Disruption
Chair: Diane B. Boivin, MD, PhD, McGill University, Montréal,
Québec.

Fatigue is a prevailing complaint in modern society. It is a complex problem that requires a comprehensive, multilevel approach, since it is affected by several factors. Working on atypical schedules is an important contributor to fatigue and has important socioeconomic impacts as it leads to worker impairment and increased risk of accidents. This symposium will address contributors to fatigue when working on atypical schedules and will discuss intervention strategies designed to alleviate it.

Learning Objectives:
1. Understand the impact of shift work and fatigue on mental and physical health.
2. Understand how irregular work schedules can lead to sleep disruption, vigilance impairment and increased risk of work injuries.
3. Understand how the history of light exposure can affect circadian adaptation to atypical work schedules.
Note: Each talk is followed by a 5-minute question period.

10:30 -10:35	Introduction
	Diane B. Boivin, MD, PhD, McGill University
10:35 -10:55	Behavioral and Physiological Adaptation to Shift Work
	Diane B. Boivin, MD, PhD, McGill University
11:00 -11:20	The Calgary Police Service Health and Human Performance Research Initiative: Sleep Deprivation and Vigilance Field Research: Application and Knowledge Transfer
	Charles H. Samuels, MD, CCFP, DABSM, University of Calgary
11:25 -11:45	Fatigue in Students who Work during the School Year
	Luc Laberge, PhD, Université du Québec à Chicoutimi
11:50 -12:10	Manipulation of Daytime Light Exposure Improves Circadian Adaptation in a Simulation of Night Work
	Marie Dumont, PhD, Université de Montréal
12:15 -12:30	Panel Discussion and Question Period
10:30 – 12:30	**SYMPOSIUM 9 (Salon D)**
	The Significance of the Sleep Spindle during Sleep: Basic and Clinical Data
	Co-Chairs: Célyne H. Bastien, PhD, Université Laval
	Carlyle Smith, PhD, Trent University

The study of phasic events during sleep has recently become a key topic in the search for markers of sleep quality. Given the new AASM scoring rules that confer phasic events a significant role, this symposium is timely as it should lead the audience toward a better understanding of the sleep spindle and its functional significance, albeit as a sleep protection mechanism, sleep learning marker or sleep pathology determinant.

Learning objectives:
1. Provide an overview of current research in the area of 'phasic events in sleep', and especially the sleep spindles, through new data on a broad array of topics, including aging, developmental disorders and insomnia.

2. Examine the significance of spindles within sleep and how they characterize/define sleep quality or are markers of daily performance.

3. Cover both basic and clinical approaches to sleep quality while using automatic and electrophysiological techniques to the understanding of brain-behaviour relationships in the context of sleep research.

Note: Each talk is followed by a 5-minute question period.

10:30 - 10:40	Introduction: The Sleep Spindle: Basic Neurophysiology and Functions Derk-Jan Dijk, PhD, University of Surrey
10:40 - 11:00	Age, Gender and Topographical Differences in Sleep Spindles Julie Carrier, PhD, Université de Montréal
11:05 - 11:25	Changes in the Number and Density of Sleep Spindles following Learning of a Simple Word-pairs Declarative Task Carlyle Smith, PhD, Trent University
11:30 - 11:50	EEG Sleep Spindles in Pedopsychiatric Disorders: Autism, ADHD and Anxiety Roger Godbout, PhD, Université de Montréal
11:55 - 11:15	Sleep Spindles in Insomnia Célyne Bastien, PhD, Université Laval
12:20 - 12:30	Panel Discussion and Question Period
10:30 – 12:30	**TECHNICAL WORKSHOP 3 (Salon A/B)** Portable Monitoring in Sleep Chair: Natalie Morin, RPSGT, CSS Member-at-large (Technologist), Sleep Strategies

Are we entering the era of portable monitoring (PM) for the diagnosis of obstructive sleep apnea (OSA)? With the growth in available devices for PM, we must ensure that the technology is robust and accurate. There is ongoing discussion and research on algorithms for the utilization of portable monitors for diagnosing OSA, including the stratification of patients, cost-benefit analysis, procedural and management strategies for how to best to employ PM as a diagnostic strategy for OSA. This workshop is designed

to provide an overview of portable alternatives to full PSG testing for situations such as: regions with long wait lists; patients with reduced mobility; pre-operative apnea screening; oral appliance follow-up. We will also have a lively discussion of how PM has been pioneered and utilized in the Canadian setting, with views on options for the future direction of PM in Canada.

Note: This workshop will be of interest for Sleep Technologist and Physicians who are interested in the utility and developments of portable monitors for sleep and screening of sleep disorders, specifically obstructive sleep apnea (OSA).

Learning Objectives:
1. Review the various levels of sleep studies and requirements for portable monitors.
2. Explanation of the AASM guidelines for at home-testing.
3. Evaluating the effectiveness of the various portable diagnostic set-up locations.
4. Explore the various options available in portable monitoring devices.
5. Compare scoring respiratory events from home studies with full-PSG.

10:30 – 11:00	Levels of Studies and Options for the Home Testing Environment Robert Safron, RRT, MBA (Covidien)
11:00 – 11:30	AASM Guidelines for at Home-testing, Scoring and Reporting Ron Fligge, RRT (Respironics, Philips Home Healthcare Solutions)
11:30 - 12:15	Debate: Yays and Nays for the use of PM in the Canadian Setting Yay-side: Kristin Fraser, MD, DABSM (Alberta) Nay-side: Judith Leech, MD, DABSM (Ontario)
12:15 - 12:30	General Discussion and Opinions
12:30 – 13:00	**LUNCH (Trinity Ballroom)** **POSTER VIEWING (York A/B)**
12:30 – 13:30	**Lunch hour DISCUSSION II – for RPSGT Educators (Bay Room)** BRPT Exam Eligibility Requirements for Canadians Hosted by Roger Godbout, PhD, RPSGT (CSS representative on the BRPT) and Helen Driver, PhD, RPSGT, DABSM (CSS President)

13:30 – 15:00   **SYMPOSIUM 10 (Salon C)**
                          Sensory and Motor Regulation of Upper Airways in Sleep: Relevance
                          to Sleep-Disordered Breathing
                          Chair: John Peever, PhD, Systems Neurobiology Laboratory, Department
                          of Cell & Systems Biology and Physiology, University of Toronto.

Disturbances in motor control and sensory integration underlie some of the major sleep disorders including obstructive sleep apnea and disordered breathing in newborns. The primary goal of this symposium is to provide an up-to-date understanding of the motor and sensory regulation of upper airway control during sleep and wakefulness. This symposium will also address important aspects of upper airway function related to the pathogenesis of sleep disordered breathing in both adults and newborns.

Objectives:
1. Understand the neurophysiology of respiratory motor regulation across the sleep cycle.
2. Discuss novel pharmacological strategies for improving upper airway motor function in sleep as it relates to treating obstructive sleep apnea.
3. Understand the sensory and motor control of laryngeal muscles in sleep and waking in the newborn.
4. Understand the role of upper airway trauma and altered sensation associated with repeated airway obstructions in the pathogenesis of OSA.
Note: Each talk is followed by a 5-minute question period.

13:30 - 13:45   Neurobiology of Upper Airway Motor Control in Sleep and Waking
                       Richard Horner, PhD, University of Toronto
13:50 - 14:05   Novel Pharmacological Strategies for Improving Upper Airway
                       Motor Function in Sleep: Relevance to Obstructive Sleep Apnea
                       John Peever, PhD, University of Toronto

14:10 - 14:25   Control of Laryngeal Muscles During Sleep in the Newborn
                       Jean-Paul Praud, MD, PhD, Université de Sherbrooke

14:30 - 14:25   Neuromuscular Function of the Upper Airway in Obstructive
                       Sleep Apnea
                       R. John Kimoff, MD, FRCP(C), McGill University

14:50 - 15:00   Panel Discussion and Question Period

13:30 – 15:00   **SYMPOSIUM 11 (Salon D)**
Children's Sleep and Daytime Functioning
Organizer / Chair: Penny Corkum, PhD, Registered Psychologist, Associate Professor, Clinical Psychology Program, Department of Psychology, Dalhousie University.

Age-appropriate quantity and quality of sleep is required for optimal development and functioning in childhood. Sleep problems in children are highly prevalent, with an estimated 25% (range 15-40%) of children experiencing difficulties with sleep (Owens et al., 2007). High rates of sleep problems in children, as well as the fact that children in general are sleeping less now than previously, underscore the importance of research examining the impact of sleep problems on child functioning. Sleep problems which result in fragmented sleep or shorter sleep duration may cause daytime sleepiness and reduced alertness, resulting in compromised daytime functioning in a number of areas. There is a growing body of research demonstrating that poor sleep in children is associated with deficits in working memory, executive functioning, and attention, as well as poorer academic achievement, more behavioural problems, difficult temperament, increased negative mood, and poor emotional regulation (Sadeh, 2007).

Learning Objectives:
1. Explore sleep issues in typically developing children as well as children with mental health disorders.
2. Describe the types and prevalence of sleep problems in children.
3. Explain the association between sleep and daytime cognitive functioning.
4. Understand if sleep helps to enhance motor skills learning and the potential negative impact of sleep disordered breathing.
5. Highlight the changes that result from a behavioural intervention for sleep problems.
Note: Each talk is followed by a 5-minute question period

13:30 - 13:35   Introduction to Symposium

13:35 - 13:50   Prevalence of Sleep Problems in Children
Dominique Petit, PhD, University of Montreal

13:55 - 14:10   The Interplay Between Sleep, Arousal and Neurobehavioral Functioning in School-Age Children
Eva Monson and Reut Gruber, Ph.D, McGill University

| 14:15 - 14:30 | Overnight Learning in Children with ADHD and Controls |
| | Mary A. Carskadon, PhD, Brown University |

14:35 - 14:50	The Benefits of Behavioural Interventions for Daytime Functioning
	in Children with Pediatric Insomnia
	Penny Corkum, PhD, Dalhousie University

| 14:55 - 15:00 | Panel Discussion and Question Period |

13:30 – 15:00   **TECHNICAL WORKSHOP 4 (Salon A/B)**
Coping with Night Work in the Sleep Lab
Organizer / Moderator: Marie Dumont, PhD, Montreal University
and Sacre-Coeur Hospital of Montreal.
Co-Chair: Lori Davis, BSc, RCPT(P), RPSGT West Park Hospital,
Ontario

Sleep technologists have to work nights. For most night workers, staying awake at night and sleeping in the daytime represent a difficult challenge, and negative health outcomes have been associated with night work. Since night work cannot be avoided, various strategies are used by night workers, and studied by researchers, to minimize the negative effects of night work. This workshop aims at putting together the field experience of night technicians with the research expertise of sleep and circadian researchers to determine 1) what can be done now to help night workers and 2) what do we need to know to improve the well-being of night workers. The format will include a review of current recommendations for night workers, the presentation of a survey among sleep technicians, commentaries from the panel participants, and a general discussion with the audience.

Learning Objectives:
1. Describe the main difficulties associated with night work.
2. Understand the conflicts between night work and the internal biological clock.
3. List recommendations from scientists and from fellow technicians to help coping with night work.
4. Identify priorities for future research and for knowledge transfer to help night workers, with specific attention to sleep technicians.

| 13:30 - 13:35 | Presentation of the Objectives and Format of the Workshop |
| | Lori Davis, BSc, RCPT(P), RPSGT, West Park Hospital, Ontario |

13:35 - 13:55	Recommendations for Night Workers – A View from Scientists Marie Dumont, PhD, Chronobiology Laboratory, Sacre-Coeur Hospital and Montreal University
13:55 - 14:15	Recommendations for Night Workers – Results of a Survey Among Canadian Sleep Technicians. Hélène Blais, BA, MEPT, Center for the Study of Sleep and Biological Rhythms, Sacre-Cœur Hospital of Montreal
14:15 - 14:55	Commentaries from the Panel Participants and Panel Discussion Charles Samuels, MD, CCFFP, DABSM, Centre for Sleep and Human Performance and University of Calgary Diane Boivin, MD, PhD, Center for Study and Treatment of Circadian Rhythms, Douglas Mental Health University Institute and McGill University, Montreal Marc Hébert, PhD, ORL-Ophthalmology Department, Université Laval, Québec
14:55 - 15:20	General discussion on suggestions and priorities
15:20 - 15:30	Concluding remarks by the co-chairs
15:00 – 15:30	**COFFEE BREAK (Trinity Ballroom)** **POSTER VIEWING (York A/B)**
15:30 – 16:15	**KEYNOTE 7 (Grand Ballroom)** Chair: Richard Horner, PhD Sleep Apnea and Cardiovascular Disorders Dr. Douglas Bradley, MD, University of Toronto, Canada
16:15 – 17:30	**POSTER DATA BLITZ (Grand Ballroom)** Chairs: Kimberly Cote, PhD, Brock University Célyne Bastien, PhD, Université Laval

To showcase the diversity and excellence of Canadian sleep research, a number of poster submissions will be selected to give a one-minute, one-slide oral presentation of their work.

17:30	**Evaluation And Closing Remarks** Meeting Ends

Notes

Poster Presentations

Sleep Deprivation

P001   EFFECTS OF SLEEP DEPRIVATION AND DAYTIME RECOVERY SLEEP ON
SELF-EVALUATION ACCURACY
Martin N.[1,2,] Dostie V. [1,2,] and Carrier J. [1,2]
[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur
de Montréal, Montréal, QC, Canada; [2]Département de psychologie, Université
de Montréal, Montréal, QC, Canada

P002   SLEEP DURING PEDIATRIC HOSPITALIZATION
Stremler, R[1, 2,] Weston, J[1,] Dhukai, Z[1,] Lumb, A[1,] Wong, L[1,] Adams, S[2,] Weiss, S[2,]
Parshuram, C2
[1] Lawrence S. Bloomberg Faculty of Nursing, University of Toronto, Toronto,
Canada; [2] The Hospital for Sick Children (SickKids), Toronto, Canada

P003   AGE-RELATED DIFFERENCES IN N-REM SLOW OSCILLATIONS: EFFECTS
OF SLEEP DEPRIVATION
Lafortune, M.[1,2,] Viens, I.[1,2,] Jacob-Lessard, A.[1,2,] Poirier, G.[1,2,]Vandewalle, G.[1,2,]
Barakat, M.[1,2,] Martin, N.1,2, Filipini, D.[1,] & Carrier J.[1,2]
[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur
de Montréal, QC, Canada; [2]Département de psychologie, Université de Montréal,
QC, Canada

P004   IMPACT OF ACUTE SLEEP RESTRICTION ON CORTISOL AND LEPTIN
LEVELS IN YOUNG WOMEN.
Omisade A.[1,] Buxton M. O.[2] and Rusak B.[1,3,4]
Departments of [1]Psychology, [3]Psychiatry, and [4]Pharmacology, Dalhousie
University, Halifax, NS, Canada; [2]Department of Medicine, Brigham and
Women's Hospital, Harvard Medical School, Boston, MA, USA

P005   SHORT DAILY SLEEP OPPORTUNITIES AMELIORATE THE EFFECTS OF
SLEEP LOSS ON ENERGY EXPENDITURE IN RATS
Caron A.M. and Stephenson R.
Department of Cell & Systems Biology, University of Toronto, Toronto, ON,
Canada M5S 3G5

Sleep & Performance

P006   LOWER SUBJECTIVE SOCIOECONOMIC STATUS IS ASSOCIATED WITH SHORTER SLEEP DURATION, POORER SLEEP QUALITY, AND MORE DAYTIME SLEEPINESS IN CHILDREN AND ADOLESCENTS
Jarrin, D.C.[1,] Silverstein, J.E. [1,] & McGrath, J.J.[1]
[1]Department of Psychology, Concordia University, Montreal, QC, Canada

P007   SUBJECTIVE EXCESSIVE DAYTIME SLEEPINESS IN A COMMUNITY-BASED SAMPLE: FREQUENCY AND ASSOCIATED FACTORS
Beaulieu-Bonneau S., Fortier-Brochu E., LeBlanc M., Vallières A., and Morin C. M.
École de psychologie, Université Laval, Québec, Canada

P008   DO DIFFERENT LEVELS OF SLEEPINESS MODULATE EFFECTS OF CAFFEINE ON VIGILANCE?
Dostie, V[123,] Robillard, R.[123] Filipini, D[3,] Selmaoui, B.[4] and Carrier, J.[123]
[1]Département de psychologie, Université de Montréal, Montréal, QC, Canada, [2]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Montréal, QC, Canada, [3]Institut universitaire de gériatrie de Montréal, Montréal, QC, Canada, [4]Immunology, Charles River Labotories-Preclinical services, Seneville, QC, Canada

P009   BENEFITS OF A DAYTIME NAP FOR STUDENTS ON A UNIVERSITY CAMPUS
Black J., Ling E., and Cote K.
Department of Psychology, Brock University, St. Catharines, ON, Canada

P010   SLEEP QUALITY AND NEXT-DAY PVT PERFORMANCE AFTER HEAVY ALCOHOL CONSUMPTION
Arnedt J.T.[1,] Almeida A.[2,] Hunt S.[2,] Gokhale M.[3,] Rohsenow D.[4,] & Howland J.[2]
[1]Sleep and Chronophysiology Lab, Department of Psychiatry, University of Michigan; [2]Department of Social and Behavioral Sciences, Boston University School of Public Health; [3]Data Coordinating Center, Boston University School of Public Health; [4]Center for Alcohol and Addiction Studies, Brown University

P011   EFFECTS OF CONSOLIDATION OF PROCEDURAL MOTOR MEMORY TRACES ON SLOW AND FAST SPINDLES
Barakat M.[1,3,] Doyon J.[1,3,] Debas K.[1,3,] Morin A.[1,3,] Poirier G.[2,] Viens I.[2,3,] Lafortune M.[2,3,] Vandewalle G.[1,2,] Carrier J. [1,2,3]

[1] Unité de Neuroimagerie Fonctionnelle, Centre de Recherche de l'Institut Universitaire de Gériatrie de Montréal, Montréal, QC, Canada; [2] Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Montréal, QC, Canada ; [3] Centre de Recherche en Neuropsychologie et Cognition, Université de Montréal, Montréal, QC, Canada

P012   POST-LEARNING REDUCTION IN SLOW-WAVE SLEEP AFTER MENTAL PRACTICE OF THE ROTARY PURSUIT TASK

Nesbitt D., Peters K.R., DeCicco T. and Smith C.T.

Department of Psychology, Trent University, Peterborough, Ontario, Canada

P013   IDENTIFICATION OF A RAPID EYE MOVEMENT SLEEP WINDOW FOR THE WIN-SHIFT RADIAL ARM MAZE TASK

Madore A.R.[1,] DeLay S.[1,] Legault M.G.[1]

[1]Department of Psychology, Laurentian University, Sudbury, ON, Canada

Neuropsychology & Psychiatry

P014   SLEEP EEG IN AUTISM AND PERFORMANCE ON THE EMBEDDED FIGURE TEST

Sophie Tessier,[1,4] Christianne Bolduc,[1,4] Élyse Limoges,[1,4] Édith Ménard,[4] Laurent Mottron,[2,3,4] Roger Godbout.[1,2,3,4]

[1]Sleep Laboratory & Clinic, Hôpital Rivière-des-Prairies; [2]Department of Psychiatry, Université de Montréal (Québec) Canada; [3]Neurodevelopmental Disorders Program, Hôpital Rivière-des-Prairies; [4]Centre de recherche Fernand-Seguin, Hôpital Rivière-des-Prairies, Montréal (Québec) Canada.

P015   DISSOCIATION BETWEEN STAGE 2 SIGMA EEG ACTIVITY AND SLEEP SPINDLE DENSITY IN HIGH-FUNCTIONING AUTISM

Annie-Claude Rochette,[1,4] Élyse Limoges[1,4,] Élyse Chevrier[1,3,4,] Laurent Mottron[2,3,4] Roger Godbout.[1,2,3,4]

[1]Sleep Laboratory & Clinic, Hôpital Rivière-des-Prairies; [2]Department of Psychiatry, Université de Montréal (Québec) Canada; [3]Neurodevelopmental Disorders Program, Hôpital Rivière-des-Prairies; [4]Centre de recherche Fernand-Seguin, Hôpital Rivière-des-Prairies, Montréal (Québec) Canada.

P016   SLEEP PATTERNS IN ADOLESCENTS WITH BIPOLAR DISORDER OR
        BORDERLINE PERSONALITY DISORDER

Huynh C.[3], Guilé J.M.[1,3,] Breton J.J.[1,3,] Cohen D.[2,] Chevrier É.[4] and Godbout
R.[1,4]

[1]Department of Psychiatry, Université de Montréal (Québec) Canada; [2]Université
Pierre et Marie Curie, Hôpital Pitié-Salpétrière, Psychiatrie de l'enfant et de
l'adolescent, Paris, France; [3]Pedopsychiatry Program, Mood disorders Clinic,
Hôpital Rivière-des-Prairies; [4]Neurodevelopmental Disorders Program,
Sleep Laboratory & Clinic, Hôpital Rivière-des-Prairies; 5Centre de recherche
Fernand-Seguin, Hôpital Rivière-des-Prairies, Montréal (Québec) Canada

P017   SLEEP/WAKE DISTURBANCE FOLLOWING SEVERE TRAUMATIC BRAIN
        INJURY; IMPACT ON RECOVERY OF COGNITIVE-COMMUNICATION
        FUNCTION: A CASE STUDY

Wiseman-Hakes, C.[1,2] , Murray, BJ, [2,3,4,] Victor, JC[5]

[1] Graduate Dept. of Rehabilitation Science, [2] Toronto Rehab Institute, [3] University
of Toronto, Dept. of Neurology, [4] Sunnybrook Hospital, [5] University of Toronto
Dalla Lana School of Public Health

P018   DAYTIME SLEEPINESS AFTER MODERATE/SEVERE TRAUMATIC BRAIN
        INJURY: PRELIMINARY FINDINGS

Beaulieu-Bonneau S., Roy M.-A., and Morin C. M.
École de psychologie, Université Laval, Québec, Canada

P019   RELATIONSHIP AMONG SUBJECTIVE SLEEP COMPLAINTS, HEADACHES,
        AND MOOD ALTERATIONS FOLLOWING A MILD TRAUMATIC BRAIN INJURY

Chaput, G[1, 2,] Giguère, J-F. [2,] Chauny, J-M.[2,] Denis, R.[2 ,] Lavigne, G.[1, 2]

[1] Facultés de médecine dentaire et de médecine, Université de Montréal, CP
6128, succursale Centre-ville, Montréal, Que, Canada, H3C 3J7. [2] Division of
Trauma Research, Department of Surgery, Hôpital du Sacré-Cœur de Montréal,
5400 Boul Gouin Ouest, Montreal, Que, Canada, H4J 1C5

P020   SPONTANEOUS K-COMPLEXES IN STAGE 2 SLEEP ARE REDUCED
        FOLLOWING TRAUMATIC BRAIN INJURY (TBI)

Milner C.E. and Cote K.A.
Department of Psychology, Brock University, St. Catharines, ON, Canada

P021   INVESTIGATION OF SLEEP IN CHRONIC TREATMENT-RESISTANT DEPRESSED PATIENTS
Ahmadi N, Shapiro CM
Psychiatry and Sleep Research Unit, University Health Network, Toronto, ON, Canada

P022   A PRELIMINARY ATTEMPT AT DEFINING 'SLEEP MARKERS OF DEPRESSION' CATEGORICALLY AND EXAMINING THEIR ASSOCIATION WITH SUBJECTIVE LOW MOOD.
Saleh, P[1, 2, 3,] Shahid, A[1, 2,] Chung, F[4] and Shapiro, CM[1, 2, 3]
[1]Department of Psychiatry, University of Toronto; [2]Youthdale Child and Adolescent Sleep Centre, Toronto, Ontario; [3]Department of Cell and Systems Biology, University of Toronto; [4]Department of Anesthesia, Toronto Western Hospital

Insomnia

P023   EPIDEMIOLOGY OF INSOMNIA IN A CANADIAN POPULATION-BASED SAMPLE
LeBlanc, M[1,2,] Bélanger, L. [1,2,] Mérette, C. [2,3,] Savard J. [1,4,] Morin, C.M. [1,2]
[1] École de psychologie, Université Laval, Québec, Canada. [2] Centre de recherche Université Laval-Robert-Giffard, Québec, Canada. [3] Département de psychiatrie, Université Laval, Québec, Canada. [4] Centre de recherche en cancérologie de l'Université Laval, l'Hôtel-Dieu de Québec, Québec, Canada.

P024   SLEEP DISTURBANCES IN CHINESE EARTHQUAKE VICTIMS
Shen J.[1,] Wang L.2, Shi Z.[2,] Zhang Y[2,] Xin Y.[3,] Wang W.[2,] Shan S.[4,] Zheng S.[5,] Shapiro C.M.[1]
[1]Department of Psychiatry, University of Toronto, Toronto, ON, Canada, [2] Institute of Psychology, Chinese Academy of Sciences, Beijing, China, [3] Department of Psychology, Southwest University of Science and Technology, Mianyang, Sichuan, China, [4]Hong Fook, Mental Health Association, Toronto, ON, Canada, [5]Mandarin Clinic, Toronto, ON, Canada

P025   ARE INSOMNIA SYMPTOMS STABLE OVER TIME? A 5-YEAR PROSPECTIVE STUDY IN THE GENERAL POPULATION
Fortier-Brochu, É., Ivers, H., Beaulieu-Bonneau, S., LeBlanc, M. & Morin, C. M.
Université Laval, Québec, Canada

P026   INSOMNIA AS A RISK FACTOR FOR HEALTH PROBLEMS: A LONGITU-
        DINAL STUDY

Perozzo, C.[1,2,] Gagnon, C.[1,2,] LeBlanc, M.[1,2,] Savard, J.[1,3,] & Morin, C. M.[1,2]

[1] École de psychologie, Université Laval, Québec, QC, Canada; [2] Centre d'étude
des troubles du sommeil, Centre de recherche Université Laval-Robert-Giffard,
Québec, QC, Canada; [3] Centre de recherche en cancérologie de l'Université
Laval, Hôtel-Dieu de Québec, Québec, QC, Canada

P027   CLASSIFICATION OF INSOMNIA SUFFERERS BASED ON LABORATORY
        PSG RECORDINGS AND SUBJECTIVE SLEEP REPORTS

St-Jean G.[1-2] and Bastien C.H. [1-2]

[1] École de psychologie, Université Laval, Québec, Canada; [2] Laboratoire de
neurophysiologie humaine, Centre de recherche Université Laval Robert-Giffard,
Québec, Canada

P028   ERP MEASURES DURING SLEEP IN PSYCHOPHYSIOLOGICAL AND
        PARADOXICAL INSOMNIA SUFFERERS

Turcotte I.[1,] Adam A-M.[1,] Lecarpentier M.[1] and Bastien C.[1]

[1] Department of Psychology, Laval University, Quebec, Quebec, Canada.

P029   EVENT-RELATED POTENTIALS (ERPs) REVEAL FAILURE TO INHIBIT
        STIMULI DURING THE PRE-SLEEP WAKING PERIOD FOR PATIENTS
        WITH SLEEP-ONSET INSOMNIA

Kertesz R. S., Cote K. A.

Department of Psychology, Brock University, St. Catharines, ON, Canada

P030   BAROREFLEX SENSITIVITY DURING SLEEP AND WAKEFULNESS IN
        PRIMARY INSOMNIA : PRELIMINARY RESULTS

Lorraine Fradette, BSc[1,2,] Pennestri Marie-Hélène, BSc[1], Jacques Montplaisir,
MD, PhD[2,3] , Charles M. Morin, PhD[4,5,] Roberto Colombo BEng6, Paola
A.Lanfranchi, MD, MSc[2,7]

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Québec, Canada; [2] Sleep Disorders Center, Hôpital du Sacré-Coeur de Montréal,
Montréal, Québec, Canada; [3] Department of Psychiatry, Université de Montreal,
Montréal, Québec, Canada.; [4] École de Psychologie, Université Laval, Québec,
Québec, Canada; [5] Centre d'étude des troubles du sommeil, Centre de recherche

Université Laval-Robert-Giffard, Québec, Québec, Canada; [6] Bioengineering Department, Salvatore Maugeri Foundation, Veruno, Italy; [7] Department of Medicine, Division of Cardiology, Hôpital du Sacré-Coeur de Montréal and Université de Montréal, Québec, Canada

P031   DOES CBT FOR INSOMNIA ALTER SLEEP MISPERCEPTIONS?
Moreau V., Gagnon C., Lamy M., Ivers H. and Morin C. M.
École de psychologie, Université Laval, Québec, QC

P032   IMPACT OF CBT FOR INSOMNIA AND CBT COMBINED WITH MEDICATIONS ON DAYTIME FUNCTIONING
Bélanger, L.[1,] Sanchez-Ortuño, M.[1,2,] Ivers, H[1,] Morin, C. M.[1]
[1] Université Laval, Québec, Canada; 2 Universidad de Murcìa, Murcìa, Spain

P033   ARE THE EFFECTS OF INSOMNIA TREATMENT ON DAYTIME MEASURES CLINICALLY IMPORTANT?
Sanchez-Ortuño, M.[1,2,] Bélanger, L.[1,] Ivers, H.[1,] Morin, C. M.[1]
[1] Université Laval, Québec, Canada; [2] Universidad de Murcìa, Murcìa, Spain

Dreaming

P034   SCHIZOPHRENIA-LIKE COGNITION IN REM SLEEP MENTATION
Stenstrom P.[1,2,] Nielsen T.[1,3,] Solomonova E.[1,2,] Lara-Carrasco J.[1,2]
[1] Sleep Research Center, Hôpital du Sacré-Coeur de Montreal, Québec; [2] Psychology Department, Université de Montréal, Québec; [3] Psychiatry Department, Université de Montréal, Québec

P035   LUCID DREAMING IS ASSOCIATED WITH SLEEP PARALYSIS BUT NOT NIGHTMARES
Solomonova, E.[1,2,] Nielsen T.[1,3,] Stenstrom, P.[1,2]
[1] Sleep Research Center, Hôpital du Sacré-Coeur de Montreal; [2] Psychology Department, Université de Montréal; [3] Psychiatry Department, Université de Montréal .

P036   BEHAVIORAL ENACTMENT OF DREAMING IN A NORMAL POPULATION

Nielsen T.[1,] Svob C.[2,] Kuiken D.[2]

[1] Department of Psychiatry, Université de Montreal, Montreal, QC, Canada; [2] Department of Psychology, University of Alberta, Edmonton, AB, Canada.

P037   EVOLUTION OF GENDER DIFFERENCES IN THE DREAMS OF UNIVERSITY STUDENTS

Ouellet D., Duchesne-Pérusse, A., Paquette-Biron, M., Sabourin C., De Koninck J.

School of Psychology, University of Ottawa, Ottawa, ON, Canada

P038   WORD ASSOCIATIONS IMPROVE AUTOMATIC ANALYSIS OF DREAM EMOTIONAL TONE

Amini R., Ouellet D., Sabourin C., De Koninck J.

School of Psychology, University of Ottawa, Ottawa, ON, Canada

Chrononiology

P039   SCREENING FOR SLEEP QUALITY AND CHRONOTYPE IN ELITE WINTER ATHLETES

Samuels C[1,2,] Fryer S[1]

Centre for Sleep and Human Performance, Calgary, AB, Canada[1]

Faculty of Medicine, University of Calgary, Calgary, AB, Canada[2]

P040   EFFECT OF CIRCADIAN AND SLEEP-WAKE STATE ON HEART RATE VARIABILITY IN HUMANS

Philippe Boudreau[1], Guy Dumont[2], Diane B. Boivin[1]

[1] Centre for Study and Treatment of Circadian Rhythms, Douglas Mental Health University Institute, McGill University, Montreal, Quebec, Canada. [2] Department of Electrical and Computer Engineering, University of British Colombia, Vancouver, BC, Canada.

P041   EFFECT OF BRIGHT LIGHT ON PERFORMANCE OF POLICE OFFICERS ON ROTATING SHIFTS

Boudreau P.[1,] Tremblay G.M.[1,] Boivin D.B.[1]

[1] Centre for Study and Treatment of Circadian Rhythms, Douglas Mental Health University Institute, McGill University, Montréal, Québec, Canada

P042   DAYTIME SLEEPINESS AND NATURAL LIGHT EXPOSURE IN STUDENTS WHO WORK DURING THE SCHOOL YEAR: PRELIMINARY RESULTS

Martin J.-S.[1,] Hébert M.[2,] Ledoux É.[3,] Laberge L.[4]

[1] ÉCOBES, Cégep de Jonquière, Saguenay, Québec; [2] Centre de recherche Université Laval Robert-Giffard, Québec; [3] Institut de recherche en santé et en sécurité du travail, Montréal; [4] Département de psychologie, Université du Québec à Chicoutimi, Saguenay, Québec, Canada

P043   SLEEP DURATION, LIGHT EXPOSURE AND BIOMARKERS OF MELATONIN AMONG ROTATING SHIFT NURSES

Grundy A.[1,] Sanchez M.[1,] Richardson H.[1,] Tranmer J.[1,2,] Graham C.[3,] Aronson K.[1.]

[1] Department of Community Health and Epidemiology, Queen's University, Kingston, ON, Canada; [2] School of Nursing, Queen's University, Kingston, ON, Canada; [3] Department of Anatomy and Cell Biology, Kingston, ON, Canada

P044   CHANGES IN CIRCADIAN RHYTHM EVIDENT DURING AN ACUTE RANDOMIZED PLACEBO-CONTROLLED TRIAL OF METHYLPHENIDATE

Davidson, F., Ironside, S., & Corkum, P.

Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada

P045   SELECTIVE INCREASE OF SLOW WAVES SLEEP (SWS) BY A NOVEL MELATONIN PARTIAL AGONIST

R.Ochoa-Sánchez [1,] G. Spadoni [3,] A. Bedini [3,] M. Mor [4,] S. Rivara [4,] F. Fraschini [5,] G.Tarzia [3,] G. Gobbi [1,2]

[1.] Neurobiological Psychiatry Unit, Dept. of Psychiatry, McGill Univ.; [2.] Centre de Recherche Fernand-Seguin, Univ. of Montreal, Canada; [3.] Inst. of Pharmaceutical Chemistry and Toxicology, Univ of Urbino, Italy; [4.] Pharmaceutical Dept. University of Parma, Italy; [5.] Dept. of Pharmacology, Chemiotherapy and Medical Toxicology, Univ.of Milan, Italy.

P046   SHORT EXPOSURE TO BLUE-ENRICHED WHITE LIGHT DOES NOT FURTHER IMPACT ALERTNESS LEVEL WHEN USED AT THE END OF THE NIGHT

Sasseville A[1,] Houle J[1,] Hebert M[1]

[1.] CRULRG, Université Laval, Quebec City, QC, Canada

P047   POLYSOMNOGRAPHIC SLEEP ACROSS THE CIRCADIAN AND
        MENSTRUAL CYCLES IN HEALTHY FEMALES

Ari Shechter [1,2] and Diane B. Boivin [1,2]

[1] Centre for Study and Treatment of Circadian Rhythms, Department of Psychiatry, McGill University, Montreal, Quebec, Canada; [2] Department of Neurology and Neurosurgery, McGill University, Montreal, Quebec, Canada

P048   QUANTITATIVE SLEEP ELECTROENCEPHALOGRAM ACROSS THE
        MENSTRUAL CYCLE IN WOMEN WITH PREMENSTRUAL DYSPHORIC
        DISORDER

Ari Shechter [1,2,] Paul Lesperance [3] and Diane B. Boivin [1,2]

[1] Centre for Study and Treatment of Circadian Rhythms, Department of Psychiatry, McGill University, Montreal, Quebec, Canada; [2] Department of Neurology and Neurosurgery, McGill University, Montreal, Quebec, Canada; [3] Centre Hospitalier de l'Universite de Montreal, Department of Psychiatry, Montreal, Quebec, Canada

Physiology & Neurophysiology

P049   SLEEP-WAKE AND CIRCADIAN CONTRIBUTION TO CHANGES IN PER2
        PROTEIN IN WHOLE LIVING MICE

Curie T.[1,] Mongrain V.[1,] Dorsaz S.[1,] Maret S.[1,] Emmenegger Y.[1,] Franken P[1].

[1.] Center for Integrative Genomics, University of Lausanne, Lausanne, Switzerland

P050   DECREASING THE STRESS RESPONSE DOES NOT RESCUE THE
        IMPAIRED HOMEOSTATIC SLEEP REBOUND IN DBA/2J MICE

Mongrain V.[1,] Curie T.[1,] Gip P.[2,] Heller H.C.[2,] Franken P.[1,2]

[1.] Center for Integrative Genomics, University of Lausanne, Lausanne, Switzerland; [2.] Stanford University, Stanford, CA, USA.

P051   INCREASED GABA-ERGIC ACTIVITY IN THE PEDUNCULOPONTINE
        NUCLEUS REDUCES REM SLEEP AND IMPAIRS LEARNING IN RATS

Fogel S.[1,] Smith C.[2] and Beninger R.[1]

[1.] Centre for Neuroscience Studies, Queen's University, Kingston, ON, Canada; [2.] Department of Psychology, Trent University, Peterborough, ON, Canada

P052   REM SLEEP INSOMNIA AND DECREASED PPT CHOLINERGIC NEURONS
FOLLOWING MYOCARDIAL INFARCTION IN THE RAT

Bah T.M.[1,] Laplante F.[4,] Kaloustian S.[1,] Sullivan R.[2,4,] Rousseau G.[1,3,] Godbout R.[1,2]

[1.] Centre de Biomédecine, Hôpital du Sacré-Cœur de Montréal; [2.] Départe-
ment de Psychiatrie, Université de Montréal (Qc), Canada; [3.] Département de
Pharmacologie, Université de Montréal (Qc), Canada; [4.] Centre de recherche
Fernand-Seguin, Hôpital Louis-H Lafontaine, Montréal (Qc), Canada.

P053   GABAB-MEDIATED INHIBITION PLAYS A CRITICAL ROLE IN MEDIATING
REM SLEEP ATONIA

Brooks, P.L. and Peever, J.H.

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ON, Canada

P054   NORADRENERGIC MODULATION OF MUSCLE TONE DURING
CATAPLEXY IN HYPOCRETIN/OREXIN KNOCKOUT MICE

Burgess C.R. and Peever J.H.

Department of Cell and Systems Biology, University of Toronto, Toronto, ON,
Canada

P055   NORADRENERGIC TRANSMISSION TRIGGERS MUSCLE TONE BY
AMPLIFYING GLUTAMATERGIC DRIVE ONTO SOMATIC MOTONEURONS

Schwarz P.B., Yee N., Mir S. and Peever J.H.

Departments of Physiology & Cell and Systems Biology, University of Toronto,
Toronto, ON, Canada

P056   ENHANCED CHOLINERGIC ACTIVITY AT THE HYPOGLOSSAL MOTOR
NUCLEUS SUPPRESSES GENIOGLOSSUS MUSCLE ACTIVITY

Grace K.[1,] Liu H.[1,] Nolan P.[2,] Horner R.[1]

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        Department of Psychology, Trent University, Peterborough, Ontario, Canada

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        Sciences, Johns Hopkins School of Medicine, Baltimore, MD, USA

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Notes

1. Sleep Deprivation

P001

Effects Of Sleep Deprivation And Daytime Recovery Sleep On Self-Evaluation Accuracy

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Introduction: The ability to assess the change in one's own performance under sleep deprivation (SD) is crucial to make adaptive behavioural decisions (e.g. drive, nap, or pursue activities). This study aims to evaluate whether subjects are able to assess their performance deterioration under sleep loss as well as their performance improvement after recovery sleep.

Methods: Twenty-four healthy volunteers (13W, 11M; 37.1y ± 1.14) spent one night of sleep deprivation in the laboratory. Daytime recovery sleep (DRS) was initiated one hour after habitual wake time (HWT). Subjects stayed in bed during DRS for their habitual sleep duration. Psychomotor vigilance task (PVT) was performed in the evening before SD (PVT-1, 12 hours before HWT), at the end of the SD in the morning (PVT-2) and in the evening after DRS (PVT-3; 12 hours before HWT). PVT was immediately followed by an analogical subjective performance scale (APS). Differences between PVT-2 and PVT-1 (SD effect) and between PVT-3 and PVT-2 (DRS effect) were calculated on APS and PVT variables (median reaction time-RT, lapses, fastest and slowest RT). Pearson correlation coefficients were used to evaluate relationships between APS and PVT variables for SD and DRS.

Results: In SD, only the fastest RT significantly correlated with the APS (R = -0.45, p = 0.03) with subjects reporting stronger deterioration of performance showing a stronger increase in fastest RT. After DRS, all PVT variables were strongly correlated with APS (all R > -0.60, p < 0.001). The subjects who reported stronger improvements of performance after DRS showed greater decreases in median RT, lapses, fastest and slowest RT.

Conclusions: These results suggest that subjects were less able to accurately evaluate the decrease of their performance during SD than its increase after DRS. This may be explained by a loss of one's points of reference as a result of higher homeostatic/circadian sleep pressure.

P002

Sleep During Pediatric Hospitalization

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Introduction: Many health care professionals believe children's sleep is affected by hospitalization; however, no objective determinations of sleep are reported for children in critical care or general medicine units.

Methods: Demographic data and information about the hospital stay and illness, and usual sleep habits (Children's Sleep Habits Questionnaire, Owens et al, 2000) were collected. Children wore an actigraph for 1-3 consecutive days and nights and completed a sleep diary. Sound and light meters were placed at the bedside. Sleep variables were averaged over nights recorded.

Results: From Oct 2007-July 2008, 124 eligible children were approached and 69 consented (84% general medical unit, 16% critical care; 51% male; 29% age 1-3, 14% age 4-7, 25% age 8-12, 32% age 13-18). Reason for admission included chronic illness (49%), acute illness/trauma (47%) and planned surgery (4%). Mean nocturnal sleep time (19h30-07h29) was 444 minutes (95%CI 137-600) for ages 1-3; 475 minutes(95%CI 357-662) for ages 4-7; 436 minutes (95%CI 238-595) for ages 8-12; and 384 minutes (95%CI 217-512) for ages 13-18. Mean number of night awakenings was 14 (95%CI 8-21) for ages 1-3; 18 (95%CI 12-23) for ages 4-7; 14 (95%CI 5-24) for ages 8-12; and 12 (95%CI 1-18) for ages 13-18. There was no relationship between usual sleep habits and sleep time or awakenings. Light and sound levels were high at night; mean minutes of light >150 lux ranged from 44-99 minutes, mean minutes of sound >46 dB ranged from 84-116, mean minutes >80 dB ranged from 32-47. Relationships between sound, light and other environmental (e.g. type of room; parental presence; type of unit) and medical (e.g. pain scores; medications) variables and sleep outcomes will be presented.

Discussion: During hospitalization children experience significant nighttime sleep restriction and frequent awakenings at a time when they most need the benefits of sleep.

P003

Age-Related Differences in N-REM Slow Oscillations: Effects of Sleep Deprivation

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Introduction: The possibility that alterations in the build-up function of the homeostatic process could explain lower N-REM sleep synchronization with increasing age is still a matter of debate. Most knowledge on how age modulates the effects of sleep deprivation on NREM sleep synchronization comes from visual scoring of sleep stages and quantitative sleep EEG (e.g. spectral analyses). Spectral analysis provides important indices on sleep EEG synchronization but it does not allow identifying N-REM sleep EEG oscillations per se.

Methods: We used an automatic algorithm to assess the effects of age, sleep loss and topography on N-REM sleep slow oscillations (SO; >75 µm). Twenty-four healthy volunteers with no sleep disorders were separated in two groups: Young (6W, 6M; 24.2y ± 3.3), and Middle-aged (6W, 6M; 53.8y ± 3.7). Each subject participated in a baseline nocturnal sleep and a daytime recovery sleep (after 25-hours of wakefulness). SO detection was performed on artefact free sections of NREM sleep for Fp1, F3, C3, P3, and O1 (linked-ears), with an automatic algorithm using published criteria (Massimini et al. 2004).

Results: Three-way ANOVAs (Age group*Sleep condition*Derivation) were performed on SO amplitude and density (nb/min). In both age groups, SO density was higher during daytime recovery sleep compared to baseline sleep and this effect was stronger in Fp1 and F3. Compared to baseline sleep, SO amplitude increased during daytime recovery sleep and this effect was more prominent in young compared to older subjects in FP1.

Conclusion: Age-related difference in the effects of the sleep deprivation was observed on SO amplitude only, and not on SO density, particularly in anterior derivations. This may be explained by age-related decline in the capacity to synchronize larger neuronal populations after sleep deprivation, especially in frontal areas.

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P004

Impact of Acute Sleep Restriction on Cortisol and Leptin Levels in Young Women.

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Introduction: Sleep loss has been shown to affect cortisol and leptin patterns and cravings for calorie-rich foods in men, and sleep loss is associated with weight gain. Women undergo fluctuations in estrogen and progesterone levels during menstrual cycles that could influence the effects of sleep restriction on cortisol and leptin. This study assessed how overnight sleep restriction affects salivary levels of cortisol and leptin, and food cravings in young women. A secondary goal was to examine the effects of menstrual phase on these responses.

Methods: Fifteen healthy women, ages 18-25, spent 3 nights and 2 days in a chronobiology laboratory. Participants were permitted to sleep 10 h/night for the first 2 nights, and 3 h/night on the third night. During both days, salivary samples were collected every 2 h after waking, and participants completed questionnaires about their cravings. Menstrual phase was estimated from self reports. Salivary samples were assayed to determine: absolute morning and evening cortisol and leptin levels; afternoon/evening areas under the curve (AUC) for cortisol; and slope of decline in cortisol concentrations from morning to evening.

Results: After sleep restriction, participants showed reduced morning cortisol levels ($p = 0.02$), elevated morning leptin levels ($p = 0.04$), elevated afternoon/evening cortisol AUC values ($p = 0.008$), and a slower rate of decline in cortisol ($p = 0.04$). Women in the follicular phase showed greater changes in morning cortisol ($p = 0.05$), afternoon/evening AUC values ($p = 0.03$), and cortisol slope of decline ($p = 0.03$) compared to women in the luteal phase. Craving scores were unaffected by sleep loss and unrelated to hormone measures.

Discussion: The effects of one night of restricted sleep on cortisol rhythms in young women were affected by menstrual phase, suggesting a modulating role for reproductive hormones on the response to sleep loss.

P005

Short Daily Sleep Opportunities Ameliorate The Effects Of Sleep Loss On Energy Expenditure In Rats

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Introduction: Sustained sleep deprivation induces a doubling of resting metabolic rate and eventual death in rats[1], suggesting that sleep has essential physiological functions linked directly or indirectly to energy metabolism. However, the relevance of these findings to humans is unclear because most affected individuals experience ongoing insufficiency of sleep (chronic sleep restriction, CSR) rather than total sleep deprivation. Despite its potential clinical relevance, little attention has yet been given to the physiological consequences of CSR. Here we describe and validate an experimental model for the study of CSR in rats, and compare changes in energy metabolism in CSR with those found in total sleep deprivation.

Methods: Biotelemetry implants and non-invasive respirometry allowed unrestrained recordings, thereby minimizing stress and facilitating long-term studies. Sleep-wake states were continuously monitored using a fully automated rat sleep autoscoring system (ratSAS) in combination with an intermittently rotating wheel-respirometer for scheduling daily sleep opportunities and wake maintenance intervals.

Results: ratSAS was shown to be >85% concordant with visual scoring. Furthermore, rats were awake for >90% of the 18h daily wake window with the wheel scheduled on an 8s on - 8s off cycle. Rats accumulated a total sleep "debt" of 63 h during 10 days of CSR, equivalent to sleep lost in approximately 6 days of TSD. However CSR, unlike TSD, caused no increase in $\dot{V}O_2$ (p=0.175; Fig. 1) despite a lack of recovery sleep in the daily 6h sleep opportunities.

Conclusions: We conclude that brief daily sleep opportunities ameliorate the effects of sleep loss on energy metabolism.
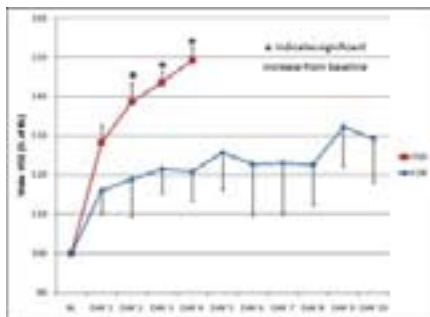
2. Sleep & Performance

P006

Lower Subjective Socioeconomic Status Is Associated With Shorter Sleep Duration, Poorer Sleep Quality, And More Daytime Sleepiness In Children And Adolescents

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Introduction: Socioeconomic status is inversely associated with many health outcomes, suggesting a socioeconomic gradient in health. In adults, lower socioeconomic status is associated with shorter sleep duration, poorer sleep quality, greater difficulty initiating and maintaining sleep, and considerably more accumulated sleep debt. While researchers have started to examine whether a socioeconomic gradient exists for sleep problems in adults, relatively little is known about this relationship in youth. The aim of the present study was to examine whether socioeconomic status was associated with sleep measures in children and adolescents.

Methods: Participants were part of the larger Healthy Heart Project and included 183 youth (47.8% female), aged 8-18 years (M=12.83, SD=2.12). The MacArthur Scale (adolescent version; Goodman et al., 2001) was used to measure subjective socioeconomic status. Sleep duration was the difference between self-reported bed- and wake-times. Perceived sleep quality was rated on a 10-point Likert scale (1=poor, 10=excellent). Daytime sleepiness was assessed with the self-report Pediatric Daytime Sleepiness Scale (Drake et al., 2003).

Results: Consistent with adult literature, after controlling for age, a linear relation between subjective socioeconomic status was associated with shorter sleep duration (ravg = .20, p<.05), poorer sleep quality (ravg=.25, p<.01), and more daytime sleepiness (ravg = -.25, p<.01).

Conclusions: Lower subjective socioeconomic is associated with greater risk for sleep problems in children and adolescents, suggesting sleep may be one pathway underlying the socioeconomic gradient in health. Future researchers should aim to elucidate how specific sleep constructs (e.g., sleep architecture) may explain how socioeconomic status "gets under the skin" to affect health.

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P007

Subjective Excessive Daytime Sleepiness in a Community-Based Sample: Frequency and Associated Factors

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Introduction: Despite the fact that excessive daytime sleepiness (EDS) is commonly reported and has been linked to several negative outcomes such as traffic accidents, epidemiological data on this subject are scarce. This study aimed at investigating the frequency of occurrence and associated factors of self-reported excessive daytime sleepiness in a community-based sample.

Methods: Participants were French-speaking adult residents of the province of Quebec who took part in an epidemiological study examining the longitudinal course of insomnia. Data used in the current project are derived from the fifth postal follow-up, which assessed sleep/sleepiness, psychological and health variables, and was completed by 633 (aged 21-87 years old; 63.8% women) of the 997 participants initially included in the longitudinal study. EDS was defined as an Epworth Sleepiness Scale (ESS) score greater than 10. Pearson chi-square tests were computed to examine associations between EDS and several sociodemographic, life habits and health-related variables.

Results: Mean ESS score was 8.3 ± 4.3, and 28.1% of the sample had a score higher than 10. Chi-square tests revealed that the presence of EDS was significantly associated ($p < .05$) with a sleep duration shorter than 7 hours, the use of two or more caffeinated beverages daily, as well as the presence of chronic pain. Nearly significant associations ($.05 <= p < .09$) were found between EDS and a body mass index greater or equal to 30, a frequency of physical activity lower than once per week, as well as the presence of headaches/migraines, moderate/severe depression symptoms, and moderate/severe insomnia symptoms.

Conclusions: The frequency of self-reported EDS was relatively high in this sample compared to other epidemiological investigations. Several variables were associated with EDS, including sleep-related variables as well as the presence of specific chronic health conditions. Future analyses should investigate additional factors potentially associated with EDS.

P008

Do Different Levels Of Sleepiness Modulate Effects Of Caffeine On Vigilance?

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Introduction: It is suggested that the initial activation level influences the effects of caffeine but no study has directly compared the effects of similar doses of caffeine in situations of varying sleepiness levels produced by homeostatic/circadian sleep pressure. Also, effects of caffeine on melatonin/cortisol levels are still a matter of debate and it is unknown whether time of caffeine administration influences these results. This study compares the effects of caffeine in two situations of varying levels of sleepiness (i.e. in the evening after a normal day and during a night of sleep deprivation).

Methods: Fifty moderate caffeine consumers (mean age: 38.3) were assigned to an Evening protocol (EP) or a Night protocol (NP). All subjects participated in both caffeine (200 mg) and placebo (lactose) conditions in a double-blind crossover design. In the EP, subjects received 100 mg of caffeine (or placebo) 3 hours and 1 hour before habitual bedtime. In the NP, subjects were sleep deprived for one night and received 100 mg of caffeine (or placebo) 2 hours before and at the habitual wake time. All measures were collected between 30 and 45 minutes after the second dose.

Results: Compared to placebo, caffeine increased subjective alertness and decreased median reaction time for psychomotor vigilance measure (PVT) similarly in the EP and NP. However, the effect of caffeine on PVT slowest reaction time was more prominent in the NP than in the EP and caffeine decreased the number of PVT laps in the NP only. Caffeine increased melatonin secretion in both protocols, but increased cortisol secretion in the NP only.

Conclusion: In conclusion, caffeine shows stronger effects on vigilance in high sleepiness conditions induced by enhanced homeostatic/circadian sleep drive. The effects of caffeine on cortisol but not on melatonin are influenced by time of day.

P009

Benefits Of A Daytime Nap For Students On A University Campus

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Introduction: Sleepiness is a problem for students at all levels of education, and naps are known to alleviate some negative effects of sleep deprivation1. We hypothesized that a 20-minute nap opportunity while on campus would lead to improvements in well-being and performance that would continue throughout the day during classes and on the commute home.

Methods: Twenty-two university students (mean age=24, 14 female), who were healthy, good sleepers, participated in a repeated-measures, counterbalanced design where they had a 20-minute nap opportunity in the Sleep Laboratory on one week, and read for 20-minutes as a control condition in another week (same day and time). Mood, sleepiness, and fatigue were measured before and after each condition. Reaction time (RT) and working memory were assessed after each condition. In the evening at home, participants retrospectively rated their sleepiness and fatigue during class (n=9) and on the commute home, as well as their current mood state.

Results: Condition (nap, no nap) by Time (pre, post) ANOVAs indicated that napping led to decreased sleepiness (p=0.045) and fatigue (p=0.003), faster mean RT (p=0.003), and improved mood in areas of happiness (p=0.029) and energy (p=0.010). Memory was not influenced by napping. Paired t-test comparisons of assessments taken in the evening showed naps led to decreased sleepiness (p=0.011) and fatigue (p=0.001) during class, decreased sleepiness (p=0.002) and fatigue (p=0.000) on the commute home, and increased positive mood in areas of happiness (p=0.005), relaxation (p=0.000), and calmness (p=0.003).

Conclusion: A 20-minute nap opportunity for students on campus led to benefits immediately following the nap, as well as later in the day during class and on the commute home. These findings suggest that daytime naps may benefit student learning in the classroom and driving safety in particular, and highlight the need for napping stations on university campuses.

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P010

Sleep Quality And Next-Day Pvt Performance After Heavy Alcohol Consumption

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Introduction: Heavy alcohol consumption close to bedtime disrupts sleep quality and may impair next-day performance. We assessed the effects of a high dose of alcohol (.11g% breath alcohol concentration [BrAC]) and of beverage content (high vs. low congener) on subjective and objective sleep quality and next-day PVT performance.

Methods: Ninety-five heavy drinking student volunteers (56 women, 24.5 ± 2.8 years) without a history of drinking or other health problems consumed alcohol or placebo at two experimental sessions in a randomized, 2x2 mixed design. Following three days of 8 hours time in bed and screening polysomnography, participants consumed placebo or were dosed (1.2 g/kg for men, 1.1 g/kg for women) with either high [bourbon] or low [vodka] congener alcohol from 8:45 -10:00 p.m. Mean peak BrAC was 0.11 ± .01g%. Sleep was monitored with polysomnography (PSG) between 11 p.m. and 7 a.m. In the morning, participants completed a validated post-sleep questionnaire and participated in a 10-minute trial of attention/sustained reaction time (PVT) at 8:30 a.m.

Results: Sleep was rated as less refreshing (p < .001) and of a worse quality (p < .05) following heavy alcohol consumption. PSG findings indicated that, compared to placebo, alcohol increased wake after sleep onset (p < .01), SWS (p < .01) and latency to REM (p < .001) while reducing total sleep time (p < .02), sleep efficiency (p < .02), REM (p < .001) and latency to SWS (p < .02). No subjective or objective differences were evident by beverage content. Next-day PVT median reaction time was longer after alcohol than after placebo (229 ± 29.9 vs. 220.1 ± 23.8 msec, p < .001), with no effect of congener content.

Conclusions: Heavy drinking produces subjective and objective sleep disturbance and slows next-day reaction time. We are evaluating whether sleep disturbance mediates decrements in next-day performance. These findings may have implications for safety sensitive occupation regulations.

P011

Effects Of Consolidation Of Procedural Motor Memory Traces On Slow And Fast Spindles

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Introduction: We reported that the expression of consolidation (i.e., "off-line" gains in performance) for a motor sequence learning task (MSL) is dependent on sleep, and associated with an increase in spindle density. Other studies have shown that fast spindles (but not slow spindles) are associated with declarative memory. Yet, the distinct contribution of these two types of spindles in consolidating procedural memories remains unknown. This study aims to identify the effect of motor memory consolidation on fast and slow spindles in a MSL and a motor adaptation (MA) task.

Methods: Two groups of young subjects participated in a counterbalanced experimental task/control study design. The MSL group (n=12) performed one task (finger MSL or sequence control task) on the first evening (around 9:00 p.m.), and was retested on the same task 12 hrs later (around 9:00 a.m.). The counterbalanced task (MSL or control) was administered one week later. The MA group (n=13) followed a similar protocol but performed an eight-target pointing MA task or an adapted control task. Polysomnographic recordings were carried out during the night and an automatic algorithm was used to detect fast and slow spindles on frontal (F3, Fz, F4), central (C3, Cz, C4), parietal (P3,Pz, P4) and occipital (O1,Oz,O2) derivations.

Results: Three-way ANOVAs (2groups*2tasks*4derivations) revealed a significant task*group interaction ($p<0.05$) on density of fast (but not slow) spindles detected on medial derivations. Contrast analyses yielded a significant task effect in MSL group only ($p<0.01$, MSL>Ctrl). In MSL group, two-way ANOVAs (2tasks*4derivations) carried out on the left, medial and right axes showed a significant task effect for fast spindle density (but not slow) in all three axes ($p<0.05$). No significant changes in slow/fast spindle density were found between tasks in the MA group.

Conclusion: These findings demonstrate that sequence learning consolidation affects slow and fast spindles differently.

P012

## Post-Learning Reduction In Slow-Wave Sleep After Mental Practice Of The Rotary Pursuit Task

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Introduction: Previous studies have reported learning related sleep changes after physical practice on a motor task. The present study assessed sleep changes occurring after acquisition of a motor task (rotory pursuit task) using mental practice. Mental practice refers to the cognitive rehearsal of a task in the absence of the physical movements used to perform the task.

Methods: Participants were 22 university students (mean age=20.5). After an acclimatisation night, participants had a baseline night of sleep recording. The following evening participants were randomly assigned to one of three conditions: (1) physical practice (n=8; performed 30 physical trials of the rotory pursuit); (2) mental practice (n=7; performed 3 physical and 27 mental trials); (3) no-practice controls (n=7; performed 3 physical trials). Then, participants had a post-learning night of sleep recording. One week later, all participants were given 30 physical trials on the rotory pursuit. Within-group comparisons (baseline vs. post-learning) were used to examine learning related changes in sleep stage duration, stage 2 spindle densities and REM densities.

Results: The physical and mental practice groups performed significantly better than the controls on the rotory pursuit at retest (p<.02), indicating that successful learning occurred in both groups. The mental practice group had a significant decrease in slow-wave sleep from the baseline to post-learning night (p<.01; figure A), with the reduced minutes manifesting primarily as increased REM sleep. None of the groups showed a significant difference between baseline and post-learning for stage 2 spindles or REM densities.

Conclusions: Mental practice was as effective as physical practice for learning the rotary pursuit task. Furthermore, the post-learning night of sleep following mental practice appears to be structurally different from that following physical or no-practice. Together, the findings point to a role for sleep in the consolidation of motor learning with mental practice.
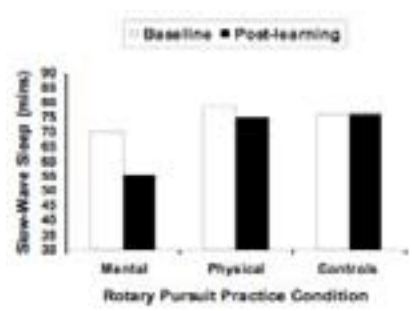
P013

Identification Of A Rapid Eye Movement Sleep Window For The Win-Shift Radial Arm Maze Task

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Introduction: Efficient rodent learning of various maze tasks has been attributed to animals experiencing REM sleep during specific periods of time (REM sleep windows (RSW)) following training on a specific task. Previous research has suggested that different RSWs exist for tasks known to require neural processing in specific neuro-anatomical structures: the amygdala, the dorsal striatum and the hippocampus. Other research identified RSWs for amygdaloid-dependent avoidance tasks and striatal-dependent win-stay radial arm maze (RAM) tasks. Our study hypothesizes that a RSW for the hippocampal-dependent win-shift RAM task exists during the 4-8 hour interval following training, consistent with that identified for the hippocampal-dependent Morris Water Maze.

Methods: To determine the RSW for the win-shift task, rats (n=71) were trained on the win-shift RAM task. Subsequently, groups of animals were deprived of REM sleep during different 4 hour intervals such that the entire 24 hour period following training was explored. Training and sleep deprivation were continued for 15 days.

Results: Three statistical analyses were used to evaluate the animals' learning of the task: ANOVA of latency data, survival analysis with the first day of maze completion as the censoring variable, and ANOVA for the rate that animals consumed baits over days of training. Analyses supported the existence of an RSW of 0-4 hours following daily training. Animals REM deprived immediately following training were slower in completing the maze (p < 0.01). Survival analysis showed this same group was delayed in completing the maze for the first time (p < 0.05). Finally, animals deprived of REM sleep immediately following training found baits at a delayed rate (p < 0.01).

Discussion: We conclude that the RSW for the win-shift RAM task is 0-4 hours after training. Our data suggests the possibility of a second less influential RSW for this task during the 4-8 hour interval.

3. Neuropsychology & Psychiatry

P014

Sleep Eeg In Autism And Performance On The Embedded Figure Test

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Introduction: Neuropsychological, EEG and brain imaging studies point toward enhanced low-level visual perception in autism, leading to a more local bias and increased performance in low-level visual stimuli than typically developing participants (TDP). Our group has recently reported a decreased contrast between primary and non-primary visual areas for EEG Beta activity during REM sleep in autism in comparison to TDP (Bolduc et al., APSS 2005). The present study aimed at verifying if EEG Beta activity during REM sleep correlates with performance in the Embedded Figure Test (EFT), a task that relies on visual search and local perception, and consistently performed at a superior level in autism.

Methods: Eight autistic (21.9 ± 4.3 years) and 11 comparison participants (19.9 ± 4.4 years) were recorded for two consecutive nights. Spectral analysis of REM sleep Beta EEG activity (13.0 to 19.75 Hz) was performed on primary (O1, O2) and non-primary (P7, P8) visual areas. In the morning of night two, participants were tested with the Embedded Figure Test. Group performance on the EFT task was compared with Mann-Whitney U-tests. The correlation between performance and EEG spectral power was estimated with Spearman's rho coefficients.

Results: HFA participants performed better than comparison participants on the EFT task (p<.03). There was a negative correlation between REM sleep EEG Beta activity and time to complete the EFT task in controls only (rho = -0.66; p=0.025), not in the HFA group (-0.19; p=0.63).

Conclusions: These results suggest that autistic individuals use an atypical visual cortical network in association with enhanced performance in local perceptions tasks. Once again REM sleep EEG Beta activity, thought to reflect the REM sleep control mechanisms, is shown to correlate with visual cognition and discriminate persons with autism from comparison groups.

P015

Dissociation Between Stage 2 Sigma Eeg Activity And Sleep Spindle Density In High-Functioning Autism

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Introduction: Adults with high functioning autism (HFA) display fewer stage 2 sleep spindles than controls (Limoges et al., 2005). Since EEG sigma activity is in the same frequency range as sleep spindles, we verified whether sigma activity is also diminished in HFA.

Methods: Sixteen adults with HFA (14 men, 2 women, 22.1±3.6 years) and 16 comparison participants (COM: 20.6±3.9 years) were recorded for two consecutive nights. Stage 2 sleep spindles of night 2 were visually identified on the C3 and Fp1 electrodes as bursts of EEG activity at 12-15 Hz, lasting 0.5-2.0 sec, with no amplitude criteria applied. Spectral amplitude of stage 2 sigma EEG activity (11.75-14.75 Hz) was computed for the first seven hours of sleep of night 2 in a subgroup of nine HFA and six comparison participants. Data is expressed as mean ± sem. Groups were compared using t-tests.

Results: Minutes of stage 2 was the same in the two groups (HFA: 275.6±2.1, COM: 283.6±2.1, n.s.). Number of spindles per hour of stage 2 at Fp1 was the same in the two groups (HFA= 46.9±11.9, COM: 62.1±9.9, n.s.) but C3 spindle density was lower in the HFA group (146.2±15.3 vs. 215.4±16.6; p<.004). The whole first 7 hours of sleep did not show group differences on stage 2 sigma activity for the Fp1 (HFA=6.±5.7, COM=3.9±2.1) nor the C3 electrode (10.4 ±4.8, COM=11.1±4.1), neither did an hour by hour breakdown of the data.

Discussion: These results show that, contrary to sleep spindles, the quantity of sigma activity during stage 2 is typical in HFA. This supports the hypothesis that visually identified sleep spindle waveforms and quantified EEG sigma spectral activity reflect two distinct processes (see also Gais et al., 2002). Since persons with Asperger syndrome are known to display fewer sleep spindles than those with HFA, further analyses will compare these two subgroups.

P016

Sleep Patterns In Adolescents With Bipolar Disorder Or Borderline Personality Disorder

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Introduction: Not only emotional instability but also sleep disorders are found both in bipolar disorders (BD) and borderline personality disorder (BPD). The aim of this research was to compare sleep patterns in adolescents with BD and BPD.

Methods: Seven adolescents with euthymic BD (2M; 5F; 16.7±1.0 years) and twelve with BPD (11F; 1M; 15.9±1.1 years) wore wrist actigraphy device and filled a sleep agenda for an average of nine days. All participants were under psychopharmacological treatments. Actigraphy data was computed using one minute epochs and groups were compared using Mann-Whitney U-tests.

Results: Actigraphy results showed that during weekdays, awake time during rest and sleep intervals was significantly longer in BPD compared to BD (p<.05), and percentage of immobility was significantly longer in BPD compared to BD (p<.05). BPD adolescents had shorter active intervals during weekends compared to weekdays (p<.05). Weekdays and weekends agendas showed significantly longer total sleep time in BD compared to BPD (p<.05). There were no differences between actigraphy and agenda data for sleep onset latency and total sleep time.

Conclusions: When both groups are compared to one another, actigraphy reveals a sleep maintenance problem in adolescents with BPD. The lack of significant difference between the times reported by the actigraphy and the one by agenda suggests that BD and BPD adolescents can make a good estimate of their sleep. Further research should determine whether these differences are due to affective symptoms, poor sleep hygiene or a circadian rhythm disorder. We are continuously enrolling new patients in the protocol in order to replicate the present results with larger groups.

P017

Sleep/Wake Disturbance Following Severe Traumatic Brain Injury; Impact On Recovery Of Cognitive-Communication Function: A Case Study

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Introduction: Traumatic brain injury (TBI) is the leading cause of disability for persons under age 45 in North America. TBI is commonly associated with physical, cognitive, communication and neuropsychiatric sequelae including sleep disorders, which are reported at a rate of 76% post injury. Cognitive-communication impairments (difficulty with listening, speaking, reading, writing, thinking and social communication as a result of underlying cognitive disturbances) occur at a rate of 81% post injury. We hypothesize that sleep and alertness disorders exacerbate deficits in cognitive-communication function and can impede the recovery process. The objective of this study was to longitudinally examine the impact of sleep/wake disturbance and changes in sleep quality on cognitive-communication function in a person with severe TBI.

Methods: The subject completed a monitoring profile, the Daily Cognitive-communication and Sleep Profile (D-CCASP), to document subjective evaluation of sleep quality, daytime sleepiness and fatigue, sustained attention, verbal memory and language processing over a period of 17 weeks. During this time period (11 to 15 mos post injury), a sleep evaluation including polysomnography, was conducted with accompanying medication changes and improvements in sleep and daytime function. A follow-up sleep study was conducted 3 years 8 mos post injury.

Results: A time series analysis demonstrated significantly increased attention, language processing, verbal memory and sleep quality associated with specific changes in medication ($p < .01$). Changes in sleep architecture over time (as evaluated by polysomnography) were also noted.

Conclusions: Clinical and research evidence suggests that timely and effective diagnosis and management of sleep/wake disturbances post TBI may facilitate recovery of maximal cognitive-communication potential. Based on results of this study, we conclude that for certain persons, pharmacological management of sleep and alertness post traumatic brain injury can result in functional subjective improvements in sustained attention, language processing and verbal memory. Further research in this area is warranted.

P018

Daytime Sleepiness After Moderate/Severe Traumatic Brain Injury: Preliminary Findings

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Introduction: Excessive daytime sleepiness has been reported following traumatic brain injury (TBI). However, most studies have focused solely on the acute phase following TBI and/or have been conducted in heterogeneous samples including both mild and moderate/severe TBI despite the differences in expected outcomes between these severity levels. The aim of this study was to document long-term subjective and objective sleepiness symptoms after moderate/severe TBI.

Methods: Preliminary data are available for 15 participants having sustained a moderate/ severe TBI (mean age = 39.7 years old; 20% women; mean time elapsed since injury = 60.7 months) and 8 age-, gender- and education-matched healthy controls (CTL; mean age = 39.1 years old; 37.5% women). All participants underwent four 40-minute Maintenance of Wakefulness Tests (MWT) and completed the Epworth Sleepiness Scale (ESS), the Functional Outcome of Sleepiness Questionnaire (FOSQ) and nine hourly visual analogue scales (VAS) of sleepiness.

Results: Mean sleep onset latency on MWT was similar between TBI and CTL groups (33.1 vs. 34.5 minutes); 53.3% of TBI and 37.5% of CTL participants had at least one sleep onset episode. The two conditions were comparable on ESS score (TBI, 7.3 vs. CTL, 6.8) but were significantly different on FOSQ total score (TBI, 7.3 vs. CTL, 5.7; p = .04). Although the difference was not statistically significant, TBI participants had higher mean ratings of sleepiness on the VAS (20.1 vs. 13.6). This trend was even more pronounced in early afternoon.

Conclusions: These results suggest that, as a group, individuals with moderate/severe TBI do not seem to be pathologically sleepy when assessed at least one year after the injury. On the other hand, according to these preliminary data, TBI patients could be more vulnerable to detrimental effects of sleepiness on activities of daily living as well as to circadian variations of alertness.

P019

Relationship Among Subjective Sleep Complaints, Headaches, And Mood Alterations Following A Mild Traumatic Brain Injury

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Background: Sleep complaints (e.g., frequent awakenings, nightmares), headaches and mood alterations (e.g., feeling depressed, irritable) can appear following a mild traumatic brain injury (MTBI). The objective of this retrospective study was to assess the relationships between the above symptoms. Our hypothesis was that sleep complaints might be among the risk factors for the development of headaches and mood alterations.

Methods: The charts of 443 patients (68.2% males vs. 31.8% female; mean age of 46.9 years) diagnosed with MTBI were randomly selected and reviewed for past medical history and above symptoms using the Rivermead Post-Concussion Symptom Assessment Questionnaire and self-report. Data were retrieved in two time courses: 10 days and 6 weeks. Distribution of variables, Odds Ratios (OR) and Chi Squares were calculated.

Results: For the 2 time courses, prevalence of sleep complaints were 13.3% and 33.5%; headaches 46.8% and 39.3%; feeling depressed 9.5% and 20.4%; and feeling irritable 5.6% and 20.2%, respectively. Reports of sleep complaints at 6 weeks were 2.9 times (p: 0.004) more likely if such symptom was reported at 10 days. Moreover, presence of sleep complaints at 10 days increased the risk of concomitant headaches, depressive symptoms, and feeling irritable by 2.3, 9.9 and 12.2 times (p: 0.0001 to 0.014); and by 2.9, 6.3, and 4.8 times (p: 0.0001) at 6 weeks, respectively.

Conclusion: Our results suggest that patients afflicted with sleep complaints are at increased risk of suffering from concomitant headaches, depressive symptoms, and irritability.

P020

Spontaneous K-Complexes In Stage 2 Sleep Are Reduced Following Traumatic Brain Injury (Tbi)

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Introduction: Individuals with TBI often complain of insomnia[1], and it has been suggested that TBI accelerates aging of the brain. Given these complaints of sleep continuity and that sleep phasic events change with age[2], we predicted that those with a TBI would have fewer K-complexes.

Method: We investigated 19 individuals with a TBI (mean age=30, 9 men) who ranged in severity (6 mild, 8 moderate, 5 severe). Sleep complaints included insomnia (n=9), excessive daytime sleepiness (EDS) (n=4), and those with both insomnia and EDS (n=3); 3 TBI participants reported being good sleepers. The comparison group was 17 healthy good sleepers (mean age=26, 6 men) who did not differ in age (p=0.376). Following an off-protocol screening night, participants' sleep was recorded on two consecutive laboratory nights. Spontaneous K-complexes were visually counted in Stage 2 sleep by two raters. K-complex density (# events/minute of Stage 2) was compared between groups on both nights.

Results: On Night 2, individuals with TBI had sleep architecture consistent with insomnia: more Stage 1 (p=0.041), less Stage 2 (p=0.022), less total sleep time (p=0.018), and reduced sleep efficiency (p=0.020) compared to good sleepers. As predicted, individuals with TBI had fewer K-complexes than good sleepers on Night 1 (p=0.001) and Night 2 (p=0.017).

Conclusion: Although sleep complaints often follow TBI, the neurophysiological underpinnings of this sequela are not understood. Consistent with the view that K-complexes play an inhibitory role in non-REM sleep, the current data provide evidence for a breakdown in sleep-protective mechanisms following TBI. Since K-complexes have not been shown to change in primary insomnia[3], these data suggest that sleep mechanisms have been altered through TBI in a unique way that cannot be explained by the presence of insomnia alone. The reduction in K-complex generation is consistent with a view of an aging brain following TBI.

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P021

Investigation Of Sleep In Chronic Treatment-Resistant Depressed Patients

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Introduction: The existing literature supports a bi-directional relationship between sleep disorders and depression. Sleep disorders are often undiagnosed in the general population and in the psychiatric population. However, once detected treatment of sleep disorders among depressed patients has been shown to improve mood and alleviate depression. Among depressed patients, about 12-15% will remain chronically depressed and will not respond to treatment. The aim of this study was to investigate the sleep architecture and prevalence of sleep disorders among a group of chronic treatment-resistant depressed patients.

Method: Nine patients who met diagnostic criteria for chronic treatment-resistant depression were recruited from local mood disorder clinics. Depression severity was assessed using the 21-item Hamilton Depression Scale (HAM-D). The patients under-went an overnight polysomnographic (PSG) study followed by Multiple Sleep Latency Test (MSLT).

Results: Seven of the participants were females and the average age for the whole group was 50 ± 7 years. The average HAM-D score ($\pm$ SD) was 24 ± 4. The average sleep onset latency and the average REM latency after sleep onset were 22 ± 14 min and 253 ± 119 min respectively (all patients were on antidepressants). The average sleep efficiency was $81\pm4\%$. The average slow wave was sleep percentage was $8\pm9\%$ and the average REM sleep percentage was $13\pm8\%$. The average arousal index was 27 ± 15/hr. Three of the nine patients had an undiagnosed moderate to severe sleep apnea. Three of the patients had a moderate to severe periodic leg movement disorder and five of the patients had "fragmented" sleep. The average mean MSLT was 10 ± 4 min and two of the patients had severe daytime sleepiness.

Conclusion: The results suggest that chronic treatment-resistant depressed patients often have disturbed sleep architecture. A significant proportion of patients in this pilot study had an undiagnosed sleep disorder that might have been the underlying cause or contributor to their chronic treatment-resistant depression.

P022

A Preliminary Attempt At Defining 'Sleep Markers Of Depression' Categorically And Examining Their Association With Subjective Low Mood.

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Introduction: Sleep is the most commonly observed physical complaint in depressed patients and polysomnographic sleep disturbances have been extensively studied as possible etiological and specific markers of depressive state. However, no previous attempt has been made to operationalize the observed macroarchitectural sleep changes observed in Major Depressive Disorder (Slow wave sleep abnormalities, REM sleep abnormalities, and decreased sleep continuity) into a categorical model which could be applied in the clinical setting.

Methods: In a sample of 2467 patients with no prior sleep complaint screened for possible sleep apnea prior to surgery, 74 patients who underwent polysomnographic sleep studies and completed a battery of questionnaires relating to their sleep and mood were studied. Using predetermined cutpoints for the Epworth Sleepiness Scale (ESS), Fatigue Severity Scale (FSS), Athens Insomnia Scale (AIS) and Center for Epidemiologic Studies Depression Scale (CES-D), we retrospectively compared categorical results of these scales to presence or absence of sleep markers of depression, which were defined in the context of previous depression literature and normative polysomnographic data.

Results: No significant associations were found between the CES-D and total sleep markers of depression. However, there was a significant trend toward subjective insomnia in those with sleep markers of depression.

Conclusions: This study does not indicate a high specificity of sleep markers of depression for low mood. However, controlled, cross-sectional prospective studies are required to clearly determine whether a more specific model can be constructed for either subjective or objective depression.

4. Insomnia

P023

Epidemiology of Insomnia in a Canadian Population-Based Sample

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Introduction: The goals of the present study were to estimate the prevalence of insomnia symptoms and syndrome in the Canadian general population and to describe the types of products used to promote sleep.

Methods: A telephone survey was conducted among Canadians aged 18 years and older. A representative sample was obtained using a random digit dialing method programmed to generate geographically stratified phone numbers. Of the 4 869 persons contacted, 2000 (41%) completed the telephone interview. Participants' mean age was 48.6 years old (range 18-99) and 60.5% were women. For the purpose of another study, the province of Quebec was over-sampled compared to the other Canadian regions (60% of the sample). Data were weighted to adjust for differences between gender and region representation in the sample and that of the last national census.

Results: Of the total sample, 39.9% presented at least one insomnia symptom (i.e., initial, middle, late insomnia) for a minimum of three nights per week and 36.5% reported non-restorative sleep. Moreover, 10.9% of the sample met the criteria for an insomnia syndrome as defined by the DSM-IV-TR and the ICD-10. French-speakers presented significantly lower prevalence rates than English-speakers both for insomnia symptoms (34.5% versus 44.3%) and syndrome (7.7% versus 11.2%). Regarding consultations for sleep problems specifically, 13.1% of the sample and 23.2% of those with insomnia (symptoms and syndrome) reported having consulted once in their lifetime, with English-speakers having consulted more often than French-speakers (25.3% versus 15%). Finally, 9.9% of the sample and 28.9% of the subsample of individuals with an insomnia syndrome reported having used prescribed medication in the year preceding the survey, while 43% reported not having used any sleep promoting products despite their difficulties.

Conclusion: These results confirm the high prevalence of insomnia in the Canadian general population and the low level of consultation for this condition.

P024

Sleep Disturbances In Chinese Earthquake Victims

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Introduction: The objective of this study was to investigate sleep problems in Chinese earthquake survivors and their relationships with posttraumatic stress disorder (PTSD) and depression.

Methods: An investigator-administered questionnaire was used to explore subjects' sleep latency (SL), number of wake (WN), wake duration (WD), wake-up time (WUT), and total sleep time (TST) and sleep efficiency (SE). Each measurement included two time frames: "last week" and "the week before the earthquake". A 17-item subscale and a 4-item subscale of the Los Angeles Symptom Checklist were used to measure the severity of PTSD and depression, respectively. Based on the mean values, subjects were divided into groups of high PTSD and low PTSD, and groups of high depression and low depression. Data collection was performed 3 months (90.3±2.8 days) after the grade-8 earthquake, which was on May 12, 2008.

Results: A total of 1603 subjects (55.8% female) completed the study and met the inclusion criteria. Age distribution was 41.5±16.9 for males and 40.4±16.0 for females. Compared with results from the week before the earthquake, during the past week of the study the subjects' SL (58.1±51.3 vs. 29.4±32.9 min), WN (1.8±1.5 vs. 0.8±1.0) and WD (43.1±52.3 vs. 19.0±36.0 min) were increased (all P<0.001), WUT (6:12 vs. 6:30) was earlier (P<0.001), and TST (6.6±2.3 vs. 7.8±1.8 hrs) and SE (%78±22 vs. %91±12) were decreased (both P<0.001). Compared with those in the low PTSD group, subjects in high PTSD group had an increased SL, WN and WD (all P<0.001), earlier WUT (P<0.001) and decreased TST and SE (both P<0.001). Results of the comparison between the groups of low depression and high depression are similar to those between the groups of low PTSD and high PTSD.

Conclusion: Sleep disturbances in Chinese earthquake victims are significant. The sleep disturbances are affected by the severity of PTSD and depression.

P025

Are Insomnia Symptoms Stable Over Time? A 5-Year Prospective Study In The General Population

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Introduction: Whether sleep onset, maintenance or mixed insomnia represent changing manifestations or distinct stable subtypes of insomnia remains unclear. The aim of this study was to examine the stability of insomnia subtypes across time.

Methods: In the context of a larger epidemiological study, participants completed questionnaires every year over a five-year period. The subset used in this project includes those who reported symptoms of insomnia on the Pittsburgh Sleep Quality Index on at least two assessments (N = 459; mean age = 43.6; 63.8% women). At each assessment, participants were classified as having either sleep onset, maintenance or mixed insomnia, or other types of insomnia (e.g., non-restorative sleep).

Results: Overall, 52.7% of participants remained classified within the same insomnia subtype over the five-year period, with 1.7% having sleep onset insomnia, 46.2% having maintenance insomnia and 4.8% having mixed insomnia. Within the remaining 47.3% of participants for whom the nature of symptoms changed over time, 36.9% did not experience a predominant insomnia subtype, 7.4% had predominant sleep onset insomnia, 38.7% had predominant maintenance insomnia and 17.1% had predominant mixed insomnia. For individuals classified as having sleep onset insomnia at a given assessment period (time X), the probability of having sleep onset insomnia the next time they had insomnia symptoms (time X + 1) was 32.3% (see figure). For those classified as having maintenance insomnia, the probability of remaining with maintenance insomnia the next time they experienced insomnia symptoms was 82.1%. Finally, for individuals classified as having mixed insomnia, the probability of having mixed insomnia the next time they experienced insomnia symptoms was 49.8%.

Conclusion: The stability of insomnia subtypes varies among individuals with insomnia and seems to differ depending on the nature of the subtype. While maintenance insomnia appears relatively stable across time, sleep onset and mixed insomnia seem more volatile.
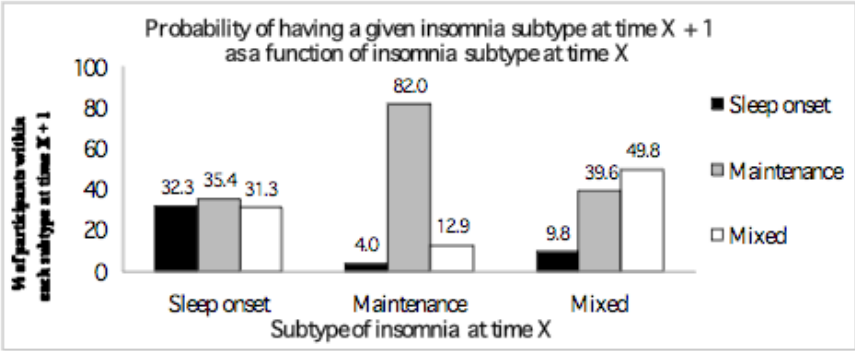
Support: Research supported by the Canadian Institutes of Health Research (#42504).

P026

Insomnia As A Risk Factor For Health Problems: A Longitudinal Study

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Introduction: Longitudinal studies indicate that health problems are associated with a higher incidence of insomnia suggesting that they may represent a risk factor. Little research, however, has addressed if insomnia is itself a risk factor for developing health problems. Therefore, the aim of the present study was to investigate the role of insomnia in the development of health problems.

Methods: Participants (n = 997 adults, mean age = 45.0 years, 51.7% women) completed a questionnaire assessing sleep, physical and mental health, lifestyle habits, personality and demographics at baseline, and at 6- and 18-month follow ups. They were divided into two groups: (a) insomnia symptoms or syndrome (n = 358) and (b) good sleepers (n = 482) according to baseline sleep. The main dependent variable was self-reported current health problems.

Results: Among participants without health problems at baseline, insomniacs did not show a significant increased risk of developing health problems 18 months later compared to good sleepers. However, good sleepers who had developed insomnia had a significantly higher risk of developing at least one health problem compared to those who remained good sleepers (OR = 2.04). Also, participants whose insomnia persisted both at 6 and 18 months after baseline exhibited a significantly greater risk of developing at least one health problem by the last follow up, compared to those who remained good sleepers for the same interval (OR = 2.14).

Discussion: The results suggest that the incidence of insomnia is associated with the development of health problems, but the direction of that link remains unclear as insomnia could either constitute a risk factor or a consequence of health problems. However, the persistence of insomnia seems a risk factor for the subsequent development of health problems.

P027

Classification of insomnia sufferers based on laboratory PSG recordings and subjective sleep reports

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Introduction: Classification of chronic insomnia sufferers (INS) as psychophysiological (PsyI) or paradoxical (ParI) subtypes usually depends on objective sleep variables and sleep perception. PsyI present below normative PSG values and an accurate perception of their sleep while ParI show normal PSG values and an underestimation of sleep. The objective of this study is to describe the distribution of INS's PSG sleep parameters and accuracy of sleep perception in order to test the validity of the actual PsyI/ParI classification.

Methods: Forty-six chronic INS ($M age$ = 42.4 years) underwent four consecutive PSG recording nights while completing sleep diaries each morning. From nights 2 and 3, subjective (S) and objective (O) sleep measures were computed (TST, SE) as well as difference scores (O-S; diffTST, diffSE). K-Means cluster analyses were performed with 2, 3 and 4 clusters using OTST, OSE, diffTST and diffSE as variables.

Results: Based on cluster size, the 2-cluster model was retained. INS were classified on the basis of sleep perception: accurate (n=31; diffTST≤82.0min, diffSE≤18.2%) or overestimation of sleep difficulties (n=15; diffTST≥102.7min, diffSE≥22.0%). Between groups ANOVAs revealed significant differences in diffTST and diffSE ($p<.001$), but similar OTST and OSE ($p>.05$). It is noteworthy that the 3 and 4-cluster models both generated a small group (n=3) of poor sleepers (OTST =363.5min, OSE=75.7%) greatly overestimating their sleep difficulties (diffTST=279.0min, diffSE=59.7%).

Discussion: These results suggest that chronic insomnia sufferers who accurately perceive their sleep and those who show large objective-subjective differences might belong to different insomnia subtypes. Contrary to our expectations, this classification is obtained regardless of objective sleep difficulties. However, according to the 3 or 4-cluster models, some individuals, having objectively poor sleep and considerably underestimating their sleep time, may represent another, although rare, subtype of chronic insomnia. Further research shall be conducted to validate this classification (ex. using cortical activation).

P028

Erp Measures During Sleep In Psychophysiological And Paradoxical Insomnia Sufferers

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Introduction: Using PSA, high cortical arousal has been reported during the night in psychophysiological insomnia sufferers (Psy-I), and even more so in paradoxical insomnia sufferers (Para-I). Although event-related potentials (ERPs) provide powerful indexes of arousal levels in sleep, studies using them are scarce. The objective of the present study is to use ERPs (N1 and P2) to document arousal levels in Psy-I, Para-I and good sleepers (GS) in stages 2, 3-4 and REM sleep.

Methods: Eight Psy-I (mean age = 40.4y), 9 Para-I (mean age = 43.1y) and 10 GS (mean age = 43.9y) underwent four consecutive nights of PSG (N1 to N4). ERPs N1 and P2 were recorded during the fourth night in stages 2, 3-4 and REM. Auditory stimuli consisted of 'standard' frequent (70 dB, 2000 Hz, .85 probability) and 'deviant' rare stimuli (90 dB, 1500 Hz, .15 probability).

Results: Mixed ANOVAs on N1 amplitude showed significant main effects of auditory stimuli $F(1, 24) = 12.99$, $p < .01$. Mixed ANOVAS on P2 amplitude resulted in significant effects for auditory stimuli ($F(1, 23) = 30.23$, $p = .00$). Furthermore, Recording Time × Auditory Stimuli ($F(2, 46) = 4.13$, $p = .04$) was also significant. Analyses on latency measures of N1 and P2 revealed no significant effects at all. No between groups differences were found.

Conclusion: These preliminary results suggest that the amplitude and latency of the different ERPs is similar during stages 2, 3-4 and REM sleep in insomnia sufferers and good sleepers. Hyperarousal might thus be limited to the awake and sleep-onset periods and not sleep in insomnia sufferers.

P029

Event-Related Potentials (Erps) Reveal Failure To Inhibit Stimuli During The Pre-Sleep Waking Period For Patients With Sleep-Onset Insomnia

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Introduction: Insomnia, difficulty falling or staying asleep, is a highly prevalent disorder which leads to problems with daytime functioning (e.g., attention, memory). Various models have been proposed to explain its etiology and pathophysiology. The Neurocognitive Model suggests that chronic insomnia occurs through conditioned central nervous system (CNS) arousal. Consistent with this, we hypothesized that patients with sleep-onset insomnia would show altered information processing during the sleep-onset period. Specifically, we expected ERP components to reveal hyper-arousal or heightened attentiveness.

Methods: Twelve good (mean age=23, 9 female) and thirteen poor sleepers (mean age=22, 10 female) participated in a two-night study. Night1 was to screen for sleep disorders. On night2, both groups were administered an auditory oddball task during 4-6 repeated sleep onset attempts (Standard tone: 1000Hz, 70dB, 50ms, 80% of trials; Target: 2000Hz, 70dB, 50ms, 20% of trials). Participants signaled detection of a higher pitch target tone with a button press as they fell asleep. Each sleep onset block ended after 5-minutes of consolidated Stage 2 sleep
.

Results: Poor sleepers had significantly smaller P2 amplitudes to standard stimuli at fronto-central sites (F3, Fz, F4, C3, Cz, C4) during the pre-sleep waking period of sleep-onset. The adjacent figure shows the Grand Average ERP at Cz for good (solid line) and poor (dotted line) sleepers. Groups did not differ for N1, N350, and P300 components.

Conclusion: Experiments show that P2 is larger when attention is disengaged. Thus, the group difference here indicates that poor sleepers did not withdraw attention to the same extent as good sleepers at sleep-onset, i.e., poor sleepers failed to inhibit or block out irrelevant stimuli. This hyper-attentiveness may explain chronic problems with sleep initiation and could be the target of behavioural and pharmaceutical treatment strategies.
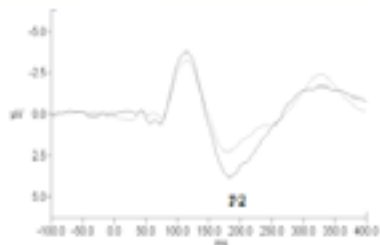
P030

Baroreflex Sensitivity During Sleep And Wakefulness In Primary Insomnia : Preliminary Results

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Introduction: The arterial baroreflex is an important mechanism implicated in the short term regulation of blood pressure (BP). We assessed baroreflex sensibility (or function, BRS) during pre-sleep wakefulness and across sleep stages in subjects with chronic primary insomnia compared to good sleepers.

Methods: We studied 11 subjects with chronic primary insomnia (7 women; 43±7 years) and 11 sex and age matched good sleepers. Subjects were free of any medical or psychiatric co-morbidity and other sleep disorders. Subjects underwent 2 week sleep diary and 3 night polysomnography (PSG) including non-invasive beat-to-beat BP recordings. BRS was assessed during pre-sleep wakefulness, stage 2 non-REM and REM sleep of night 3 by calculating: 1) total slope of the regression line between R-R interval and systolic BP (SBP) changes occurring spontaneously (sequence method) and 2) a -coefficient index in the low frequency band (a LF), high frequency (a HF) and the a lumped ((a LF + a HF)/2), obtained by cross spectral analysis of R-R interval and SBP variabilities. Measures of BRS and changes in RR variability from wakefulness through stage 2 and REM sleep were compared between groups by *2 X 3* ANOVA with repeated measures. Between groups comparison was performed by unpaired t-test or by Mann-Whitney U-test.

Results: Total slope, a LF and a lumped were highly similar between insomniacs and good sleepers during wakefulness, stage 2 and REM sleep. However, these measures of BRS during stage 2 tended to be lower in insomniacs with impaired sleep efficiency

(SE) at PSG (SE<85%, N=6 subjects) versus those with preserved SE (≥85%, N=5 subjects). Values were respectively: total slope 10.3±5.7 vs 19.5±9 ms/mmHg, p=0.1; a LF 7.2±3.9 vs 12.6±4.4 ms/mmHg; p=0.06; and a lumped 10.2±6.3 vs 18.8±8.6 ms/mmHg; p=0.1.

Conclusions: Our preliminary results suggest that BRS mechanisms are preserved in subjects complaining of insomnia. Nevertheless, certain impairment may occur in insomniacs as a function of objective measures of poor sleep.

Does Cbt For Insomnia Alter Sleep Misperceptions?

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Introduction: Individuals with insomnia tend to overestimate the severity of their sleep disturbances and to underestimate total sleep time (TST) relative to polysomnographic (PSG) recordings. Cognitive behavioral therapy (CBT) for insomnia may indirectly reduce this discrepancy by addressing distorted beliefs and perceptions about sleep and insomnia symptoms. This secondary analysis of data from an insomnia treatment trial aimed at investigating the impact of CBT for insomnia on sleep misperception.

Methods: A total of 160 individuals with chronic insomnia (mean age = 50.3 years, 60.6% women) were randomized to CBT alone or CBT combined with hypnotic medication. Pre- and post-treatment assessments included sleep diaries, questionnaires, and PSG recordings (three baseline and two post-treatment nights). Participants completed morning questionnaires estimating several sleep-wake variables after each PSG recording. Percentage of accurate estimation of TST was computed from PSG data as : (estimated TST – objective TST)/objective TST X 100 (a negative score means an underestimation of TST). Values from the second and third baseline nights and the two post-treatment nights were averaged for each participant and analyzed using repeated-measures ANOVA.

Results: Overall, TST was underestimated by 17.5% (SD = 21.9) at baseline and underestimated by 7% (SD = 15.4) at post-treatment. A significant time effect showed an overall reduction of the magnitude of sleep misperception. A significant group X time interaction effect indicated that the combined group had a larger reduction in sleep misperception than the CBT alone group. Exploratory analysis suggested that the improvement of sleep perception was positively associated with improvement reported on sleep diary measures, but not on the other questionnaires.

Conclusions: Results suggest that CBT for insomnia helps reduce sleep misperception in individuals with chronic insomnia, despite the fact that this issue was not directly addressed in the course of treatment. Larger reduction of sleep misperception in the combined group may reflect a drug-induced improvement of sleep perception.

P032

Impact Of Cbt For Insomnia And Cbt Combined With Medications On Daytime Functioning

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Introduction: This study examined the effects of CBT for insomnia delivered alone to CBT combined with hypnotic medications (CBT+Med) on daytime functioning variables. A second objective was to compare the effects of different extended treatment strategies on daytime functioning. **Methods.** One hundred and sixty individuals (61% women; mean age: 50.3 years) with persistent insomnia were randomized either to a CBT alone or CBT+Med condition. After initial treatment, patients treated with CBT alone were then randomized either to extended CBT (6 months) or to "no additional treatment". Those treated with CBT+Med were randomized to CBT combined with intermittent medication use or CBT with medication taper. Daytime functioning variables included fatigue (Multidimensional Fatigue Inventory; MFI), anxiety (Beck Anxiety Inventory; BAI), depression (Beck Depression Inventory; BDI), health perception (SF-36) and beliefs about sleep (Dysfunctional Beliefs about Sleep, DBAS-16).

Results: In the CBT alone group, significant pre-post improvements were observed on all measures but two SF-36 subscales, while in the CBT+Med group, significant changes were observed only on the DBAS-16 scores. After the extended phase, further improvements on the DBAS-16, MFI and SF-36's Vitality subscale scores were observed in the CBT alone group while in the "no additional treatment" group, SF-36's Mental Health and Physical Functioning subscale scores showed significant worsening. In the CBT+Med group who continued receiving medication, significant positive changes were observed on most measures, while in the medication tapering group, significant positive changes were observed only on the MFI and SF-36's Social Functioning and Vitality subscales.

Conclusions: Results suggest that CBT for insomnia has some positive effects on daytime functioning. However, adding medication to this therapy may hinder its positive effects in the initial treatment phase. Nevertheless, benefits associated with intermittent medication use seemed to appear in the extended treatment phase. Further research is needed to examine ideal combinations of CBT and medication, especially in those who benefit less from either approach alone.

Keywords: Insomnia, treatment, CBT

P033

Are The Effects Of Insomnia Treatment On Daytime Measures Clinically Important?

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Introduction: Clinical significance of treatment outcomes is seldom reported in insomnia studies. This study assessed if change experienced by individuals on daytime variables after CBT for insomnia was clinically meaningful. An additional goal was to evaluate if adding medication to CBT may enhance the proportion of participants experiencing a meaningful impairment reduction.

Methods: One hundred and sixty individuals (61% women; mean age: 50.3 years) with persistent insomnia were randomized to one of two 6-week insomnia treatment conditions, CBT alone or CBT plus medication (CBT+Med; zolpidem). Daytime variables assessed included fatigue (Multidimensional Fatigue Inventory; MFI), anxiety (Beck Anxiety Inventory; BAI), depression (Beck Depression Inventory; BDI), health perception (SF-36) and beliefs about sleep (Dysfunctional Beliefs about Sleep, DBAS-16). Participants were classified as "dysfunctional" or not on each measure at baseline based on descriptive data from normative or insomnia-free samples. At post treatment, participants were reclassified as recovered, improved, unimproved or deteriorated on each of the measures. Proportions of participants experiencing meaningful impairment reduction, i.e. recovered or improved, in each treatment condition were then compared.

Results: Depending on the daytime measure considered, the proportion of recovered participants ranged from 31.2% to 83.3% in the CBT alone condition and from 9.4% to 71.0% in the CBT+ Med condition. CBT alone yielded significantly higher proportions of improved and recovered subjects on the following variables: Depression (x^2_1=3.62, p=.05), SF-36 Vitality scale (x^2_1=6, p<.05), SF-36 Social functioning scale (x^2_1=4.7, p<.05) and SF-36 Role-Emotional scale (x^2_1=4.5, p<.05).

Conclusions: Unlike statistical significance testing, clinical significance analyses provide information regarding the standing of treated and untreated individuals relative to healthy controls. These findings suggest that some daytime deficits associated with insomnia may remit and return to normative levels with treatment. The results provide further evidence that CBT alone may be more effective than CBT+Med at ameliorating some of these daytime deficits.

Support: National Institute of Mental Health (MH60413)

Keywords: Insomnia, treatment, CBT

5. Dreaming

P034

Schizophrenia-Like Cognition In Rem Sleep Mentation

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Introduction. There is growing evidence that schizophrenia and REM sleep share similar neurophysiological processes. The notion that schizophrenia also shares cognitive features with REM sleep mentation is widely believed but has not been empirically tested. The present study examines whether three key cognitive characteristics of schizophrenia are also found in REM sleep mentation, i.e., 1) diminished logical thinking, 2) diminished ability to appreciate bizarreness and 3) inappropriate emotional reactivity.

Methods. Fourteen healthy participants aged 21-32 years (M=23.4) slept an adaptation and 2 experimental nights in a sleep laboratory. For each experimental night they were awakened for mentation reports four times each after 10 minutes of REM sleep. They rated the mentation on 9-point Likert scales for the presence and awareness of bizarreness, the logical rigor of thinking and the appropriateness of emotional responses.

Results. One-sample t-tests revealed that ratings of logical rigor (M=7.94, SD=0.87) were significantly higher than the middle value of the measurement scale (5; $t(8)=10.11$, $p=.001$) while ratings of awareness of bizarreness (M=2.70 SD=2.27) were significantly lower (5; $t(11)=.-3.64$, $p=.003$). Thoughts were considered very logical (7, 8 or 9 out of 9) in 19 cases (91%). Of the 35 mentation reports containing bizarreness, in only 2 cases (6%) was it fully appreciated. In all cases (100%) emotional reactions were characterized as entirely appropriate to the dreamed situation.

Discussion. The results suggest that some cognitive deficiencies commonly characterizing schizophrenia, i.e. inappropriate emotional reactivity and diminished logical thinking, are not found in REM sleep mentation. Nonetheless, subjects reported a diminished ability to appreciate bizarreness during REM sleep mentation, a phenomenon which closely resembles the diminished ability for people with schizophrenia to appreciate bizarreness. This may stem from common neurophysiological characteristics shared by REM sleep and schizophrenic pathophysiology.

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P035

Lucid Dreaming Is Associated With Sleep Paralysis But Not Nightmares

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Introduction: Lucid dreaming is understood as an intrusion of wake-like cognition (self-awareness) into REM sleep. In contrast, sleep paralysis (SP) is believed to consist of an intrusion of REM psychophysiology (muscle atonia and vivid dreamlike hallucinations) into wakefulness. Research indicates that the two phenomena are associated, but it remains unclear whether LD is related specifically to sleep disturbances involving state dissociation, such as SP, or to sleep disturbances in general. It is hypothesised that LD frequency will be correlated with the frequency of SP (which involves state dissociation), but not with the frequency of nightmares (which do not involve state dissociation).

Methods: 245 participants (141 female, 85 male, 15 not specified, mean age=30.9, sd=13.5) completed an online questionnaire. LD, SP and nightmare frequency were measured on 7-point Likert scales; SP- and nightmare-related distress were measured on 5-point Likert scales.

Results: LD frequency positively correlated with SP frequency ($r=.24$; $p<.001$), SP distress ($r=.21$; $p=.001$) and SP intensity ($r=.29$; $p<.001$), but not with NM frequency ($r=.03$; $p=.67$), or NM distress ($r=.09$; $p=.18$).

Discussion: Results of the present study confirm the hypothesis that LD is associated with SP but not nightmares. While the phenomena of LD and SO are in many respects entirely different, their manifestations may depend on a common propensity for state-dissociation which is not implicated in nightmare production. LD may not be associated with sleep disturbances more generally.

P036

Behavioral Enactment Of Dreaming In A Normal Population

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Introduction: The behavioral enactment of dreaming (BED)—movements/emotional expressions occurring while dreaming—is prevalent (57%) among new mothers.[1] However, little is known about its prevalence in other populations.

Methods: 492 undergraduates (M: 182; F: 286; unspec.: 24; Mn_{age}: M: 19.2±1.73; F: 19.0±1.55, p=.180) completed a 7-item questionnaire about dream-enactment behaviors: speaking, crying, smiling/laughing, fear, anger, other motor activity, sexual arousal (0=never, 1=rarely, 2=sometimes, 3=often), and about sleep-walking and sleep-talking (with no clear recall of a dream).

To determine whether BED is distinct from sleep-walking/sleep-talking, exploratory factor analysis (principal-components extraction, varimax rotation) was conducted. To determine gender effects, a MANOVA with a gender variable and 7 dream-enacting behaviors as dependent measures was conducted, followed by a MANCOVA adding covariates sleep-walking and sleep-talking.

Results: BED was prevalent and frequent. 79% reported at least one of four emotional behaviors 'sometimes' or 'often'; 87% reported at least one of all 7 behaviors. A 3-factor solution (54.2% VAF) distinguished BED from other parasomnias. Factor 1 (25.8%) grouped the 4 emotional behaviors (loadings: .650-.751) and motor activity (.521). Factor 2 (17.3%) grouped sleep-walking (.779) and sleep-talking (.840). Dream-speaking loaded equally on Factors 1 (.487) and 2 (.481). Factor 3 (11.1%) grouped sexual arousal (.827) and age (.824).

There was a multivariate gender effect (Hotelling-T=.366, $F_{7,470}$=24.570, p<.0000001) and univariate effects for speaking (p<.042), crying (p<.0000001), fear (p<.0001), smiling/laughing (p<.058) and sexual arousal (p<.0000001; Figure). Controlling covariates did not diminish the multivariate effect (T=.363, $F_{7,457}$=23.693, p<.0000001) but rendered all 7 univariate effects significant at p<.001.

Discussion: BED is prevalent in an undergraduate population. Except for sexual arousal, females report all behaviors more often than do males—especially crying and fear. Dream-enacting behaviors form an entity distinct from sleep-walking/sleep-talking, though are mildly intercorrelated. Dreamed sexual arousal is a distinct, highly prevalent, behavior correlated with age and male gender.

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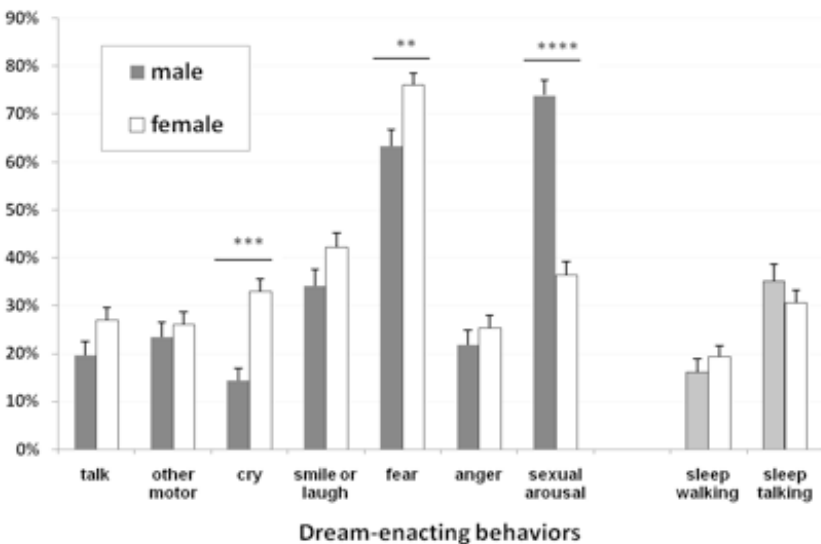
Figure 1. Percent of subjects reporting dream-enacting speaking, motor activity, emotions and sexual arousal 'sometimes' or 'often'. Females reported more frequent behaviors on all items except sexual arousal, which was characteristic of males (unpaired t-tests for gender: **p<.005; ***p<.000001; ****p<.0000001). Sleep-walking and sleep-talking formed a distinct factor that did not account for the observed gender differences.

P037

Evolution Of Gender Differences In The Dreams Of University Students

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Introduction: In the 1950s and 1980s, significant gender differences in the dream content of American college students were reported[1,2]. However, it has been suggested that these differences may be affected by significant changes towards homogenization of gender roles in Western society. We examined this hypothesis with a sample of Canadian students.

Methods: So far, two morning home diary dreams were collected from each of 152 Canadian university students (76 males, 76 females, age range 18 to 24). Each dream was then coded by two raters using the Hall and Van de Castle scales of characters, social interactions, settings, self-concepts, misfortunes, good fortune, success and failure as well as striving. Gender differences were calculated using Cohen's h for the 25 variables previously compared.

Results: Of the 14 variables that previously showed significant differences, only 4 differences remained in our sample: male to female ratio of dream characters (M>F, h=0.23, p=0.04), percentage of characters that were animals (F>M, h=0.21, p=0.001), aggression to friendliness ratio of social interactions (M>F, h=0.27, p=0.021) and amount of indoor settings (F>M, h=0.21, p=0.042). In addition 3 variables showed significant differences that were not previously observed: amount of aggressions initiated by dreamer (M>F, h=0.37, p=0.021), percentage of bodily misfortunes (F>M, h=0.29, p=0.049) and percentage of self-negativity (M>F, h=0.23, p=0.032). Other variables showed no significance. Consequently, while previous studies showed 14 gender differences in the 25 variables studied, ours only found 7.

Discussion: It will be interesting in future studies to determine if the alteration in gender differences is attributable to changes in social roles and/or cultural differences between Americans and Canadians or simply to sample size.

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P038

Word Associations Improve Automatic Analysis Of Dream Emotional Tone

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Introduction: We previously demonstrated that Logistic Regression used in dream negative emotional tone classification achieved 59% agreement with a human judge using the following attributes: text mining, word-correlation, affect progression and dreamer's experience of Joy, Happiness, Apprehension, Anger, Sadness, Confusion, Fear and Anxiety (Razavi, 2008). Here we attempt to improve the automatic analysis by adding a new class of attributes: word-association. With word-association we attempt to include those words that might be intended but not explicitly expressed.

Methods: 458 English dream reports were used to construct a list of words and their definitions from Wordreference.com and Wikipedia.org. The word frequencies of definitions were used to construct the word-association matrix. The normalized matrix produced a vector relating a word to all words. The vectors of all words were summed and used as attributes for each dream. All attributes were subjected to the weka.attributeSelection. BestFirst algorithm for attribute selection. 66% of the dreams were used for training the weka.classifiers.functions.SimpleLogistic model for both positive and negative affect. The remaining 34% were used to test the model with a 10 fold cross-validation.

Results: On the negative affect scale, Simple Logistic Model achieved a moderate machine-human judge agreement of 62%, kappa 0.466, *MSE* of 0.388. Here we find a 3% improvement over the previous model. The model for the positive affect scale, tested for the first time, produced a moderate agreement of 77%, kappa 0.520, *MSE* of 0.317.

Discussion: Word-association attributes improved the machine-human agreement for the negative affect scale and was associated with a promising agreement on the positive affect scale. This suggests that word-association is a set of attributes that may not be explicitly communicated but is a significant contributor to automated classification. This also suggests that other forms of implicit communication such as symbols and themes may be strong contributors for affect classification.

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6. Chronobiology

P039

Screening For Sleep Quality And Chronotype In Elite Winter Athletes

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Introduction: The prevalence of poor sleep quality in elite athletes has been described in a previous pilot study (Samuels 2008). The pilot study used the Pittsburgh Sleep Quality Index (PSQI), Athlete Morningness/Eveningness Scale (AMES) and the Adjusted Neck Circumference (ANC). A larger sample of athletes was screened in a similar fashion as the pilot study to determine the consistency and validity of the pilot study findings.

Methods: Elite winter athletes (N=118) were screened using three standardized questionnaires; PSQI, AMES, ANC. Average age was 24.2 years old, with a range of 17-38. Fifty-eight percent were male (68/117).

Results: Results of the PSQI were consistent with the pilot study, the prevalence of poor sleep quality using a standard cutoff score of 5 was 56% (65/116). In the pilot study 83% (54/65) scored > 5. Using a more conservative cutoff score of 8 yielded a prevalence of 15% (17/116). In the pilot, 23% (15/65) scored > 8. Eighty-five percent of athletes (100/117) were identified as being either "Moderate Morning or Mid Range" chronotype, whereas in the pilot 80% (53/66) were identified as "Moderate Morning or Mid Range" chronotype. Prevalence of moderate to high risk for sleep apnea was 15% (18/117), similar to the pilot study, 13% (3/24).

Pittsburgh Sleep Quality Index (PSQI): Global Score Distribution

Conclusion: The results of this follow-up study support and confirm the results of the pilot study that indicates a high prevalence of poor sleep quality in this population. Contrary to the investigators expectation the prevalence of eveningness chronotype is low, consistent with the results of the pilot study. The prevalence of moderate to high risk for sleep apnea is low. The results of the pilot study and this follow-up study are consistent. The validity of the findings reinforces the need to develop a sleep screening tool for elite athletes that discriminates good from poor quality sleepers and screens for chronotype.

References: Samuels C.H. Sleep, Recovery, and Performance: The New Frontier in High-Performance Athletics. Neurologic Clinics 2008; 169-180.

P040

Effect Of Circadian And Sleep-Wake State On Heart Rate Variability In Humans

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Introduction: Recent evidence suggests that a complex interaction between sleep and the endogenous circadian oscillator modulates heart rate (HR) in humans. Using an ultra-rapid sleep-wake (URSW) procedure, we investigated the effect of circadian and homeostatic processes on heart rate variability (HRV).

Methods: Five healthy participants (3 men, 2 women, mean age ± SD: 27.1 ± 3.2 years), were studied for 5 days in time isolation. After an 8-hour baseline sleep episode, participants underwent a 72-hour URSW consisting of 60-min waking episodes in dim light (<10 lux) alternating with 60-min nap episodes in total darkness. During the procedure, participants remained in a semi-recumbent position and were served balanced iso-caloric snacks. HR was monitored continuously throughout the procedure. RR interval, high and low frequencies (HF, LF) and LF/HF ratio were calculated using wavelet transform. Data were binned per waking and napping episode then folded per day. Two-way ANOVA (factors: Time of day x Sleep-wake state; i.e. wake and nap episodes) was used to analyse HRV data.

Results: For all parameters analysed, there was a significant main effect of sleep-wake state (RR, HF, LF/HF: $F_{(1,4)} \geq 9.79$, $p<0.05$), and of time of day (RR, HF, LF/HF: $F_{(11,44)} \geq 3.78$, $p<0.05$). However, none of these parameters showed a significant interaction of time of day and sleep-wake state.

Conclusions: HRV parameters have been shown to be affected by time of day and sleep-wake state. Naps promote parasympathetic modulation and reduction of HR, and this effect seems to be constant throughout the day. Sleep stages will be analysed to further address the interaction between these factors on HRV.

P041

Effect Of Bright Light On Performance Of Police Officers On Rotating Shifts

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Introduction: Working on atypical schedules can lead to a reduction in performance and vigilance as a result of circadian misalignment[1]. The aim of the present study was to assess changes in performance of police officers working nights throughout a phototherapy field trial.

Methods: Police officers (age ± SD; 30.1 ± 5.2) working rotating shifts were assigned to 2 groups (control, n=9; intervention, n=8) and were studied before, after (in laboratory) and during (ambulatory) a series of 7 consecutive night shifts. Participants in the intervention group were instructed to expose themselves to bright light during their night shifts (Litebook device, Medicine Hat, Alberta, Canada), to wear orange-tinted glasses from sunrise until bedtime and to sleep for 8 hours following their night shifts. A psychomotor vigilance task[2] (PVT) was scheduled every 2 hours in the laboratory and at start, middle and end of shifts during each night shifts. Three-way ANOVA (factors: Group X Day X Time of Day) was used to analyse PVT data.

Results: Police officers had greater performances during the 1st laboratory visit vs. the 2nd visit (p≤0.008). At the 2nd visit, they had more lapses with increased time awake (p=0.006) whereas performance was more stable during the 1st visit (p=0.19). A similar interaction was observed for the slowest reaction speed (p=0.053). No between-group difference was observed. In ambulatory conditions, the average of the fastest reaction speed significantly decreased throughout the 7 night shifts in the control group (p≤0.04) but not in the intervention group (p≥0.35). Similarly, median speed was reduced throughout the night shifts in the control group (p=0.0008), but not in the intervention group (p=0.47).

Conclusions: Exposure to portable lamps can stabilized psychomotor performances of night shift workers in field operations. A faster circadian adjustment and/or a direct stimulant effect of light could contribute to these effects.

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P042

Daytime Sleepiness And Natural Light Exposure In Students Who Work During The School Year: Preliminary Results

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Introduction: Adolescents and young adults were acknowledged as a population at increased risk of daytime sleepiness. Recent studies showed that bright light exposure decrease subjective sleepiness. The objective was to assess whether excessive daytime sleepiness (EDS) in students who work during the school year relates to their corresponding environmental light exposure.

Methods: Twenty students (1 secondary, 15 college, 4 university) aged 19-20 years (12 males) wore an actigraph with a light sensor (Actiwatch-L, MiniMitter) and completed the Social Rhythm Metric (SRM-5) for two consecutive weeks during the winter/spring semester as well as the Epworth Sleepiness Scale (ESS) and the morningness- eveningness questionnaire (MEQ). EDS was defined by ESS≥11. Ambulatory light measurements averaged over two weeks included daily pattern of light exposure and number of minutes per 24-h at different light intensity levels (0-15, 15-100, 100-500, 500-1000, >1000 lux). Chi-square and t-tests were used for statistical comparisons.

Results: EDS was present in 30% of subjects. Subjects with and without EDS did not differ with gender, school level, weekly hours of school (23.0 vs. 23.8) and work (24.8 vs 21.2), timing of work time (day, evening, night), sleep onset (01h18 vs 00h36), offset (8h36 vs 8h30) and sleep duration (362 vs 386 min), and SRM-5 (2.8 vs 2.6) and MEQ scores (48.9 vs 50.0). In subjects with EDS, averaged light exposure was significantly lower at 9h and between 12h and 17h (p<0.05; figure 1). Similarly, total bright light (>1000 lux) exposure was significantly lower in EDS than in non-EDS subjects (12 vs 38 min; p<0.01).

Discussion: These results suggest that EDS cannot be ascribed to a difference in circadian phase associated with light exposure. Reduced bright light exposure may contribute to EDS but further analyses must verify which of morning or afternoon bright light is more important as well as the seasonal effect.

Figure 1.
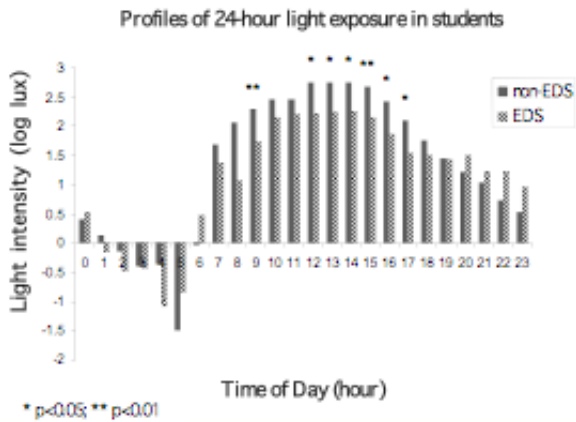
Profiles of 24-hour light exposure in students

P043

Sleep Duration, Light Exposure And Biomarkers Of Melatonin Among Rotating Shift Nurses

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Introduction: Long-term night shift work has been identified as a potential carcinogenic risk factor. It is hypothesized that increased light at night exposure during shift work reduces melatonin production, which is associated with increased cancer risk. Sleep duration has been hypothesized to influence both melatonin levels and cancer risk and it has been suggested that sleep duration could be used as a proxy for melatonin production.

Methods: A cross-sectional study of light intensity exposure during sleep and work, sleep duration, and melatonin levels was conducted among 61 rotating shift nurses at Kingston General Hospital. Light was measured using a light intensity data logger, and melatonin concentrations were measured from urine and saliva samples, collected over a 24-hour period. Sleep duration was assessed from a one-day diary.

Results: Light intensity was significantly higher during sleep for night workers ($p < 0.0001$), while mean sleep duration for day workers (8.27 hours) was significantly longer than for night workers (4.78 hours, $p < 0.0001$). An inverse association ($p = 0.002$) between light exposure and urinary melatonin levels was observed; however, the relationship was no longer significant when stratified by shift group. Analysis of salivary melatonin levels demonstrated that circadian rhythms of melatonin production in night workers were not shifted, such that peak melatonin production occurred at night during work and not in the day during sleep. No significant correlation was observed between sleep duration and urinary melatonin levels.

Discussion: Since peak melatonin production occurred at night among all workers, to avoid false conclusions regarding the associations of sleep duration and light intensity with melatonin levels, confounding by circadian rhythm must be taken into account in future studies. In this study sleep duration was not correlated with urinary melatonin levels, suggesting it may not be a good proxy for melatonin production.

P044

Changes in circadian rhythm evident during an acute randomized placebo-controlled trial of methylphenidate

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Introduction: Attention-deficit/hyperactivity disorder (ADHD) is a highly prevalent disorder occurring in approximately 3-5% of school-aged children. It is commonly treated with stimulant medications such as methylphenidate. Stimulant medications have been found to have negative side effects including appetite suppression and insomnia. It has also been suggested that they may alter the timing or regularity of circadian motor activity levels. The current project aimed to investigate the impact of stimulant medication on the strength and timing of circadian rhythms in children with ADHD.

Methods: Twenty stimulant medication-naïve children with ADHD were monitored for changes in motor-activity during a 3-week blinded placebo-controlled medication trial to examine the impact of immediate-release methylphenidate hydrochloride (MPH-IR). Motor activity was measured by actigraphy and twenty-four hour activity profiles were analyzed using three mathematical techniques to identify measurable changes in circadian rhythms.

Results: MPH-IR was not found to affect the strength of the daily rhythm but was related to a measurable phase-delay in the timing of the daily rhythm. Children on medication were found to experience night-time increases in motor activity during the sleep-onset latency period. The children in this sample also had a strong and stable daily rhythm over each week of the medication trial, as measured by the autocorrelation analysis.

Conclusions: Children taking MPH-IR have been found to experience prolonged sleep-onset latency. Clinicians and parents of children being treated with stimulant medication for ADHD should be aware that it may cause disruption of sleep/circadian rhythms. Behavioral strategies to improve sleep may be useful for children experiencing these negative effects from medication.

P045

Selective Increase Of Slow Waves Sleep (Sws) By A Novel Melatonin Partial Agonist

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Introduction: Melatonin is a neurohormone implicated in the regulation of sleep and circadian rhythms and binds two G-protein coupled brain receptors, MT1 and MT2. The differential role of these two receptors in sleep function is completely unknown. Here we propose a novel drug acting as an MT2 receptor partial agonist, called UCM765 (N-{2-[(3-Methoxyphenyl)-phenylamino]ethyl}acetamide), with a 100-fold higher affinity for the human recombinant receptor MT2 (pKi = 10.18) than for MT1 (pKi = 8.38) .

Methods: In order to test the sleep-promoting properties of UCM765, we recorded electroencephalogram (EEG) and electromyogram (EMG) in freely moving rats (n=7-11) from 6:00-9:00 PM following a subcutaneous injection of vehicle, diazepam (2mg/kg) or UCM765 (40mg/kg).

Results: Rats treated with UCM765 exhibited a shorter latency (min) of onset of the first slow wave sleep (SWS) episode compared to control ($p < 0.05$) similar to diazepam. The UCM 765 also increased the total duration (min) of the SWS ($p < 0.05$). Then, we administered the selective MT2 antagonist 4-PPDOT (10mg/kg) prior to UCM765. The antagonist 4-PPDOT blocked the effects on the latency of SWS (p=0.02) as well on the duration of SWS (p=0.006). Results for paradoxical sleep (PS, or REM sleep) indicate that UCM765, prolonged the latency of the first episode of PS ($p < 0.05$), similar to diazepam, but did not change the duration or quantity of PS episodes. Next, we assessed the properties of UCM765 (40 mg/kg, six times per day, every 4 hours) across 24 hours. Remarkably, the 24-hour EEG and EMG showed that UCM765 increases SWS only during the inactive or light phase (p=0.006) and not during the active or dark phase.

Conclusion: These results suggest that the MT2 partial agonist UCM765 may be a novel and selective hypnotic drug that increases SWS (restorative sleep) during the sleeping period with no effect during active time.

P046

Short Exposure To Blue-Enriched White Light Does Not Further Impact Alertness Level When Used At The End Of The Night

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Objectives: In night shift workers, it has been suggested that blocking blue light to avoid the resynchronizing effect of morning sunlight, could be detrimental to vigilance. But so far, the effect of blue light on vigilance has been tested only at the beginning of the night using dim light (12 μW/cm^2/s). We therefore challenged the impact of a bright light of 500 μW/cm^2/s (with or without blue) on the second half of the night shift, when vigilance is expected to be at its lowest.

Methods: Ten participants (5 M, 5 F) were tested on two consecutive nights, during which the VAS and the Conners' continuous performance test 2 (CPT II) were performed at 23h30, 1h30, 3h30 and 5h30. First night served as baseline where subjects were maintained awake and tested under less than 5 lux. On the second night, they were exposed to two 500 μW/cm^2/s 30 minutes pulses of light at 3h00 and 5h00, produced by a blue-enriched white LED source (Litebook™). Since participants wore blue blockers for the first pulse, the light source was set so that 500 μW/cm^2/s of light was received by the eyes behind the glasses.

Results: Repeated measures ANOVA revealed a night [$F(1,7) = 6.12$, $P = 0.04$] and a time [$F(3,21) = 11.35$, $P = 0.003$] effects, but only for the VAS with subjective vigilance decreasing with time on both nights, although being better on the second night during both light conditions.

Discussion: The better subjective vigilance on the second night may happen because participants were more tired on the first night, due to a longer sleep deprivation period. However, it is also possible that short pulses of bright light can improve vigilance, but that at an intensity of 500 μW/cm^2/s, blue wavelengths are not essential.

P047

Polysomnographic Sleep Across The Circadian And Menstrual Cycles In Healthy Females

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Introduction: Sleep propensity and organization vary across circadian phase[1]. We hypothesize that the expression of core body temperature (CBT) and melatonin, two reliable circadian markers, may influence changes in sleep at different phases of the menstrual cycle.

Methods: Eight women with regular menstrual cycles participated in an ultra-rapid sleep-wake cycle (URSW) designed to assess the circadian variation of sleep during the mid-follicular (MF) and mid-luteal (ML) phases of the menstrual cycle. After a 3-week stabilization of the sleep-wake cycle to an 8-hr sleep episode, participants entered the laboratory for a nocturnal polysomnographic (PSG) sleep recording, followed by a 72-hr URSW (36 cycles of 60-min wake episodes in constant conditions alternating with 60-min naps) at MF and ML. PSG sleep and CBT were recorded, and salivary melatonin was sampled (1x/hr).

Results: During nocturnal sleep episodes in ML compared to MF, sleep efficiency (SE) and REM sleep decreased significantly, whereas significant increases were observed in sleep onset latency (SOL) and non-REM sleep. Throughout the URSW, total sleep time, SE, SOL, REM onset latency (ROL), stage 2, SWS, REM and non-REM sleep showed a significant circadian variation. A significant main effect and a trend for a menstrual phase difference was observed for ROL and REM sleep, respectively. A trend for a menstrual phase x time interaction was observed for ROL. During ML compared to MF, ROL in naps at 15h00 and 05h00 was significantly lengthened, whereas REM sleep was significantly decreased in naps at 01h00 and 05h00. The circadian profile of salivary melatonin was similar at both menstrual phases; however in ML, participants demonstrated significantly increased CBT and decreased CBT amplitude.

Conclusions: We observed moderate but significant sleep changes across the menstrual cycle in healthy women. The variation of body temperature and/or sex hormones across the menstrual cycle may interact with circadian processes to alter sleep characteristics.

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P048

Quantitative Sleep Electroencephalogram Across The Menstrual Cycle In Women With Premenstrual Dysphoric Disorder

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Introduction: Hormone fluctuation across the menstrual cycle can affect body temperature, mood and sleep. Sleep EEG varies across the menstrual cycle in healthy women, with increased spindle frequency activity (SFA) observed during the luteal phase (LP)[1]. A report of women with severe premenstrual syndrome studied at two menstrual phases determined that these women experience a similar increase in SFA during the LP[2]. Out current aim was to investigate quantitative sleep EEG across eight phases of the menstrual cycle in women diagnosed with PMDD based on DSM-IV criteria.

Methods: Seven PMDD participants entered the laboratory every third night throughout an entire menstrual cycle for nocturnal PSG recordings. Visits were allocated into eight menstrual phases including: menses (ME), early follicular (EF), mid-follicular (MF), late follicular (LF), peri-ovulatory (PO), early luteal (EL), mid-luteal (ML) and late luteal (LL). The C3-A2 EEG signal (sampling rate: 250 Hz) was subjected to spectral analysis, and non-REM sleep (Stages 2-4) data within the 0-25 Hz range was separated into 1-Hz bins, and subsequently combined into 1-5 Hz and 12-16 Hz bins, reflecting slow wave activity (SWA) and SFA, respectively.

Results: Activity in the 13-14, 14-15, and 15-16 Hz bands varied significantly across the menstrual cycle (p<0.001). Menstrual phase had no effect on SWA (p=0.86), whereas a significant main effect of menstrual phase was observed for SFA (p=0.001), with significantly increased SFA observed in all post-ovulatory phases (i.e. EL, ML and LL) compared to MF and LF (p<0.05).

Conclusions: This report detailing sleep EEG across an entire menstrual cycle in PMDD women confirms that these women, like healthy participants, experience LP-associated increases in SFA. These subtle but consistent changes likely result from the modulation of $GABA_A$-receptors by neuroactive progesterone metabolites, whose levels increase after ovulation[1]. We also lend support to the hypothesis that sleep homeostasis is unaffected by menstrual phase[1].

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7. Physiology & Neurophysiology

P049

Sleep-Wake And Circadian Contribution To Changes In Per2 Protein In Whole Living Mice

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Introduction: Sleep is regulated by homeostatic and circadian processes which are thought to act independently. We have shown, however, that expression of clock genes, in particular *Per2*, increases during waking and decreases during sleep. Here, we investigate the dynamics of PER2 protein levels as a function of time-of-day and time-spent-awake in Per2: Luciferase knock-in (Per2Luc) mice.

Methods: Brain mRNA levels for *Per2* were measured by qPCR in sleep-deprived (6h SD starting at ZT0, -6, -12, or -18) and time-matched C57BL/6J controls. The sleep of another group of animals, implanted with EEG/EMG electrodes, was recorded during baseline and during recovery after the same 4 different timing of SD and relative delta power during sleep was calculated. PER2 protein levels were measured in brain, liver, and kidney of Per2Luc mice using an *in vivo* Imaging system (IVIS-3D, Xenogen). Diurnal changes in PER2 were sampled at 3h intervals and sleep-wake dependent changes after 6h SD and 2h of recovery sleep.

Results: Expression of the circadian *Per2* gene followed the diurnal sleep-wake distribution in control conditions and increased with SD but remained rhythmic due to a time-of-day modulation of the SD-induced increase. The decay of delta power during the first 6-h of recovery sleep differed between the timing of SD and the higher initial level was observed after the SD occurring at ZT6 and ZT12 which corresponds to the timing of the maximum SD-dependent increase in *Per2* observed after ZT6 SD. PER2 protein equally varied with time-of-day in brain, liver, and kidney. SD increased PER2 protein in all three tissues albeit with different dynamics.

Conclusion: We show that PER2 protein can be followed around the clock in whole living mice. As mRNA, PER2 protein also increased with sleep loss, supporting a role for *Per2* in the homeostatic regulation of sleep.

P050

Decreasing The Stress Response Does Not Rescue The Impaired Homeostatic Sleep Rebound In Dba/2j Mice

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Introduction: We have previously shown that both the change in EEG delta power and in clock-gene expression after sleep deprivation (SD) in mice depends on genetic background. Among several inbred strains, DBA/2J (D2) mice showed the smallest increase in delta power and the largest changes in clock-genes expression in the forebrain (Franken et al., 2001, 2007). SD in rodents induces a stress response as it increases corticosterone secretion (Tobler et al., 1983; Hairston et al., 2001). This study aimed at determining the contribution of changes in corticosterone to the homeostatic response of EEG delta power and clock-gene expression.

Methods: *Experiment 1:* Mice from C57BL/6J, AKR/J, and D2 inbred strains were submitted to SD by gentle handling (ZT0-6) and killed immediately after for plasma corticosterone measurements. *Experiment 2:* Sleep of adrenalectomized (ADX) and sham-lesioned (sham) D2 mice was recorded for a baseline day and during recovery from SD, before and after surgery. *Experiment 3:* ADX and sham D2 mice were submitted to either baseline condition or SD and killed at ZT6 when brains were collected for analysis of forebrain clock-gene expression using qPCR.

Results: SD induced a higher increase in corticosterone secretion in D2 mice compared to the other two strains. Nevertheless, the delta power rebound after SD was similar in ADX and sham D2 mice (Fig.1A). ADX attenuated the SD-mediated increase in *mPer1-3* expression but did not change the SD-dependant decrease in *mDbp* mRNA (Fig.1B).

Discussion: While the larger SD-mediated increase in corticosterone observed in D2 mice could partly explain the larger increase in *mPer* expression in this strain, a higher stress response seems not to underlie the lower EEG delta power rebound after SD in this strain. The findings also indicate that stress does not importantly contribute to the homeostatic regulation of EEG delta power.
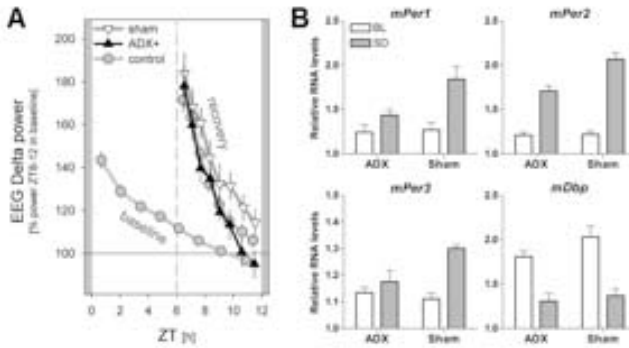
Figure 1: A) Dynamics of EEG delta power during the Light periods of baseline and recovery after 6h SD before and after ADX or sham surgery. B) Forebrain expression of mPer1-3 and mDbp at ZT6 after ADX or sham surgery for baseline conditions (BL) or after 6h SD.

References: Franken et al., *J Neurosci*, 21, 2610-21, 2001; *BMC Neurosci*, 8, 87; 2007; Hairston et al., *Neurosci Lett* 315, 29-32, 2001; Tobler et al., *Neurosci Lett* 35, 297-300, 1983.

P051

Increased Gaba-Ergic Activity In The Pedunculopontine Nucleus Reduces Rem Sleep And Impairs Learning In Rats

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Introduction: Newly formed memories are initially fragile, and require "consolidation" to be transformed into an enduring state. Memory consolidation may occur during intensified post-learning rapid eye movement (REM) sleep. REM deprivation during these periods impairs subsequent performance (termed REM sleep windows; RSW). The pedunculopontine nucleus (PPT) has been implicated in both the generation of REM sleep and memory processes.

Methods: Twenty-five male Sprague-Dawley rats (250-300g) were implanted with EEG and EMG electrodes. Guide cannulae were implanted bilaterally 2mm above the PPT. After recovery, and 24 hours baseline recording, animals were trained on the two-way avoidance task for 100 trials over two days (50 trials/day) and re-tested (25 trials) on day 3. EEG was recorded for 22h after training on training day 1 (TD1) and TD2. Rats were injected with either the $GABA_B$ agonist baclofen (n=8) or saline (n=16) into the PPT at 0300h to coincide with the start of a known RSW. Saline rats were subdivided into a learning group (LG) (n=8) that avoided the footshock on 60% of the last 20 trails. The remaining rats (n=8) were assigned to the non-learning group (NLG).

Results: The LG increased avoidances ($F(2,14)=13.09$, $p<.0001$) whereas the NLG and baclofen group did not change significantly (**figure 1A**). Groups differed in the change in REM (percentage of total recording time; %TRT) from baseline on TD1 ($F(10,95)=4.21$, $p<.001$) during the 5th 4hr post-training block (hours 17-20) (**figure 1B**; $F(2, 21)=15.29$, $p<.0001$). The LG had significantly more REM than the NLG ($t(19)=2.79$, $p<.05$) and baclofen groups ($t(19)=7.39$, $p<.01$).

Discussion: PPN infusions of baclofen decreased REM and impaired subsequent memory performance. Rats receiving saline infusions were distinguishable into learners and non-learners, whereas no baclofen rats were able to learn the task. Normal GABA-ergic transmission is necessary for REM to occur and for the consolidation of incentive learning.

P052

Rem Sleep Insomnia And Decreased Ppt Cholinergic Neurons Following Myocardial Infarction In The Rat

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Introduction: We have already shown that myocardial infarction (MI) in the rat is followed within a few weeks by cell loss in the limbic system due to apoptosis, together with a "post MI behavioral syndrome" characterized by signs of anxiety and depression[1]. Here we show that the post MI syndrome is accompanied by selective losses of Paradoxical (or REM) sleep (PS) and of cholinergic neurons in the pedunculopontine tegmental area (PPT).

Methods: Ten adult Sprague-Dawley rats were implanted with chronic EEG and EMG electrodes; baseline sleep was recorded seven days after, for 24h. The following morning MI was induced by occluding the left coronary artery for 40 minutes in four rats while the six other rats were used as sham controls. Sleep was recorded again two weeks after MI. At the end of the protocol, the rats were perfused and quantification of choline acetyltransferase (ChAT) expressing neurons was carried out in the pedunculopontine tegmental area (PPT) and the laterodorsal tegmental area (LDT), using immunohistochemistry. Results in both groups of rats were compared using t-tests for independent samples.

Results: Compared to sham rats, MI rats displayed less total sleep time and less time in PS, particularly at the light-dark transition. We also found that, compared to sham rats, MI rats showed a significant reduction (20%) of ChAT neurons in the PPT area, not in the LDT.

Conclusion: The present results extend the apoptotic effects of MI in the limbic system to brainstem cholinergic area known to control PS. The selective loss of PS at the light-dark transition is not typical of anxiety and depression models and this needs to be further investigated.

Reference: Wann BP, Bah TM, Boucher M, Courtemanche J, Le Marec N, Rousseau G, Godbout R. Vulnerability for apoptosis in the limbic system following myocardial infarction in the rat: a possible model for human post-infarct major depression. J Psychiatry Neurosci. 32:11-6, 2007.

P053

GABA$_B$-mediated inhibition plays a critical role in mediating REM sleep atonia

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Introduction: Skeletal muscle tone is potently suppressed during REM sleep. We recently found that REM sleep atonia could not be prevented in masseter muscles by blockade of glycine and GABA$_A$ receptors at the trigeminal motor pool. REM atonia persisted even when glycine- and GABA$_A$-mediated inhibition was blocked and high doses of AMPA were simultaneously applied. Accordingly, we concluded that REM atonia is triggered by an unidentified inhibitory mechanism(s). Although GABA$_B$ receptors are present on somatic motoneurons and postsynaptically inhibit them, the role of these receptors in mediating REM atonia is unknown. The aim of this study was to determine if GABA$_B$ receptors play a role in suppressing muscle tone during REM sleep.

Methods: Rats (n=6) were implanted with microdialysis probes in the trigeminal motor pool for application of candidate drugs across the sleep-wake cycle. GABA$_B$ receptors were antagonized on trigeminal motoneurons (via CGP52432) and the resulting effects on masseter EMG tone were determined.

Results: We found that GABA$_B$ receptor blockade at the trigeminal motor pool increased masseter tone during waking and NREM sleep, but had no effect on REM atonia. However, when GABA$_B$ receptors as well as both GABA$_A$ and glycine receptors were simultaneously blocked (via 0.2mM CGP52432, 0.1mM bicuculline, and 0.1mM strychnine), this not only increased masseter muscle tone during waking and NREM sleep ($p<0.001$ for both states), it also triggered a robust increase in basal muscle tone during REM sleep ($p=0.024$).

Discussion: We show for the first time that an endogenous GABA$_B$ drive inhibits motoneurons and suppresses masseter tone during both waking and sleep. While GABA$_B$-mediated inhibition itself does not trigger REM sleep atonia, blockade of GABA$_B$ as well as GABA$_A$ and glycine receptors is capable overriding REM atonia, indicating that GABA$_B$ receptors play a critical role in mediating this motor phenomenon.

P054

Noradrenergic Modulation Of Muscle Tone During Cataplexy In Hypocretin/ Orexin Knockout Mice

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Introduction: Cataplexy is a major symptom of narcolepsy and is characterized by the involuntary loss of postural muscle tone during waking. Reduced noradrenergic excitation of motoneurons may trigger cataplexy because cells in the locus ceoruleus cease firing during cataplectic attacks in narcoleptic dogs. Although noradrenergic reuptake inhibitors improve cataplexy, it is unknown if such effects are mediated by increasing noradrenaline levels within somatic motor pools. The aim of this study was to determine whether restoring noradrenergic drive to the trigeminal motor pool would reverse loss of masseter muscle tone during cataplexy in hypocretin knockout (KO) mice.

Methods: We used reverse microdialysis to apply phenylephrine (an 1-noradrenergic receptor agonist) onto trigeminal motoneurons during cataplectic episodes in freely-behaving hypocretin KO mice. We quantified levels of masseter muscle tone during cataplexy (and across the sleep-wake cycle) before and after noradrenergic excitation of trigeminal motoneurons. Cataplexy and sleep-wake state were determined using EEG, EMG (neck and left/right masseters) and videography.

Results: We quantified 14 episodes of cataplexy while applying either aCSF (n=7) or 1mM phenylephrine (n=7) at the trigeminal motor pool in 4 hypocretin KO mice. We showed that masseter muscle tone was potently suppressed below waking levels during individual cataplectic attacks (p=0.003). Importantly, we found that increasing noradrenergic drive by activating 1-noradrenergic receptors at the trigeminal motor pool significantly increased masseter tone during cataplexy (p=0.004); however, such activation did not restore masseter tone to waking levels (p=0.008).

Discussion: We conclude that reduced noradrenergic drive onto somatic motoneurons contributes, at least in part, to loss of muscle tone during cataplexy. However, as we could not increase muscle tone to waking levels changes in the release profiles of other neurotransmitters must also be involved in triggering loss of muscle tone during cataplexy.

P055

Noradrenergic Transmission Triggers Muscle Tone By Amplifying Glutamatergic Drive Onto Somatic Motoneurons

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Introduction: Skeletal muscle tone is markedly reduced during sleep, particularly in REM sleep. Noradrenergic cells project to somatic motoneurons and their discharge activity is correlated with state-dependent changes in muscle tone, thus implicating the noradrenergic system in driving the pattern of muscle activity across the sleep-wake cycle. It is unknown whether noradrenaline modulates muscle tone by directly altering motoneuron excitability or whether it functions to amplify excitatory synaptic transmission. Since noradrenaline has previously been demonstrated to act synergistically with glutamate to strengthen its synaptic activity, we hypothesize that noradrenaline regulates motoneuron excitability by indirectly potentiating glutamate-mediated excitation.

Methods: To test this hypothesis, we used reverse-microdialysis to apply an α1-adrenoceptor agonist (phenylephrine) and glutamate-receptor agonist (AMPA) and antagonist (CNQX) onto trigeminal motoneurons in both anaesthetized and freely-behaving rats. This allowed us to determine whether noradrenergic neurotransmission onto trigeminal motoneurons affects masseter muscle tone by increasing spontaneous motoneuron activity or whether it acts to amplify prevailing glutamate-driven excitation.

Results: Perfusion of 1 mM phenylephrine alone had no significant effect on masseter muscle tone in anaesthetized rats ($p=0.095$). Both exogenous and endogenous glutamate-driven excitation of muscle tone, however, were significantly amplified during simultaneous perfusion of phenylephrine ($p<0.05$ in both groups). Thus, noradrenaline's stimulatory effects were unmasked and rapidly switched-on only in the presence of glutamatergic transmission. Blockade of AMPA receptors with CNQX abolished this enhanced excitatory effect and returned muscle tone to baseline levels in both anaesthetized rats ($p<0.05$) and during active wake ($p=0.027$), quiet wake ($p=0.028$) and REM sleep ($p=0.003$) in freely-behaving rats, indicating that noradrenergic drive requires ongoing glutamatergic activity to trigger muscle tone.

Conclusions: Our data indicate that noradrenaline does not directly modulate masseter mucle tone, but instead acts to indirectly trigger muscle activity by amplifying prevailing glutamate-driven excitation of trigeminal motoneurons.

P056

Enhanced Cholinergic Activity at the Hypoglossal Motor Nucleus Suppresses Genioglossus Muscle Activity

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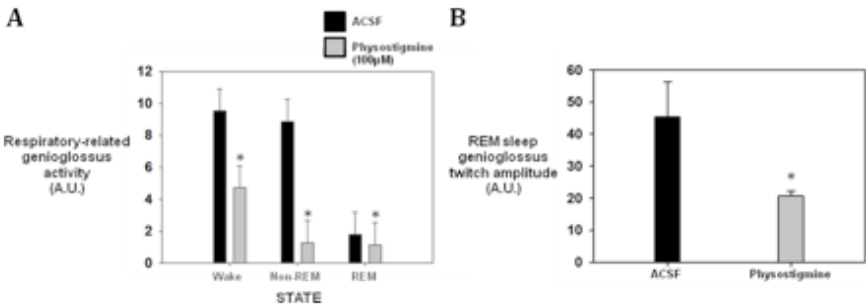1Departments of Medicine and Physiology, University of Toronto, Toronto, ON, Canada; 2 The Conway Institute of Biomolecular and Biomedical Research, University College Dublin, Dublin, Ireland

Introduction: Sleep-related breathing disorders have a failure to maintain adequate breathing during sleep as their root cause. For example, obstructive sleep apnea (OSA) is caused by suppression of pharyngeal muscle tone in sleep, especially rapid-eye-movement (REM) sleep, which leads to repeated cycles of upper airway obstruction, hypoventilation and arousals from sleep. Given that OSA occurs *only* during sleep it is important to determine the neural mechanisms underlying the impact of sleep on the activity of respiratory motoneurons that innervate the relevant pharyngeal muscles, such as the genioglossus muscle of the tongue. The genioglossus muscle is innervated by the hypoglossal motor nucleus (HMN) which is itself innervated by acetylcholine-containing neurons which are primarily active during REM sleep. Accordingly there is appropriate neural circuitry for alterations in acetylcholine release to influence genioglossus muscle activity. However, the effects of increasing endogenous acetylcholine at the HMN in the intact freely-behaving organism *in-vivo* have never been determined.

Methods: In ten freely behaving rats, endogenous acetylcholine concentration at the HMN was enhanced by local microdialysis perfusion of an acetycholinesterase inhibitor (physostigmine) while sleep-wake states, diaphragm and genioglossus muscle activities were recorded.

Results: Physostigmine significantly reduced respiratory-related genioglossus muscle activity (P=0.020; figure A). This decrease in genioglossus activity with physostigmine did not depend on the prevailing sleep-wake state (P=0.073), i.e., the suppression of genioglossus activity occurred in wakefulness, non-REM and REM sleep. Additionally the amplitude of phasic genioglossus muscle twitching during REM sleep was significantly reduced (P=0.047; figure B).

Discussion: These results show for the first time that endogenous acetylcholine at the hypoglossal motor nucleus suppresses genioglossus muscle activity in wakefulness and sleep. Since REM sleep is produced by increased activity of pontine acetylcholine-containing neurons that project to the HMN, this effect of increased acetylcholine may contribute to the major suppression of genioglossus muscle activity in REM sleep.

Support: CIHR

P057

Effect Of Ethanol On Medullary Respiratory Activity And Sleep

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Introduction: Ethanol exacerbates sleep-related breathing disorders in humans. This effect may be due to central depression of respiratory motor activity and/or delayed arousal from sleep but the distinction has not been formally tested.

Methods: Sleep-wake states, genioglossus and diaphragm muscle activities were recorded in freely-behaving rats (n=10) following intra-peritoneal injection of 1.25 g/kg ethanol or saline (control), administered 48 hours apart and in random order. In isoflurane-anesthetised rats (n=10), ethanol (0.025, 0.05, 0.1, 0.3, and 1 M) was microdialysed locally into the hypoglossal motor nucleus, and genioglossus and diaphragm muscle activities were measured.

Results: Ethanol injections resulted in maximum blood levels of 140 mg/dl, i.e., a physiologically relevant level for producing impairment of cognitive and motor behaviour in rats and humans. Ethanol decreased wakefulness (40.1 to 34.0 %, P=0.033), increased non-rapid eye movement (NREM) sleep (47.4 to 54 %, P=0.020), but had no effect on REM sleep (12.5 to 11.9 %, P=0.808). Ethanol also decreased respiratory-related genioglossus activity in wakefulness (P=0.02), but did not affect diaphragm amplitude or respiratory rate in any state. Application of ethanol directly to the hypoglossal motor nucleus in anesthetised rats did not suppress genioglossus muscle activity at any concentration.

Conclusion: Ethanol at physiologically relevant concentrations promoted sleep indicative of a sedating effect. The lack of effect on genioglossus activity when ethanol is applied directly to the hypoglossal motor nucleus suggests that the suppression observed during wakefulness with systemic administration was mediated via effects operating outside this motor pool.

P058

PKA-mediated modulation of respiratory-drive transmission to hypoglossal motoneurones in vivo

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Introduction: The genioglossus, innervated by the hypoglossal motor nucleus (HMN), is an important respiratory muscle that helps maintain the upper airway open for effective lung ventilation. *In-vitro* studies have shown that protein kinase A (PKA) modulates the transmission of the respiratory-drive signal at the level of the HMN. We first tested the hypothesis that local delivery of PKA activators into the HMN would increase respiratory-related genioglossus activity *in vivo*, and that application of a PKA inhibitor would suppress genioglossus activity, indicative of constitutive PKA activity. Additionally, since PKA is implicated in models of long-term augmentation of neuronal activity, we tested the hypothesis that prolonged stimulation of the HMN with PKA activators would result in the long-term facilitation of genioglossus activity.

Methods: Experiments were performed in 20 isoflurane-anaesthetised, tracheotomised, and spontaneously-breathing adult Wistar rats. Electromyographic electrodes were implanted into the diaphragm and genioglossus. Microdialysis perfusion of artificial cerebrospinal fluid (control) and of membrane-permeant PKA modulators into the HMN was used to determine the role of PKA in modulating genioglossus muscle activity *in vivo*.

Results: Application of 8-Br-cAMP (direct PKA activator) into the HMN significantly increased genioglossus muscle activity ($211 \pm 21\%$). Application of forskolin (indirect PKA activator) had a similar excitatory effect on genioglossus activity ($185 \pm 18\%$). Genioglossus activity steadily decreased back to pretreatment levels during a 90-minute washout period with artificial cerebrospinal fluid. Application of Rp-8-Cl-cAMPS (direct PKA inhibitor) into the HMN had no significant effects on genioglossus activity.

Discussion: These results demonstrate that PKA activation at the HMN increases respiratory-drive transmission *in vivo* and, therefore, genioglossus muscle activity. However, these results also show that PKA does not constitutively facilitate the transmission of the respiratory-drive signal to hypoglossal motoneurones *in vivo* and that prolonged PKA activation in the HMN does not induce long-lasting augmentations of genioglossus activity.

P059

Novel Mechanism Underlying Opioid-Induced Respiratory Depression: Suppression Of Motor Drive From The Medulla To Upper-Airway Muscles

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Introduction: Upper-airway obstruction and respiratory depression are serious clinical hazards associated with the administration of opioid analgesics. However, it has not been determined whether opioids directly suppress the activity of the central respiratory motoneurones that ultimately constitute the source of motor drive to the respiratory muscles. Furthermore, it has also recently been demonstrated that opioids induce acetylcholine release at the medullary hypoglossal motor nucleus, the source of motor outflow to the genioglossus, an important lingual respiratory muscle that helps maintain the upper airway open for effective breathing. Acetylcholine at the hypoglossal motor nucleus has an inhibitory effect on motoneuronal activity via muscarinic-receptor stimulation, but the physiological relevance of this opioid-induced acetylcholine release has not been established. We hypothesized that (1) local delivery of the μ-opioid-receptor agonist fentanyl into the hypoglossal motor nucleus will suppress genioglossal muscle activity in vivo, and that (2) a component of this suppression is mediated by acetylcholine.

Methods: Microdialysis probes were implanted into the hypoglossal motor nucleus of 37 isoflurane-anaesthetised, tracheotomised rats for the local delivery of (1) fentanyl (0, 1, 10, and 100 μM) and naloxone (μ-opioid-receptor antagonist, 100 μM), and (2) fentanyl (100 μM), either in the presence or absence of the muscarinic-receptor antagonist atropine (10 μM).

Results: Fentanyl at the hypoglossal motor nucleus caused a significant dose-dependent suppression of genioglossal muscle activity, which was subsequently reversed with naloxone. Atropine did not affect the decrease in genioglossal activity caused by fentanyl.

Discussion: These results show that the μ-opioid-receptor agonist fentanyl suppresses the activity of the central respiratory motoneurones that activate the genioglossus, and that any effect on acetylcholine release does not play a significant role in this suppression. This suppressant effect on central respiratory motoneuronal activity may explain a major component of the upper-airway obstruction and respiratory depression observed clinically with opioid analgesics.

P060

Opioid-Sensitive Site In The Medulla Underlying Suppression Of Breathing In The Sleeping Adult Rat

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Introduction: Respiratory rhythm is generated in vitro by the pre-Bötzinger Complex (PBC), a small structure of the ventrolateral medulla. In neonatal rodents in vitro, inhibition of PBC neurons by activation of μ-opioid receptors decreases respiratory rate, and in adult rats in vivo, destruction of PBC neurons produces ataxic breathing and apneas during sleep. To understand the mechanisms underlying inhibition of PBC neurons and sleep-disordered breathing in adult rats, we hypothesized that local activation of μ-opioid receptors at the PBC will depress respiratory rate and reduced respiratory muscle activity in vivo in anaesthetized and sleeping rats.

Methods: We recorded activities of the diaphragm muscle and the genioglossus muscle of the tongue - an important muscle involved in upper airway patency - in anaesthetized or freely-behaving adult rats with exogenous drugs applied to the PBC by microdialysis probes.

Results: In anaesthetized rats (n=7), application of the μ-opioid receptor agonist fentanyl (100 μM) to the PBC (panel A) decreased respiratory rate (p=0.004, panel B), without affecting diaphragm muscle amplitude (p=0.81, panel C), and suppressed genioglossus muscle activity (p=0.007, panel D), with this depression reversed by the antagonist naloxone (100 μM). Preliminary data in 2 freely behaving rats showed that fentanyl (150 μM) at the PBC also reduced respiratory rate in non-REM sleep (~30%) but not wakefulness (~3%).

Conclusion: Opioid-sensitive neurons in the PBC mediate the reduction of respiratory rate, as well as the suppression of genioglossus muscle activity in vivo, with responses to μ-opioid receptor agonists being prominent in non-REM sleep and anaesthesia. This is the first evidence that the PBC is involved in the generation of respiratory rate in vivo and that inhibition of PBC neurons leads to sleep-disordered breathing.
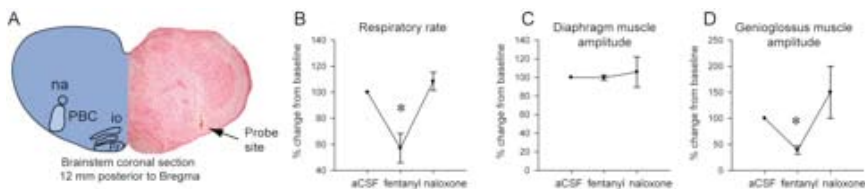
P061

Sleep Loss Reduces Apnea-Induced Respiratory Neuroplasticity

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Objectives: Sleep loss leads to deficits in neuroplasticity that underlie long-term potentiation (LTP) and important physiological functions such as learning and memory. Long-term facilitation (LTF) is a form of respiratory neuroplasticity that serves to enhance breathing and shares common features with LTP. However, the affect of sleep loss on respiratory neuroplasticity is unknown. We previously showed that repeated airway obstructions, as experienced in obstructive sleep apnea (OSA), trigger LTF of genioglossus muscle tone in rats. The goal of this study was to determine if short-term sleep loss affects apnea-induced respiratory LTF.

Methods: LTF of genioglossus EMG tone was measured in anesthetized, tracheostomized, spontaneously breathing adult male rats. Protocol-1 (n=12), control rats: Respiratory activity was recorded for 60 minutes before and after exposure to ten, 15-second apneas, each separated by one minute. Protocol-2 (n=6), sleep deprived rats: At the onset of the light phase, rats were sleep deprived for 6 hours by gentle handling. They were then anesthetized and the same protocol as the control rats was performed. LTF was quantified as an increase in genioglossus EMG from baseline for at least 60 minutes after repeated apneas.

Results: Repeated apneas triggered LTF of genioglossus EMG tone, increasing it by 61 ± 11% above baseline levels ($p<0.05$; 60-min after apneas). Similarly, repeated apneas also enhanced genioglossus EMG tone in sleep deprived rats (30 ± 9% increase at 60 minutes; $p < 0.05$), but to a significantly lesser degree (RM ANOVA; $p < 0.05$).

Conclusions: Apnea-induced LTF of genioglossus muscle tone could help maintain upper airway patency. However, sleep loss potently suppresses the beneficial effects of apnea-induced LTF of genioglossus activity. Therefore, lack of LTF in OSA patients could be caused by the sleep loss/fragmentation associated with apnea-induced arousals. Triggering LTF by pharmacological mechanisms could be a potentially useful strategy for improving airway patency in OSA patients.

P062

N-Rem Sleep Slow Oscillations Amplitude And Density In The Young And Middle-Aged Men And Women

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Introduction: High level of neural synchronisation in N-REM sleep is detectable with the EEG as large amplitude slow-waves (SO). Aging is associated with lower slow-wave activity (SWA; spectral power between 0.5-4.5 Hz). Compared to men, women show higher SWA. However, it is still unknown whether age/sex-related differences in SWA are associated to changes in SO density, SO amplitude or both. We used an automatic detector to assess age and sex differences in SO.

Methods: Eighty-seven healthy volunteers with no sleep disorders were separated in two groups: Young (22W, 26M; 23.3y ±2.4), and Middle-aged (21W, 18M; 51.9y ±4.6). SO on Fp1, F3, C,3 P3 and O1 were automatically detected during N-REM using published criteria (Massimini et al. 2004). ANOVAs were performed on SO density (nb/min) and amplitude.

Results: Compared to young subjects, middle-aged subjects showed lower SO density in all derivations but this effect was less prominent in FP1. Age-related decrease in SO density was more prominent at beginning of the night. For SO amplitude, middle-aged men showed lower SO amplitude than young men in all derivations but this effect was less prominent in O1. Middle-aged women showed lower SO amplitude than young women and this effect did not differ between derivations. Men showed lower SO amplitude than women in the frontal derivation only.

Conclusion: In conclusion, effects of aging and sex differed on SO amplitude and density. While age-related decrease in SO density was less prominent in anterior area, age-related decrease in SO amplitude was less pronounced in posterior area (O1) in middle-aged men. Age effects on SO density were more prominent early in the night while age effects on SO amplitude were constant across the night. Sex differences were only observed on SO amplitude and constant across the night. These results suggest different neuro-physiological mechanisms underlying age and sex effects on SO.

P063

Ethnic Differences In Sleep Architecture In Healthy, Normotensive Young Adults Are Asscociated With Nocturnal Heart Rate Variability

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Introduction: Recent preliminary work from our group1 has found ethnic disparities in sleep architecture, notably, healthy African-Americans demonstrated decreased slow-wave sleep relative to Caucasian-Americans. These disparities exist even in young, healthy sleepers, self-reported as healthy sleepers. A common hypothesis points to the increased global stress accompanying minority status. While subjective stress reports and hypothalamic-pituitary-adrenal axis measures have failed to explain the effect, the use of heart rate variability (HRV) analyses, known to have marked ethnic disparities, offers new insight into both differences in sleep architecture and how sleep may relate to greater cardiac health and outcome.

Methods: 59 good sleepers (Mean Age=26.25±6.11, African-American n=15, Asian-American n=14, Caucasian-American n=30), as previously reported, completed two laboratory polysomnographic (PSG) studies, including the electrocardiogram, used to derive the HRV outcomes of the Standard Deviation of Normal RR Intervals (SDNN) and the Low-Frequency/High-Frequency Ratio (LF/HF): a measure of overall autonomic sympathetic tone.

Results: As previously reported, no sleep continuity differences existed between the groups (p's>.05), however, there was a marked disparity in slow-wave sleep (SWS) (p=.002) between the groups when controlling for sex, age, BMI and blood pressure using linear mixed effects modeling (LMEMs). Subjecting HRV analyses to LMEMs revealed that: African-Americans showed a 22 ms decrease of SDNN, with an HR increase of 6.1 BPM, compared to Caucasian-Americans (p=.05, p=.02, respectively). Across all the nights SDNN was positively correlated with SWS (r=.243, p=.015), particularly in Stage 2 (r =.313, p=.001.)

Discussion: Sleep and ethnicity both have effects on cardiac health. Nocturnal Heart Variability is a new window into this relationship. While ethnic minority sleepers report healthy habits, a latent lightening of sleep coupled with increased nocturnal sympathetic drive may increase risk for later cardiac pathology. Ethnic disparities in sleep may serve as early biomarkers for known cardiac disparities.

References: 1. Saletin JM, Kronfli TR, Peterson SC, Smith MT. Ethnic differences in sleep architecture and continuity in healthy self-reported good sleepers. Sleep 2008; Abstract Supplement.

8. Respiratory - and Movement - related Sleep disorder

P064

Sleep-Related Breathing Problems Affect Blood Pressure Regulation In Children

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Introduction: Sleep-related breathing problems (SRBP) and short sleep duration are linked with obesity among adults and children. In adults, SRBP and sleep duration are associated with increased sympathetic nervous system activity and high blood pressure (BP); this change occurs via decreased vagal baroreflex gain (BRG), a measure of BP regulation. It is unknown if SRBP and sleep duration influences BP regulation in children. Moreover, it is unclear if the effect of SRBP on BRG is a consequence of excessive body mass. Examine the relationship between SRBP and short sleep duration and BP regulation among children.

Methods: Analyses included 225 grade 6-8 children who were randomly selected to undergo BRG assessment in a laboratory-based protocol from an original sample of 1,285 children whose BP and body mass were measured at school. BRG was measured using 5 minutes of continuous beat-to-beat BP (Finapres) and RR interval (RRI) recordings (standard ECG). High frequency (HF) and low frequency (LF)-BRG were calculated. Parent-reported child sleep duration on weekdays and SRBP was measured using the Pediatric Sleep Questionnaire. Generalized regression analyses were conducted with 198 children with complete data.

Results: In correlations, HF-BRG (r = -0.19; p=0.006) and LF-BRG (r = -0.18; p=0.011) were both negatively related to SRBP. BRG and sleep duration were not significantly related. In regression analyses, increases in SRBP were related to lower HF- and LF–BRG (p<.001), after adjusting for sleep duration and body mass index. That is, breathing problems were independent of body mass. Further, higher body mass was independently associated with lower HF- and LF-BRG (p<.001) adjusting for SRBP.

Conclusion: Reductions in autonomic activity in children are due, in part, to SRBP, independent of body mass. Despite their young age, baroreceptor function is already altered demonstrating a reduction in BP regulation.

P065

The Impact Of Slow Wave Sleep (Sws) And Sleep Fragmentation On Fatigue Verus Daytime Sleepiness In Patients With Obstructive Sleep Apnea (Osa)

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Introduction: Fatigue and daytime sleepiness are common complaints in OSA patients. Although fatigue can be a result of many pathophysiological conditions, it has been documented that more patients report fatigue, as compared to sleepiness, in this population. The aim of this study was to determine if fatigue changes after CPAP treatment and whether or not changes in fatigue (as opposed to daytime sleepiness) can be correlated with amount of SWS and degree of sleep fragmentation. This finding could suggest management strategies of fatigue in sleep clinic patients.

Methods: This was a retrospective chart review of 60 (out of 130 charts) sleep clinic patients who were diagnosed with OSA and underwent CPAP treatment. Patients' Fatigue Scale scores, Stanford Sleepiness Scale scores, percentages of SWS, Arousal Indices (AI) and Apnea Hypapnea Indices (AHI) were extracted from the charts before and after CPAP treatment.

Results: Of the 60 patients, 40 were males (age=56 ± 14) and 20 were females (age=57 ± 12). Statistical analysis was performed for males and females separately as well as males and females together. After CPAP treatment, there was a significant ($P<0.05$) decrease in fatigue, daytime sleepiness and arousal index (sleep fragmentation). SWS percentage, however, did not increase significantly after CPAP treatment. The decrease in fatigue was moderately correlated with the decrease in arousal index only in males ($P<0.05$, r=0.4, r²=0.16). No significant correlations were observed in females and when the males and females were mixed. Daytime sleepiness did not have any significant correlation with neither SWS nor sleep fragmentation before and after treatment in any of the populations.

Conclusion: Fatigue seems to be moderately associated with sleep fragmentation rather than SWS in OSA patients and this association seems to be specific to male patients.

P066

Frequency Of Obstructive Sleep Apnea In Paediatric Patients With Adenotonsillar Hypertrophy

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Introduction: Adenotonsillar hypertrophy is one of the main causes of Obstructive Sleep Apnea (OSA) in children. The consequences of OSA are particularly severe in children, including failure to thrive, enuresis, attention deficit/ behavioural disorders, and poor academic performance. The aim of this study is to explore to the relationship between large tonsils and OSA in children.

Methods: A retrospective study of 45 children (33 with enlarged tonsils and 12 controls) from the Youthdale Child and Adolescent Sleep Centre was conducted. Tonsil size was rated on a 5-point scale ranging from 0 (tonsillectomy or no enlargement) to 4+ ($\geq$75% airway blocked). The children underwent overnight polysomnography and questionnaire assessment of sleepiness and fatigue.

Results: Children with enlarged tonsils had significantly higher total AHIs (3.3±5.5 vs. 0.3±0.3, p=0.004) and AHIs in REM sleep (7.7±17.5 vs. 1.0±1.0, p=0.04) when compared to controls. For those with enlarged tonsils, there was no correlation between tonsil size and AHI. No significant differences were found in daytime sleepiness (2.8±1.6 vs. 3.0±0.6, p=0.43) or fatigue (3.0±1.8 vs. 3.2±1.2, p=0.64) between children with enlarged tonsils and controls. Average PO_2 saturation did not differ between study groups (98.0±0.8 vs. 97.9±0.5, p=0.511), but there was a trend for lower minimum PO_2 saturation in children with enlarged tonsils (79.3±13.5 vs. 86.9±11.6, p=0.077). Lastly, for children with enlarged tonsils, the incidence of OSA was over forty times greater than for children with normal sized tonsils (OR=40.9, 95%CI: 4.5-372.7).

Conclusion: Adenotonsillar hypertrophy in paediatric patients immensely increases their likelihood of having OSA. These children should be sent for overnight sleep assessment as the degree of tonsillar enlargement did not predict the severity of OSA in those with enlarged tonsils. Further, these children do not exhibit typical daytime symptoms of fatigue or sleepiness as a consequence of disturbed sleep, so their OSA may be masked.

P067

Obstructive Sleep Apnea Is Highly Prevalent Among Kidney Transplanted Patients: Results Of The Slept Study

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Introduction: The prevalence of obstructive sleep apnea (OSA) is much higher in patients on chronic dialysis than in the general population. Here we used a large, randomly selected sample of kidney transplanted patients to assess for the first time the prevalence of OSA and its clinical correlates.

Methods: Data from 100 kidney transplanted and 50 waitlisted patients obtained in a cross-sectional survey (SLeep disorders Evaluation in patients after kidney Transplantation (SLEPT) Study) were analyzed. Socio-demographic data, history of renal disease, medication, co-morbidity and laboratory parameters were collected at enrolment. Patients underwent one-night full polysomnography. Definition of moderate and severe OSA was an apnea-hypopnea index (AHI) higher than 15/hour.

Results: The prevalence of mild ($5/h \leq AHI < 15/h$), moderate ($15/h \leq AHI < 30/h$) and severe OSA ($AHI \geq 30/h$) in the Tx group was 18%, 11% and 14% versus 28%, 16% and 10%, respectively, among waitlisted patients. The apnea-hypopnea index was significantly correlated with age (rho=0.34), body mass index (rho=0.45), neck- (rho=0.4) and abdominal circumference (rho=0.51) and hemoglobin (rho=0.24) in the transplanted group. The proportion of males was significantly higher among OSA patients versus those without OSA (80% vs 49%; p<0.01). The average systolic blood pressure was higher in OSA vs non OSA patients (147±21 mmHg vs 139±18 mmHg; p=0.059). A significantly higher proportion of patients used three or more antihypertensive drugs in the OSA group versus the non-OSA group (56% vs 31%; p<0.05). In multivariate logistic regression analyses only abdominal circumference independently associated with OSA. The ten-year Framingham coronary heart disease risk and risk for stroke was twice as high in OSA versus non OSA patients.

Discussion: Obstructive sleep apnea is highly prevalent in the kidney transplanted population. The prevalence of OSA is similar in transplanted and waitlisted patients. OSA may contribute to increased cardio-cerebro-vascular risk in transplanted patients.

P068

Periodic Limb Movements In Sleep And Cardiovascular Risk In Kidney Transplanted Patients

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Introduction: Periodic Limb Movements in Sleep (PLMS) is more prevalent in chronically dialyzed patients compared to the general population. Recent reports suggested an association between PLMS and mortality both in the general population and in patients with chronic kidney disease. Here we assess the prevalence and clinical correlates of PLMS in kidney transplanted (Tx) patients. Finally, we wanted to test if PLMS is associated with cardiovascular risk in transplanted population.

Methods: Data from 100 Tx and 50 waitlisted patients (WL) obtained in a cross-sectional survey (SLeep disorders Evaluation in patients after kidney Transplantation Study) were analyzed. Socio-demographic data, history of renal disease, medication, co-morbidity and laboratory parameters were collected at enrolment. Patients underwent one-night polysomnography.

Results: The proportion of males was 57% vs 54% and mean age was 51±13 vs 50±13 years in the Tx vs the WL groups, respectively (p=NS for both). The prevalence of diabetes was 19% vs 16% in the Tx vs the WL group (p=NS). Median (Interquartile range IQR) PLMS index (periodic leg movements/hour) was similar in the Tx and the WL groups: 6.20 [15.44] vs 6.19 [45.15], (p=NS). Interestingly, the prevalence of PLMS (defined as PLMS-index > 5/hour) was 52% in both groups. The prevalence of severe PLMS (PLMS-index > 25/hour) was 16% vs 32% in the Tx vs the WL group, respectively (p=0,024). In the Tx group PLMS was more frequent in males vs females (65% vs 35%, p=0.03) and also in diabetics vs non-diabetics. Finally, PLMS-index was significantly correlated with the 10-year Framingham coronary-risk (rho=0,255; p=0,013) and also with the 10-year stroke-risk (rho=0,274; p=0,006).

Discussion: PLMS is very frequent both in WL and in Tx patients. Severe PLMS, however, is less prevalent in the Tx group. Importantly, PLMS was associated with increased cardiovascular risk in the Tx group.

P069

Evaluation Of A Portable Monitor Compared With Polysomnography For The Diagnosis Of Obstructive Sleep Apnea

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Introduction: Validated portable monitors (PM) are a viable tool to assist in the diagnosis of obstructive sleep apnea (OSA) in the estimated 1 in 15 middle-aged adults with OSA of moderate or worse severity. We assessed the utility of a Level 3 PM for OSA, Medibyte® (Braebon® Medical Corporation) by pairing it with attended overnight polysomnography (PSG) in the sleep laboratory (Level 1).

Methods: A series of patients, not all of whom were suspected to have OSA, wore the PM with PSG. Hypopneas were scored based on a 50% reduction or more in airflow on the nasal cannula pressure transducer signals from baseline, or a reduction in oxygen saturation of ≥ 3% on the PM record, and on PSG when associated with arousals. The number of apneas and hypopneas for the PM were calculated per hour of recording time - called the respiratory disturbance index (RDI), and for the PSG per hour of sleep time to provide the apnea-hypopnea index (AHI).

Results: For 53 patients (20M/33F) aged 20 to 73 years (mean ± SD: 52 ± 12) and BMI 33.4 ± 7.3 kg/m2 (range 21.4 - 52.7), the AHI was 28.7 ± 28.3 while the RDI was 21.5 ± 20.1. There was good correlation between the RDI and AHI (Pearson correlation r = 0.93) which accounted for 87% of the variance (R2 = 0.872). The mean difference AHI - RDI (PSG versus PM) showed under-reporting using the PM by 7.2 ± 11.9 events per hour. For an AHI ≥10 the sensitivity (true-positive), as well as the specificity (true-negative), of the PM were 92% . For severe OSA (AHI ≥ 30), the PM sensitivity was 82% and specificity 100%. All 17 cases of severe OSA had an RDI > 15 on the PM.
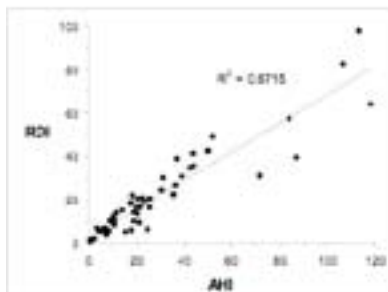
Conclusion: Tested in the laboratory, the PM was highly sensitive and specific in evaluating moderate to severe OSA.

P070

Analysis Of Hospital Discharge Data To Determine The Use Of Continuous Positive Airway Pressure In Canadian Hospitalized Patients With Sleep Apnea

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Introduction: Diagnosed obstructive sleep apnea affects 2-4% of middle-aged Americans and represents a substantial healthcare burden. Despite its prevalence little is known about the demographic characteristics or clinical management of hospitalized sleep apnea patients in Canada. The objectives of this study were to: (1) Describe the prevalence of hospitalized, sleep apnea individuals according to age, gender, and co-morbidities. (2) Estimate the prevalence of the use of continuous positive airway pressure (CPAP) therapy during hospitalization in the individual provinces and territories.

Methods: A retrospective analysis of data of hospitalized patients with obstructive sleep apnea using the Canadian Institute for Health Information's (CIHI) 2007 Discharge Abstract Database was completed. Cases of obstructive sleep apnea were obtained from discharge records coded according to the International Classification of Diseases, Tenth Revision, Canadian version (ICD-10-CA) and were identified using the ICD-10-CA code G47.30. A subset of these patients, those receiving CPAP therapy, was further identified using the Canadian Classification of Health Interventions code 1.GZ.31.CB-ND.

Results: Review of the discharge data identified 8,823 estimated cases of obstructive sleep apnea. Approximately 52.4% of these individuals were 40-69 years old with a gender distribution for all ages of 66.5 % males. The most common diagnoses in hospitalized sleep apnea patients included hypertension, congestive heart failure, type II diabetes mellitus and obesity. Sleep apnea was managed using CPAP therapy in 4.3 % of patients and use was most often reported in the provinces of Prince Edward Island (8.8% of 34), Manitoba (7.1% of 382), and Ontario (5.4% of 3,704).

Conclusions: In conclusion, only a small percentage (4.3%) of patients coded with obstructive sleep apnea in the 2007 Discharge Abstract Database were abstracted as being provided with CPAP therapy, and there appear to be provincial and territorial differences in the reporting of CPAP utilization. These findings suggest the possibility that management of obstructive sleep apnea in hospitalized patients in Canada is deficient. It is possible deficiencies in the coding of OSA and CPAP therapy exist.

P071

The Mouth Leak Syndrome In Patients Initiating Ncpap For Osas

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Introduction. We have observed that patients with a poor response to nasal continuous positive airway pressure (nCPAP) for obstructive sleep apnea syndrome (OSAS) have developed a mouth leak syndrome (MLS) of air rushing out the mouth, nasal congestion and premature removal of the nCPAP during the night. We propose that this is a reaction to nCPAP mouth leak. We sought to test this hypothesis with custom polysomnography for continuous monitoring of mouth leak in patients treated with nCPAP.

Methods. Consecutive new patients with obstructive sleep apnea (n = 40; age 52.5, SD 12.0) were studied with validated prospective questionnaires and a download every week of fixed pressure nCPAP therapy (mean pressure 9.1 SD 2.0; REMStar, Respironics, USA) which was adjusted to extinguish any residual sleep apnea. After 4 weeks, patients were monitored with custom polysomnography to quantify the time spent in mouth leaks and any associated interruptions in sleep continuity associated with mouth leaks. Correlation was made with symptoms, quality of life measures with the Sleep Apnea Quality of Life Index (SAQLI) as well as the Quebec Sleep Questionnaire (QSQ), satisfaction, & compliance.

Results. Of 40 patients, 16 met pre-defined criteria for the MLS. At baseline, patients who later developed MLS had similar total scores on the SAQLI (MLS mean 57 SD 14% vs 65 SD 17%; p = 0.19) and the QSQ (MLS mean 57 SD 21% vs 62 SD 19%; p = 0.41). With nCPAP, their scores improved to levels inferior to those who did not develop MLS on SAQLI (MLS mean 66 SD 15% vs 83 SD 9%; p = 0.0040) and the QSQ (MLS mean 71 SD 20% vs 82 SD 13%; p = 0.045). These patients demonstrated less satisfaction and compliance with nCPAP. Polysomnography demonstrated no residual sleep apnea yet more mouth leak with respect to time. Mouth leaks were also more often terminated with sleep disruption.

Polysomnographic Findings

Characteristics	No Syndrome	Mouth Leak Syndrome
Apnea-hypopnea index (/hr)	4.9 (5.0)	4.0 (3.4)
Desaturation index (/hr)	3.2 (4.8)	3.1 (3.2)
Mouth leak event index (/hr)	31.9 (11.1)	46.0 (19.0)**
Mouth leak time (% sleep time)	32 (18)	55 (17)**
Leaks terminating in arousals (% sleep time)	14 (11)	30 (22)**

Means and standard deviations are shown. ** $p<.01$ * $p<.05$

Conclusions: Patients with OSAS treated with nCPAP who develop mouth leak syndrome demonstrate less clinical improvement in OSAS measures as well as more mouth leaks and more sleep disruption due to these mouth leaks. Support: OSR Medical & the Mount Sinai Hospital Research Foundation

9. Technology & Proceedures

P072

What Is The Level Of Sleep Knowledge In Family Medicine Residents?

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Introduction: Sleep education is lacking in medical school and residency training programs. Medical trainees are commonly exposed to sleep deprivation and disturbance with consequences of degradation of human performance and safety risk. A study looking at sleep education of residents as a stand alone intervention with the *Sleep, Alertness, and Fatigue Education in Residency (SAFER) Program* did not improve sleep outcomes (*Arora 2007*). The purpose of this study is to evaluate sleep knowledge in a sample of Family Medicine Residents.

Methods: Family Medicine Residents (N=56) were surveyed using the 15-item Sleep Knowledge questionnaire from the SAFER program which assesses knowledge of basic concepts of sleep and circadian biology. Fifty-five percent (21/38) were first year residents; average age was 29 years (range of 25-40), and majority were female (77%). Participants indicated True, False or Don't Know for each item. The authors reviewed and categorized the items into four domains to better differentiate the scope of sleep knowledge. The four domains are 1) Sleep Deprivation (*items 1,3,4,7,8,9,14)* 2) Sleep Hygiene (*items 2,3,6,10,13)* 3) Counter Measures (*items 2,5,10,11,13)* 4) Circadian Biology (*items 11,12,15)*. Items relevant to more than one domain were counted within each appropriate domain. The number (percent correct) of correct responses for each item were tabulated.

Results: Table 1. Represents the group response rates of all items in each domain.

Table 1 Sleep Knowledge Domains

Domain	Correct (%)	Incorrect (%)	Don't Know (%)
1. Sleep Deprivation	68	26	6
2. Sleep Hygiene	67	18	15
3. Counter Measures	59	25	16
4. Circadian Biology	43	32	25

The group average for the total number of items correct was 9/15 (61%), range 5/15 (33%) to 13/15 (86.67%).

Conclusion: The results suggest that in this group of residents, basic sleep knowledge is limited. It has been proposed that sleep knowledge may lead to better sleep outcomes. Further research looking at sleep knowledge, attitudes and behaviors of medical trainees is proposed to identify sleep education learning objectives to help create an efficacious sleep education intervention.

Reference: Arora V.M, Georgitis E, Woodruff J.N et al. Improving sleep hygiene of medical interns. *Arch Intern Med* 2007; 167(16):1738-1744.

P073

What Makes Some Patients Want To Investigate Their Sleep?

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Introduction: Sleep problems and daytime fatigue are commonly experienced but rarely reported by family practice patients to their physicians. But what happens when patients are spontaneously offered the possibility of investigating their sleep? To answer this question we examined symptom severity and health conditions in two patient groups: (1) those who voluntarily underwent one overnight polysomnographic (PSG) testing at a sleep laboratory (Completers Group) and (2) those who declined the offer to go to the sleep laboratory for testing (Decliners Group).

Methods: The Completer sample comprised 62 family practice patients and the Decliners sample comprised 66 family practice patients. All completed the Sleep Study Checklist (SSC) in family practice waiting areas. The SSC includes 19 self reported symptoms of sleep disorder, insomnia, and daytime functioning. In addition, at the end of the patient's visit with his/her physician, the physician completed a brief checklist identifying the patient's recent history of cardiovascular disease, hypertension, hyperlipidemia, diabetes and obesity. After the SSC and the physician checklist were completed participants were offered a sleep evaluation, including questionnaires, medical assessment, and PSG testing in a sleep lab.

Results: We found that (1) the Completers had significantly higher scores on all SSC subscales: Sleep Disorder, Insomnia, and (impaired) Daytime Functioning compared to those of the Decliners; (2) participants' willingness to complete a night of PSG testing was related to the presence of hypertension.

Conclusion: Sleep-related symptoms appear to be related to specific health conditions. Identification of this profile permits a better understanding of sleep disturbances and daytime complaints in primary care patients. This information can be used to alert physicians to ask patients about their sleep and to refer them for further PSG testing, where indicated.

P074

Comparison Of Synchronized Level 1 And Level 3 Sleep Studies

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Introduction: A breath by breath comparison of Level 1 and Level 3 studies allows insight to potential errors in the interpretation of sensitivities and specificities and Altman Bland comparison.

Method: Synchronized full and partial in-lab polysomnography with a split flow catheter was done using the Harmonie (Stellate) and Stardust (Respironics) units. Scoring of 18 full, 13 split, and 3 partial-split studies was done according to AASM standards.

Results: Distinction between normal and abnormal using a Level 3 cut-off point of RDI =5/hr gives a sensitivity=1.0, and specificity 0.71.   For severity subgroups <5/hr, >5-15/hr, >15-30/hr, and >30/hr, the sensitivities and specificities are 0.7 & 1.0, 0.86 & 0.82, 0.57 & 0.88, and 0.75 & 0.97. Altman Bland comparison for RDI(total) yields a normally distributed cluster around a bias line(L1-L3) of -1.38 with 95% limits of agreement -18.9 to16.1 (SE=1.3, p=.29).   For obstructive, central and mixed apneas, and hypopneas the clusters are skewed with bias and 95% limits of agreement are respectively: 4.64, -11.73 to 21; 0.83, -6.5 to 8.14; -0.12, -9.4 to 9.1, and -6.92, -28.1 to 14.27. Qualitative breath by breath analysis of individual events indicates a large variation in the contribution to Level 3 RDIs due to event duration differences, exclusion of hypopneas with arousals, poor to moderate concordance of events (as exemplified by scoring of events during wake) and use of TIB vs. TST. Level 3 hypopneas are underscored and apneas overscored.

Conclusions: RDIs from corresponding studies may be artificially close or dissimilar due to cancelling and compounding factors. Significant proportions of events scored may be from different populations. Use of uncorrected RDIs for comparison of studies may lead to artificially high sensitivities and specificities and may still contribute to large variations in the degree of agreement between between the two methods of measurement.

Support: This study was supported by Medigas and the Atlantic Health Sciences Research Fund.

P075

A Retrospective, Observational Study Showing Patients With A Normal Level Iii Sleep Study And Normal Osa Pretest Probability Factors May Still Require Additional Investigations

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Introduction: Level III portable monitoring is the standard of care for screening and diagnosing sleep disordered breathing (SDB). Level III testing has shown to have limited sensitivity and specificity in patients with mild SDB and Upper Airway Resistance Syndrome (UARS) with clinical symptoms (daytime sleepiness and non-restorative sleep). The study purpose is to do a retrospective analysis of patients seen in a community sleep centre. Patients were clinically evaluated by a sleep physician, referred for level III testing and sent for level I testing.

Methods: A retrospective review of clinical charts (N=113) was performed. Inclusion criteria: all patients who had both level III and level I testing and history of snoring. Patients were excluded based on the following: Body Mass Index (BMI) $\geq$ 30, Adjusted Neck Circumference ANC $\geq$ 42, Level III Respiratory Disturbance Index (RDI) $\geq$ 15, Epworth Sleepiness Scale (ESS) < 10.

Preliminary Results: Analysis was performed on 9/113 patients (8%) who met inclusion criteria. There were 3 female and 6 male patients. The average age was 40.3 years. The average BMI was 25.4, average ANC was 38.2, mean RDI was 6.5 (level III) and mean RDI (Level I) was 20.3. Five patients (1%) ended up on Continuous Positive Airway Pressure (CPAP) Therapy. Of those, 2 patients had severe OSA (RDI > 30), 1 moderate (RDI 15-30) and 1 mild (RDI 5–15). One was given a trial of Auto Positive Airway Pressure (APAP). Remaining 4 (3.5%) patients received on-going care.

Table 1

ID #	RDI(III)	RDI(1)	Snoring History	Outcome
1	7.0	10.1	Yes	CPAP therapy
2	2.8	0.8	Yes	On-going care
3	15.0	47.3	Yes	CPAP therapy
4	1.0	0.4	Yes	On-going care
5	4.0	3.6	Yes	On-going care
6	14.9	84.8	Yes	CPAP therapy
7	8.0	6.5	Yes	Treatment
8	3.0	27.0	Yes	CPAP therapy
9	3.1	0.2	Yes	APAP therapy

Conclusions: The results were limited due to a small sample size. However, the results did show that 5/113 (5%) of patients having only a level III study would not have been identified and treated for SDB. This would have had an impact on the patients' quality of life. More work is needed in this area. Further chart review is underway.

P076

Specialized New Programming To Facilitate Research Oriented Analysis Of Polysomographic Studies

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Introduction: Commercial Sleep analysis software is designed for the needs of routine clinical practice. They often do not allow for specific analysis that may be required for research protocols. As part of a research study on sleep in preeclamptic women we wished to assess the temporal relationships between obstructive respiratory events and oxygen saturation, with fetal heart rate, maternal pulse and maternal blood pressure parameters. We wrote software which analyzes the raw study data parameters to output these special variables.

Methods: The program, termed "SANDIGGER" (manipulates the studies from the Sandman© Sleep system) allowed for an almost instantaneous access to any portion of the overnight study. Two specialized databases of 1): text and binary output files and 2): the "scored" events list, permitted the computerized processing of all raw data from the overnight sleep study. The research variables were automatically generated, and output directly to a standard Excel© spreadsheet. No manual entry to the spreadsheet was required.

Results: After rigorous testing to confirm the workings of the program, we estimated a timesaving of about 200 man-hours and eliminated tedious manual extraction of research variables. The program also removes human error from the process.

Discussion: The program works extremely well for the intended purpose. It could be easily altered to suit any research study involving the manipulation of specialized re-search parameters not within the scope of standard sleep variables. The program is Windows© XP based and runs on any standard PC which has the Microsoft Excel© application installed. Other capabilities, such as graphing could be added.

P077

Artificial Neural Network Scoring Of Human Sleep-Wake Stages Combining Short-Epoch Feature Extraction And Post-Processing Inference Rules

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The use of learning machines in the automatic analysis of human sleep-wake stages has shown performance near to inter-expert agreement. However, automatic classifiers are sensitive to small differences in the signal conditioning inherent to the existence of various polysomnographic collection systems and digital file formats. In addition, automatic analysis yields some incoherent results and doesn't always provide the requested flexibility in time resolution, which may vary between countries and species.

Using candidate features selected for their relative independence to biosignal collection parameters, we have developed a new method allowing 1) to train artificial neural network (ANN) from a database of short duration prototypic sleep/wake stage epochs and 2) to infer final scores at a variable duration using a set of implemented expert rules. The PRANA biosignal processing software was used to compute and extract a set of 16 different features from the electroencephalographic and electromyographic signals collected during 48 night recordings performed in 11 healthy adults using ambulatory recorders. Two independent human experts scored sleep/wake stages into 20-s epochs according to the conventional criteria. A database of 1 029 125 2-s epochs including the expert scores and the computer-extracted features was created. Five hundred and six 2-s epochs representative of each sleep/wake stage were manually selected by one expert from a subset of 7 individuals and further used for ANN learning.

Simulation of the automatic scoring system using 20-s epochs showed a 30±10% error rate as compared to consensual expert scores and a Cohen's kappa of 0.56±0.16. The global performance of the sleep-wake stage classification system ranges slightly below inter-scorer agreement (82.8±3.3%).

This new classification method can perform automatic sleep/wake staging with various epoch durations. Improved performance can reasonably be expected by selecting a larger amount of learning epochs and by introducing additional rules mimicking expert decision-making strategies.

Keywords: Human, Sleep, automatic analysis

P078

The Influence Of Sleep Quality On Mental And Physical Health In The Canadian Community Health Survey

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Introduction: The purpose of the current study was to examine the influence of sleep quality and quantity on the perceived mental and physical health of Canadians.

Methods: Data from 36984 Canadian residents (20211 women, 16773 men) who completed the Canadian Community Health Survey (2001) was analyzed using multiple regression analyses.

Results: Descriptive analyses highlighted that many Canadians have poor sleep quality and may not be getting enough sleep. Fifty percent and 60% of Canadians reported symptoms of insomnia and excessive sleepiness, respectively. Seventy-five percent reported sleep that is not refreshing, occasionally. Multiple regression analysis demonstrated the linear combination of sleep quality variables (insomnia, sleepiness, and refreshing sleep) were significantly related to perceived mental and physical health ($p<.001$). The sleep quality variables were each significant individual predictors ($p<.001$) and cumulatively accounted for 12% and 13% respectively, of the variance in the perceived mental and physical health of Canadians. Insomnia, sleepiness, and un-refreshing sleep predicted perceived mental health even after controlling of the effects of gender and the presence of a mood or anxiety disorders ($p<.001$). The sleep quality variables predicted perceived physical health even after controlling for the effects of age, body mass and chronic health conditions ($p<.001$). Although sleep quantity (hours of sleep per night) was a significant individual predictor, it did not contribute additionally to the models of perceived mental and physical health above beyond the variance accounted for by the sleep quality measures.

Discussion: Findings indicate that sleep quality plays an influential role in the perceived mental and physical health of Canadians. Preventative health-care measures should include education about improving sleep quality as an important component of the overall health of Canadians.

P079

The Discriminant Validity Of The Sleep Problems Inventory

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Introduction: The Sleep Problems Inventory (SPI) is a multidimensional self-report measure for the assessment of sleep problems in adults. In previous research, the SPI has demonstrated good test-retest reliability and a replicable 4-factor structure in large samples of undergraduate students, community-based adults, and sleep disorder patients. The present study examined the predictive validity of the SPI.

Methods: Adult patients (N=211) from a sleep disorder clinic in central Ontario completed the SPI prior to their PSG assessment. Fifty-three percent (N=111) of these patients (66 men, 45 women) were diagnosed with a breathing-related sleep disorder. The second sample consisted of 240 community-based adults (104 men, 135 women) from central Ontario.

Results: To explicitly examine the ability of the SPI to predict individuals diagnosed with breathing-related disorders and community samples, a discriminant function analysis (DFA) was conducted with the 4 SPI subscales (insomnia, sleepiness, nightmares and movement) to predict group membership (community, N=240 vs. breathing-related disorder, N=111). Collectively, the four subscales could significantly discriminate between groups, Wilks' Lambda= .94, F (4, 346) = 5.97, p < .0001]. The overall correct classification rate was 70.08%. The addition of the two items specifically designed to assess breathing related sleep disorders improved the overall classification to 80.91%. The DFA was then repeated at the item level and collectively, the 28 SPI items could significantly discriminate between groups, Wilks' Lambda= .55, F (28, 322) = 3.41, p < .0001. The overall correct classification rate was 76.07%. The addition of the two items specifically designed to assess breathing related sleep disorders improved the overall classification to 83.48%.

Discussion: Findings provide preliminary validation for the SPI by demonstrating the new measure can satisfactorily discriminate between patients with a confirmed breathing-related sleep disorder and individuals from a community-based sample.

P080

There May Be No First Night Effect For Ambulatory Polysomnography In Older Adults

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Introduction: The first-night effect (FNE), characterized by poor quality sleep on the first of multiple sleep studies, is a well-documented hallmark of in-laboratory polysomnography (PSG). However, the strength and presence of the FNE during ambulatory, in-home PSGs remains inconclusive. It is predicted that ambulatory PSG requires less adaptation and, therefore, the FNE may be reduced. Some research indicates a decreased FNE in healthy children or young adults recorded with in-home ambulatory PSGs. However, the presence of the FNE in older adults is less certain. The study presented here aims to determine the strength and presence of the FNE in older adults, including those with difficulty sleeping due to insomnia and/or chronic pain.

Methods: The sleep of 11 older adults (8 females; mean age=59.8±11.4), was studied using two in-home ambulatory PSG studies. Participants were screened for good general health and consist of a broad sample of older adults with and without insomnia and/or osteoarthritis. PSG studies were acquired and scored according to AASM 2007 criteria.

Results: Paired t-tests revealed no statistically significant differences in sleep quality or architecture variables between the first and second ambulatory PSGs (p-values > .05). There were no differences in the duration of wake ($t(10)=1.01$, $p=.34$), stage 1 ($t(10)=.81$, $p=.44$), or rapid eye movement (REM) ($t(10)=1.03$, $p=.33$) sleep between the two nights. Additionally, there were no differences in latencies to slow wave sleep (SWS) ($t(9)=.81$, $p=.44$) or to REM ($t(10)=.47$, $p=.65$) between the PSGs.

Discussion: These data corroborate that the FNE seen during in-laboratory studies is reduced, if not absent, by using ambulatory PSG. This study suggests that the FNE may be minimal, if present, for older adults recorded by ambulatory PSG. Ambulatory, in-home PSG may provide the advantage of being more reliable than in-laboratory PSG across repeated sleep studies.

P081

Systematic Optimization Of Automated Sleep Spindle Detection

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Introduction: The sleep spindle has been implicated in memory consolidation[1] and is thought to serve as a physiological index of intelligence[2]. To identify spindles, most studies utilize manual or automatic spindle detection. Manual scoring methods are labour intensive and may be less reliable due to subjectivity or the expertise of the scorer. To be reliable, automatic methods should have adequate sensitivity (true positive/true positive + false negative) and specificity (true negative/true negative + false positive). Only a few methods for automatic spindle detection are commercially available and validation data are limited[3]. This warrants the need for validation of automatic spindle detection methods. The goal of the current investigation was to develop a systematic method to optimize the use of automated spindle detection.

Methods: Ten polysomnograms (adults: 19-22yr, 5 females) were randomly selected. Spindles were manually scored in the first 25 consecutive epochs of stage 2 sleep for each quartile of the night. Spindles were identified at the C3 scalp location based on two criteria: 1) minimum of 0.5sec in length, and 2) between 12-16Hz. Subsequently, the PRANA® software (PhiTools, Strasbourg, France) spindle detection algorithm was employed to automatically detect spindles at least 0.5sec in length for the same epochs using six different minimum amplitude thresholds (11, 15, 17, 18, 19 & 23μV).

Results: Figure1A shows the percent agreement between manual and automatic spindle detection at varying amplitude thresholds. Percent agreement was highest (95.9%) at 19μV. The sensitivity (70.1%) and specificity (78.2%) were most balanced at this point (figure1B).
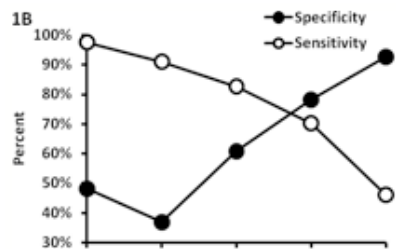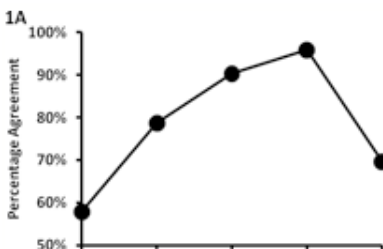
Conclusions: Across thresholds, an inverse relationship between sensitivity and specificity was observed. Sensitivity and specificity were balanced at the point where manual and automatic detection totals had the highest percent agreement. Selecting amplitude thresholds for spindle detection based on systematic benchmarking data may validate methods and improve reproducibility of experimental results.

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Notes