Mot de bienvenue du Président / Welcome from the President

Gilles Lavigne, DMD, MSc, PhD, FRCD
Président / President

C'est avec un immense plaisir que nous vous accueillons au 3ième Congrès de la Société Canadienne du Sommeil. Le comité scientifique, sous la gouverne énergique de notre vice-présidente, Helen Driver, en collaboration avec la représentante des techniciens, Carol Mously et les représentants des étudiants, Stuart Fogel et Tonya Bauermann ont concocté un programme exceptionnel. L'objectif a été de vous faciliter l'acquisition de nouvelles connaissances en médecine du sommeil en sélectionnant des conférenciers de grande qualité. Je tiens à remercier sincèrement ces derniers; aucun ne reçoit d'honoraires et certains ont subit un très long voyage afin de venir vous rencontrer. Le programme est donc diversifié, touchant autant la recherche fondamentale que la recherche clinique. Cette année, les principaux thèmes sont le sommeil chez l'enfant, le sommeil chez la femme, les mouvements anormaux au cours du sommeil, le sommeil et la conduite automobile, le sommeil et la santé cardiaque. Ces choix ont été faits suite à un appel de symposium à laquelle vous avez répondu avec un grand enthousiasme. De plus, tout comme à Québec en 2004, nous avons inclus deux conférences pour le grand public, jeudi soir, sur les thématiques de l'insomnie et du sommeil chez les adolescents.

Cette année, nous avons le plaisir de décerner 2 prix à de jeunes scientifiques en médecine du sommeil, le prix « Roger Brougthon » est décerné au docteur Penny Corkum, Dalhousie University et au docteur John Peever, University of Toronto. Nous inaugurons aussi la remise de la « Distinction honorifique pour une contribution exceptionnelle en recherche sur le sommeil » à un clinicien ou à un chercheur ayant particulièrement marqué la médecine du sommeil par ses activités. Cette année, le récipiendaire est le docteur Jacques Montplaisir de l'Hôpital Sacré-Cœur de Montréal.

Enfin, nous espérons que cette programmation saura stimuler vos recherches et influencer votre pratique de la médecine du sommeil. Le réseautage des forces cliniques et recherche en sommeil au Canada est essentiel afin de maintenir une voix qui fera écho auprès des décideurs et planificateurs des soins de santé. Je tiens tout spécialement à remercier les participants de l'exécutif de la Société Canadienne du Sommeil ainsi que le personnel de Felicissimo et associés ainsi que Carmen Remo qui ont organisé la logistique de notre congrès afin de rendre ces 2 jours inoubliables. Un immense merci à nos commanditaires sans qui la réalisation de cet événement aurait été impossible.

Bonne vigilance pour les 2 prochains jours.

Gilles Lavigne DMD, Ph.D.
Président
Société Canadienne du Sommeil

Gilles Lavigne, DMD, MSc, PhD, FRCD
Président / President

We are thrilled to welcome you to the Canadian Sleep Society's 3rd annual congress. The scientific committee, passionately led by our vice-president, Helen Driver, in collaboration with the technologist representative, Carol Mously, and student representatives, Stuart Fogel and Tonya Bauermann, have devised an exceptional program. Their goal was to make it easier for attendees to acquire new sleep medicine knowledge by selecting first-rate speakers. I am very grateful to them. None of these individuals received payment for their work and some travelled thousands of miles to be here. The program is diversified, touching upon both basic and clinical research. The main themes this year are children and sleep, women and sleep, abnormal movements during sleep, sleep and driving and finally, sleep and heart health. These topics were chosen following a call for symposium proposals that you answered with great enthusiasm. Moreover, as we did in Québec City in 2004, we included two conferences for the general public, to be held on Thursday evening. These will explore insomnia and adolescent sleep patterns.

We are pleased to be handing out two awards to young sleep medicine scientists this year. The Roger Brougthon Young Investigator Award goes to Dr. Penny Corkum of Dalhousie University and Dr. John Peever of the University of Toronto. We are also giving our first "Honorary Distinction Award for Exceptional Contribution to Sleep Research" to a clinician or researcher who had a specific impact on the field of sleep medicine. This year's recipient is Dr. Jacques Montplaisir of the Hôpital Sacré-Cœur de Montréal.

Finally, we hope that this congress will stimulate your research and positively influence your practice of sleep medicine. Pooling clinical strengths and sleep research in Canada is essential to maintaining a voice that will echo among the health care planners and decision-makers. Special thanks to the members of the Canadian Sleep Society's executive committee, Felicissimo & associates' staff and Carmen Reno for organizing the logistics of our congress, ensuring that these two days will be truly memorable. Thanks also to our sponsors, without whom this event could not have taken place.

Enjoy the congress!

Mot de bienvenue du Vice-Président / Welcome from the Vice – President

Helen Driver, Ph.D., RPSGT, D.ABSM

Smile and the world smiles with you. Snore and you sleep alone…. While you are here to continue your education about sleep and the practice of sleep medicine, I hope that you will be smiling at the chance to greet friends and colleagues. I'd especially like to thank the great team who have worked with us to host this conference.

Thank you for your participation.

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Comités / Committees

Exécutif CSS / CSS Executive

Gilles Lavigne, DMD, Ph.D., / President
Charles M. Morin, Ph.D. / Past-President
Judith A. Leech, M.D., FRCPC / Vice-President (Clinical)
Helen S. Driver, Ph.D., RPSGT, D.ABSM. / Vice-President (Research)
Paola Lanfranchi, M.D., / Secretary/Treasurer
James G. MacFarlane, Ph.D., D.ABSM / Member-at-Large
Kimberly A. Cote, M.Sc., Ph.D. / Member-at-Large
Shelly K. Weiss, M.D., / Member-at-Large
Carol Mously, B.Sc., RPSGT / Member-at-Large (Technologist)
Najib Ayas, M.D., MPH / Member-at-Large
Tonya M. Bauermann, M.Sc., RPSGT / Member-at-Large (Student)
Stuart Fogel, B.Sc., MA / Member-at-Large (Student)

Comité scientifique / Scientific Committee

Helen Driver, Ph.D., / Chair
Gilles Lavigne, DMD, Ph.D.
Charles Morin, Ph.D.
Carol Mously, B.Sc., RPSGT
Paola Lanfranchi, Ph.D.
Judith Leech, M.D.
Shelley Weiss, M.D.

Comité local d'organisation / Local Organizing Committee

Gilles Lavigne, DMD, Ph.D.
Christiane Manzini, Research Coordinator
Charles Morin, Ph.D.
Lucy Felicissimo, Jason Rossie, June Viau, and Associates
Paola Lanfranchi, Ph.D.
Carmen Remo

Évaluateurs / Reviewers

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

General Abstract Reviewers

1. Kimberly Cote, Ph.D.
2. Helen Driver, Ph.D.
3. Charles George, M.D.
4. Paola Lanfranchi, Ph.D.
5. Gilles Lavigne, DMD, Ph.D.
6. Judith Leech, M.D.
7. James MacFarlane, Ph.D.
8. Charles Morin, Ph.D.
9. Benjamin Rusak, Ph.D.
10. Shelly Weiss, M.D.

Student and Technologist Competition Reviewers

1. Todd Arnedt, Ph.D.
2. Helen Driver, Ph.D.
3. Judith Leech, M.D.
4. Charles Morin, Ph.D.
5. Carol Mously, RPSGT.
6. John Peever, Ph.D.
7. Dominique Petit, Ph.D.
8. Carlyle Smith, Ph.D.
9. Shelly Weiss, M.D.

Roger Broughton Young Investigator Award Judges

1. Kimberly Cote, Ph.D. (Winner 2004)
2. Joseph De Koninck, Ph.D. (Past-President CSS)
3. Charles George, M.D. (Past-President CSS)
4. Charles Morin, Ph.D. (Past-President CSS)

2007 Congress Organizers

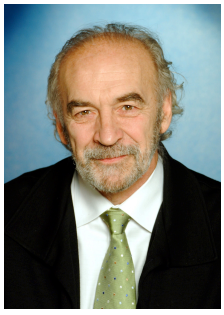
Felicissimo & Associates Inc.
Edifice Place du Quartier
1111 St. Urbain Street, Suite 116
Montréal, Québec, Canada H2Z 1Y6
Tel: (514) 874-1998
Fax: (514) 874-1580
info@fa-events.com
www.fa-events.com

Prix / Awards

Distinguished Scientist Award for an Exceptional Career Contribution in Sleep Research / Distinction honorifique pour une contribution exceptionnelle en recherche sur le sommeil

This new award is the Canadian Sleep Society's highest award for scientific advances in the field of sleep research. The award is given for significant, original and sustained contributions of a basic, clinical or theoretical nature.

As a distinguished scientist and for his significant contributions to the field of sleep research and research in Canada, the Canadian Sleep Society is extremely proud to present the inaugural award for his achievements to Professor Jacques Montplaisir.



Dr. Jacques Montplaisir completed his medical training at the University of Montreal where he also obtained his PhD in Physiology (Neuroscience) under the supervision of Professor Herbert Jasper. He then underwent a three-year postdoctoral training at the California Institute of Technology and at the Stanford University in California. Upon his return to Quebec, he completed clinical training in psychiatry at McGill University and was appointed professor in Psychiatry and in Neuroscience at the University of Montreal in 1977. The same year, he put together the Sleep Disorders Center at the Sacré-Coeur Hospital of Montreal. This Center now includes 10 full-time researchers and 25 research trainees (MSc, PhD and postdoctoral fellows). In 2001, Jacques Montplaisir obtained a Government of Canada Senior Chair for the study of sleep disorders.

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 Roger Broughton Young Investigator Award is made on the basis of an assessment by a distinguished panel of judges. This year the four judges included three former Presidents of the CSS, namely Dr. Charles Morin, Dr. Joseph DeKoninck, and Dr. Charles George, and the past winner of this award – Dr. Kimberly Cote. Eligibility was restricted to candidates up to seven years after completion of a terminal degree (Ph.D. or M.D.) at the time of application, who are members of the CSS and conducting research at a Canadian institution.

We received applications and letters of reference from excellent candidates. It is encouraging to see this because it bodes well for the future of Canadian Sleep Research; the judges were keen to say that those candidates who are recent PhD graduates will have a chance to apply next time. The Award this year is made to two young scientists for important early career research contributions, rather than a single submitted abstract or paper. The winners will each receive an award of $1,000.
The winners are:

Dr. Penny Corkum from Dalhousie University for her contributions to paediatric sleep research and clinical services for children with ADHD and their families.

Dr. John Peever from the University of Toronto for his work on the neurobiology of sleep with an emphasis on physiological mechanisms by which the brain regulates motor activity during sleep.

Both awardees are chairing symposia at this meeting - Penny Corkum is talking in the General Symposium (Paediatric Theme) on Thursday afternoon, and John Peever will be giving a talk in Symposium II (Motor Disorders of Sleep) on Friday morning.

Student and Technologist Abstract Prize Winners

This year we have two student prizes and one technologist prize. The winners of the student abstract competition for 2006 and 2007 are giving an oral presentation during the Student Session on Thursday morning. Congratulations!

Technologist Prize: Sonia Frenette

Gender differences on topographical sleep EEG in midlife.
Frenette S.,[1,2] Paquet, J.[1] Carrier J.[1,2]
[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Québec; [2]Centre de recherche en neuropsychologie et cognition, Département de psychologie, Université de Montréal, Québec.

Two Student Prizes:

- **Patti Brooks**

Impaired GABAergic and glycinergic neurotransmission induces REM-sleep behaviour disorder (RBD) in transgenic mice
Brooks P.L. [1], Tse G. [1] and Peever J.H. [1,2]
Depts. of [1]Cell & Systems Biology and [2]Physiology, University of Toronto, Toronto, Ontario.

- **Marie St. Hilaire**

Laryngeal Stimulation by an Acid Solution in Preterm Lambs during Quiet Sleep
St-Hilaire M., Samson N., Duvareille C., Praud J-P.
Departments of Physiology and Pediatrics, Université de Sherbrooke, Québec.

Trainee Travel Grants

With the CSS conference this year, we made eight Trainee Travel Awards available. There were 15 applications. Awards were given based on rankings in the student abstract competition and by location.

Funding for these travel awards were made through the CSS student fund – *Thank you to CSS members who made contributions to the student fund with their CSS registration* – and a donation from the World Congress of Sleep Apnea care of Professor Jacques Montplaisir.

Travel funds went to assist the two CSS student representatives who co-ordinated and chaired the Student Session - Tonya Bauermann and Stuart Fogel.

6 x $300 travel fellowships were awarded:

1x Halifax (Kimberly Woodford)
1x Kingston (Ariel Le Huquet)
2x Quebec City (Isabelle Turcotte, Simon Beaulieu-Bonneau)
2x Toronto (Neema Kasravi, Christian Burgess)

Présidents, Société canadienne du sommeil /
Presidents, Canadian Sleep Society

Roger Broughton, M.D., Ph.D. (1986-88)
Robert D. Ogilvie, Ph.D. (1988-90)
Meir H. Kryger, M.D. (1990-93)
Alistair MacLean, Ph.D. (1993-96)
Charlie George, M.D. (1996-99)
Joseph De Koninck, Ph.D. (1999-2002)
Charles Morin Ph.D. (2002-2005)
Gilles Lavigne, DMD, PhD (2005-2008)

CME and CEC Information and Certificates of Attendance

This event is approved for up to 15 credits, for Continuing Medical Education.
This event is an accredited group learning activity (Section 1) as defined by the Maintenance of Certification Program of the Royal College of Physicians and Surgeons of Canada. This program meets the accreditation criteria of the College of Family Physicians of Canada for MAIN-PRO-M1 credits. The Centre for CME, Faculty of Medicine, Université de Montréal designates this activity for Category 1 credit towards the AMA Physicians Recgnition Award. Each physician should claim only those hours of credit that he/she actually spent at the education activity.

This two-day congress has BRPT and Educational Advisory Committee recognized Continuing Education Credits for 15.45 hours.

Please complete the evaluation forms.

Certificates of attendance are available through the registration desk.

General Information

Driver's Licences
American state driver's licenses are valid in Canada for varying periods of time as legislated by individual provinces and territories. The International Driving Permit is also valid but it must be accompanied at all times by the visitor's state or national driver's license.

Public Transportation
The *Métro* is the name of Montréal's subway system. Maps are located in each station. Operating hours are from 05:30 to 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. Most stores are open Sundays from 12:00 to 17:00.

Taxes
The Federal Goods and Services Tax (GST) (TPS in French) of 6% applies to purchases. In addition, the Québec Sales Tax (QST) (TVQ in French) adds 7.5% to the total after the GST is included. Non-residents can apply for a GST refund on most goods and hotel accommodations. Tax rebate forms will be available at the hotel and at the airport.

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 expect $1 CDN per piece of luggage.

Metric System
Canada uses the metric system. Visitors will find weather temperature reports given in degrees Celsius, gasoline sold by the liter, milk and wine by milliliter and liters, grocery items in grams and kilograms, and road speeds posted in kilometers per hour.

Voltage
Electricity in Canada is supplied at 110 volt, 60Hz AC alternating current. All non-North American appliances require an adapter for the current as well as for the plug.

Child Care
Most major hotels will gladly arrange childcare upon request.

Speaker Disclosure

Speakers will be 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.

Congress Venue

Centre Mont-Royal / Mount Royal Centre

2200, rue Mansfield
Montréal (Québec)
Tel: (514) 844-2000
Toll free: 1-866-844-2200
www.centremontroyal.com

Horaire d'inscription / Registration Opens

Jeudi 19 avril / Thursday, April 19th 7:00
Vendredi 20 avril / Friday April 20th 7:00

Horaire des exposants / Exhibit Hall Hours

Jeudi 19 avril / Thursday, April 19th 10:00 – 21:00
Vendredi 20 avril / Friday April 20th 7:00 – 17:00

****Don't miss the Opening Mixer on Thursday from 18:00-20:00****

Exposants / Exhibitors

Astromed **(Kiosque / Booth # 22)**
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.

Apnair **(Kiosque / Booth # 24)**
Apnair est une entreprise spécialisée dans les soins de l'apnée obstructive du sommeil assurant une approche personnalisée à la fine pointe de la technologie en matière d'aide respiratoire. Nous offrons des services sur rendez-vous dans nos cliniques du sommeil.

Apnair specializes in a treatment of obstructive sleep apnea. We are devoted to offer you a personalized approach using the latest respiratory aid technology available. You can make an appointment in one of our sleep clinics.

Banque Nationale du Canada **(Kiosque / Booth # 25)**
National Bank launches new Financial Package for Health Professionals

On the heels of its successful Financial Package for Students, National Bank is launching its new Financial Package for Health Professionals. This all-inclusive program, one of the best on the market, gives doctors access to various financial products and services tailored to their needs, all at attractive rates.

Whether clients are looking for personal or business banking solutions, National Bank's Financial Package for Health Professionals gives them preferred rates on selected products and services, including business financing, investments, insurance, legal aid, tax solutions, estate planning, lines of credit, RRSP lines of credit, and more.

Self-employed professionals also have access to services especially for them, including help starting up a business and managing its growth, support with acquisitions, business transfers, purchases and sales of ownership interests and adopting innovative health technologies.

For more information, please contact Mehdi Perrault, at (514) 394-6235.

Braebon **(Kiosque / Booth # 26)**

For years you've known us for our high-quality sleep sensors with a proven track record of reliability and durability. During the last year, BRAEBON has evolved into more than just a sensor company and now offers the following new products for 2007: PURSUIT Sleep, a second generation 22-channel Full PSG system featuring the MediPalm® amplifier; PURSUIT Outcomes®, the industry standard core management business software package for efficiently operating sleep laboratories; and also new for 2007 is the powerful and simple MediByte®, a miniature 10-channel snoring & sleep apnea screener. For more information contact BRAEBON at 1-888-462-4841 or sales@braebon.com.

Fisher **(Kiosque / Booth # 8 and 9)**

At Fisher & Paykel Healthcare, we believe everyone should enjoy a good night's sleep. We've based our business on this belief. To those who suffer from Obstructive Sleep Apnea and those who provide treatment for them, we bring SleepStyleTM - an innovative family of Continuous Positive Airway Pressure and interface solutions.

Medigas-Praxair **(Kiosque / Booth # 14 and 15)**

As a leading provider of sleep diagnostics and sleep apnea therapy, Medigas is well positioned to meet the ever-changing needs of the sleep medicine community. Serving Canadians from over 50 locations, Medigas delivers highly reliable and caring service to improve the quality of life of those affected by sleep apnea.
Medigas – Breathe well. Sleep well. Live well.

OSR Medical Inc. **(Kiosque / Booth # 17 and 18)**

OSR Médical possède et opère un centre de troubles de sommeil réparti sur deux sites à Montréal avec une capacité de six lits. Agrée par le Conseil Canadien d'agrément des Services de Santé CCASS depuis 2002 nous offrons des services intégrés de thérapies respiratoires pour traiter l'apnée du sommeil, l'asthme et les maladies pulmonaires obstructives chroniques.

OSR Médical owns and operates a sleep disorders center at two locations in Montreal with a capacity of six beds. Accredited by the Canadian Council on Health Services Accreditation CCHSA since 2002 we offer integrated therapeutic respiratory services to treat sleep apnea, asthma, and chronic obstructive pulmonary disease.

Quadromed Inc. **(Kiosque / Booth # 7)**

Quadromed Inc. is a Canadian distributor with service and support of sleep diagnostic equipment and supplies.
Our key products in sleep include Compumedics sleep diagnostic, Salter Labs and Smith Medical. Compumedics manufactures wireless ambulatory and wireless PSG sleep devices as well as fixed systems. The Summit IP is Gold Standard Respiratory Effort Measurements device using True Inductive Plethysmographic technology. Salter labs manufactures a full line of nasal pressure and Co2 cannula for sleep. And finally, Smith Medical manufactures End-tidal C02 monitors for sleep diagnostics.

Feel free to come by our booth asking us any question about our products and services.

ResMed **(Kiosque / Booth # 19)**

ResMed is a global leader in medical equipment for the screening, treatment, and management of sleep-disordered breathing and other respiratory disorders. Our new S8™ series of compact flow generators and our popular Mirage Swift™ nasal pillows system are redefining the SDB treatment market. Our VPAP™ Adapt SV is the first bilevel device FDA cleared for the treatment of central sleep apnea, mixed apnea and Cheyne-Stokes respiration. We offer a range of high-quality products across the treatment spectrum for lasting results for our customers and their patients.

Respironics **(Kiosques / Booths # 2,3,4 and 5)**

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. Respironics markets its products in more than 131 countries and employs over 4,700 associates worldwide. Further information can be found on the Company's Web site: www.respironics.com.

Roxon Medi-Tech **(Kiosque / Booth # 23 and 23A)**

Roxon has been providing quality medical equipment in Canada since 1975.
Our Sales Reps will show you the latest in sleep diagnostic equipment including the New Easy III EEG system from Cadwell, Sleeplab Sensors from SLP, and the revolutionary SleepStrip disposable sleep apnea screener.
Visit us at booth 23-23A.

Roxon fourni de l'équipement médical de qualité au Canada depuis 1975.
Nos représentants vous montreront les nouveautés en étude du sommeil, incluant le système EEG Easy III de Cadwell, les capteurs SLP et le révolutionnaire Sleepstrip, un dispositif de dépistage d'apnée du sommeil.
Nous vous attendons au kiosque 23-23A.

Summit Technologies **(Kiosque / Booth # 16)**
Summit Technologies has been providing Canadian Hospitals and clinics with medical capital equipment since 1983. Over these years we have provided the highest quality of products, top notch service and support, and have now added clinical education to our portfolio. All of the above assisted us in becoming one of the most respected suppliers in Canada. Summit is committed to be a major contender in the sleep market, with the new complete line of sleep diagnostics and sleep therapy products from Viasys. Please come by our booth to see what is new with the latest generation of Viasys Sleep Products and a revolutionary new screener from BodyMedia, called the Armband.

Sleep Strategies **(Kiosque / Booth # 1)**
Sleep Strategies Inc. is a leading provider of professional scoring and consultation services for hospitals, private sleep facilities, homecare organizations and pharmaceutical companies worldwide. As a pioneer of sleep scoring services, Sleep Strategies strives to continually offer customers low cost, tailored sleep analysis services of level 1, 2 & 3 sleep studies that meet and exceed industry standards. Our team of registered technologists and flexible scoring solutions give clients easy access to industry experts which assists in increasing productivity and supporting in-house resources optimization. For more information on our sleep scoring services, visit www.sleepstrategies.com or call 1.800.905.0348.

Stellate **(Kiosque / Booth # 6)**

Founded in 1986, Stellate is a leading global supplier of advanced solutions for Sleep Diagnostics, EEG 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 conforms to ISO 13485:2003, ISO 9001:2000 and CE quality requirements.
www.stellate.com
info@stellate.com

Tyco **(Kiosque / Booth # 20 and 21)**

Tyco Healthcare is a leading marketer, manufacturer, distributor and servicer of medical devices and drug products. Its product portfolio includes disposable medical supplies, diagnostic imaging agents, monitoring equipment, medical instruments, bulk analgesic pharmaceuticals and nuclear medicines.

Tyco Healthcare's major business segments include Medical, Surgical, Respiratory, and Imaging. Leading brand names include: Kendall, U.S. Surgical, Auto Suture, Mallinckrodt, Nellcor, Valleylab, and Puritan Bennett.

Tyco Médical est l'un des plus grands commerçants, fabricants, distributeurs et fournisseurs de services de l'industrie des appareils médicaux et des médicaments. Sa gamme de produits comprend des fournitures médicales jetables, des agents d'imagerie diagnostique, des appareils de surveillance, des instruments médicaux, des analgésiques en vrac et des produits de médecine nucléaire.

Les principaux segments commerciaux de Tyco Médical sont le Médical, le Chirurgical, le Respiratoire et l'Imagerie. Ses principales marques comprennent : Kendall, U.S. Surgical, Auto Suture, Mallinckrodt, Nellcor, Valleylab et Puritan Bennett.

Valeant **(Kiosque / Booth # 12 and 13)**

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, de la dermatologie et des maladies infectieuses

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, dermatology and infectious diseases.

VitalAire **(Kiosque / Booth # 10 and 11)**

VitalAire provides one of the largest selections of CPAP Therapy in Canada with full product lines from ResMed, Respironics and Fisher&Paykel. Our focus on Compliance, Communication and Convenience ensures a quick and professional response to your needs and those of your patients. Serving Sleep Specialists and Sleep Professionals across Canada through over 70 offices from coast to coast, visit www.VitalAire.com for an office near you.

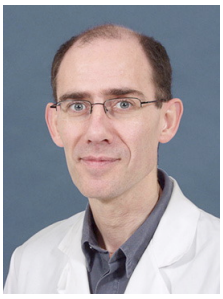
Xltek **(Kiosque / Booth # 27)**

XLTEK is an established market leader in the monitoring and diagnosis of conditions affecting the central and peripheral nervous systems. XLTEK is an innovative developer and manufacturer of quality products and services for EMG, EEG, LTM, IOM and PSG. We also offer a full portfolio of Neurodiagnostic and Sleep Accessories.

Conférenciers invités / Keynote speakers

Dr. Alistair MacLean was born in Aberdeen, Scotland and educated at the University of Aberdeen and Queen's University at Kingston where, as a Commonwealth Scholar, he graduated with his Ph.D. in 1969. After two years at the University of Edinburgh working with Ian Oswald as a Post-Doctoral Fellow, he returned to the Department of Psychology at Queen's in 1971 where he is currently a Professor and Dean of the Faculty of Arts and Science.

From 1986 to 2000 he held an appointment as Staff Scientist at The Toronto Hospital (Western Division). In addition to clinical work with sleep disordered patients - Dr. MacLean has carried out research in a number of different aspects of sleep including: the effects of hypnotic drugs on sleep; the effects of alcohol on sleep; the relationship between sleep and depression; the effect of the menstrual cycle on sleep; the effects of age on sleep; the effects of sleep loss and sleep displacement on subjective state and performance; the measurement of sleepiness; and the nature of the basic mechanisms controlling sleep. Most of his current work is on sleepiness and performance.



Le **Docteur Pierre Maquet** a reçu son diplôme de Médecine de l'Université de Liège (Plus grande distinction avec félicitations du jury) en 1986 et a été agréé Neuropsychiatrie (orientation Neurologie) en 1991. Docteur en Sciences Biomédicales expérimentales depuis 1990, il est Agrégé de l'enseignement supérieur depuis 1998.

Le Docteur Pierre Maquet est actuellement Directeur de Recherches au F.N.R.S. depuis octobre 2003 , attaché au Service de Neurologie du CHU Sart Tilman ainsi qu'au Centre de Recherches du cyclotron de l'Université de Liège où il dirige une équipe d'une dizaine chercheurs (neurologues, neuropsychologues, biologistes ingénieurs et physiciens). Ses travaux portent sur l'étude des états de conscience modifiée chez l'homme en conditions physiologiques (sommeil) ou pathologiques (syndrome de pointe-onde continu du sommeil lent, coma, état végétatif, état de conscience minimale) par les techniques de neuroimagerie fonctionnelle (tomographie à émission de positons, imagerie par résonance fonctionnelle).

Il a été le premier a caractériser la neuroanatomie des sommeils lent profond et paradoxal chez l'homme. Il a démontré, également pour la première fois chez l'homme, l'influence d'un apprentissage sur l'activité cérébrale régionale, suggérant ainsi un rôle du sommeil dans les phénomènes de consolidation mnésiques. Ces travaux ont fait l'objet d'une centaine de publications dans les meilleures revues (Nature, Nature Neuroscience, Science…).

Dr. Judith Owens is an internationally recognized authority on pediatric sleep. Her particular research interests are in the neurobehavioral and health consequences of sleep problems in children, pharmacologic treatment of pediatric sleep disorders, sleep health education, and cultural and psychosocial issues impacting on sleep. As a recipient of a 5-year NIH grant in sleep education, the Sleep Academic Award, she has developed educational materials for the Brown Medical School, as well as the American Academy of Sleep Medicine (AASM). Dr. Owens received the AASM 2006 Excellence in Education Award, and is currently the Chair of the AASM Section on Childhood Sleep Disorders and Development.

Dr. Owens is the Director of the Pediatric Sleep Disorders Clinic at Hasbro Children's Hospital and the Learning, Attention, and Behavior Program at Rhode Island Hospital. She received her undergraduate and medical degrees from Brown and a Master's in Maternal and Child Health from the University of Minnesota. She completed pediatric residency training at Children's Hospital of Philadelphia, and fellowships in Behavioral Pediatrics at Minneapolis Children's Medical Center and in Child Psychiatry at Brown University. She is board certified in Developmental/Behavioral Pediatrics and Sleep Medicine, and is an Associate Professor Pediatrics at the Brown Medical School.

Dr. Owens has recently co-authored a book for physicians on pediatric sleep disorders: "A Clinical Guide to Pediatric Sleep; Diagnosis and Management of Sleep Problems" (2003) and a parent's book on sleep with Jodi Mindell, PhD: "Take Charge of Your Child's Sleep: The All-in-One Resource for Solving Sleep Problems in Kids and Teens".

Virend Kristen Somers, M.B.Ch.B., received his Medicine Degree *cum laude* from the University of Natal, South Africa. He was awarded a Nuffield Dominion Scholarship to Oxford University, United Kingdom where he received his Doctor of Philosophy Degree in Cardiovascular Physiology. He completed a Post-Doctoral Fellowship and Internal Medicine Residency and Cardiology Fellowship at the University of Iowa, where he served as Director of the Cardiovascular Neurophysiology Laboratory. He is presently Professor of Medicine in the Division of Cardiovascular Diseases at the Mayo Clinic and Mayo Foundation, and Director of the GCRC Sleep Core Laboratory. He is Board Certified in Internal Medicine and in Cardiovascular Diseases and is an American Society of Hypertension certified Consultant in Hypertension.

Dr. Somers is a prior Sleep Academic Awardee of the NIH, a Fellow and Established Investigator of the American Heart Association (AHA). Twice he was awarded the Demuth Prize for excellence and originality in hypertension research by the International Society of Hypertension. Among other awards, he received the First Prize of the National Young Investigator Competition of the Heart Institute for Children, the Cournand and Comroe Young Investigator Award from the AHA. He serves on the Editorial Boards of Circulation, Sleep, Hypertension, Italian Heart Journal, is Associate Editor for Chest, and is Consulting Medical Editor for the American Journal of Physiology.

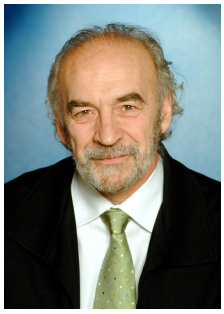
His work examines the physiologic and genetic mechanisms influencing the brain's regulation of the heart and blood vessels in both health and disease. His primary research focus is in sleep and its interaction with circulatory control. This research is funded by the NIH, the Dana Foundation, the AHA and the Mayo Foundation.

Sonia Ancoli-Israel, Ph.D. is a Professor in the Department of Psychiatry at the University of California San Diego (UCSD) School of Medicine, Research Director of the Sleep Medicine Clinic at UCSD, Co-Director of the UCSD GCRC Gillin Laboratory of Sleep and Chronobiology and Co-Director of the Education Unit of the VA VISN-22 Mental Illness Research, Education and Clinical Center (MIRECC). Dr. Ancoli-Israel received her Bachelor's Degree from the State University of New York, Stony Brook, a Master's Degree from California State University, Long Beach and a Ph.D. in Psychology from the University of California, San Francisco.

Dr. Ancoli-Israel is one of the nation's predominant experts in the field of sleeping disorders and sleep research in aging. She was the first at UCSD to be board certified in Sleep Medicine and is the only faculty member at UCSD to be board certified in Behavioral Sleep Medicine. Her current interests include the longitudinal effect of sleep disorders on aging, the effect of circadian rhythms on sleep, therapeutic interventions for sleep problems in dementia, and fatigue, particularly the relationship between sleep, fatigue and circadian rhythms in cancer and other chronic illnesses.

Dr. Ancoli-Israel is Past-President of the Sleep Research Society, past-President of the Society for Light Treatment and Biological Rhythms, and was on the founding Executive Board of the National Sleep Foundation. She has been a guest on television and radio programs including NPR's Morning Edition and Fresh Air with Terry Gross.

Dr. Ancoli-Israel is published regularly in medical and psychiatric journals with over 300 publications in the field.



Jacques Montplaisir completed his medical training at the University of Montreal where he also obtained his PhD in Physiology. His postdoctoral training was at the California Institute of Technology and Stanford University. On returning to Quebec, he completed clinical training in psychiatry at McGill University and was appointed professor in Psychiatry and in Neuroscience at the University of Montreal in 1977. The same year, he put together the Sleep Disorders Center at the Sacré-Coeur Hospital of Montreal. This Center now counts ten full-time researchers and 25 research trainees (MSc, PhD and postdoctoral fellows). In 2001, Jacques Montplaisir obtained a Government of Canada senior Chair for the study of sleep disorders.
To name a few of his awards, he received the Heinz Lehman Award in 1989 for his contribution to research in psychiatry, the *Réalisation de l'année* 1998 Award from the Psychiatrists Association of Quebec for the creation of the Psychopharmacology Chair, and the *Excellence 2000 Award* from the same association. He was honored by the CSS during its inaugural meeting in 2001 and by the Venice Club in 2002 for his research on RLS. In 2004, he received the Léo Parizeau Award from the French Canadian Association for the Advancement of Science for biomedical research. In June 2005, he was the recipient of the Distinguished Scientist Award from the Sleep Research Society.
His laboratory has been funded continuously by the MRC/CIHR for more than 30 years (1978-2010). He has published 3 books, 55 book chapters, over 250 papers and 450 abstracts in peer-reviewed journals and has given 94 invited lectures since 1995. Last year, he was president of the World Congress of Sleep Apnea held in Montreal and he was awarded a major infrastructure grant from the Canadian Innovation Funds for the creation of the multidisciplinary Center for Advanced Research in Sleep Medicine, which will be affiliated with the University of Montreal.

THURSDAY - APRIL 19, 2007

07:00
Registration Opens / Continental Breakfast **(3rd Level Foyer)**

07:15 – 10:00
Exhibit Set-Up **(4th Level Foyer)**
Poster Set-Up **(3rd Level Cartier I)**

08:00 – 09:00

Student Session I (Cartier I)
Chairs: Stuart Fogel; Tonya Bauermann

Opportunities in Behavioral Sleep Medicine: Past, Present, and Future
Keynote Speaker: Charles A. Morin
8:00 - 8:30 am

Scholarship and Grant Applications
Keynote Speaker: B. Rusak
8:30 - 9:00 am

Technologist Session I (Mont Royal II)

Chair: Carol Mously
Welcome Introduction

Influence of Medications on PSG
Speaker: J. MacFarlane
8:00 - 8:30 am

Scoring of Sleep Bruxism
Speaker: G. Lavigne
8:30 - 9:00 am

09:00 – 10:00

Workshop 1 (Cartier I)
Best Foot Forward: Preparing for the Job Interview
Speaker: A. MacLean
9:00 - 9:30 am

Presentation & Workshops (Mont Royal II)
Taking the "Art" out of Artifacts
Speaker: M. Eden
9:00 - 9:30 am

Student Presentation:
(10 minutes, followed by 5 minute question period)
-2006 Abstract Winner
9:30 - 9:45 am
-2007 Abstract Winner
9:45 - 10:00 am

Discussion Group
BRPT: RPSGT Exam and Continuing Education Credits
Speaker: R. Godbout
9:30 - 10:00 am

10:00-10:15
Break & Opening of Exhibits **(4th Level Foyer)**

10:15-11:45

Student Session II (Cartier I)
Chairs: Stuart Fogel & Tonya Bauermann

Workshop 2
The Big Picture: Designing a Research Plan
Speaker: Julie Carrier
10:15 - 10:45 am

Student Presentation:
(10 minutes, followed by 5 minute question period)
- 2006 Abstract Winner
10:45 – 11:00 am
- 2007 Abstract Winner
11:00 – 11:15am

Workshop 3

Presentation Skills: The Good, the Bad and the Ugly
Speaker: John Peever
11:15 – 11:45 am

Technologist Session II (Mont Royal II)
Chair: Carol Mously, B.Sc., RPSGT

Paediatric Sleep Studies
Speaker: K.J. Gibbons
10:15 - 10:45am

AASM Scoring Rules Update
Speaker: S. Ancoli-Israel
10:45 - 11:15am

Canadian Thoracic Society Guidelines for Sleep-Disordered Breathing Diagnosis and Treatment in Adults
Speaker: M. Fitzpatrick
11:15 - 11:45am

11:45-12:45

Lunch **(4th Level Foyer & 4th Level Mont Royal I)**
Visit Exhibitors **(4th Level Foyer)**
Posters **(4th Level Mont Royal I)**

Welcome Remarks **(4th Level Salon Mont-Royal II)**
Gilles Lavigne and Helen Driver

12:45-13:30 **(Salon Mont-Royal II)**
Sleep and Driving
Keynote Speaker: Alistair MacLean, Queen's University, Kingston, Canada

13:30-14:15 **(Salon Mont-Royal II)**
Memory Consolidation, as Assessed by Functional Neuroimaging
Keynote Speaker: Pierre Maquet, Centre de Recherches du cyclotron de l'Université de Liège, Belgium

14:15-15:00 **(Salon Mont-Royal II)**
Cultural Differences in Sleep and Naps in Children
Keynote Speaker: Judith Owens, Brown Medical School, Rhode Island, USA

15:00-15:15
Break –
Visit Exhibits **(4th Level Foyer)**
Posters **(4th Level Mont Royal I)**

15:15-17:15 **(Salon Mont-Royal II)**
General Symposium
Childhood Psychopathology and Sleep Problems
Chairs: Penny Corkum; Roger Godbout
Speakers: Graham Reid, Mélanie St-Onge, Christine Chambers, Penny Corkum, Roger Godbout, Roseanne Armitage

17:15-18:00 **(4th level Mont Royal II)**
Annual General Meeting and Awards Ceremony

18:00-20:00 **(4th level Foyer)**
Opening Mixer

19:30-21:00 **(4th level Mont Royal II)**
Public Lecture

Thursday, April 19th from 08:00 to 11:45
Student Session

Chairs: Stuart Fogel, BSc., MA., Centre for Neuroscience Studies, Queen's University, Ontario
Tonya Bauermann, MSc., Department of Psychology, Queen's University, Ontario

Welcome and Introduction: Stuart Fogel / Tonya Bauermann

08:00 Opportunities in Behavioral Sleep Medicine: Past, Present, and Future
Charles Morin, Ph.D., Université Laval, Québec

This presentation will discuss behavioral sleep medicine as a new subspecialty emerging from a blend of sleep research/medicine and health psychology/behavioral medicine. We will present a brief historical perspective, with examples of ongoing clinical research to illustrate the scope of this new field. Education and training opportunities will be discussed, as will opportunities for career developments in behavioral sleep medicine.

08:30 The Art and Science of Writing Competitive Grant Applications
Ben Rusak, Ph.D., Dalhousie University, Halifax, Nova Scotia

1) How to write strong applications.
2) Common problems to avoid.
3) Where to apply?
4) Future areas of interest.

09:00 Workshop 1: **Best Foot Forward: Preparing for the Job Interview**
Alistair MacLean, Ph.D., Queen's University, Kingston, Ontario

Learning Objectives:
1) Writing a good job application.
2) Being an excellent interview candidate.
3) How to negotiate the terms of your position.
4) Career management.

Student Presentations

09:30 2006 abstract winner: Pennestri M-H., Montplaisir J., Richard M., Rompré S., Colombo R. , Lanfranchi P.
Differential Rise of Blood Pressure in Periodic Leg Movements Associated or not with Micro-Arousals in Patients with Restless Legs Syndrome
Marie-Hélène Pennestri, BSc.

09:45 2007 abstract winner: Brooks P.L., Tse G. and Peever J.H.
Impaired GABAergic and Glycinergic Neurotransmission Induces REM-Sleep Behaviour Disorder (RBD) in Transgenic Mice
Patti Brooks, MSc.

10:15 Workshop 2: **The Big Picture: Designing a Research Plan**
Julie Carrier, Ph.D., University of Montreal, Québec

Student Presentations

10:45 2006 abstract winner: Mongrain V., Dumont M.
Effect of Sleep Fragmentation on Spectral Activity of Sleep EEG in Morning-Type and Evening-Type Individuals
Valérie Mongrain, PhD.

11:00 2007 abstract winner: St-Hilaire M., Samson N., Duvareille C., Praud J-P.
Laryngeal Stimulation by an Acid Solution in Preterm Lambs During Quiet Sleep
Marie St-Hilaire, MA.

11:15 Workshop 3: **Presentation Skills: The Good, The Bad and The Ugly**
John Peever, Ph.D., University of Toronto, Ontario

Learning Objectives:
1) Importance of presenting a winning presentation.
2) What is a winning presentation?
3) The 5 steps to success.

Concluding Comments: Stuart Fogel / Tonya Bauermann

Thursday, April 19th from 08:00 to 11:45
Technologist Session

Chair: Carol Mously, B.Sc., RPSGT University of Toronto, Ontario

Welcome and Introduction: Carol Mously

08:00 The Influence of Medications on Polysomnography (PSG)
James MacFarlane Ph.D., DABSM, Department of Pediatrics and Psychiatry, University of Toronto, Ontario

Learning Objectives:
1) Review the objective effect of various sleep promoting medications on the PSG.
2) Review the objective effect of other (non-sleep promoting) medications on the PSG.
3) To outline the pharmacokinetic principles in relationship to effects on sleep and daytime functioning.

08:30 Scoring of Sleep Bruxism
Gilles Lavigne, Ph.D., DMD, Université de Montréal and Hôpital du Sacré-Cœur de Montréal, Québec

Learning Objectives:
1) Review current standards on the scoring of sleep bruxism.
2) To be able to recognize and score sleep bruxism
3) Future trends in bruxism research/treatment.

09:00 Workshop: **Taking the "art" Out of Artifacts**
Michael Eden, RPSGT, Toronto, Ontario

Learning Objectives:
1) To be able to recognize artifact in the overnight PSG recording.
2) To be able to take corrective action to eliminate artifact from PSG recordings.
3) Review actions that can be taken to prevent the presence of artifacts in the overnight PSG.

09:30 BRPT Representative: RPSGT Examination and Continuing Education Credits
Roger Godbout, Ph.D., RPSGT, Université de Montréal, Québec

Learning Objectives:
1) Provide an overview about the Board of Registered Polysomnographic Technologists.
2) Review education an training required to sit the Registered Polysomnographic Technologist (RPSGT) exam.
3) Discuss the new RPSGT recertification guidelines.

10:15 Paediatric Sleep Studies
K. Jeremy Gibbons, RPSGT, Hospital for Sick Children, Toronto, Ontario

Learning Objectives:
1) Identify and recognize the challenges of conducting overnight PSGs on the paediatric population.
2) Specialized equipment and procedures for conduction PSGs on a paediatric population.
3) Considerations for scoring paediatric sleep PSGs.

10:45 American Academy of Sleep Medicine (AASM) Scoring Rules Update
Sonia Ancoli-Israel, Ph.D, DABSM, University of California San Diego, USA

Learning Objectives:
1) Discuss the specific AASM scoring rules for characterizing sleep and sleep-related events.
2) Recognize the underpinnings of evidence and consensus for the AASM rules.
3) Review the input of industry and technical panels and the responses from task forces and the steering committee.

11:15 Canadian Thoracic Society Guidelines: Diagnosis and Treatment of Sleep Disordered Breathing in Adults
Michael Fitzpatrick, M.D., DABSM, Kingston General Hospital and Queen's University, Kingston, Ontario

Learning Objectives:
1) Discuss the process whereby the Canadian Thoracic Society – Sleep Disordered Breathing (SDB) Committee developed the guidelines.
2) Review the clinical SDB syndromes and severity definitions.
3) Discuss the referral process and diagnostic criteria for SDB.
4) Review appropriate therapeutic interventions for SDB syndromes.

11:45 Concluding Comments: Carol Mously

Note for all RPSGT: To obtain your Continuing Education Credits, please make sure that you sign in for each day and that you complete the evaluation forms.

Thursday, April 19th from 12:45 to 15:00
Keynote Speakers

12:45 - 13:30 Sleepiness and Driving
Alistair MacLean, Ph.D, Queen's University, Kingston, Canada

Learning Objectives:
1) Be familiar with the extent of the problem of sleepiness and driving.
2) Comprehend the mechanisms underlying sleepiness and driving.
3) Know the principal manifestations of sleepiness in driving.
4) Be aware of the issues relating to the assessment of sleepiness, particularly with respect to driving.
5) Understand what countermeasures are effective.

13:30 - 14:15 Memory Consolidation, as Assessed by Functional Neuroimaging
Pierre Maquet, MD, Ph.D, Cyclotron Research Centre, University of Liege, Belgium

Learning Objectives:
1) Sleep takes part in memory consolidation
2) Memory consolidation does not take place exclusively during sleep
3) The brain is redundant: similar behavioral performances can be achieved using different cerebral networks.

14:15 - 15:00 Cultural Differences in Sleep and Naps in Children
Judith Owens, MD, D.ABSM, Brown Medical School, Rhode Island, USA

Learning Objectives:
1) Appreciate how culturally determined factors impact on children's sleep.
2) Understand important cultural differences in sleeping practices around the world, including napping, use of transitional objects, sleep amounts, and treatment of sleep problems.
3) Differentiate between causes and consequences of the practice of co-sleeping in different cultures.

Thursday, April 19th from 15:15 to 17:15
General Symposium: ***Childhood Psychopathology and Sleep Problems***

Chairs: Penny Corkum, Ph.D., Department of Psychology, Dalhousie University, Halifax
Roger Godbout, Ph.D., Sleep Laboratory and Clinic, Hôpital Rivière-des-Prairies, Montréal

The regulation of sleep is closely related with behavioural and emotional disorders in childhood. The exact nature of this relationship between sleep and psychopathology is unknown; however, at a minimum there appears to be bi-directional influences. The focus of the symposium is to explore these relationships using different methodologies and within the context of different childhood disorders.

Learning Objectives:

1) Understand the inter-relationships between childhood sleep problems and psychopathology.
2) Learn about naturalistic and lab-based research methodology designed to address the impact of sleep on childhood functioning, as well as a range of technologies used to assess sleep in pediatric populations.
3) Gain knowledge about assessment and treatment of childhood sleep problems in the context of mental health disorders.

15:15 Introduction
Penny Corkum and Roger Godbout

15:20 Contributions of Sleep Problems to Psychopathology in 2 and 3 Year Old Children
Graham J. Reid, Ph.D., The University of Western Ontario, Ontario

15:35 Dreams, Nightmares and Sleep Terrors in Children
Mélanie St-Onge, Ph.D., Centre Hospitalier, Universitaire de Québec, Québec

15:50 Impact of Sleep Restriction on Pain Responses in Healthy Adolescents
Christine T. Chambers, Ph.D., Dalhousie University, Halifax, Nova Scotia

16:05 Impact of Stimulant Medication on Sleep in Children with Attention-Deficit/ Hyperactivity Disorder
Penny Corkum, Ph.D., Dalhousie University, Halifax, Nova Scotia

16:20 Sleep in Pervasive Developmental Disorders
Roger Godbout, Ph.D., Université de Montréal, Québec

16:35 Sleep, EEG and Depression in Adolescents
Roseanne Armitage, Ph.D., University of Michigan, Ann Arbor, Michigan, USA

16:50 General Discussion

17:10 Concluding Remarks - Penny Corkum and Roger Godbout

FRIDAY - APRIL 20, 2007

07:00
Registration / Continental Breakfast **(3rd Level Foyer)**

7:30 - 8:30 **(Salon Mont-Royal II)**
Early Morning Session
Insomnia Task Force
Chairs: Charles Samuels, Centre for Sleep and Human Performance, University of Calgary
James MacFarlane, University of Toronto

8:30 - 10:00

Symposium I (Cartier I)
Title: *Sleep and Psychopathology*
Chair: Eileen Sloan
Speakers: Ruzica Jokic, Alan Douglass
James MacFarlane, Roseanne Armitage

Symposium II (Mont Royal II)
Title: *The Motor Disorders of Sleep: Neurophysiological and Clinical Perspectives*
Chair: Richard Horner
Speakers: John Peever, Jacques Montplaisir, Gilles Lavigne, Brian Murray

10:00 - 11:00
Break –
Visit Exhibits **(4th Level Foyer)**
Poster Sesssion **(4th Level Mont Royal I)**

11:00 - 12:30

Symposium III (Cartier I)
Title: *Sleep, Performance and Cognition*
Chair: Kimberly Cote
Speakers: Carlyle Smith, Julie Carrier, Kimberly Cote, Marc Lavoie

Symposium IV (Mont Royal II)
Title: *Upper Airway Physiology and the Pathogenesis of Obstructive Sleep Apnea*
Chair: John Peever
Speakers: Richard Horner, Frédéric Sériés, John Kimoff, Michael Fitzpatrick

12:30 - 13:30
Lunch –
Visit Exhibits **(4th Level Foyer)**
Posters **(4th Level Mont Royal I)**

13:30-14:15 **(Salon Mont-Royal II)**
Sleep, Death and the Heart
Keynote speaker: Virend Somers, Mayo Medical School, Mayo Clinic, Rochester, USA

14:15-15:00 **(Salon Mont-Royal II)**
Sleep and Fatigue in Women with Breast Cancer
Keynote speaker: Sonia Ancoli-Israel, University of California San Diego, USA

15:00 - 15:15
Break –
Visit Exhibits **(4th Level Foyer)**
Posters **(4th Level Mont Royal I)**

15:15 - 16:00 **(Salon Mont-Royal II)**
Movement Disorders
Keynote speaker: Jacques Montplaisir, University of Montreal, Canada

16:00 - 17:00 **(Salon Mont-Royal II)**
Inovations in Sleep Medicine
(5-10 minute update on new findings / Novelties)
Chairs: Helen Driver and Gilles Lavigne

- Mayer / Altitude experience (10 minutes)
- Laverdure Dupont / Placebo and Sleep (5 minutes)
- Dumont / Circadian Rhythms (5 minutes)
- Weiss / Paediatric (5 minutes)
- Boivin / Women (5 minutes)
- Carrier / Aging (5 minutes)
- Smith / Dreams, memory (5 minutes)
- Sériès / Sleep Medicine Practice (5 minutes)

17:00 **(Salon Mont-Royal II)**
Closing Remarks from Helen Driver

Friday, April 20th from 7:30 to 8:30

Early Morning Session: ***Insomnia Clinical Practice Guidelines: Development, Dissemination and Implementation***

Chairs: Charles Samuels, M.D., CCFP, DABSM, University of Calgary, Calgary, Alberta
James MacFarlane, Ph.D., DABSM, Asst Professor of Pediatrics and Psychiatry, University of Toronto, Ontario

The complaint of difficulty initiating and maintaining sleep as well as non-restorative sleep is prevalent in a primary care setting. Family physicians receive limited training in sleep medicine during medical school and family practice residency training. At the 2001 APSS in Seattle a group of Canadian sleep specialists met to begin the process of developing an Insomnia Clinical Practice Guideline. Following this meeting the University of Calgary Department of Continuing Medical Education and Professional Development identified a need for educational programs addressing the diagnosis and management of Insomnia in the Primary Care setting. Two nationally accredited Insomnia programs were developed by the U of C in conjunction with the University of Laval. These programs were run as 3-hour problem-based, case-oriented workshops and as a 3-week online CME program through the Memorial University online MDcme program. Since the Seattle meeting a sub-group of the original steering committee enlisted the assistance and support of the Alberta Medical Association, Toward Optimized Practice (TOP) Program to develop an evidenced based clinical practice guideline. This symposium will outline the development process, dissemination strategies and implementation of the guideline.

Learning Objectives:

1) To discuss the process of developing an appropriate evidenced based clinical practice guideline.
2) To explain dissemination strategies and discuss the limitations of various dissemination strategies.
3) To discuss implementation strategies and research methodology to evaluate the efficacy of the guideline.

07:30 Introduction
Charles Samuels, M.D., CCFP, DABSM, University of Calgary, Calgary, Alberta

07:45 Development
James MacFarlane, Ph.D., DABSM, Department of Pediatrics and Psychiatry, University of Toronto, Ontario

08:00 Dissemination
Charles Samuels, MD, CCFP, DABSM

08:15 Implementation
Charles Samuels, MD, CCFP, DABSM
James MacFarlane, PhD, DABSM

08:30 Discussion

Friday, April 20th from 08:30 to 10:00
Symposium I: ***Sleep and Psychopathology***

Chair: Eileen P. Sloan, Ph.D., M.D., Assistant Professor of Psychiatry, University of Toronto, Mt. Sinai Hospital, Toronto, Ontario

There is a strong correlation between sleep disorders and psychiatric illness, essentially all psychiatric disorders having a profound impact on sleep. The most common complaint is insomnia. Conversely, it is now evident that insomnia can predispose sufferers to psychiatric conditions, the most commonly studied condition being major depressive disorder. Furthermore, the medications used to treat psychiatric disorders often affect sleep detrimentally. It is essential that the assessment of all patients in the sleep clinic take into account the fact that psychiatric illness may be playing an important role in the presentation. This symposium is intended to provide an overview of the relationship between psychiatric illness and sleep disorders; to provide a template for determining whether or not psychiatric illness is contributing to the sleep complaint; and to look at the impact of psychotropic medications on sleep quantity and quality.

Learning Objectives:

1) Describe the role that sleep disorders play in the etiology of psychiatric illness.
2) Outline how sleep is affected by various psychiatric illnesses.
3) Understand the impact of psychotropic medications on sleep.
4) Determine when a patient's complaint of sleep disruption is influenced by a psychiatric disorder.

08:30 Introduction
Eileen P. Sloan, M.D., Ph.D.

08:35 Psychiatric Illness and Sleep - Approach to the Assessment of Sleep Complaints in a Patient with a Mental Disorder
Ruzica Jokic, M.D., Queen's University, Kingston, Ontario

08:55 REM Density: A New Standard for Predicting Depression
Alan Douglass, M.D., University of Ottawa, Ontario

09:15 Psychiatric Medications: How Do They Affect Sleep?
James MacFarlane, Ph.D., University of Toronto, Ontario

09:35 The Interaction Between Sleep, Gender and Depression
Roseanne Armitage, Ph.D., University of Michigan, Ann Arbor, USA

09:55 Concluding Comments
Eileen P. Sloan

Friday, April 20th from 08:30 to 10:00

Symposium II: ***The Motor Disorders of Sleep: Neurophysiological and Clinical Perspectives***

Proposer: John Peever, Ph.D., Systems Neurobiology Laboratory, Department of Cell & Systems Biology and Physiology, University of Toronto, Ontario

Chair: Richard L. Horner, Ph.D., University of Toronto, Onatrio

Disturbances in motor control underlie most of the major sleep disorders including obstructive sleep apnea, bruxism, REM-sleep behaviour disorder, periodic limb movement disorder and narcolepsy. The goal of this symposium is two-fold. The first is to provide an up-to-date review of the neurophysiological basis of motor control during sleep in health and disease. The second is to provide a clinical overview of the pathophysiology and treatments for REM-sleep behaviour disorder, bruxism and narcolepsy/cataplexy.

Learning Objectives:

1) Understand the neurophysiology of motor regulation across the sleep cycle.
2) Understand the clinical and physiological basis of REM sleep behaviour disorder and its treatments.
3) Understand the clinical and physiological aspects of bruxism.
4) Understand the clinical features and treatments for narcolepsy and cataplexy.

08:30 Introduction
Richard Horner, Ph.D., University of Toronto, Ontario

08:35 Neurophysiology of Motor Control Across the Sleep Cycle
John Peever, Ph.D., University of Toronto, Ontario

08:55 Understanding REM-Sleep Behaviour Disorder
Jacques Montplaisir, M.D., Ph.D., Université de Montréal and Hôpital du Sacré-Cœur de Montréal, Québec

09:15 The Pathophysiology of Bruxism
Gilles Lavigne, Ph.D., Université de Montréal and Hôpital du Sacré-Cœur de Montréal, Québec

09:35 Diagnosis and Treatment for Narcolepsy/Cataplexy.
Brian Murray, M.D., University of Toronto and Sunny Brook Hospital, Ontario

09:55 Discussion

Friday, April 20th from 11:00 to 12:30
Symposium III: ***Sleep, Performance and Cognition***

Proposers: Kimberly Cote, Ph.D., Brock University, St. Catherines, Ontario
Celyne Bastien, Ph.D., University of Laval, Québec City, Québec

Chair: Kimberly Cote, Ph.D., Brock University, St. Catherines, Ontario

In our aging society, where work demands are higher, people are intentionally reducing sleep time and sleep disorders are more prevalent. This symposium includes four presentations that will examine the bi-directional relationship between sleep and wakefulness. Smith will present human data to support the idea that cognitively challenging motor procedural tasks invoke REM sleep systems to achieve consolidation, while familiar motor tasks involve Stage 2 sleep spindles. Carrier will discuss age-related difference in the ability of sleep to adapt to various challenges (e.g., sleep deprivation, caffeine), and the impact on vigilance and performance. Next, data on the effects of varying levels of sleep restriction on CNS arousal and attention will be presented by Cote. Lastly, Lavoie will report on the effects of 24hrs total sleep deprivation on both frontal and parietal ERPs to examine compensatory mechanisms in attention and motor processes.

Learning Objectives:

1) Provide an overview of current research in area of "sleep, performance, and cognition" through new data on a range of topics, including sleep deprivation and restriction, learning, and aging.
2) Examine the bi-directional relationship between sleep and wakefulness, that is, how sleep impacts waking function, and how waking experience alters sleep physiology.
3) Illustrate how electrophysiological techniques (e.g., EEG and event-related potentials) may be applied to understanding brain-behaviour relationships in the context of sleep research.

11:00 Introduction
Kimberly Cote, Ph.D., Brock University, Ontario

11:05 Sleep states and memory processes: Roles for REM and Stage 2 sleep
Carlyle Smith, Ph.D., Trent University, Peterborough, Ontario

11:25 Challenges to the sleep-wake cycle in aging: effects on sleep, alertness, vigilance and performance.
Julie Carrier, Ph.D., Université Montreal, Québec

11:45 Arousal, attention and performance during short-term sleep restriction
Kimberly Cote, Ph.D., Brock University, Ontario

12:05 Event-related potentials reflecting brain compensatory mechanisms following total sleep deprivation
Marc Lavoie, Ph.D., Université Montreal and Louis-H Lafontaine Hospital, Québec

12:25 Discussion

Friday, April 20th from 11:00 to 12:30

Symposium IV: ***Upper Airway Physiology and the Pathogenesis of Obstructive Sleep Apnea***

Proposer: Richard L. Horner, Ph.D., University of Toronto

Chair: John H. Peever, Ph.D., University of Toronto

Obstructive sleep apnea (OSA) is a common and serious sleep-related breathing disorder characterized by repeated episodes of upper airway closure occurring exclusively during sleep. This symposium will provide an up-to-date review of current research related to the physiology of upper airway muscle control across sleep and awake states. The symposium will also address important aspects of upper airway function related to the pathogenesis of OSA.

Learning Objectives:

1) Understand the mechanisms underlying the effects of sleep on upper airway muscle activity and the clinical implications.
2) Understand the factors promoting upper airway collapsibility and stability in humans.
3) Understand the role of upper airway trauma and altered sensation associated with repeated airway obstructions in the pathogenesis of OSA.
4) Understand the effects of route of breathing on upper airway function and the pathogenesis of OSA.

11:00 Introduction
John H. Peever, Ph.D., University of Toronto

11:05 Sleep and Upper Airway Muscle Activity: Mechanisms of Control and Clinical Implications
Richard L. Horner, PhD, University of Toronto, Ontario

11:25 Upper Airway Collapsibility and Stability in Humans
Frédéric Sériès MD, Université Laval, Québec

11:45 Upper Airway Trauma and Sensation in Obstructive Sleep Apnea
R. John Kimoff, MD, McGill University, Montreal

12:05 Route of Breathing and Obstructive Sleep Apnea
Michael Fitzpatrick, MD, Queen's University, Kingston

12:25 Discussion

Friday, April 20th from 13:30 to 16:00
Keynote Speakers

13:30 - 14:15 Sleep, Death and the Heart
Virend Somers, MD, D.Phil, Mayo Medical School, Mayo Clinic, Rochester, USA

Learning Objectives:
1) Understand the physiology of sleep
2) Review the early morning preponderance of cardiac and vascular events
3) Examine interactions between blood pressure control during sleep and cardiovascular risk
4) Identify disease conditions predisposing to sleep related sudden death
5) Explore the role of obstructive sleep apnea in cardiovascular events occurring during sleep.

14:15 - 15:00 Sleep and Fatigue in Women with Breast Cancer
Sonia Ancoli-Israel, Ph.D, D. ABSM, University of California San Diego, USA

Learning Objectives:
1) Understand the relationship between fatigue and sleep in breast cancer
2) Understand the correlates of poor sleep in women with breast cancer
3) Knowledge of the literature on treatment of insomnia in breast cancer.

15:15 - 16:00 Movement Disorders During Sleep
Jacques Montplaisir, MD, Ph.D, University of Montreal, Canada

Learning Objectives:
1) Recognize the symptoms of restless legs syndrome (RLS) and periodic limb movements during sleep as compared to the loss of motor inhibition during REM sleep seen in REM sleep behaviour disorder (RBD)
2) Review diagnostic procedures for sleep-related movement disorders
3) Review the evidence-based and clinical practices regarding management of RLS and PLMS for various age groups
4) Assess the treatment options for RBD.

Friday, April 20th from 16:00 to 17:00
Innovations in sleep medicine

Welcome to our final session of this conference. The idea for this session is to have researchers and clinicians give a 5 to 10 minute update on new findings or novelties in their area of expertise. These brief presentations may be done with or without slides.

Chairs:
Helen Driver, Ph.D., RPSGT,
DABSM, Kingston General Hospital and Departments of Medicine and Psychology, Queen's University, Kingston, Ontario
Gilles Lavigne, Ph.D., DMD, Université de Montréal and Hôpital du Sacré-Cœur de Montréal, Québec

- Pierre Mayer / **Altitude experience** (10 minutes)
- Danièle Laverdure-Dupont / **Placebo and Sleep** (5 minutes)
- Marie Dumont, Ph.D., Université Montreal / **Circadian Rhythms** (5 minutes)
- Shelley Weiss, M.D., Hospital for Sick Children, Toronto **/ Paediatric** (5 minutes)
- Diane Boivin, M.D., Ph.D., McGill University / **Women** (5 minutes)
- Julie Carrier, Ph.D., Université Montreal / **Aging** (5 minutes)
- Carlyle Smith, Ph.D., Trent University / **Dreams, memory** (5 minutes)
- Frédéric Sériès, M.D., Université Laval / **Sleep Medicine Practice** (5 minutes)

Poster Presentations

General Neuroscience

P101 ***Effects of Hypercapnia and Hypoxia on Non-Nutritive Swallowing in Newborn Lambs***
Duvareille C., St-Hilaire M., Samson N., Micheau P.[1], Bournival V., Praud JP.

P102 ***Special Charateristics Surrounding the Spontaneous K-Complex in Good Sleepers***
Forget D., Morin C.M. & Bastien C.H

Circadian Rhythms

P103 ***A Non-Invasive Cerebral Temperature Sensor for the Assesment of Circadian Rhythms***
Boudreau P., Shechter A., Dittmar A., Gehin C., Delhomme G., Nocua R, Dumont G., Boivin D.B.

P104 ***Effect of an Intervention on Melatonin Production in Police Officers Working Rotating Shifts***
Tremblay G.M., Bourdhouxe M. and Boivin D.B.

P105 ***Emergence of Physiological Rhythmicity in Term and Preterm Neonates in a Intensive Care Unit***
Esmot ara B, Motoki B , Makoto O, Hatsumi Y, Kawai M, Yoshihiro K

P106 ***Age-Related Effects of 200MG of Caffeine on Daytime Recovery Sleep***
Girouard, L., Fernandez-Bolanos, M., Roy, J., Paquet, J., Filipini, D., Carrier, J.

P107 ***Clock Gene Expression in Peripheral Blood Mononuclear Cells Following Simulated Night Shift Work***
James F.O., Cermakian N., Boivin D.B.

P108 ***Clock Gene Expression in Human Peripheral Blood Mononuclear Cells Throughout an Uninterrupted 72-Hour Period***
James F.O., Charbonneau S., Bélanger V., Boivin D.B., Cermakian N

P109 ***Evidence that Wearing Blue-Blockers after the Night Shift Improves Day Time Sleep***
Sasseville A. Charron M-C, Hebert M

Sleep Deprivation

P110 ***Effects of Ovariectomy and Estrogen Replacment on Spontaneous Sleep and Recovery Sleep after Sleep Deprivation in Rats***
Deurveilher S., Wilkinson M., Rusak B., Semba K.

P111 ***The Effects of Sleepiness, Incentive and Distraction on the P300 and Contingent Negative Variation (CNV)***
Murphy, T.I., Segalowitz, S.J.

P112 ***Effects of Sleep Deprivation, Attention Load and Visual Information on Postural Control in Young Healthy Adults (Preliminary Results)***
Robillard R., Boissoneault M., Filipini D., Prince F., Carrier J.

Behaviour & Cognition

P113 ***Too Much of a Good Thing? REM Sleep Theta Activity Predicts Two-Way Active Avoidance (TWAA) Performance and Increases Prior to Maladaptive Learning in Rats***
Fogel, S. M., Smith, C. T. & Beninger, R. J.

P114 ***REM Sleep Duartion Predicts Two-Way Shuttle Active Avoidance (TWAA) Performance and Increases 17-20 Hours Following Learning in Rats***
Fogel, S.M., Smith, C.T. Beninger, R.J.

P115 ***Effects of CPAP Treatment of Obstructive Sleep Apnea on Daily Functions, Mood and Quality of Life***
Lau E.Y.Y., Eskes G.A., Morrison D.L., Rajda, M., Spurr K.F.

P116 ***Alteration of REM Sleep Duration in Placebo Responders Depending on Evening Placebo Analgesia Manipulations***
Laverdure-Dupont, D., Rainville, P., Montplaisir, J., Lavigne, G.

P117 ***Event-Related Potentials Differentiates the Processes Involved in the Effects of Sleep on Recognition Memoryt***
Mograss M, Guillem F, Godbout R

P118 ***Dual Target Processin During Continuous Sleep Restriction: The Attentional Blink Task***
Smith B.A., Arnell, K.M., Cote K.A.

P119 ***Decreases in Number of Sleep Spindles Following Acquisition of a Declarative Task Using a Retroactive Interference Paradigm***
Smith CT, McGilvray MP, Moran CR, Peters KR

P120 ***Decreases in Stage 2 Sleep Following Acquisition of a Declarative Task Using a Retroactive Interference Paradigm***
Smith, CT, Moran CR, McGilvray MP, Peters KR

Instrumentation & Methodology

P121 ***Instrumentation and Scoring Validity of Polysomnographic Mouth Leak Events During Treatment with Nasal Continuous Positive Airway Pressure***
Baltzan M.A., Garcia-Asensi A., Sully J., Tanzimat G., Kassissia I., Wolkove N.

P122 ***The Sleep Hygiene Inventory (SHI): A New Self-Report Measure***
Bauermann T.M , MacLean A.W., Parker J.D.A

P123 ***Feasibility of Unattended Overnight Cardio-Respiratory Monitoring in Screening for Sleep-Disordered Breathing in a Chronic Hemodialysis Population***
Champagne K.A.,Tangri N., Kimoff R.J., Barre P., Iqbal S.

P124 ***Minimal Impact of Inadvertent Sleep Between Naps on the MSLT AND MWT***
Kasravi N., Legault G., Jewell D., Murray B.J.

P125 ***Reliability of the French Version of the Occupational Fatigue Exhaustion Recovery (OFER) Scale in College Students with Part-Time Jobs***
Laberge L., Ledoux É., Perron M., Bourdouxhe M., Gaudreault M., Laberge, M., Arbour N., Hébert M., Winwood P.C., Veillette S

P126 ***Is Actigraphy Able to Detect Wakefulness During Sleep?***
Paquet J., Kawinska A., Carrier J.

P127 ***Development and Validation of a Novel Scale for Measuring Sleepiness and Fatigue Concurrently***
Shen J., Chatoo K., Streiner D.L., Chung C.A., Huterer N., Shapiro C.M.

Paediatrics

P128 ***Do Better Nights Mean Better Days? Impact of a Behavioural Sleep Treatment Program on Health-Related Quality of Life in School-Aged Children***
Woodford K.M., Corkum P., McGrath P.

P129 ***Sleep Problems and Performance on a New Test of Attention in Children with ADHD***
Corkum P., Mullane J.C., Bower J.L

P130 ***Sleep Difficulties and Behavioral Problems in Young Children: A Community Study***
Hall W.A., Scher A., Warnock F., Clauson M., Espezel H., Zaidman-Zait A.

P131 ***A Retrospective Review of Sleep Studies in Children with Cleft Palate***
MacLean JE, Fitzsimmons D, Hayward P, Waters K, and Fitzgerald DA

P132 ***Longitudinal Study of Bad Dreams in Preschool Children: Risk and Protective Factors***
Simard V., Nielsen T.A., Tremblay R.E., Boivin M., Montplaisir J.Y.

P133 Award ***Laryngeal Stimulation by an acid Solution in Preterm Lambs During Quiet Sleep***
St-Hilaire M., Samson N., Duvareille C., Praud J-P.

P134 ***Policies Practices and Provisions for Parents Sleeping Overnight with a Hospitalized Child***
Stremler R., Wong, L., Parshuram, C

P135 ***Longitudinal Short Sleep Duration and Risk of Obesity in Early Childhood***
Touchette E., Petit D., Boivin M., Tremblay R., Montplaisir J.

P136 ***Self –Reported Sleep Difficulties in a Community Based Sample of Children and Adolescents with Recurrent Pain***
Vincent N., Chambers C.T., Corkum P., Rusak, B

Aging

P137 Award ***Gender Differences on Topographical Sleep EEG in Midlife***
Frenette S., Paquet, J., Carrier J.

P138 ***Stage 2 Sleep Spindles, Motor Learning and Aging***
Peters K.R., Ray L., Smith V., Smith C

P139 ***Age Differences in the Temproal Organization of Stage 2 Sleep Spindles***
Peters K.R., Vlaskalin M., Ray L., Smith V., Smith C.

Dreams

P140 ***Decreaded Nightmare Frequency and Improved Sleep Quality Following Treatment with Interactive Voice Mail and Cognitive-Behavioral Drawing Techniques***
Alain, G & Nielsen, T.A.

P141 ***Evaluating the Usefulness of a Multidimensional Approach to Attitudes & Beliefs About Dreams***
Charneau-Simard, C., Beaulieu-Prévost, D., Zadra, A

P142 ***Dream Emotion is Associated with Cross-Night Adaptation to Negative Picture Stimuli***
Lara-Carrasco J., Nielsen T.A., Paquette T., Solomonova E., Stenstrom P.

P143 ***Late-Night REM Sleep Rebound is Reduced but Sleep Efficiency is Normal in Subjects with Idiopathic Nightmares***
Nielsen T, Paquette T, Solomonova E

P144 ***Active But Not Passive, Virtual Reality and TV Maze Tasks Produce Bimodal Patterns of Dream Incorporation Over 14 Days***
Nielsen T, Saucier S, Stenstrom P, Solomonova E, Lara-Carrasco J

P145 ***First-Night Eye Movement Density is Lower in Subjects With vs Without Nightmares*** Nielsen T, Solomonova E, Lara-Carrasco J, Paquette T

P146 ***Increased REM Latency and NREM/REM Cycle Length in Idiopathic Nightmare Sufferers***
Paquette T., Nielsen, T.A., Stenstrom P., Lara-Carrasco J., Solomonova E., Bourdeau, G., Dallaire S

P147 ***Differential Impact of Two Prospective Log Measures on the Frequency of Nightmares***
Robert, G., Zadra, A

P148 ***The Exploration of Dream's Emotional Content Using Automatic Analysis***
Sabourin C., Nadeau D., De Koninck J., Matwin S., Turney P.D.

P149 ***Sleep Paralysis and Nightmares Are Both Related To Affect Distress***
Solomonova, E., Nielsen, T.A., Stenstrom, P., Frantova, E., Lara-Carrasco, J., Donderi, D., Popova, A

P150 ***Dream Like Quality of Sleep Onset and REM Mentation is Reduced in REM Sleep-Deprived Nightmare Sufferers***
Stenstrom, P., Nielsen T.A., Solomonova E., Lara-Carrasco J., Polotskaia A.

P151 ***Logical Thought and the Detection of Bizarreness in Sleep Mentation***
Stenstrom, P., Nielsen T.A., Lara-Carrasco J., Solomonova E., Osmani A.

Women's health & Gender

P201 ***The Effect of Napping on Mood in Women with Sever Premenstrual Symptoms***
Lamarche L., Driver H.S., De Koninck J.

P202 ***Subjective Alertness and Cognitive Performance Across the Menstrual Cycle***
Shechter A., Boivin D.B

Sleep Disorders: Breathing

P203 ***Symptom Pattern of Sleep-Disorder Complaint Influences Physician Referral***
Bailes S., Baltzan M., Rizzo D., Fichten C., Sully J., Libman E.

P204 ***Can One or More Questionnaires be Used To Detect Sleep Apnea In Sleepy Individuals?***
Chung S.A, Ahmadi N., Shapiro C.M

P205 ***At Home vs In Sleep Laboratory Diagnosis and Treatment of Obstructive Sleep Apnea: Effect on Mood, Anxiety Level and Cognitive Function***
Jokic R., Cotton D., Reid J., Gjevre J., Stiles M., Ward H., Skomro R.

P206 ***Jaw Position During Sleep***
Le Huquet A.L., Christensen J, Fitzpatrick M.F.

P207 ***Cognitive Difficulties in Sleep Apnea Syndrome: Vigilance and Attention are Impaired – A New Comparative Study of Younger And Older Patients***
Mathieu A., Mazza S., Petit D., Décary A., Massicotte-Marquez J., Gosselin N., Malo J., Montplaisir J.Y.

P208 ***Neonatal Caffeinc Has Long-Term Impact On Sleep and Breathing in Freely-Behaving Adult Rats***
Montandon G., Bairam A., Horner R., Kinkead R

P209 ***Differentiation of Cheyne-Stokes Respiration From Obstructive Sleep Apnea By Pattern Recognition On Finger Oximetry***
Sharif Z., McKim D., Dales R., Keays T., Leech J.

P210 ***Clustered Obstructive Apneas Induce Long-Term Facilitation of Upper Airway Motor Outflow in Spontaneously Breathing Rats In-Vivo***
Tadjalli A., Duffin J. and Peever J.H

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P211 Award ***Impaired Gabaergic and Glycinergic Neurotransmission Induces REM-Sleep Behaviour Disorder (RBD) In Transgenic Mice***
Brooks P.L., Tse G. and Peever J.H.

P212 ***Glutamatergic Control of Somatic Motoneurons In Freely-Behaving Rats***
Burgess C., Mir S., Lai D., Siegel J. and Peever J.

P213 ***REM Sleep Behavior Disorder Predicts Cognitive Impairment in Parkinsons's Disease***
Gagnon J.-F., Vendette M., Doyon J., Décary A., Postuma R.B., Massicotte-Marquez J., Panisset M. and Montplaisir J

P214 ***EEG Spectral Analysis in Wakefulness REM and NREM Sleep Following Sport-Related Concussions***
Gosselin N., Lassonde M., Petit D., Leclerc S., Mongrain V., Montplaisir J.

P215 ***Strong Association Between Cyclic Alternating Pattern A1 and SWA in Sleep Bruxers and Control Subjects***
Huynh N.T., Macaluso G.M., Rompré P.H., Guilleminault C., Parrino L., Terzano G.M., Lavigne G.J

P216 ***The Prevalence of Headaches, Neck Pain and Fatigue in Sleep Bruxism***
Khoury S., Huynh N., Rompré P.H., Montplaisir J.Y., Lavigne G.J.

P217 ***Cognitive Impairments In REM Sleep Behavior Disorder Patients***
Massicotte-Marquez J., Décary A., Vendette M., Mathieu A., Petit D., Rompré S., Carrier J., Montplaisir J.Y.

P218 ***Periodic Leg Movements During Sleep Are Associated With Cardiovascular Changes in healthy Normal Subjects***
Pennestri M.H., Lanfranchi P.A., Fradette L., Richard M., Colombo R., Montplaisir J.

P219 ***Somnambulistic Episodes in Sleepwalkers With A High Periodic Leg Movements in Sleep (PLMS) Index are Rarely Associated With PLMS***
Pilon M., Zadra A., & Montplaisir J

P220 ***Does Melatonin Affect The Circadian Variation of RLS Symptoms?***
Whittom S., Dumont M., Adam B., Selmaoui B., Montplaisir J

Sleep Disorders: Insomnia

P221 ***Daytime Consequences of Insomnia: A factor Analysis***
Beaulieu-Bonneau S, Fortier-Brochu E, Ivers H, Morin CM

P222 ***Meta-Analysis of Sleep Changes During Placebo Administration Compared To Untreated Groups in Insomnia Treatment Trials***
Bélanger L., Vallières A., Ivers H., Moreau V., Lavigne G., Morin C.M.

P223 ***Utilization of Hypnotic Medications Among Cancer Patients***
Casault L., Savard J., Simard S., Ivers H

P224 ***Sleep and Fatigue in Individuals with Insomnia***
Fortier-Brochu E., Beaulieu-Bonneau S., Ivers H., Morin C.M.

P225 ***Cardiovascular Changes Associated with Micro-Arousals in Primary Insomniacs and Normal Subjects***
Fradette L., Pennestri MH., Pelletier S., Montplaisir J., Morin C., Lanfranchi PA

P226 ***Incidence and Risk Factors of Insomnia in a Population-Based Sample***
LeBlanc M., Mérette C., Savard J., Morin C.M

P227 ***Development and Dissemination of an Insomnia Clinical Practice Guideline: The Alberta Experience***
Samuels C.H. MacFarlane J., Dirks-Farley S.,Fidler H.,Moorehouse R., Fraser K

P228 ***Cerebral Asymmetry in Sleep: Relation with BDI and BAI Scores in Chronic Primary Insomnia***
St-Jean G., Bastien C.H

P229 ***Mechanisms Explaining The Effect of Cognitive-Behavioural Therapy For Chronic Insomnia Comorbid with Breast Cancer***
Tremblay V., Savard J., Ivers H., Simard S

P230 ***Chronic Primary Insomnia: Relation Between Quality of Sleep and Cognitive Evoked Potentials Measures***
Turcotte, I., St-Jean, G. and Bastien, C.H.

P231 ***Perceived Health and Psychological Consequences Associated with Work Schedules for Regular Night and Rotating Shift Workers: The Moderating Effect of Insomnia Symptoms*** Vallières A., Morin C.M., Ivers H., LeBlanc M

Sleep in Medical Disorders

P232 ***Insomnia and Increased REM Sleep Pressure in a Rat Model of Post Myocardial Infarction Depression***
Bah T.M., Wann B.P., Chebli M, Le Marec, N, Rousseau G. and Godbout R

P233 ***Evolution of Sleep Disturbances and Other Concomitant Symptoms Following Mild Traumatic Brain injury: A Preliminary Report***
Chaput G, Manzini C, Denis R, Demers A, Giguère J-F, Lavigne G

P234 ***Does Having Sleep Apnea Preclude The Diagnosis of Chronic Fatigue Syndrome?***
Creti L, Rizzo D, Bailes S, Baltzan M, Fichten C, Libman E

P235 ***How To Make Insomnia Interventions Available To People With Cancer: Insights From Patients***
Davidson, J.R., Feldman-Stewart, D., Brennenstuhl, S., Ram, S.

P236 ***Sleepiness And Depression Contribute To Fatigue In Systemic Lupus Erythematosus (SLE)***
Iaboni A., Ibanez D., Gladman D.D., Urowitz M.B., Moldofsky H

P237 ***Tobacco Smoking, Sleep and Clinical Symptoms In Patients With Schizophrenia***
Poulin J., Chouinard S., Guillem F., Stip E., Godbout R.

P238 ***Medication, Perceptions Of Sleep And Polysomnography Results***
Rizzo, D., Bailes, S., Sully, J., Baltzan, M., Fichten, C., Creti, L., Libman, E

P239 ***The Relationship Between Insomnia and Depression In Prostate Cancer Patients Receiving Androgen-Deprevation Therapy***
Savard J., Ivers H., Simard S

P240 ***Insomnia and Androgen-Deprevation Therapy for Prostate Cancer***
Savard J., Ivers H., Simard S

P241 ***REM Sleep is Reduced on an Ultra-Rapid Sleep-Wake Cycle Procedure in Bipolar Affective Disorder***
Waddington Lamont E., Lalinec M., Beaulieu S., Boivin D.

Abrégés scientifiques / Abstracts

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Effects of Hypercapnia and Hypoxia on Non-Nutritive Swallowing in Newborn Lambs

Duvareille C., St-Hilaire M., Samson N., Micheau P.[1], Bournival V., Praud JP. Neonatal Respiratory Research Unit, Depts of Pediatrics and Physiology; 1: Dept of Mechanical Engineering; University of Sherbrooke, Quebec.

Non-nutritive swallowing (NNS) is essential for clearing upper airway secretions and gastro-pharyngeal reflux in the neonatal period. Precise NNS-breathing coordination is essential to prevent apneas and aspirations probably implicate in few cases of sudden infant death syndrome during sleep, and to ensure a normal maturation of both functions. Common respiratory challenges, such as hypercapnia or hypoxia can potentially disrupt that coordination. The aim of the present study was to investigate the effect of hypercapnia and hypoxia on sleep architecture, NNS frequency and on the coordination between NNS and the respiratory cycle in newborn lambs in each state of alertness.

METHODS: nine lambs were chronically instrumented for recording states of alertness, respiratory movements, thyroarytenoid muscle (a glottal constrictor) activity and electrocardiogram. The non-sedated lambs underwent 3 polysomnographic recordings while in a plexiglas chamber flushed with air (control, FiO_2 0.21), hypercapnic (FiO_2 0.21, $FiCO_2$ 0.05) and a hypoxic mixture (FiO_2 0.10). The three recordings were performed randomly, using a custom-made radiotelemetry system.

RESULTS:

	State	Sleep Architecture	RR	NNS freq	iNNS/eNNS	ieNNS/eiNNS
Normoxia (Nx)	W	44 ± 8	63 ± 21	53 ± 14	17±10/3±2	22±12/6±5
	QS	43 ± 4	60 ± 22	29 ± 10	11±7/2±1	11±8/4±5
	AS	12 ± 4	61 ± 10	92 ± 27	39±25/6±3	33±19/9±6
Hypercapnia (Hc)	W	**53 ± 9***	**90 ± 30***	**74 ± 32***	14±12/5±7	**44±20***/7±7
	QS	**38 ± 4***	**89 ± 29***	**42 ± 21***	**6±6***/3±5	**29±19***/3±4
	AS	9 ± 5	**77 ± 17***	113 ± 47	25±18/4±6	**73±34***/5±5
Hypoxia (Hx)	W	42 ± 7.5†	**79 ± 22***	46 ± 17†	15±12/4±2	20±10†/3±3†
	QS	**49 ± 6 *†**	**77 ± 26***	23 ± 11†	**6±6***/2±2	13±9†/2±2
	AS	9 ± 4	**77 ± 12***	89 ± 43†	37±26/6±8	37±26†/**3±4***

Sleep architecture (%) time in each state of alertness; RR: respiratory rate (h^{-1}); NNS frequency (h^{-1}); iNNS/eNNS: NNS frequency occurring in inspiration or expiration (h^{-1}); ieNNS/eiNNS: NNS frequency occurring at the transition between inspiration-expiration or expiration-inspiration (h^{-1}); W: wakefulness; QS: quiet sleep; AS: active sleep. *: $p < 0.05$, Hx and Hc *vs.* Nx, † $p < 0.05$, Hx *vs*. Hc.

CONCLUSION: Our results suggest that contrarily to hypoxia, hypercapnia stimulates NNS. This increase in NNS is observed in the three states of alertness and is specific to the ie-type NNS. Overall, no changes in NNS frequency were observed in hypoxia. The mechanisms and potential consequences of such observations on aspirations and apneas, and on swallowing maturation, need to be addressed in further studies.

Supported by the Canadian Institutes of Health Research (NRF15558), the Fonds de la Recherche en Santé du Québec and the Foundation for Research into Children's Diseases.

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Special Charateristics Surrounding the Spontaneous K-Complex in Good Sleepers

Forget D., Morin C.M. & Bastien C.H.
École de psychologie, Université Laval, Québec, QC, Canada

Objectives: The spontaneous k-complex (KC) is a hallmark of sleep and is visually identifiable in stage 2 sleep. Although traditionally perceived as an arousal response, recent studies rather tend to support a "sleep-protective" role of the KC. The objective of the present study is to investigate the role of the KC in good sleepers by examining and comparing the spectral features of EEG segments prior to and following their presence.
Methods: Ten adults (mean age = 43.4 years; range = 25 to 55 years) without insomnia complaints underwent four consecutive nights of PSG recording. The present data are based on the third recording night. KCs were scored during continuous (> 5 minutes) early stage 2 sleep (2E) and late stage 2 sleep (2L) using R&K criteria. Relative spectral power (delta [1-4 Hz], theta [4-8 Hz], alpha [8-12 Hz], sigma [12-14 Hz], beta [14-35 Hz], gamma [35-60 Hz] and omega [60-125 Hz]) was calculated for each 1 second EEG segments prior to (PRE) and following (POST) all identified KCs.
Results: A total of 1300 KCs were scored and analyzed. Average KC density was 1.00 KC/minute. Repeated measure ANOVAs showed significant changes in relative EEG power from PRE to POST: relative delta power increased (d = 1.72), whereas relative power for theta, alpha, sigma and beta bands decreased following KCs (d = -1.17; range = -0.59 [alpha] to -1.40 [theta]). No differences were found between 2E and 2L.
Conclusions: An increase in relative delta power combined with a decrease in relative power of theta to beta bands suggests that EEG activity is lower following KC than at baseline level prior to KC. Thus, KC could be involved in the lowering of EEG activity, therefore temporarily deepening the sleep state in good sleepers. Changes in EEG relative power surrounding KC were constant throughout the whole night for stage 2 sleep. These results support the "sleep-protective" role hypothesis for the KC.

Research supported by the Canadian Institutes of Health Research (# 49500) to C. H. Bastien.

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A Non-Invasive Cerebral Temperature Sensor for the Assesment of Circadian Rhythms

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Dumont G.[3], Boivin D.B.[1]

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Objectives: The human circadian phase and amplitude is commonly assessed with the core body temperature cycle in the laboratory. However, in ambulatory conditions, invasive measurements are unsuitable. To overcome this issue, we tested a newly developed cerebral temperature sensor which is based on the idea of zero-heat flux regulation. In this study, we compared the assessment of circadian phase and amplitude drawn from a rectal temperature (CBT) sensor and from a non-invasive cerebral temperature (BT) sensor using an ultra-rapid sleep-wake cycle procedure (URSW).
Methods: Three healthy young women (mean age ± SD: 27.0 ± 2.5 years), with regular menstrual cycles (mean length ± SD: 28.1 ± 1.0 day) were studied individually for 5 days in time isolation for a total of four visits (one subjects came twice during each phases of the menstrual cycle). After an 8 hour baseline sleep episode, participants underwent an URSW. This procedure consists of 60 minutes waking episodes in dim light (<10 lux) alternating with 60 minutes nap episodes in total darkness. During the procedure, participants remained in a semi-recumbent position and meals were replaced by balanced snacks during the waking episodes. The BT and CBT were monitored throughout the procedure. A dual harmonic regression model was used to assess circadian phase and amplitude from both body temperature series.
Results: The dual harmonic regression revealed a significant variation of CBT and BT (95% CI not including the zero axis). Comparison of circadian phases and amplitudes revealed no significant difference between BT and CBT (p=0.85 and p=0.86, respectively). The mean value of BT across all waking episodes was significantly lower than that of CBT (minus 0.23°C, p=0.034), while no significant difference was found for the mean values of CBT and BT during all nap episodes.
Conclusion: This study suggests that BT can be an acceptable alternative to CBT for the assessment of circadian phase and amplitude. While the BT is lower than CBT during waking episodes, it is not the case during napping episodes. This finding suggests a different masking effect of sleep on temperature levels recorded rectally versus by our brain sensor.

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Effect of an Intervention on Melatonin Production in Police Officers Working Rotating Shifts

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Objectives: In a prior study we looked at the effect of an intervention based on the timing of light/darkness in permanent night shift workers. This study aims at determining the effect of a similar intervention on melatonin secretion in rotating shift workers.

Methods: Eight police officers (4 per group) were studied in the laboratory before and after a series of 7 consecutive night shifts (8 to 8.5 hours). Saliva melatonin was sampled every 1 to 2 hours over a 24 hour period. Participants in the "intervention" group exposed themselves to bright light (Litebook 1.2, Litebook Company Ltd.) for the first 6 hours of their shifts. They wore orange-tinted glasses from sunrise until bedtime (Blue-blockers, Telemedoptique Inc.). An 8 hour daytime sleep episode was planned 2 hours after the end of their shift. Participants in the "control" group did not receive any instructions regarding their light exposure and sleep schedule.

Results: One factor ANOVA (factor: group) was used to analyze the percentage of the 24 hour AUC of salivary melatonin that occurred during the work period. This ANOVA revealed a significant difference ($F(1)= 18,1851; p=0.0053$), with the "intervention" group having lower values than the "control" group.

Conclusions: Our preliminary results indicate a better circadian adjustment to working nights of participants exposed to our intervention. "Control" participants secreted more melatonin during their work period then participants from the "intervention" group.

Support: This study was supported by the Institut de recherche Robert-Sauvé en santé et en sécurité du travail (IRSST). GT is supported by a fellowship from the IRSST. DBB is supported by a career award from the Canadian Institutes of Health Research (CIHR). Equipment grant from Litebook Company Ltd..

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Emergence of Physiological Rhythmicity in Term and Preterm Neonates in a Intensive Care Unit

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Objective: Biological rhythmicity, particularly circadian rhythmicity, is considered to be a key mechanism in the maintenance of physiological function. Very little is known, however, about biological rhythmicity pattern in preterm and term neonates in neonatal intensive care units (NICU). In this study, we investigated whether term and preterm neonates admitted to NICU exhibit biological rhythmicity during the neonatal period.
Methods: Twenty-four-hour continuous recording of four physiological variables (heart rate: HR recorded by electrocardiogram; pulse rate: PR recorded by pulse oxymetry; respiratory rate: RR; and oxygen saturation of pulse oxymetry: SpO_2) was conducted on 187 neonates in NICU during 0-21 days of postnatal age (PNA). Rhythmicity was analyzed by spectral analysis. The Fisher test was performed to test the statistical significance of the cycles. The amplitudes and amplitude indexes for each dominant cycle were calculated.
Results: Circadian cycles were observed among 23.8% neonates in HR, 20% in PR, 27.8% in RR and 16% in SpO_2 in 0-3days of PNA. Percentages of circadian cycles were the highest (40%) at <28 wks of gestational age (GA), decreasing with GA, and the lowest (14.3%) at >= 37 wks GA within 3 days of PNA in PR and were decreased in the later PNA. An increase of the amplitude with GA was observed in PR, and significant group differences were present in all periods. Amplitudes and amplitude indexes were positively correlated with postconceptional age (PCA) in PR ($p < 0.001$). Among clinical parameters, oxygen administration showed significant association ($p < 0.05$) with circadian rhythms of PR in the first 3 days of life.
Conclusion: Whereas circadian rhythmicity in neonates may result from maternal influence, the increase of amplitude indexes in PR with PCA may be related to physiological maturity. Further studies are needed to elucidate the effect of oxygenation on physiological rhythmicity in neonates.

Table: Descriptive profiles for significant cycles of HR, PR, RR and SpO_2.

Period (postnatal days)		Period 1(0 - 3)	Period 2(4 - 6)	Period 3(7 - 13)	Period 4(14 - 21)
Significant cycle*	HR	80 (98)	64 (100)	89 (98)	67 (100)
n (%)	PR	100 (99)	87 (99)	104 (98.1)	83 (99)
	RR	90 (91)	84 (99)	97 (93.3)	79 (94)
	SpO2	94 (91.3)	86 (97)	103 (97)	78 (92)
Circadian cycle**	HR	19 (23.8)	11 (17.2)	20 (22.5)	13(19.4)
n (%)	PR	20 (20)	16 (18.4)	20 (19.2)	16 (19.3)
	RR	25 (27.8)	28 (33.3)	21 (21.6)	11 (13.9)
	SpO2	15 (16)	10 (11.6)	17 (16.5)	15 (19.2)

* significant cycles in all eligible samples, and ** circadian cycles in significant cycles.

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Age-Related Effects of 200MG of Caffeine on Daytime Recovery Sleep

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We reported that caffeine produces almost similar effects on nocturnal sleep in young and middle-aged subjects. Recently, we have shown that the effects of caffeine on sleep are more prominent when caffeine is taken during the night before daytime recovery sleep than in the evening before nocturnal sleep. We aimed to evaluate the effects of caffeine on daytime recovery sleep after a 25-hour sleep deprivation in young (Y) and middle-aged (MA) subjects. Twenty-four subjects participated in both caffeine (200 mg) and placebo (lactose) conditions in a double-blind crossover design (12 Y, 20-40 y.; 12 MA, 40-60 y.; one month between conditions). For each condition, subjects came for a baseline night after which they were sleep-deprived for one night. Recovery sleep started the next morning after 25 hours of wakefulness. Subjects received a 100 mg capsule of caffeine (or placebo) 3 hours before daytime recovery sleep (around 5AM), and the remaining dose 1 hour before (around 7AM). Three-way ANOVAs (Age, Night, and Condition) were performed. Sleep latency was shorter during daytime recovery sleep than during baseline sleep, but this effect was reduced in the caffeine condition. SWS was higher in young compared to middle-aged subjects, and its total amount increased in daytime recovery sleep compared to baseline. Caffeine reduced SWS rebound during daytime recovery sleep. Sleep efficiency, sleep duration, stage 2 and REM sleep were lower in daytime recovery sleep compared to baseline, and these effects were enhanced by caffeine. Importantly, the reduction of sleep duration and sleep efficiency by caffeine was more prominent in the MA than in the Y. These results support the notion that caffeine highly disrupts daytime sleep recovery, especially in the MA. With increasing numbers of the MA population now facing challenges such as shift work and jet lag, these questions are more than simply of academic interest.

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Clock Gene Expression in Peripheral Blood Mononuclear Cells Following Simulated Night Shift Work

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Objectives: Judicious light and darkness exposure throughout the day can promote the appropriate alignment of endogenous hormonal rhythms to night shift work. However, the synchronization of human peripheral circadian oscillators to shifted sleep-wake schedules is currently unknown. We evaluated *HPER2* expression in peripheral blood mononuclear cells (PBMCs) with respect to the simultaneous resetting of plasma melatonin and cortisol rhythms throughout simulated night shift work.
Methods: Five healthy candidates (4 male, 1 female in follicular phase) aged (mean ±SD) 24.9 ± 4.8 years maintained stable sleep and meal schedules before the study start. Upon admission to the laboratory, sleep/wake schedules were delayed by 10 hours to simulate nighttime "work". The light intervention included exposure to full-spectrum white light of (mean ±SEM) 6,036 ± 326 lux during 8-hour night shifts and dim light exposure after each night shift with the use of sunglasses (5% visual light transmission, gray filter, Litebook, Medicine Hat, AB). *HPER2* expression in PBMCs, and plasma melatonin and cortisol concentration were estimated from 24-hour blood sampling periods performed before and after nine simulated night shifts. The expression of *HPER2* in isolated PBMCs was determined relative to *HCDK4* via real-time PCR.
Results: Following nine simulated night shifts, plasma melatonin and plasma cortisol rhythms were adjusted to the shifted schedule given that peak values occurred 4.7 ± 0.2 hours and 11.5 ± 0.7 hours after the start of the imposed daytime sleep/darkness period, respectively. Dual-harmonic regression analyses revealed that all participants demonstrated significant circadian rhythmicity in *HPER2* expression. Significant inter-individual variability was present in the initial phase of clock gene expression. Following night shifts, peak *HPER2* expression occurred 8.7 ± 0.7 hours after the start of the imposed daytime sleep/darkness period, thus at the beginning of the active period on the shifted schedule. The calculated phase angle in the final condition was less variable after the period of light intervention and revealed that clock gene expression was in a conventional temporal relationship with the sleep/wake cycle, despite the 10-hour delay in the imposed sleep schedule.
Conclusions: This is the first report to describe the response of peripheral circadian oscillators in PBMCs to an atypical sleep-wake schedule. Considering recent evidence implicating peripheral oscillators and tissue function, this line of investigation has important implications for understanding the health consequences of the persistent circadian misalignment experienced by many night shift workers.
Support: *Fonds de Recherche en Santé du Québec,* Canadian Institutes of Health Research.

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Clock Gene Expression in Human Peripheral Blood Mononuclear Cells Throughout an Uninterrupted 72-Hour Period

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Objectives: Circadian rhythmicity in the levels of clock gene expression underlies the function of the central circadian pacemaker of the suprachiasmatic nucleus. Recent evidence of a circadian variation in levels of clock gene mRNA from peripheral blood mononuclear cells (PBMCs) suggests the presence of functional circadian oscillators in these cells. We used an uninterrupted 72-hour sampling period to compare the expression of peripheral circadian oscillators in PBMCs in the presence of a habitual sleep/wake cycle (LD) and under constant routine (CR) conditions.
Methods: Six healthy men (n=4) and women (n=2, follicular phase) aged 18-30 years (mean age ± SEM: 23.7 ± 1.6 years) maintained a regular 8-hour sleep episode for two weeks prior to the study. Repeated whole blood samples were drawn from an indwelling catheter during a 72-hour period including 40 hours of LD where subjects were exposed to 144 ± 28 lux of full-spectrum light during daytime wake periods and slept in darkness, and a 32-hour CR of sustained wakefulness and limited activity under dim light. Real-time PCR was used to assess *HPER1* expression in PBMCs isolated from whole blood samples every ~120 minutes. Plasma melatonin sampled every ~60 minutes was used as a marker of the central circadian pacemaker.
Results: Dual-harmonic regression analyses revealed that all six subjects displayed a statistically significant expression of *HPER1* under LD conditions and mean peak expression occurred (±SEM) 2.6 ±1.8 hours after awakening. During the CR, four of six participants demonstrated significant amplitudes of expression with peak expression occurring 1.8 ± 2.2 hours after the time of habitual awakening. The pattern of clock gene expression in PBMCs was comparable under LD and CR conditions (p=0.2).
Conclusions: These results demonstrate that the pattern of *HPER1* expression in PBMCs can be evaluated over extended periods. Moreover, the expression of *HPER1* in PBMCs is comparable in the presence of a habitual sleep/wake cycle and under constant conditions. Despite controlled investigative conditions, the phase of clock gene expression in PBMCs remains more variable than markers of central circadian pacemaker.
Support: *Fonds de Recherche en Santé du Québec,* Canadian Institutes of Health Research.

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Evidence that Wearing Blue-Blockers after the Night Shift Improves Day Time Sleep

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Objectives: Night shift workers complain of disturbed sleep during the day. This could partly be the result of morning bright light exposure during the commute home which would tend to maintain the body clock synchronized to a daytime schedule. But, the biological clock is most sensitive to the blue-green portion of the visible spectrum (400-540 nm). Our aim was to test if blocking all these wavelengths (blue-blockers) which should impede the biological effect of light in the morning, could improve daytime sleep of permanent shift workers.
Methods: 8 permanent night shift workers (2H, 6F, aged between 32-56) of a Canada Post distribution center of Quebec City completed the study during summer and 20 others (11H, 9F, aged between 24-55) during winter. Their day-sleep time, efficacy and fragmentation were analysed over a four weeks period with a wrist activity monitor (Actiwatch-L). The first two weeks served as baseline. The two remaining experimental weeks, workers had to wear blue-blockers either before leaving the workplace at the end of their shift (7:00AM) for the summer group, or 2 hours before the end of the nightshift for the winter group (since there is barely no light outside in the morning in winter). All had to wear the glasses when outside during the day until 4:00PM.
Results: The average duration of time spent in bed was not different between conditions. When wearing the glasses, workers slept on average 32 ±29 ($p=0.02$) and 34 ±60 ($p=0.02$) minutes per day, increased their sleep efficacy by 1.95 ±2.17% ($p=0.04$) and 4.56 ±6.1% ($p=0.004$) and lowered their sleep fragmentation by 1.74 ±1.36% ($p=0.01$) and 4.22 ±9.16% ($p=0.05$) in summer and winter group respectively.
Conclusions: Blue-blockers seem to improve daytime sleep of our workers by an average of 30 min/day. Polysomnographic recordings will be needed to confirm the latter observation.

Key words: Shiftwork, Biological clock, Wavelengths, Sleep, Actigraphy.

Funding support : FRSQ, IRSST

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Effects of Ovariectomy and Estrogen Replacment on Spontaneous Sleep and Recovery Sleep after Sleep Deprivation in Rats

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Objectives: The influence of ovarian hormones on sleep-wake states and sleep homeostasis is not well characterized, although there is evidence that hormone withdrawal (e.g., during menopause) alters sleep characteristics. We investigated the effects of ovariectomy and of estrogen (E) replacement on spontaneous sleep, and on recovery sleep after total sleep deprivation (SD) by "gentle handling" for 6 h in female rats. Intact male rats were also studied for comparison.
Methods: Adult female and male Wistar rats were housed under a 12:12 light:dark cycle. Female rats were ovariectomized (OVX) and implanted subcutaneously with silastic capsules containing oil vehicle (Oil), 10.5 µg of 17β E to mimic diestrus levels of E (Low E), or 60 µg of 17β E to mimic proestrus levels of E (High E). Male rats were intact but implanted with oil-filled silastic capsules. All animals were also implanted with EEG and EMG electrodes. Two weeks after surgery, EEG/EMG were recorded during a 24 h baseline period, followed by 6 h SD in the second half of the light phase, and an 18 h recovery period beginning at the onset of the next dark phase.
Results: During the 24 h baseline recording, Low E and High E OVX rats spent less time than those in the Oil group in both slow wave sleep (SWS; 39, 37 and 44%, respectively) and rapid eye movement (REM) sleep (9, 8 and 12%, respectively). During the 18 h recovery period after SD, the three groups showed similar amounts of SWS (46 to 50%) and REM sleep (11 to 12%). The rate at which lost SWS recovered was also similar across groups. OVX rats treated with Oil, however, recovered very little of their lost REM sleep, and the rate of recovery of REM sleep was markedly reduced in this period, compared to Low E and High E rats (4 vs. 89, 98%, respectively). Male rats resembled E-treated females in terms of baseline sleep parameters and in the amount and rate of recovery of SWS and REM sleep after SD.
Conclusions: Ovariectomy without hormone replacement increased levels of baseline SWS and REM sleep in female rats relative to those receiving E replacement and to intact males. It also decreased the rate of recovery of REM sleep, but not SWS, during the 18 h recovery period after 6 h SD. Collectively, the results indicate that the presence or absence of E can affect sleep regulation and REM sleep homeostasis in female rats. We are also examining the effects of progesterone with or without E on sleep and sleep homeostasis and the effects of both hormones on EEG power spectra during spontaneous and recovery sleep.
Funding Support: CIHR (MOP 67085)

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The Effects of Sleepiness, Incentive and Distraction on the P300 and Contingent Negative Variation (CNV)

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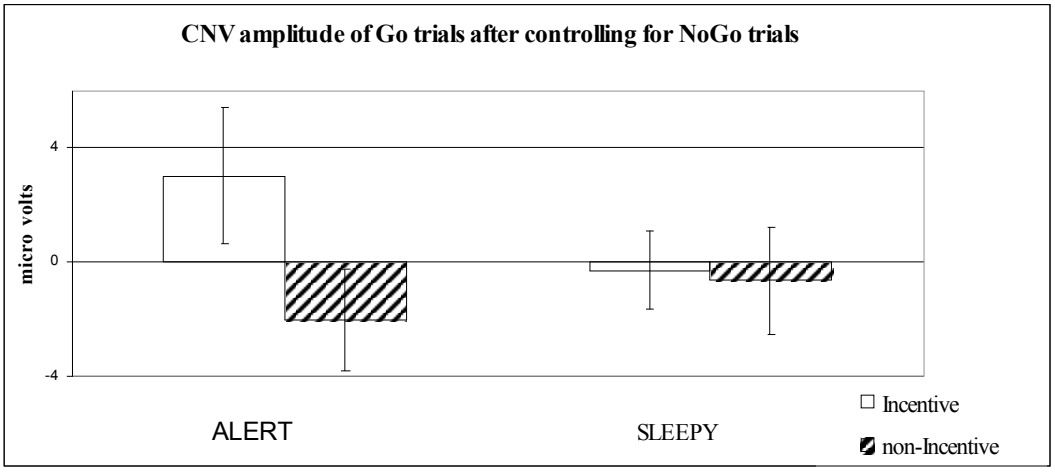
Objectives: To investigate the effect of sleepiness, distraction and incentive on the P300, CNV and behavioural performance.
Methods: Participants (17 F) completed an auditory oddball task, with and without a distracting secondary task and a Go/NoGo CNV, with and without financial incentives for fast responses under alert and sleepy conditions (awake 3 and 20 hours respectively).
Results: Response times were faster in the incentive, and slower in the distraction conditions, but not affected by sleepiness. The distracting task decreased the P300 amplitude without an interaction with sleepiness; however, the P300 latency was increased more in the sleepy/distracted than alert/distracted condition. In the incentive condition of the Go/NoGo task the P300 amplitude to the first stimulus in the Go trials was larger than for NoGo trials; however, without incentives this distinction was not maintained. After controlling for NoGo trials, CNV amplitudes were larger during the incentive trials when alert but not when sleepy (see figure).
Conclusions: The P300 latency increase during the sleepy/distracted condition indicates that processing efficiency may be considerably reduced in multitask situations when tired. The P300 and CNV amplitudes (electrocortical correlates of attention) are more affected by incentives when alert. These effects indicate a lowered ability to distinguish between important versus unimportant information when tired and occur in the absence of any change in response times attributable to sleepiness. Therefore, even though simple behaviours may appear normal during sleepiness, cortical functioning is already impaired. This has serious implications for people required to work extended hours where sustained attention, multitasking, and the ability to distinguish between relevant and irrelevant information is required. This work supported by an NSERC grant (#122222) to SJS. There are no conflicts of interest. Portions of this work were presented at the Society for Psychophysiological Research 2005 and 2006.



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Effects of Sleep Deprivation, Attention Load and Visual Information on Postural Control in Young Healthy Adults (Preliminary Results)

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Introduction: Postural control (PC), even in simple quiet standing, seems to require attention. Because of its effect on attention, sleep deprivation (SD) might decrease attentional resources available for PC. Recent results have suggested that SD may affect PC directly, or may impinge on PC only when attentional resources are mobilized by a concurrent cognitive task. Moreover, by decreasing attentional resources, SD may also alter integration of visual information in PC. The aim of this study was to determine how sleep deprivation, restricted attentional resources and withdrawal of visual input intermingle to influence PC.

Methods: Six young healthy adults (mean age=24,2±1,8) performed quiet standing on a force plate in two counterbalanced sleep conditions: after a night of sleep (baseline) and after 25h of SD. In both sleep conditions, center of pressure (CoP) displacements were measured 2h after habitual wake time in six conditions: eyes open and eyes closed while doing an interference task, a control task, and no task. T-tests were executed on CoP range and speed with a significance threshold of 0.05.

Results: In our baseline sleep condition, the absence of visual information increased CoP range in the anterio-posterior (AP) direction (p=0.013), whereas the interference task increased medio-lateral (ML) CoP speed (p=0.045). While no significant difference between sleep and SD conditions was found when the control task was done with closed eyes, SD decreased ML CoP speed when subjects did the interference task without visual input (p=0.018).

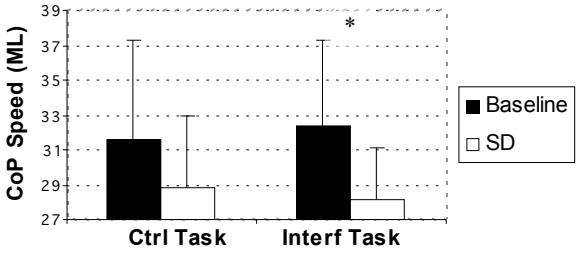

Fig1. ML CoP speed for closed eyes control task and interference task conditions in both sleep conditions.

Conclusion: SD interfered with postural sway in the most challenging condition. The central nervous system has been shown to prevent disrupted motor performance by increasing muscle stiffness when exposed to stressors. Therefore, under SD, when attentional resources are mobilized elsewhere and visual information is absent, stiffness could be increased to reduce sway speed.

This study was supported by the CIHR, the FQRNT and the IRSST.

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Too Much of a Good Thing? REM Sleep Theta Activity Predicts Two-Way Active Avoidance (TWAA) Performance and Increases Prior to Maladaptive Learning in Rats

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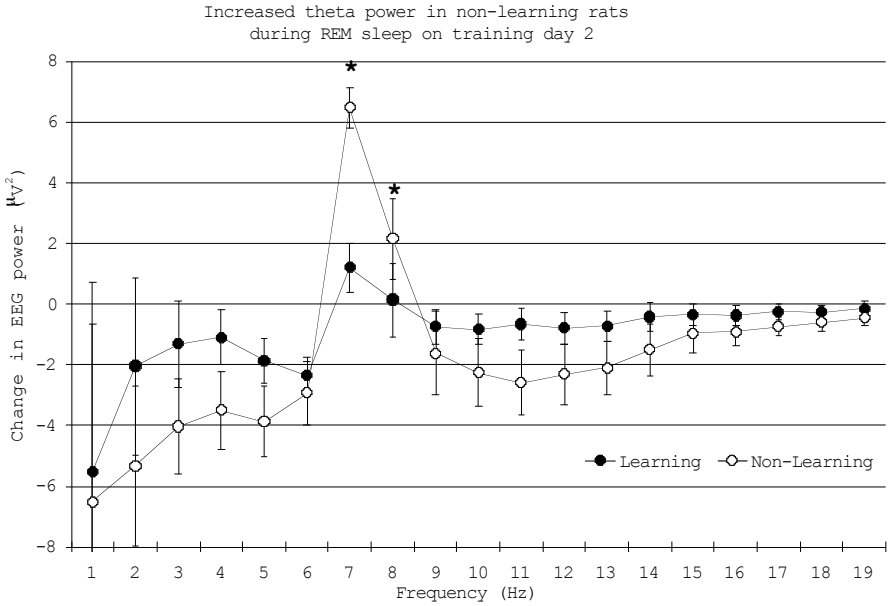
REM sleep increases following learning in the TWAA task during specific time periods termed REM sleep windows. Hippocampal theta predominates REM sleep. Theta rhythms are considered to be a mechanism for inducing hippocampal long-term potentiation. This study investigated if theta during REM is related to memory consolidation.
Methods: Twenty male Sprague-Dawley rats (250-300g) were implanted with four EEG and two EMG electrodes. After recovery, 3 days of acclimatization, and 24 hours of baseline recording, animals were trained on the TWAA task for 100 trials (50 trials/day) from 9-10AM and re-tested for 25 trials on day 3. EEG was recorded for 23 hours after training on both training day 1 (TD1) and TD2. Rats in the learning group (LG) (n=10) avoided the footshock on 70% of the last 20 test trails. The remaining rats (n=10) were assigned to the non-learning group (NLG).
Results: The LG had an increase in correct avoidances over 5 blocks of 25 trials ($F(4,36)=9.54$, $p< 0.00001$). Surprisingly, the NLG had significantly more failures to cross on the last block of trials ($Z(19)=2.12$, $p=0.034$). TWAA performance was negatively correlated with theta from hr 17-20 on the baseline day during REM sleep ($r(17)=-0.81$, $p<0.005$) and wake ($r(17)=-0.77$, $p<0.005$), but not during SWS. REM theta increased during hr 17-20 on TD2 in the NLG ($F(1,16)=8.88$, $p=0.009$) but not on the baseline or TD1, or during SWS or wake.
Conclusions: Theta during hr 17-20 on the baseline day predicted performance, and increased in hr 17-20 on TD2 for non-learning rats. Surprisingly, the NLG began to fail to escape from the footshock on the test day after the increase in theta was observed. Failures to cross may be indicative of learned-helplessness. The results suggest that theta was a good predictor of maladaptive learning and increased prior to the development of escape failures.

Supported by grants from the Natural Sciences and Engineering Research Council of Canada to CTS and RJB



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REM Sleep Duartion Predicts Two-Way Shuttle Active Avoidance (TWAA) Performance and Increases 17-20 Hours Following Learning in Rats

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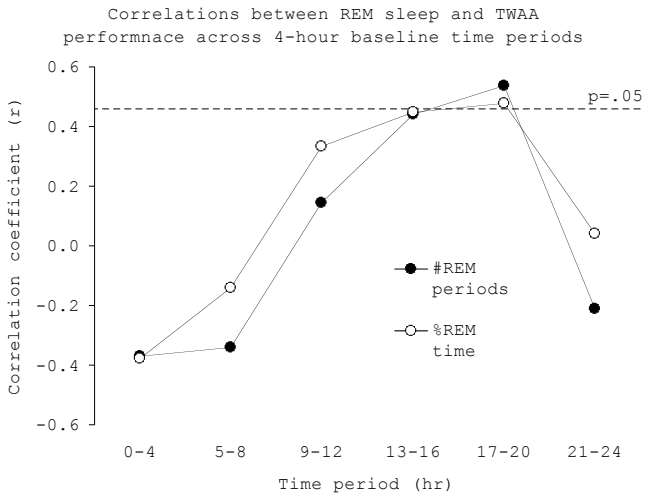
REM sleep increases following learning in the TWAA task. Subsequent memory impairment is observed if REM sleep deprivation is applied during periods of increased REM. These post-training intervals have been termed REM sleep windows. Little is known about the relation between baseline sleep characteristics and learning in rats. Human studies have demonstrated that sleep parameters can predict learning potential as measured by IQ tests. This study investigated the ability to predict memory performance from individual differences in sleep in rats.
Methods: Twenty male Sprague-Dawley rats (250-300g) were implanted with four EEG and two EMG electrodes. After recovery, 3 days of acclimatization, and 24 hours of baseline recording, animals were trained on the TWAA task for 100 trials (50 trials/day) from 9-10AM and re-tested for 25 trials on day 3. EEG was recorded for 23 hours after training on both training day 1 (TD1) and TD2. Rats in the learning group (LG) (n=10) avoided the footshock on 70% of the last 20 test trails. The remaining rats (n=10) were assigned to the non-learning group (NLG).
Results: The LG had an increase in correct avoidances over 5 blocks of 25 trials ($F(4,36)=9.54$, $p<0.00001$). There was no change in correct avoidances in the NLG. TWAA performance correlated with the %REM sleep from hr 17-20 of the 24-hr period prior to training ($r(15)=.48$, $p=0.05$) and with the number of REM periods ($r(15)=.54$, $p=.026$). The %REM sleep was higher in the LG ($F(5,85)=3.21$, $p=0.01$) on TD1 during hr 17-20 following training (post-hoc $t(17)=3.21$, $p=0.005$).
Conclusions: REM sleep during hr 17-20 on the baseline day predicted TWAA performance. Learning-dependent increases in REM sleep were observed when REM sleep already predominated. Results suggest that the duration of REM sleep is a good predictor of learning and increased in a learning-dependent manner during specific post-training windows of time.

Supported by grants from the Natural Sciences and Engineering Research Council of Canada to CTS and RJB.



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Effects of CPAP Treatment of Obstructive Sleep Apnea on Daily Functions, Mood and Quality of Life

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Introduction: Obstructive sleep apnea (OSA) is characterized by disrupted breathing during sleep, leading to hypoxemia and fragmented sleep at night, daytime sleepiness, and changes in cognition and mood. Here, we report preliminary analyses of the effects of CPAP treatment on sleepiness and daily functions, and relationships among sleep quality, daytime sleepiness, daily functions, mood, and quality of life. We hypothesized that CPAP treatment would improve sleep quality and sleepiness, and that post-treatment quality of sleep and residual sleepiness would be related to functioning.

Methods: Twenty-four patients with moderate to severe OSA and compliant on CPAP treatment for at least 3 months were studied with an overnight polysomnographic sleep study, measures of sleep quality (Epworth Sleepiness Scale (ESS), Pittsburg Sleep Quality Index (PSQI)), mood (Beck Depression Inventory (BDI), Profiles of Mood States (POMS)), daily functioning (Functional Outcomes of Sleep Questionnaire (FOSQ)), cognition (Cognitive Failure Questionnaire, (CFQ)), and quality of life (Visual Analogue Scale (QOL)). Pre-treatment Apnea Hypopnea Index (AHI), mean oxygen saturation (SaO_2) and ESS, and retrospective ratings on FOSQ and QOL were also available.

Results: CPAP improved ESS ($t(22)=5.062$, $p=0.000$), daily social interactions (FOSQ)($t(17)$, $p=0.03$), and QOL ($t(18)=-6.176$, $p=0.000$). The number of patients with abnormal scores fell from 70% to 25% on ESS and from 67% to 39% on FOSQ (Total) after treatment. Seventy five percent of treated patients reported good sleep (global PSQI >5), and global PSQI was significantly correlated with ESS ($r=0.43$). Correlations between sleep variables and daily functions are presented in Table 1.

Conclusion: CPAP appears effective in improving daytime sleepiness and social interactions, as well as quality of life. Severity of OSA as measured by pre-treatment hypoxemia, was not related to daily function after CPAP treatment. Sleep quality and sleepiness, however, are correlated with daily cognitive errors, reduced daily function, and poor emotional health.

Table 1 Correlations between sleep variables (ESS, PSQI, AHI, SaO_2) and Daily Functions

	CFQ	FOSQ (Total)	FOSQ (Activity)	FOSQ (Social Interactions)	BDI	POMS (Total)
ESS (Post-CPAP)	0.471*	-0.218	-0.252	-0.129	0.445*	0.622**
PSQI (Post-CPAP)	0.198	-0.383	-0.433*	-0.412*	0.189	0.254
AHI(Pre-CPAP)	0.185	0.006	0.120	-0.361	-0.262	-0.174
SaO_2(Pre-CPAP)	0.068	-0.092	-0.104	-0.005	0.147	0.343

*significant at the 0.05 level (2-tailed)
**significant at the 0.01 level (2-tailed)

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Alteration of REM Sleep Duration in Placebo Responders Depending on Evening Placebo Analgesia Manipulations

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Objectives: Pain perception can be modulated in some subjects (Placebo-responders, i.e. 20% or more pain intensity reduction) by heighten expectation of pain relief induced by suggestions of analgesia and sensory conditioning. The aim of this study was to evaluate the differences in sleep architecture between Placebo responders (PR) and Placebo non-responders (PNR), after evening Placebo analgesia manipulations.
Methods: 25 healthy young subjects were exposed to experimental heat pain after an inert cream was applied to the stimulation site on the left or right arm. Participants were told that the cream applied to one site was analgesic (placebo) while the other was a neutral cream (control). In the evening, all subjects were exposed to a conditioning procedure, in which the temperature of stimulation was surreptitiously lowered on the placebo site. The placebo analgesic effect was then evaluated by comparing pain ratings (visual analog scale) to the same stimulus temperatures across stimulation sites. Group 1 (n=12) was tested only in the next morning after reapplying the placebo and neutral cream while group 2 (n=13) was tested both 30 minutes after conditioning and in the next morning. Polysomnographic recording was performed in all subjects overnight.
Results: In group1, PR had reduced REM sleep compared with PNR (14.9% vs 21.47%; $F(1,21)=4.69;p=0.042$), whereas in group2, this difference was absent (22.54% vs 20.82%;ns). Additionally, group1 PR showed elevated stage 2 sleep ($p=0.031$) and reported more pain relief retrospectively ($p=0.022$), compared to group2 PR.
Conclusions: Results suggest that the induction of placebo effects may be associated with changes in sleep architecture. Specific changes observed in Group1 are consistent with previous studies suggesting that non-operant conditioning (here placebo conditioning) is associated with decrease in REM and increased non-REM sleep. Furthermore, increased stage 2 sleep in group1 PR might reflect better sensory learning and enhanced pain relief recall.

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Event-Related Potentials Differentiates the Processes Involved in the Effects of Sleep on Recognition Memory

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Introduction: Sleep is thought to be involved in memory consolidation. This study examined the influence of sleep on event-related potentials (ERPs) indicators of recognition memory following nocturnal sleep and daytime wake.

Methods: Eight participants (9 males) between 18-39yrs old were tested twice in a counterbalanced manner. During the acquisition session, subjects were presented with a series of stimuli (faces) on a computer screen and asked to memorize them for a subsequent test. At the test session, the subjects were asked to recognize the 40 previously learned "Old" items intermixed with 40 "New". Data was analyzed by a 2 sessions (Sleep/Wake) x 2 ERPs (Old/New) x EEG sites (midline/lateral). ERP data were analyzed separately using repeated measure ANOVAs for midline (Fz, Cz, Pz) & lateral regions (inferior frontal: Fp1-2, F7-8; superior frontal: F3-4; FC3-4; parietal: CP3-4, P3-4; anterior temporal: FT7-8, T3-4; posterior temporal: TP7-8, T5-6). This analysis allowed us to verify the "Old/New Memory Effect" (i.e., the amplitude difference between the old and new items) and to determine the ERP processes influenced by sleep.

Results: Participants made significantly more accurate responses following sleep compared to the wake. No significant difference on RTs across session. ANOVA performed within the early time window resulted in a larger anterior negativity following wake. For the N4, ANOVA revealed a posterior memory effect without any interactions involving session. The later time windows revealed two bilateral effects that were larger after sleep compared to wake, i.e. late frontal component (LFC) and late posterior positive component (LPC).

Conclusion: These findings suggest that ERP indices are differentially effect by sleep. The data shows that sleep influences two frontal processes (early interference inhibition and late contextual integration effort) and a later posterior process that represent facilitation of episodic memory retrieval.

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Dual Target Processing During Continuous Sleep Restriction: The Attentional Blink Task

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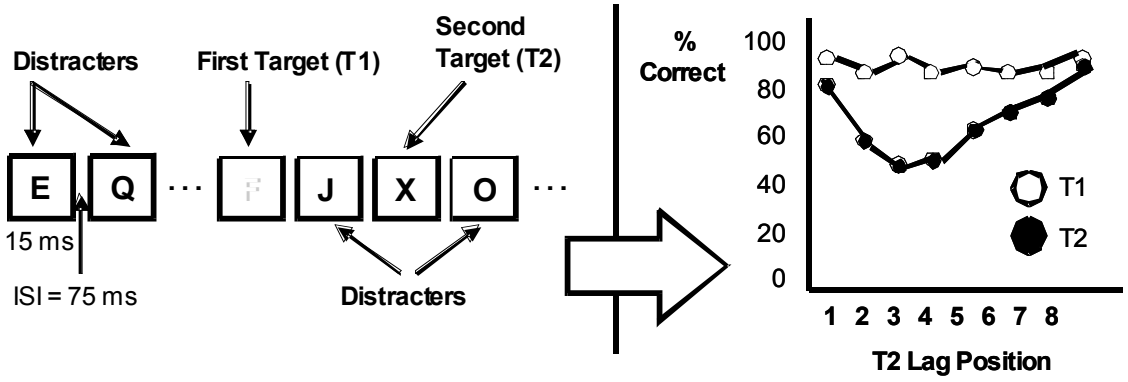
Objectives: Sleep restriction leads to reliable, dose-dependent deficits in reaction time and increases in sleepiness and negative mood. We examined the impact of sleep restriction on the 'Attentional Blink' (AB) task, where two targets are presented within a rapid stream of distractor stimuli. Typically, report accuracy is 90% for the first target (T1), while accuracy to the second target (T2) depends on its relative stimulus position to T1 (i.e., 'lag' position); when T2 occurs soon after the first target, its detection is impaired. Given that sleep loss impairs sustained attention (e.g., reaction time), the limits of dual target processing observed in the AB are expected to change following sleep restriction.
Methods: In a 96hr laboratory study, participants (n=36, mean age=21yrs) were permitted 8hrs in bed on the 1st and 4th night (baseline and recovery, respectively) and randomly assigned to a sleep restriction group for 2nd and 3rd nights (8, 5, 3hrs in bed). The 20min AB task was administered daily at 11am. The AB stream contained 19 stimuli including: distractors (random black letter), T1 (white letter at 7th/10th position), and T2 on half of trials (black 'X' at lag 1-8). Participants reported the T1 letter and the presence/absence of T2 for 160 trials.
Results: *Group* (8, 5, 3hrs) x *Condition* (Nights 1 to 4) ANOVAs yielded the expected interactions and quadratic changes across *Condition* for the 3 and 5hr groups (but not 8hr) for many of the sleepiness, mood, and reaction time variables. Typical AB results were also found, with no group differences at baseline: T2 accuracy varied as a function of lag position. However, no *Group x Condition* interaction was found at early lag positions (2-4) for T1 or T2 accuracy.
Conclusions: The null findings could be due to AB task parameters (e.g., length, non-speeded task), repeated measures, and/or the degree of sleep loss.

Figure: *Left:* Task Diagram (X at lag 2). *Right:* Typical Results



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Decreases in Number of Sleep Spindles Following Acquisition of a Declarative Task Using a Retroactive Interference Paradigm

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Objectives: The protective effect of sleep on declarative memory from associative interference has been reported. The present study examined the changes in number and density of NREM sleep spindles in participants following acquisition of lists of word pairs.
Method: Female adults were asked to learn a paired associate list (A-B). Training continued until 100% recall was obtained. After 12 hours which either included sleep (SLP, n=10) or an equal period of awake (AW, n=10), participants were given a second list of paired associates (A-C). Then after a 15 minute distractor task, participants were asked to recall both list A-B and A-C (Test 1). SLP subjects were sleep recorded for 3 consecutive nights, an acclimatization night (discarded), a baseline night, and a post-training night. All participants were given a retest 1 week later (Test 2). Spindles were counted manually at both Central (C3, C4) and Frontal (F3, F4) sites.
Results: *Behaviour and Sleep State Changes:* The SLP group had superior memory recall ($p < .002$) and showed a reduction in minutes of Stage 2 from baseline to post-training night ($p < .05$) (See accompanying poster).
Sleep Spindles: The total number of Stage 2 sleep spindles at F3, F4 was significantly decreased from baseline to post-training night [$t(9) = 2.97$, $p<.02$]. On the other hand, there was no change in the number of Stage 2 spindles observed at the central sites. The same pattern was seen for total NREM sleep. There was a very modest increase in number of spindles during Stage 3/4 ($p < .08$) at the central sites.

MEAN NUMBER OF SPINDLES

	Baseline Night		Post-training Night	
Sleep Stage	*Frontal (F3,F4)*	*Central (C3,C4)*	*Frontal (F3,F4)*	*Central (C3,C4)*
Stage 2	1291 (SD = 416)	1095 (SD = 333.7)	*1105 (SD = 398)	1018 (SD = 114)
Stage 3/4	286 (SD = 188)	211 (SD = 123)	296 (SD = 207)	278 (SD = 170)

* = significance @ $p < .02$

Conclusions: The post-training time that included a night of sleep was clearly more beneficial to memory consolidation than an equal time awake. The results are unique in reporting reductions in numbers of sleep spindles at frontal sites following successful declarative task acquisition.
Support: Natural Sciences and Engineering Research Council of Canada

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Decreases in Stage 2 Sleep Following Acquisition of a Declarative Task Using a Retroactive Interference Paradigm

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Objectives: The protective effect of sleep on declarative memory from associative interference has recently been reported. The present study examined the changes in sleep states associated with acquisition of lists of two word pairs, separated by 12 hours that either included a night of sleep or a period of waking.
Methods: Female adults were asked to learn a paired associate list (A-B). Training continued until 100% recall was obtained. After 12 hours which either included sleep (SLP, n=10) or an equal period of awake (AW, n=10), participants were given a second list of paired associates (A-C). Then, after a 15 minute distractor task, participants were asked to recall both list A-B and A-C (Test1). SLP subjects were sleep recorded for 3 consecutive nights, an acclimatization night (discarded), a baseline night, and a post-training sleep night. All participants were given a second retest 1 week later (Test 2).
Results: *Behavior:* The SLP group had superior memory recall for the A-B list compared to the AW group at both Test 1 and Test 2. [$F(1,18) = 13.58$, $p < .002$].
Sleep Recording: Comparison of times spent in each of the sleep stages between baseline and post-training night showed no differences in Stages 1, Stages 3/4, or REM. However, there was a significant *drop* in the number of minutes of Stage 2 sleep from Baseline to Post-training night [$t(9) = 2.269$, $p < .05$].

MEAN NUMBER OF MINUTES OF SLEEP

Sleep Stage	*Baseline Night*	*Post-training Night*
1	18.4 (SD = 7.80)	16.7 (SD = 8.65)
2	208.6 (SD = 24.79)	*184.2 (SD = 29.56)
3	46.2 (SD = 14.64)	52.4 (SD = 16.18)
4	52.9 (SD = 28.35)	54.5 (SD = 33.30)
REM	103.0 (SD = 20.56)	104.7 (SD = 19.64)
Total Sleep Time	429.0 (SD = 29.93)	412.3 (SD = 42.40)

* = significance @ $p < .05$

Conclusions: The post-training time that included a night of sleep was clearly more beneficial to memory consolidation, despite interference, than an equal amount of time spent awake. The decreases in Stage 2 sleep were not predicted, but suggest, in the absence of any other sleep changes, that reductions in certain sleep stages are also part of the complex consolidation process which takes place over the night of sleep following declarative task acquisition.
Support: The Natural Sciences and Engineering Research Council of Canada

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Instrumentation and Scoring Validity of Polysomnographic Mouth Leak Events During Treatment with Nasal Continuous Positive Airway Pressure

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Objectives. Treatment with nasal continuous positive airway pressure (nCPAP) is common but adaptation to nCPAP is often difficult, leading to inadequate treatment. Mouth breathing in sleep has been observed before and during use of nCPAP and has been associated with reduced nCPAP compliance. We sought to characterize mouth breathing with attention to signal instrumentation, event definition, inter scorer reliability, distribution according to sleep stages and associated polysomnographic events.

Methods. Seven patients with obstructive sleep apnea (OSA) were studied with custom nocturnal polysomnography. Tidal volume was measured with a pneumotachometer in line with their nCPAP (range 7 to 15 cm H_2O). Oral breath signal was generated with a simultaneous peri-oral pressure transducer as well as a polyvinylidene fluoride (PVDF) film airflow sensor. Two independent certified scorers reviewed the polysomnographic record.

Results. Mouth breathing during calibration and in sleep shows both a relatively fast frequency 'puffing' waveform and a slow waveform in phase with tidal breathing. Scoring rules were developed based upon categorization of the observations. "Fast" events are defined as those with definite oscillations with a frequency of 1 Hz or more. "Slow" mouth leaks were defined as lower frequency events of less than 1 Hz in phase with tidal breathing with concomitant increase of the nCPAP leak signal of at least 3 liters per minute over baseline. The fast 'puffing' appeared during stable REM and NREM sleep, as well as immediately before and during cortical arousals from sleep. Slow events occurred more frequently and were associated with stable sleep, as well as persistent hypopneas in sleep despite nCPAP. Loss of the peri-oral pressure transducer signal occurred in every study where interpretable signal lasted at best for 16% of the night's epochs. Overall agreement was 0.95 for fast and 0.90 for slow events. Kappa values were 0.90 for fast and 0.80 for slow events.

Frequency of Sleep Epochs With Mouth Leak Events

Sleep Stage	Fast Mouth Leak (% epochs)	Slow Mouth Leak (% epochs)
1	26.4	22.2
2	25.8	47.2
3 & 4	7.0	42.8
REM	7.3	30.5

Conclusions. The mouth leak events are clearly discernible on the polysomnographic record. These events may be part of stable breathing in sleep, may accompany arousals from sleep, or may be a source of sleep disruption in patients treated with nCPAP.

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The Sleep Hygiene Inventory (SHI): A New Self-Report Measure

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Objectives: Sleep hygiene encompasses behaviours and lifestyle choices (e.g., drinking alcohol, shift-work) that result in sleep disturbance. Poor sleep hygiene is associated with poor sleep quality, daytime sleepiness, and poor academic performance. Presently, there are no established measures of sleep hygiene. The few research studies that have examined sleep hygiene have used measures with unknown psychometric properties and questionable reliability and validity. The present study focused on developing a new self-report measure to assess sleep hygiene.
Methods: The initial item pool contained 80 items that were completed by 350 undergraduates (70 men, 267 women, and 13 participants that did not report their gender). The mean age of the sample was 19.82 years (*SD*=3.84).
Results: A series of principal component analyses were conducted to examine the structure of the sleep hygiene items. The exploratory factor analyses provided an interpretable factor structure for the sleep hygiene items (*n*=32 remained) and suggest that the new measure assesses five distinct sleep hygiene dimensions: Sleep routine (eigenvalue of 4.75), fitness (eigenvalue of 4.25), alcohol use (eigenvalue of 2.90), medications (eigenvalue of 2.45), and disruptive bed partner (eigenvalue of 2.05). The relationships among the sleep hygiene factor scales were low (range .03 to .25), providing some evidence of multidimensionality of the new measure. The five factors accounted for 51.28% of the variability in the 32 remaining sleep hygiene items.
Conclusions: The exploratory factor analyses provided a thematically coherent structure and suggest that the new measure assesses five distinct dimensions of sleep hygiene. The new measure identifies behaviours consistent with the International Classification of Sleep Disorders (ICSD) conceptualization that are important components of sleep hygiene.

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Feasibility of Unattended Overnight Cardio-Respiratory Monitoring in Screening for Sleep-Disordered Breathing in a Chronic Hemodialysis Population

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Objectives: Sleep-disordered breathing is very prevalent in patients with end-stage renal disease (ESRD). Sleep disturbances in ESRD may be a marker of inadequate dialysis. Sleep-disordered breathing is associated with an increased cardiovascular mortality. Home cardio-respiratory monitoring (Stardust, Respironics Inc.) has been effective in screening for sleep disordered breathing in patients with a high pre-test probability of sleep-disordered breathing. We tested the feasibility of Stardust recording in screening hemodialysis patients for sleep-disordered breathing as part of recruitment for a randomized controlled trial on the effect of hemodiafiltration on sleep-disordered breathing.
Methods: Adults without treated sleep-disordered breathing, stable on in-centre hemodialysis for a period > 3 months were eligible. Subjects underwent home cardio-respiratory monitoring after approximately 2 hours of verbal instruction at the hospital on use of the device. The recorder was then installed by the patient at home. Complete unattended polysomnography with the equipment installed by a sleep technician at home was performed on a separate night.
Results: 8 patients (7 men) were screened for sleep-disordered breathing. Home cardio-respiratory monitoring resulted in a fair or better study quality as per the Sleep Heart and Health Study criteria (Quan SF, Sleep. 1997 Dec;20(12):1077-85) in only 2/8 patients. In contrast, overnight polysomnography resulted in fair to excellent studies in 7/8 patients. Based on the complete polysomnography, the prevalence of sleep-disordered breathing was 100%, 95%CI 69-100%, p=0.008. The mean apnea-hypopnea was 26, SD 28. All had obstructive sleep apnea (AHI>10) and none had central sleep apnea.
Conclusions: Sleep-disordered breathing is highly prevalent in our hemodialysis population. Self-installed home cardio-respiratory monitoring may not be a feasible screening method in these patients. The success with complete unattended polysomnography suggests that a degree of technical support is required among ESRD patients. Furthermore, given the high prevalence of sleep-disordered breathing among ESRD patients, consideration to perform directly a diagnostic test should be given, foregoing a screening test such as cardio-respiratory monitoring.

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Minimal Impact of Inadvertent Sleep Between Naps on the MSLT AND MWT

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Introduction: Sleepiness is often neurophysiologically assessed using the multiple sleep latency test (MSLT) or the maintenance of wakefulness test (MWT). We examined the frequency of incidental intersession napping during MSLT and MWT testing to see if there was a relationship between intersession napping, mean sleep latency and subjective sleepiness on the Epworth Sleepiness Scale (ESS).
Methods: We conducted a retrospective analysis of 24 subjects who had underwent either a MSLT or a MWT as a component of their clinical assessment and had coincidental wireless telemetry recording of their sleep in between scheduled naps.
Results: We found that 15.8% of the MSLT patients and 28.6% of the MWT patients slept inadvertently between test sessions. The group of patients who napped between sessions had shorter sleep latencies on the MSLT. No statistically significant group-wise difference between the sleep latencies of those who napped between MWT sessions and those who did not was found. There was no significant difference between the ESS of those who did and those who did not sleep between sessions.
Discussion: We found that brief inadvertent intersession napping was common during the MSLT and MWT, but there was no evidence to suggest that this significantly alters clinical test results.

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Reliability of the French Version of the Occupational Fatigue Exhaustion Recovery (OFER) Scale in College Students with Part-Time Jobs

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Objectives: To assess the reliability of the French version of the Occupational Fatigue Exhaustion Recovery (OFER) scale in adolescents and young adults, a population at high risk for both problem sleepiness and injury at work.
Methods: A sample of 74 college students (45 female; *M*=19.8±2.1 years) with part-time jobs (*M*=16.6±6.9 hrs/week) filled the OFER scale twice at a 1-month interval. The 15 item OFER scale which comprises three subscales (chronic fatigue-CF, acute fatigue-AF, and intershift recovery-IR) has been developed and validated in three study populations specifically to measure work-related fatigue.
Results: Cronbach's alpha reliability coefficients (degree of item relatedness) are 0.83 for the OFER-CF, 0.76 for the OFER-AF, and 0.80 for the OFER-IR. Intraclass correlation coefficients (instrument reproducibility overtime) are 0.86 for the OFER-CF, 0.80 for the OFER-AF, and 0.82 for the OFER-IR. Finally, Goodman-Kruskal's Gamma coefficients (test retest reliability for individual items) were significant for all items ($p<0.05$).
Conclusions: Reliability of the French version of the OFER-15 was high, allowing its reliability in adolescents and young adults. The OFER-15 is suggested as a potentially valuable new tool for use in research addressing the role of fatigue in the increased risk of occupational injury among adolescents and young adults.

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Is Actigraphy Able to Detect Wakefulness During Sleep?

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Objectives: Actigraphy measures of sleep usually showed good epoch-by-epoch accuracy and good sleep parameters correlations with PSG. However, the ability to detect wakefulness, especially in population with fragmented sleep, has been very low and questioned the validity of that measure. This study evaluated the ability of actigraphy to detect wake compared with PSG when the same subjects are submitted to three sleep conditions with different amount of wakefulness. Another objective was to compare wake detection of four scoring algorithms in these three sleep conditions.
Methods: Fifteen healthy subjects aged between 20 and 60 years were monitored with actigraphy and PSG recorded at the same time in three sleep conditions: a nocturnal sleep episode, a daytime recovery sleep episode after the administration of a placebo and a daytime recovery sleep episode after the administration of 200 mg of caffeine. Four scoring algorithms for estimating sleep parameters derived from actigraphy counts were compared; two thresholds based algorithms (low; 20 and medium; 40) and two algorithms based on regression analysis.
Results: Epoch-by-epoch comparison between actigraphy and PSG showed a significant decrease of accuracy of actigraphy as the sleep conditions involved more wakefulness (table1). Low specificity of sleep by actigraphy (generally around 50%) in all sleep conditions and with the four scoring algorithms confirms the relative inability of actigraphy to detect wakefulness. Actigraphy overestimated total sleep time and sleep efficiency more strongly in conditions involving more wakefulness. The threshold algorithms were more affected in their ability to detect wake and showed an overestimation of the number of awakenings compared to the two regression algorithms.
Conclusions: The very low sensitivity to wake of actigraphy questions it's validity to measure sleep quality in clinical populations with fragmented sleep or in situations in which the sleep-wake cycle is challenged such as in jet lag and shift work.

Table 1. Means (±SEM) sleep sensitivity, specificity, and accuracy of epoch-by-epoch comparison with PSG of four actigraphy scoring algorithms in three sleep conditions (N=15)

Statistical parameter	Sleep[1] conditions	Scoring algorithms[2] Act40	Act20	LötEq	LötMt
Sensitivity (%)	NS	95.3 (0.7)	91.4 (1.0)	94.6 (0.9)	94.8 (0.8)
	DRS	96.0 (0.6)	92.3 (0.9)	96.0 (0.7)	95.8 (0.7)
	CDRS	96.0 (0.7)	93.3 (0.9)	95.3 (0.6)	95.0 (0.8)
Specificity (%)	NS	54.3 (5.6)	65.3 (4.7)	47.3 (4.7)	52.6 (5.4)
	DRS	45.1 (5.2)	54.9 (5.6)	47.1 (6.3)	49.8 (6.1)
	CDRS	37.3 (4.0)	47.8 (4.3)	48.5 (5.3)	49.4 (5.0)
Accuracy (%)	NS	90.7 (1.2)	88.2 (1.0)	90.3 (0.9)	90.6 (0.9)
	DRS	84.0 (2.4)	83.3 (1.7)	85.6 (1.6)	86.2 (1.5)
	CDRS	71.7 (4.0)	74.2 (3.2)	76.8 (3.2)	77.5 (3.0)

[1] NS, night sleep; DRS, day recovery sleep; CDRS, caffeine day recovery sleep
[2] Act40, Actiware® medium threshold algorithm; Act20, Actiware® low threshold algorithm; LötEq, Lötjönen et al's equation regression algorithm; LötMt, Lötjönen et al's method regression algorithm

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Development and Validation of a Novel Scale for Measuring Sleepiness and Fatigue Concurrently

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Objective: To develop a novel questionnaire to measure both sleepiness and fatigue.
Methods: This study was comprised of 2 phases: development and validation of the scale. During the development phase, a set of 31 questions was developed. This 31 item preliminary questionnaire, as well as the Epworth Sleepiness Scale (ESS) and the Fatigue Severity Scale (FSS), was tested in 114 sleep disorder patients (patient group, PG) and 26 normal controls (control group 1, CG 1). Based on item correlations and clinical impact evaluations, 10 items were selected. This 10 item, self report instrument is the final version of the Toronto Sleepiness and Fatigue Scale (TSFS), which includes sleepiness (TSFS S) and fatigue assessments (TSFS F) related to a common stem. We then further evaluated the concurrent validity, internal consistency, and test retest reliability of the scale in 30 normal participants (control group 2, CG 2). The efficiency of diagnostic utility was also estimated.
Results: In the CG 2, the Pearson correlation coefficients (*r*) between scores on the TSFS and those of the validated scales measuring sleepiness (the ESS and the Stanford Sleepiness Scale) and fatigue (the FSS and the Fatigue Impact Scale) were between 0.54 and 0.69 on Day 0 and between 0.46 and 0.71 on Day 7. Cronbach's alphas were between 0.84 and 0.87 for the TSFS S and TSFS F on Days 0 and 7. Intraclass correlation coefficients were 0.87 for the TSFS S and 0.80 for the TSFS F. Analyses using both original data and age restricted data found that the mean scores of the both measurements (TSFS S and TSFS F) in the sleep patient group were significantly higher than those in the control groups.
Conclusion: The TSFS has been shown to be a useful instrument to measure sleepiness and fatigue concurrently.

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Do Better Nights Mean Better Days? Impact of a Behavioural Sleep Treatment Program on Health-Related Quality of Life in School-Aged Children

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Objectives: The purpose of this study is to investigate how treating sleep problems, through the use of a behavioural intervention, impacts on elementary school-aged children's health-related quality of life (HRQL). HRQL is defined as well-being in multiple domains, including physical, social, emotional, and family. In previous research it has been found that children with sleep disorders showed significantly poorer functioning across most HRQL domains. However, there are no data examining whether HRQL improves as a result of effective treatment of sleep problems in this age group.
Methods: The current study is an analysis of a subsample of the larger study, *Better Nights, Better Days: Treatment for Sleep Difficulties*. The larger study is ongoing and examines treatment effectiveness for sleep disorders in school-aged children with and without disruptive behaviour disorders. The current study involves an analysis of a subsample of 25 participants (mean age = 9.76 years, SD = 2.05; 10 males, 15 females). Children participating in the larger study were experiencing sleep disturbances in one or both of the following areas: bedtime resistance and sleep onset delay. All participants have completed their random assignment to either the treatment group (n=13) or the waitlist control group (n=12). The treatment group received five sessions, based on a manualized behavioural treatment, which took five to eight weeks to complete. The control group was placed on a six-month waitlist, therefore not receiving treatment, but completed the same assessments as the children in the treatment group. The dependent variables regarding HRQL were taken from the *Child Health Questionnaire – Parent Form* (*CHQ-PF50*). We examined the data collected from the *CHQ-PF50* at three time points (i.e., prior to treatment, end of treatment, and six months after baseline).
Results: A series of repeated ANOVAs indicated that the treatment group demonstrated improved sleep based on the *Child Sleep Habits Questionnaire*. The treatment group demonstrated significant improvement in the overall sleep disturbances scale (Wilks = .71, $F(2,21) = 4.20$, $p = .03$). In terms of the primary focus of the intervention, there was a significant change in sleep onset (Wilks = .72, $F(2,21) = 4.07$, $p = .03$), but no difference in terms of bedtime resistance (Wilks = .82, $F(2,21) = 2.30$, $p = .12$), although the means changed in the expected direction. On the *CHQ-PF50*, our primary outcome measure regarding HRQL, the treatment group demonstrated improved functioning on the Psychosocial Summary Scale (Wilks = .75, $F(2,21) = 3.44$, $p = .05$), but not the Physical Summary Scale (Wilks = .80, $F(2,21) = 2.70$, $p = .09$).
Conclusions: These results, although preliminary due to small sample size, indicate that HRQL improves in children whose sleep problems were reduced after completion of a behavioural treatment. This was particularly evident in the psychosocial health domain, which consists of subscales scores measuring well-being in the areas of mental health, behaviour, emotion, and self-esteem.

The presented research is an interim data analysis of Kimberley Woodford's MA dissertation in the department of Child and Youth Study at MSVU. The larger study is funded by Nova Scotia Health Research Foundation.

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Sleep Problems and Performance on a New Test of Attention in Children with ADHD

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Objectives: Few studies have examined the relationship between sleep disturbances and attention in children and even fewer have investigated this association in children with Attention-Deficit/Hyperactivity Disorder (ADHD). The present pilot study sought to explore the effects of sleep problems on three types of attention, as proposed by the Attention Network Theory, using a new test known as the Attention Network Test (ANT). The ANT is a newly developed experimental task that measures three kinds of attention simultaneously. These include: Alerting (sustained attention), Orienting (automatic "capture" of attention), and Executive (attention for problem-solving tasks).
Methods: Twenty children rigorously diagnosed with ADHD (15M and 5F) between the ages of 6 and 12 (M = 115 months, SD = 16.59) participated in this study. Children completed the ANT as part of a larger battery of cognitive tests. All children were stimulant medication free for a minimum of 48 hours prior to participation. Children's parents completed the Children's Sleep Habits Questionnaire, a parent-report measure designed to assess a wide range of sleep disturbances in childhood. Parents also reported the number of hours of sleep the children had on the night prior to completing the ANT.
Results: Results of the partial correlation analyses, with age and IQ entered as covariates, indicated that acute sleep problems (as assessed by the number of hours the child slept the night prior to taking the test) were not associated with poorer accuracy on the ANT (Alerting: r=.02, p=.47; Orienting: r=.07, p=.40; Executive: r=.18, p=.24). However, a measure of more chronic sleep problems (i.e., nightly hours of sleep over a typical one week period) was associated with increased errors on the ANT in the area of alerting efficiency (r=.60, p=.004). On the Child Sleep Habits Questionnaire there were a number of significant associations found between sleep variables and performance on the ANT. In particular, orienting accuracy was related to sleep duration (r=.43, p=.04) and daytime sleepiness (r=.45, p=.03).
Conclusions: While these results are preliminary and the sample size is small, it appears that performance on the ANT, particularly on the Alerting and Orienting Networks, is associated with sleep problems in children with ADHD. Future research must replicate and further investigate these findings using a larger sample of children with ADHD and objective measures of sleep such as actigraphy.

Funding for this project included a doctoral student award to Jennifer Mullane from the Nova Scotia Health Research Foundation and a Category A grant from the IWK Health Centre awarded to Penny Corkum and Jennifer Mullane.

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Sleep Difficulties and Behavioral Problems in Young Children: A Community Study

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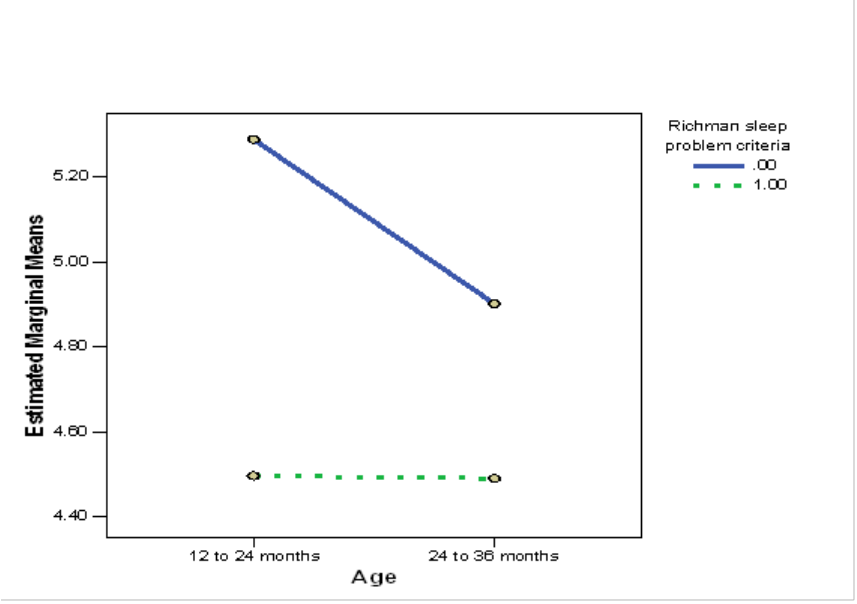
Background: The prevalence of sleep problems in infants and young children ranges between 14% and 25%. Behavioral sleep problems in infancy have been associated with pre-schoolers' inattentive and aggressive behavior.
Objectives: The aim of this community-based, cross-sectional study was to examine associations between children's sleep problems and behavioral characteristics.
Methods: Sixty parents representing a range of cultures, education, and ages completed questionnaires. Their children were between 12 to 36 months and recruited through 18 daycares. Parents were included who read and spoke English and had a child between the ages of 12 and 36 months who attended a group day care a minimum of two days a week. Excluded were children with severe developmental delays or medical conditions. Surveys included: the Infant Sleep Questionnaire [ISQ]; Toddler Behavior Assessment Questionnaire [TBAQ]; and Child Behavior Checklist [CBCL]. Children with and without sleep problems [using Richman's criteria for the ISQ] were compared using analysis of variance (2 X 2 ANOVA), with interactions examined between children's ages, (young 12-24 months) and older (>24 – 36 months) and sleep problems.
Results: Fifty-nine percent of parents reported no concerns about their children's sleep; however, 28% of the children were categorized with sleep problems. Soothability [TBAQ Scale], defined as ability to self-soothe, was significantly lower in the sleep problem group (F (1, 54) = 13.2, p =.001). On the CBCL, older children with sleep problems were significantly more likely to demonstrate somatic complaints (F (1, 54) = 5.4, p = .02) and internalizing behavior (F (1, 54) = -4.53, p = .04). **Conclusions:** Compared with the younger group, older children's sleep problems engendered more parental concerns about somatic complaints and internalizing behavior. Among children aged 2 to 3 years, sleep problems are associated with specific behavioral difficulties.
[Funding] The British Columbia Ministry of Child and Family Development and the Human Early Learning Partnership.

Figure 1: TABQ Soothability by sleep problem and age



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A Retrospective Review of Sleep Studies in Children with Cleft Palate

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Cleft palate is the most frequently occurring congenital malformation. It is associated with an increased risk of sleep disordered breathing (SDB) but the magnitude of this risk is unclear. There is increasing evidence to suggest an association between SDB and neuro-cognitive compromise in children. Children with cleft palate are at increased risk of developmental delay and, therefore, attention to other factors that impact on development is imperative.
Objective: To describe the results of sleep studies undertaken in children with cleft palate at the Children's Hospital at Westmead over the last 10 years.
Methods: The hospital medical records, the Crux Cleft Recording System and Sleep Laboratory records were reviewed to ascertain sleep studies.
Results: A total of 69 studies from 50 children with a mean age of 61.17+/-58.9 mos were reviewed. The mean time of primary cleft palate closure was 13.5+/-10.4 mos. A recognized syndrome was identified in 43.5% of the children studied. The awake average oxygen saturation was 95.2+/-4.35% with 20.6% of the sample showing awake oxygen saturations less than 95%. The average Respiratory Disturbance Index (RDI) was 6.6 events/hr with a range of 0 to 34.4 events/hr. Events were predominantly obstructive and occurring during REM with an average Apnea Hypopnea Index of 14.6+/-18.4 events/hr in REM compared to 3.8+/-4.8 events/hr in NREM. Further review of RDI results showed that 18% of RDIs were consistent with the absence of SDB (less than 1 event/hr), 41% were consistent with mild SDB (0-5 events/hr), 19% with moderate SDB (5-10 events/hr), and 22% with severe SDB (greater than 10 events/hr). RDI showed a significant negative correlation with age (Pearson -0.30, p=0.015). RDI was greater in those who were studied prior to closure of cleft palate (11.98 vs 3.54, p=0.04) and in those with an identified syndrome (9.8 vs 4.6, p=0.032).
Conclusion: Children with cleft palate have a significant risk of SDB. Younger age, pre-palatal closure and a syndrome diagnosis may confer an increased risk of SDB in this group. A prospective study of children with cleft palate is needed to further investigate this issue.
Dr. MacLean is supported by the University of Sydney International Research Scholarship.

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Longitudinal Study of Bad Dreams in Preschool Children: Risk and Protective Factors

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Introduction. Little research exists on the development of children's bad dreams (BD). Adult BD are associated with psychopathology and sleeplessness, while among children, parental sleep practices favoring emotional nurturance (e.g. co-sleeping) predict sleeplessness. This study thus investigated whether parental practices, psychopathology and sleeplessness are risk factors for children's future BD.

Methods. 987 Quebec children were assessed at 5, 17, 29, 41 and 50 mo, 5 yr and 6 yr using parental self-administered questionnaires. BD predictors assessed were: 1) sleep-related (BD frequency, sleeplessness symptoms, parental practices) and 2) psychological (difficult temperament, anxiety, separation anxiety, emotional distress). Logistic regressions conducted at each time point for both groups of predictors assessed presence/absence of BD as dependant measure.

Results. 1) Sleep-related measures. The best predictor of BD at 41 and 50 mo was BD presence the preceding yr**;** at 5 and 6 yr it was BD presence earlier at 29 mo (Table). Parental practices favoring nurturance after night awakenings (at 29 and 41 mo) were protective for BD at 50 mo (Table). No sleeplessness symptoms were risk factors. 2) Psychological measures. The best predictors of BD at 29 mo were mother's rating of child anxiety at 17 mo (OR=1.21, 95% CI: 1.07-1.37), father's rating of child anxiety at 17 mo (OR=1.18, 95% CI: 1.06-1.30), and mother's rating of difficult temperament at 5 mo (OR=1.11, 95% CI: 1.01-1.23).

Conclusion. Unexpectedly, parental practices favoring emotional nurturance after awakenings are protective in BD development, suggesting that sleeplessness and BD are distinct phenomena requiring differential parental management. That BD, starting at age 5, are best predicted by earlier BD (29 mo) supports a diathesis-stress model in which BD at 29 mo index heightened vulnerability to future stress. Early anxiety and difficult temperament may contribute to this vulnerability because their appearance as young as 5-17 mo predicts later BD.

Table. Significant predictors of BD presence or absence for sleep variables at 41 mo, 50 mo, 5 yr and 6 yr

		Regressor statistics			
Model	Significant predictors	Beta (S.E.)	Wald (df=1)	p	Exp(B) (95% CI)
1. BD at 41 mo	BD at 29 mo	1.92 (0.20)	92.95	***	6.80 (4.60-10.03)
2. BD at 50 mo	BD at 29 mo	0.95 (0.35)	7.58	**	2.59 (1.32-5.11)
	Parental practice at 29 mo: Take child out of bed to provide comfort, when he/she awakes at night[a]	-1.70 (0.45)	14.29	***	0.18 (0.08-0.44)
	BD at 41 mo	2.43 (0.34)	50.15	***	11.32 (5.79-22.17)
3. BD at 5 yr	BD at 29 mo	1.43 (0.32)	20.43	***	4.17 (2.24-7.74)
	Parental practice at 29 mo: Let child sleep in mother's bed, when he/she awakes at night[a]	-0.98 (0.36)	7.18	**	0.38 (0.19-0.77)
4. BD at 6 yr	BD at 29 mo	1.25 (0.39)	10.57	**	3.50 (1.64-7.43)
	Parental practice at 41 mo: Let child sleep in mother's bed, when he/she awakes at night[a]	-1.17 (0.42)	7.68	**	0.31 (0.14-0.71)

[a]*Reference parental practice was "Comfort him/her but leave him/her in his/her bed"; **p < 0.01; ***p < 0.001*

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Laryngeal Stimulation by an acid Solution in Preterm Lambs During Quiet Sleep

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In a mature organism, the contact between various liquids and the laryngeal mucosa triggers lower airway protective responses (cough, swallowing, arousal). These laryngeal chemoreflexes (LCR) are essential for preventing tracheal aspiration. However, previous studies in neonatal mammals have suggested that immature LCR are merely responsible for apnea and bradycardia. Consequently, LCR, especially when triggered by acid oesophageal reflux, seem to be responsible for apneas of prematurity, life-threatening events and some cases of sudden infant death syndrome. Recently, we have revisited LCR in full-term lambs during quiet sleep. Results show that LCR triggered by HCl (pH 2), mimicking an acid gastroesophageal reflux, were consistently alike the mature LCR in adult mammals, without apneas or bradycardias (St-Hilaire, 2005). This prompted us to question whether premature birth alters LCR.

Objectives; the aim of the present study was to assess the cardio-respiratory components of the LCR in response to acid solution in 6 preterm lambs.

Methods: Preterm lambs were born by caesarean section after 132 days of gestation (term = 147 days), 48 hrs after IV injection of beta-methasone. Electrodes were surgically inserted at D3 to record states of consciousness, respiration, electrocardiogram and arterial oxygenation, together with a supra-glottal catheter for liquid instillation. LCR were induced by 0.5 ml of saline and HCl (pH 2) during quiet sleep in non-sedated lambs, at post-natal day 7 (D7) and D14.

Results:

	Saline		**HCl (pH 2)**	
	D7	**D14**	**D7**	**D14**
% dec HR (%)	27.5 (SD 16)	12 (SD 4)[a]	34.9 (SD 14)	28.5 (SD 11)[b]
Bradycardia duration (sec)	2.1 (SD 5)	0 (SD 0)	10.5 (SD 22)	1.3 (SD 2.9)
% dec RR (%)	66 (SD 15)	44 (SD 17)[a]	71.3 (SD 20)	64.7 (SD 22)[c]
Apnea duration (dec)	7.2 (SD 7)	1.5 (SD 3)	17.5 (SD 22)	9.9 (SD 15)

% dec HR or RR: percentage of decrease in heart rate or respiratory rate from baseline; SD: standard deviation; [a,b]: $p < 0.005$ *vs*. [a] saline or [b] HCl, D7; [c]: $p < 0.005$ *vs*. saline, D14.

HCl triggered clinically significant apneas and bradycardias in preterm lambs at D7 only, including repetitive apneas for more than 90 seconds, severe desaturation and bradycardia.

Conclusions: laryngeal stimulation by HCl can lead to life-threatening apneas-bradycardias in preterm lambs, suggesting that LCR triggered by acid gastroesophageal refluxes can be involved in apneas of prematurity. In addition, normal postnatal maturation seem to be involved in the disappearance of apneas of prematurity. Supported by CIHR (MOP 15558).

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Policies Practices and Provisions for Parents Sleeping Overnight with a Hospitalized Child

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Objective: To describe, for the first time in the literature, practices and policies affecting parents' overnight stays, provisions and supports for parents sleeping overnight and parents' involvement in overnight care of their hospitalized child in North American pediatric hospitals.
Methods: A descriptive, cross-sectional design was used. Telephone surveys were completed with senior administrative staff using a list of 265 US and Canadian pediatric hospitals. Included pediatric hospitals had ≥50 acute care beds and ≥2 wards. Survey questions were developed based on review of the existing literature and clinical experience. Data were analyzed with descriptive statistics.
Results: From July-September 2006, 192 of 265 hospitals were contacted. 16 hospitals were ineligible, 41 did not reply to phone messages, and 135 (77% response rate) interviews were completed. Hospitals had a median of 107 beds (range 50-439) and 4 units (range 2-17). 135 (100%) general pediatric units allowed parents to sleep at the bedside overnight; only 85 (66%) PICUs and 22 (18%) NICUs allowed parents to stay. 84 (62%) hospitals limited overnight visitors at the bedside to one, 46 (34%) hospitals to two visitors, and only 5 (4%) hospitals allowed ≥3 people to stay. Only 11 (8%) hospitals routinely allowed siblings to sleep overnight. 57 (42%) hospitals allowed parent-child bed-sharing. Overnight stays by parents were routinely limited based on number of patients in the room in 24 (17%) hospitals, and by acuity of the child in 26 (19%) hospitals. Although 134 (99%) hospitals provided a bed for the parent at the child's bedside, for parents not allowed at the bedside, only 44 (33%) hospitals provided surfaces intended for sleep, and with limited access and availability. 133 (99%) hospitals reported parental involvement in their child's care at night, with 52 (39%) stating this was an expectation. 133 (99%) respondents stated their hospital made provisions to improve sleep for parents, including reduced lighting and noise, and minimal visits to the room.
Conclusion: In general, parents are given the opportunity to stay at the bedside overnight, but barriers exist that limit opportunities for sleep during their child's hospitalization. Restrictions on parental stays based on type of unit, acuity of the child and number of beds in the room creates an environment that separates families who have a hospitalized child. In future, parents' experiences of sleeping in hospital with their child, and parents' views on barriers and facilitators of sleep in hospital should be explored.
Funding support: This study was supported by the Samuel Lunenfeld Research Summer Student Program at SickKids and a Randomized Controlled Trial Mentoring Salary Award to Dr. Stremler.

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Longitudinal Short Sleep Duration and Risk of Obesity in Early Childhood

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Objective. Childhood obesity is a public health problem in many industrial countries. The aim of the study was to verify to what extent short sleep duration during early childhood is an independent risk factor for obesity at school entry.

Methods. Body mass index was measured at 6 years of age in a sample of children born in a Canadian province (N=1138). Nocturnal sleep duration was reported yearly from 2.5 to 6 years of age by their mothers. Prenatal, postnatal (5 months), and lifestyle (6 years) potentially confounding factors for excess weight were assessed by interviews, questionnaires, and hospital records. The association between BMI at 6 years and sleep duration pattern was assessed by a Kruskal-Wallis test. Logistic regressions evaluated whether sleep duration patterns is an independent risk of excess weight while controlling for a variety of obesogenic factors.

Results. The prevalence of excess weight was 13.8% for the 6-year-old children. We identified 4 sleep patterns: a *short persistent* pattern (6.0%,n=109) where children slept less than 10 hours per night until age 6; a *10-hour persistent* pattern (50.3%,n=920); a *11-hour persistent* pattern (38.9%,n=712); and a short increasing pattern (4.8%,n=88) where children slept fewer hours in early childhood but increased their sleep duration at around 2.5 and maintained it until 6 years of age. A significant difference in the distribution of BMI as a function of sleep duration pattern (P<.001). The effect of sleep duration pattern on excess weight remained significant after adjusting for confounding variables. The odds ratio of being overweight was almost threefold higher for both *short persistent* sleepers and *short increasing* sleepers compared to 11-hour persistent sleepers (P=.01). **Conclusions.** Short sleep duration (less than 10 hours) during early childhood significantly increases the risk of excess weight or obesity in childhood, and appears to be independent of other obesogenic factors.

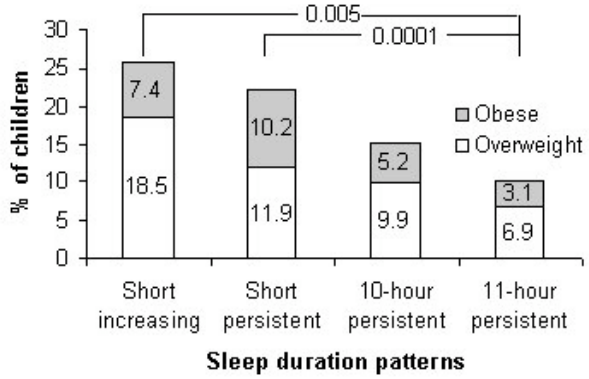

Figure 2. Percentage of obese and overweight children as a function of longitudinal sleep duration patterns. A Kruskal-Wallis test was used to measure the distribution of BMI categories in different sleep duration patterns (normal: N=989, overweight: N=105 and obese: N=54)

Research supported by "Institut de la statistique du Québec, direction Santé Québec", "Canadian Institutes of Health Research" (CIHR) (grant to J. Montplaisir) and "Fonds de la recherché en santé du Québec" (doctoral studentship to E. Touchette).

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Self –Reported Sleep Difficulties in a Community Based Sample of Children and Adolescents with Recurrent Pain

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Objectives: There is increasing interest in the association between sleep disturbances and pain in pediatric populations. In clinical samples of children and adolescents who experience recurrent pain, there is some evidence of more disturbed sleep compared to those without chronic pain complaints. This study examined self-reported sleep difficulties in a community-based sample of children and adolescents with recurrent pain in comparison to a pain-free control group.
Methods: Participants were ninety children and adolescents (53 males, 37 females) 8-15 years of age (M=10.93, SD=2.25), who experienced headaches and/or recurrent abdominal pain (n=60) or were healthy and pain free (n=30). They completed a self-report version of the Children's Sleep Habits Questionnaire, which yields a total score for sleep difficulties. Participants also completed a detailed pain questionnaire, assessing pain duration, frequency and intensity.
Results: Participants with recurrent pain reported significantly more sleep difficulties (M=20.18, SD=3.22) than did those in the pain-free control group (M=18.53, SD=2.66; $t(88)=2.42$, $p<.05$). For children in the pain group, there was a significant positive association between increased reports of sleep difficulties and higher scores for pain frequency ($r = .36$, $p<.01$), but not for pain duration ($r = .17$) or intensity ($r = .22$). **Conclusions**: These results indicate that children and adolescents in a community-based sample with recurrent pain report more sleep difficulties than their pain-free peers. In addition, the frequency of pain is related to sleep difficulties. The links between disturbed sleep and recurrent pain have important implications for the assessment and treatment of these disorders in children and adolescents.

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Gender Differences on Topographical Sleep EEG in Midlife

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We reported that women between 20 and 60 years old show higher N-REM sleep spectral power for C3 in delta, theta, low alpha, and high sigma frequencies than men. Studies have shown that age-related effects on sleep EEG vary across topographical derivations. We aimed to evaluate how gender effects interact with aging and EEG sleep topography in the middle years of life.

The sleep of eighty-seven healthy volunteers with no sleep disorders was analyzed. Subjects were separated in two groups: A) Young (22F, 26H; 23,3y ±2,4) and B) Middle-aged (21F, 18H; 51,9y ±4,6). Spectral analyses were performed on N-REM sleep for Fp1, F3, C3, P3, and O1 (linked-ears). Three-way ANOVAs (Age, Gender, Derivations) were performed on 1-Hz bins (1-25 Hz).

Polysomnographic sleep was similar in men and women. Compared to young subjects, middle-aged subjects showed lower sleep efficiency, sleep duration, SWS%, and higher Stage 2%. Women showed higher power than men between 2-4 Hz, 13-14 Hz and 23-25 Hz. Significant interactions between gender and derivations were found between 4-6Hz, 7-11Hz, and 14-19Hz. Women showed higher power than men; this effect was more prominent in C3, P3 and O1 than in Fp1 and F3. Middle-aged subjects showed lower power between 23-25Hz. Significant interactions were found between age and derivations between 2-6 Hz and 7-21 Hz, with middle-aged subjects showing lower power. Between 2-6 Hz, this effect was more prominent in Fp1 and F3, while it was more prominent in FP1, F3 and O1 between 7-21 Hz.

In the middle years of life, gender and age effects on N-REM sleep EEG show different topographical distributions. Gender effects are more prominent in posterior derivations, while age effects are more prominent in anterior and occipital areas. We found no interactions between age and gender, suggesting that aging does not influence men and women differently.

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Stage 2 Sleep Spindles, Motor Learning and Aging

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Objectives: Previous studies have established a relationship between sleep spindles and memory consolidation. The majority of these studies, however, have involved young adults. The objective of this investigation was to compare the changes in spindle density following motor learning in younger and older adults.
Methods: In-home sleep recordings were performed on 14 younger (17-24 yrs) and 14 older adults (62-79 yrs) for three consecutive nights. The first night was discarded, while the second and third nights served as the baseline and post-acquisition nights respectively. Stage 2 spindle density was determined by dividing the number of Stage 2 spindles by the number of minutes of Stage 2 sleep. Subjects performed the pursuit rotor on two occasions: between the baseline and post-acquisition nights and one week later. For behavioural data, a 2(Session: Acquisition, Retest) x 2(Group: Younger; Older) ANOVA was conducted to assess changes in performance on the pursuit rotor task. For sleep data, a 2(Night: Baseline, Post-Acquisition) x 2(Group: Younger, Older) ANOVA was conducted to assess changes in Stage 2 spindle density.
Results: Regarding pursuit rotor performance, the session by group interaction was significant [$F(1,26) = 7.73$, $p = .010$]. The magnitude of learning across sessions was greater in the younger adults [$t(13) = 9.08$, $p < .001$] than in the older adults [$t(13) = 2.85$, $p = .014$]. For Stage 2 spindle density, the night by group interaction was significant [$F(1,26) = 5.30$, $p = .030$]. The increase in spindle density was significant in the younger subjects [$t(13) = 3.30$, $p = .006$] but not the older subjects [$t(13) = .33$, $p = .747$].

	Younger		Older	
	M	(SD)	M	(SD)
Pursuit Rotor: Total Time on Target (sec)				
-Acquisition Session	357.5	(76.8)	275.2	(87.4)
-Retest Session	488.0***	(78.4)	335.1*	(122.2)
Stage 2 Spindle Density				
-Baseline Night	6.0	(1.6)	2.5	(1.7)
-Post-Acquisition Night	6.7**	(1.6)	2.6	(1.9)

*p < .05 **p < .01 ***p < .001 (Retest vs. Acquisition Session OR Post-Acquisition vs. Baseline Night)

Conclusions: The results of this investigation suggest that differences in motor learning across sessions in younger and older adults may be related to the changes in sleep architecture that occur following initial acquisition in these two age groups.
Funding Support: Canadian Institutes of Health Research and the Alzheimer Society of Canada

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Age Differences in the Temproal Organization of Stage 2 Sleep Spindles

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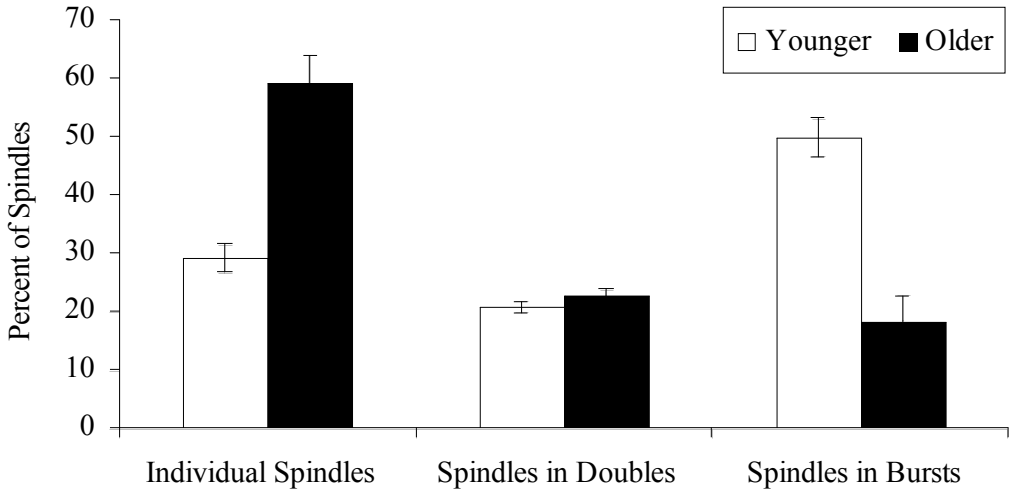
Objectives: Previous studies have shown that the number and density of sleep spindles decreases with age. The objective of this investigation was to determine whether there were age differences in the temporal organization as well as the number of Stage 2 sleep spindles.
Methods: In-home sleep recordings were performed on 14 younger (17-24 yrs) and 14 older adults (62-79 yrs) for two consecutive nights. The first night was discarded and the second night served as the baseline night. There were four key spindle-related variables: (a) the total number of spindles in Stage 2 sleep, (b) the percentage of spindles within a cluster (i.e., three or more spindles with an inter-spindle-interval less than 5 seconds), (c) the percentage of spindles within a double (i.e., two spindles with an inter-spindle-interval of less than 5 seconds), and (d) the percentage of individual spindles (i.e., isolated spindles with an inter-spindle-interval more than 5 seconds).
Results: Consistent with previous studies, the number of Stage 2 spindles was significantly greater in younger subjects (M = 1337.07; SD = 383.98) than older subjects (M = 613.21; SD = 414.88), $t(26) = 4.79$, $p < .001$. The percentage of spindles within clusters was also significantly greater in younger subjects (49.73%) than in older subjects (18.28%), $t(26) = 5.67$, $p < .001$. Younger and older subjects did not differ significantly in the percentage of spindles within doubles (20.72% and 22.70% respectively), $t(26) = 1.33$, $p = .194$. The percentage of individual spindles was significantly greater in older subjects (59.02%) than in the younger subjects (29.14%), $t(26) = 5.55$, $P < .001$.



Conclusions: These results suggest that not only do younger and older adults differ in their overall number of Stage 2 sleep spindles, they also appear to differ in how their spindles are organized temporally within Stage 2 sleep.
Funding Support: Canadian Institutes of Health Research and the Alzheimer Society of Canada

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Decreased Nightmare Frequency and improved sleep quality following Treatment with Interactive Voice Mail and Cognitive-Behavioral Drawing Techniques

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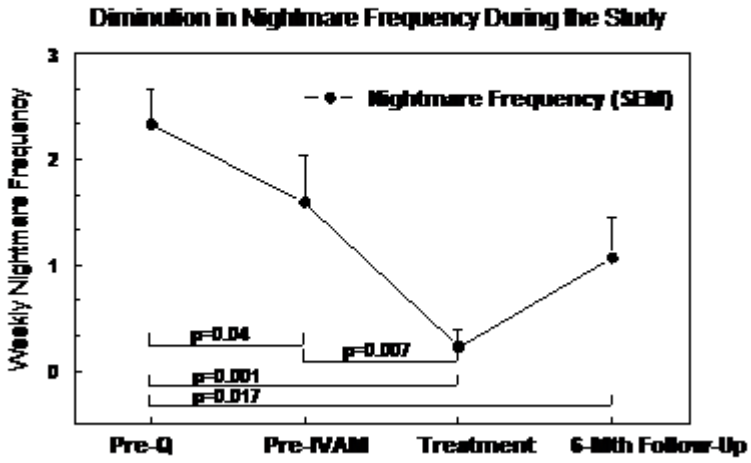
Objectives: Epidemiological studies indicate that 5 to 8% of adults report suffering from nightmares (Klink & Quan, 1987). Major psychological and sleep disorders affect nightmare sufferers. However, techniques used to treat this parasomnia are few and their application is costly and time-consuming. This study examines the efficacy of a new interactive voice-mail acquisition (IVMA) system and a simple cognitive-behavioral drawing technique (CBDT) on nightmare frequency and sleep quality.

Methods: 16 patients (12W; M=31.3±7.1years) suffering from at least one weekly nightmare over 6 months participated. They were screened by phone, evaluated by a psychologist and randomly assigned to an immediate (N=11) or a delayed treatment group (N=5). The study lasted respectively 8 and 14 weeks during which patients had to complete the IVMA system daily allowing a weekly measure of nightmares. For the last 6 weeks of the study (treatment period), they also drew their nightmares whenever they were recalled. Mean weekly nightmare estimates for all patients for four time periods were assessed with Friedman non-parametric tests: 1) pre-treatment questionnaire (Pre-Q), 2) Pre-IVMA), 3) treatment, and 4) 6-month follow-up. To evaluate sleep quality, paired t-tests were performed on pre- and post-treatment global scores using the Pittsburgh Sleep Quality Index (N=14).

Results: A significant effect was found for time (Chi-sq(3)= 23.23; $p<0.0001$). Wilcoxon post-hoc tests revealed that weekly nightmare frequency significantly decreased ($p<.05$) from pre-treatment (combined M=2.1; SEM=0.33; Range=0.68 to 5.6) to treatment (M=0.23; SEM=0.16; Range=0 to 2) to 6-month follow-up (M=1.1; SD=0.38; Range=0 to 4). Global sleep quality on the PSQI also improved after treatment [t(13)=2.65; p=0.020].

Conclusions: Although the sample size is modest, results support the efficacy of the IVMA and drawing techniques in treating nightmares, with nightmare frequency decreasing by half into 6-month follow up. Both techniques are economical, applied at home and require minimal interventions by a therapist.



Klink, M., & Quan, S. F. (1987). Prevalence of reported sleep disturbances in a general adult population and their relationship to obstructive airways diseases *Chest, Vol, 91*(4), 540-546.

Research supported by the *Fonds de la recherche en santé du Quebec (FRSQ) and the Canadian Institutes of Health Research.*

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Evaluating the Usefulness of a Multidimensional Approach to Attitudes & Beliefs About Dreams

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Objectives : Attitude towards dreams (ATD), a very popular construct in dream research, is traditionally defined as a global trait representing a general interest in dreams. However, a recent study (Schredl, Ciric et al., 2003) suggests that this operationalization is too simplistic and that it doesn't take into account the fact that different aspects of this construct have different relationships to dreaming. The purpose of this study was to evaluate the pertinence and usefulness of a new questionnaire that assesses attitudes and beliefs about dreams in a multidimensional way (the *Montreal Inventory of Dream Attitudes : MIDA)*.
Methods : The 352 participants were recruited in an undergraduate course in psychology. They first had to fill up questionnaires about dreams (e.g. the *McGill Sleep & Dream Questionnaire* and the *MIDA*), personality (e.g. the *Boundary Questionnaire* and the *Differential Personality Questionnaire : Absorption scale*) and well-being (e.g. the *State Trait Anxiety Inventory : Trait scale*). Afterward, participants were asked to record their dreams in a daily dream log provided for 3-4 consecutive weeks.
Results : Ten main factors were identified in the MIDA from a previous analysis : F1-Interest towards dreams, F2-Positive value of the dream content, F3-Quality of dream recall, F4-Attitude towards dream interpretation, F5-Pleasure from dreaming, F6-Similarity with reality, F7-Preference of dreams over waking life, F8-Quality of long-term recall, F9-Belief in a latent content and F10-Fear of dreaming. Dream recall frequency (log) was correlated to F3 (r=0.35), Nightmare frequency (log) was correlated to F10 (r=0.26), the Boundary Questionnaire was correlated to F7 (r=0.43), Absorption was correlated to F7 (r=31) and F8 (r=0.31) and Anxiety was correlated to F2 (r=-0.41) and F10 (r=0.32). Only correlations above r=0.25 are considered.
Conclusions : The results suggest that *Interest towards dreams* is not the only aspect of ATD worth evaluating to better understand dreaming.

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Dream Emotion is Associated with Cross-Night Adaptation to Negative Picture Stimuli

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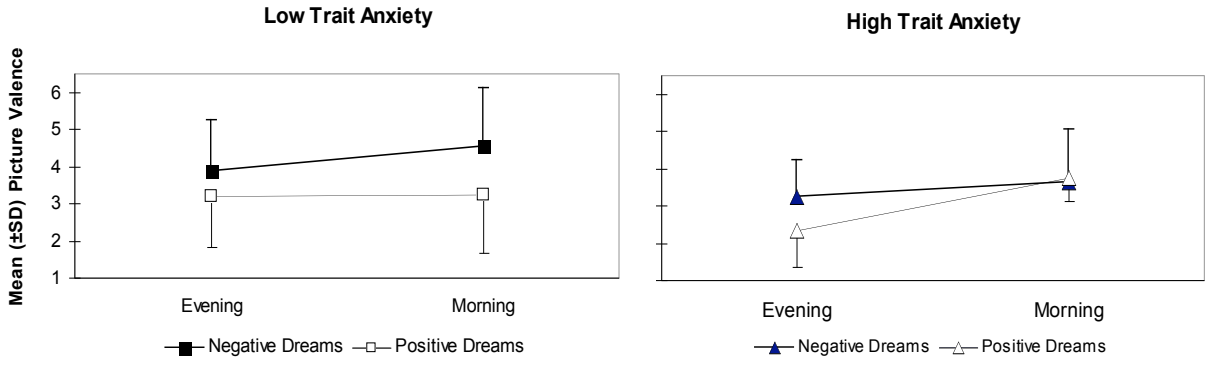
Objectives: Although growing evidence implicates REM sleep in cognitive functions, such a role for dreaming remains controversial. We therefore assessed a possible functional role for dreaming in cross-night emotional adaptation.
Methods: 33 subjects (24W; 25.67±4.46yrs) slept two nights (adaptation, experimental). They completed the State-Trait Anxiety Inventory and, prior to sleep, viewed 36 neutral and 36 negative pictures and evaluated their emotions (1-9 scales) for valence (negative-to-positive), arousal (weak-to-strong) and 9 specific types (1-5 scales). The task was repeated after morning awakening. Intervening REM sleep was disrupted by repeated awakenings (not reported here). Intervening dreams were rated using the same emotions scales. Subjects were split into low (LAnx; M=32.69±2.30; N=16) and high (HAnx; M=41.53±5.34; N=17) trait anxiety groups, and into negative (NegD; M=3.59±0.66; N=18) and positive (PosD; M=5.81±0.66; N=15) mean dream valence groups. Two ANOVAs assessed 'adaptation' to negative picture valence and arousal for *time* (evening, morning) X *anxiety* (HAnx, LAnx) X *dream valence* (NegD, PosD).
Results: A three-way interaction for picture valence (Trace=.78, F(1,29)=8.35, p=.007) was decomposed as follows. For the **HAnx group**, a *time* X *dream valence* interaction for picture valence (T=.27, F(1,15)=5.59, p=.032) showed that emotions improved for PosD (2.33 vs. 3.76, p=.010) and marginally for NegD subjects (3.23 vs. 3.63, p=.140). For the **LAnx group**, the same interaction (T=.17, F(1,14)=2.88, p=.112) showed improvement for NegD (3.88 vs. 4.54, p=.032) but not PosD subjects (3.21 vs. 3.25, p=.882). A *time* X *valence* interaction (T=.41, F(1,13)=9.10, p=.010) further clarified that emotions improved to some extent for all but the LAnx/PosD subjects. A marginal three-way interaction for picture intensity (T=.89, F(1,29)=3.78, p=.062) gave a similar pattern, with a cross-night reduction in intensity paralleling improved emotional valence.
Conclusions: Dreams are associated with cross-night emotional adaptation. Their emotional valence and intensity may be functional factors, depending upon the individual's habitual anxiety state.



Research was supported by the Canadian Institutes of Health Research and the Natural Sciences and Engineering Research Council of Canada.

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Late-Night REM Sleep Rebound is Reduced but Sleep Efficiency is Normal in Subjects with Idiopathic Nightmares

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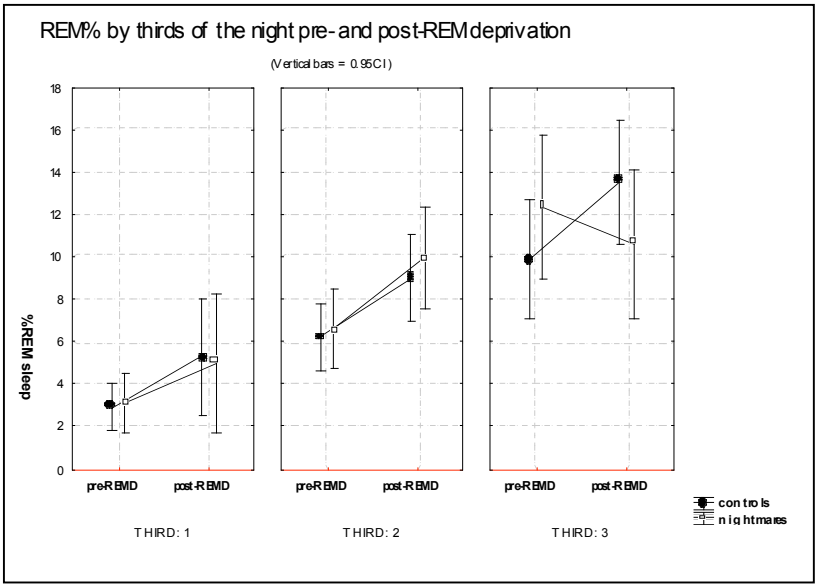
Introduction: Dysregulation of REM sleep pressure is a likely explanation for idiopathic nightmares (NM). NM sufferers and controls were therefore subjected to partial REM deprivation to increase REM sleep pressure and allow assessment of the extent and distribution of their REM sleep rebound.

Methods: Non-traumatized NM sufferers (NMs: n=13; 10 W; M=26.5±9.5 yrs) and controls without nightmare complaints (CTL: n=12; 8 W; M=25.6±7.0 yrs) slept 3 consecutive nights (adaptation, REMD, recovery) in the laboratory. Tracings were scored by highly trained polysomnographers using standard criteria. Total REM% was examined for nights 1 and 3 with a Group (NM, CTL) x Nights (N1, N2) ANOVA and contrasts. REM% distributed by thirds of the night was similarly assessed with 3 identical analyses. REM efficiency and sleep efficiency were also examined.

Results: For Total REM%, a Group x Nights interaction ($F(1,23)=6.07$, $p=.021$) revealed that CTLs displayed a REM rebound (N1: 17.9% ; N3: 26.6%; $t(23)=6.44$, $p=.000007$) that was much greater than that for NMs (N1: 18.8%; N3: 22.9%; $t(23)=3.15$, $p=.0045$). REM rebounds occurred for the 1st and 2nd thirds of the night for both Groups (all $p<.05$)—and for the 3rd third rebound for CTLs ($t(23)=2.33$, $p=.029$) but not NMs ($t(23)=0.13$, $p=.895$) (see Figure). No group differences were found for REM efficiency or sleep efficiency for either N1 or N3.



Conclusion: These results converge with results in accompanying abstracts on the same subjects to suggest that a dysregulation of REM sleep pressure exists in idiopathic NM disorder. NM subjects seem to fully rebound from partial REM deprivation during the 2nd third of the night.

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Active But Not Passive, Virtual Reality and TV Maze Tasks Produce Bimodal Patterns of Dream Incorporation Over 14 Days

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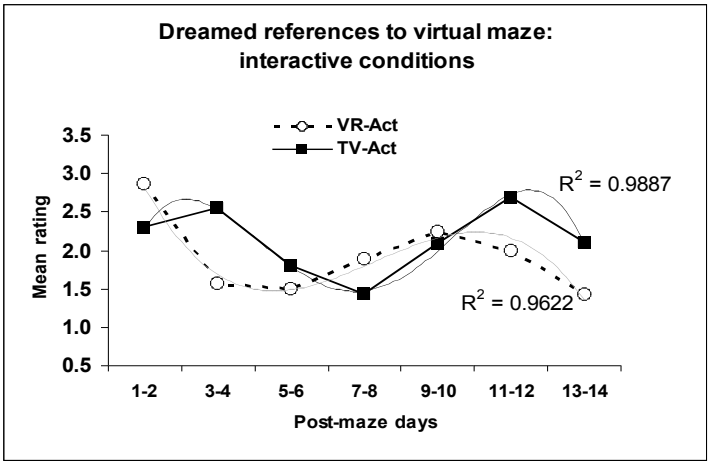
Introduction. Spatial events can influence dream content on the subsequent night (day residue effect) and about a week later (dream-lag effect). Using a virtual maze task, we studied the effects of a spatial stimulus on both of these effects by varying the stimulus attributes of *interactivity* (active, passive) and *visual display* (virtual reality: VR, wide-screen television: TV).

Methods. 57 healthy subjects (45W; 24.5±3.25 yrs) were randomly assigned to 1 of four 20-min 3D maze tasks: 1) VR-Act (n=15): wore VR goggles and interacted with the maze; 2) VR-Pas (n=14): wore VR goggles and passively viewed maze movements; 3) TV-Act (n=15): watched TV and interacted with maze (via mouse); 4) TV-Pas (n=13): watched TV and passively viewed maze movements. Subjects then rated task-related sense of presence and cybersickness and, for 14 days, wrote their dreams and rated each (on 9-pt scales) for references to maze elements. To maximize N, dream scores were averaged over successive pairs of days, producing 7 post-maze time periods per subject: D1-2, D3-4, D5-6, D7-8, D9-10, D11-12 and D13-14. A grand mean reference score was also calculated for D1-14. 2 X 2 ANOVAs (interactivity X display type) were used to assess changes in maze references by condition; polynomial curve-fitting was used to assess fluctuations in references over time.

Results. An Interactivity effect ($F(1,53)=3.9493$, $p=0.052$) revealed higher D1-14 scores for Active ($M=2.10 \pm 1.42$) than for Passive ($M=1.50 \pm 0.64$) groups. Bimodal polynomials with approximate circaseptan morphology characterized both VR-Act (3rd-order; $R2=.989$) and TV-Act (4th-order; $R2=.962$) groups. VR-Act peaks were D1-2 and D9-10; TV-Act peaks were D3-4 and D11-12 (see Figure). Lagged cross-correlation between the two curves ($r=.566$, $p<.05$) suggested a 1-day delay of the entire circaseptan process for TV-Act. The TV-Act group was also associated with more cybersickness symptoms ($p<.07$). Day-residues (D1-2), but not dream-lags, were found for both VR-Pas and TV-Pas groups.



Conclusion. Interactivity in a spatial maze task facilitates delayed dream incorporations with an approximate circaseptan morphology. This bimodal profile may reflect two steps in the hippocampus-mediated consolidation of new spatial memories. Over-stimulation sufficient to produce cyber-sickness symptoms (TV-Act group) may cause a 1-day phase delay in this process.

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First-Night Eye Movement Density is Lower in Subjects With vs Without Nightmares

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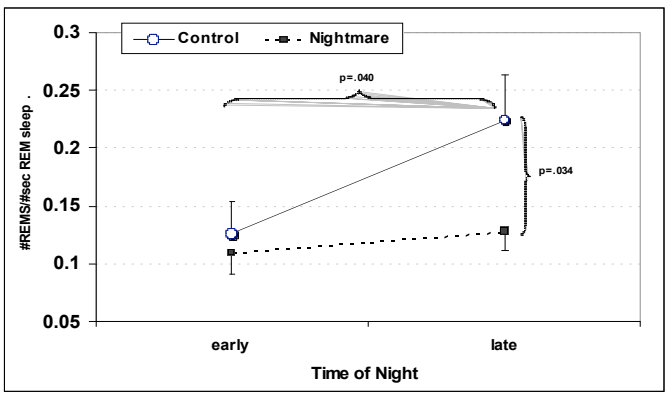
Introduction: The pathophysiology of idiopathic nightmares remains unclear although a dysregulation of REM sleep propensity is likely. Anomalies of REM sleep are more apparent on the first night of laboratory recording than on other nights—the so-called 'first night effect'. We therefore examined the first night PSGs of subjects with and without nightmares for evidence of group differences in REM sleep propensity.

Methods: Non-traumatized nightmare sufferers (NM: n=10; 6W; 24.9±8.91 yrs), who reported at least 1 NM/wk for at least 6 mo, and controls without nightmare complaints (CTL: n=10; 7W; 23.4±5.75 yrs) slept in the laboratory for 3 consecutive nights (adaptation, REM deprivation, recovery). Sleep from night 1 was scored by a trained polysomnographer. REM density for each REM period was calculated as the #individual eye movements/#seconds of REM sleep. NM and CTL groups were compared for whole night REM density as well as for early (REMP 1 or 2) and late (last REMP of night) night REM sleep using independent and paired t-tests. Times elapsed between sleep onset and the start of each early and late night REMP were also computed and compared.

Results: Whole night REM density was lower for the NM (M=.101±.021) than the CTL (M=.153) group (t(18)=1.924, p=.070). Late night density was also clearly lower (t(18)=2.299, p=.034) for NM than CTL group (see Figure). However, there was no group difference for early night density (t(17)=0.456, ns). By group, density increased from early to late night for the CTL (t(9)=2.395, p=.040) but not the NM (t(8)=1.477, p=.178) group. Mean time elapsed since sleep onset did not differentiate NM and CTL groups for either the early or the late night REMP densities (all p>.60).

Conclusion: Findings converge with other measures (REM sleep latency, NREM-REM cycle length, mentation dreamlike quality) reported in companion abstracts in supporting the possibility that NM sufferers have lower REM sleep propensity on the first—and subsequent—laboratory recording nights. The hypothesis of REM sleep dysregulation in idiopathic NMs is thus supported.

Figure. Mean (sem) REM density for early and late REM periods for subjects with and without nightmares.



Supported by the Canadian Institutes of Health Research and the Natural Sciences and Engineering Research Council of Canada

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Increased REM Latency and NREM/REM Cycle Length in Idiopathic Nightmare Sufferers

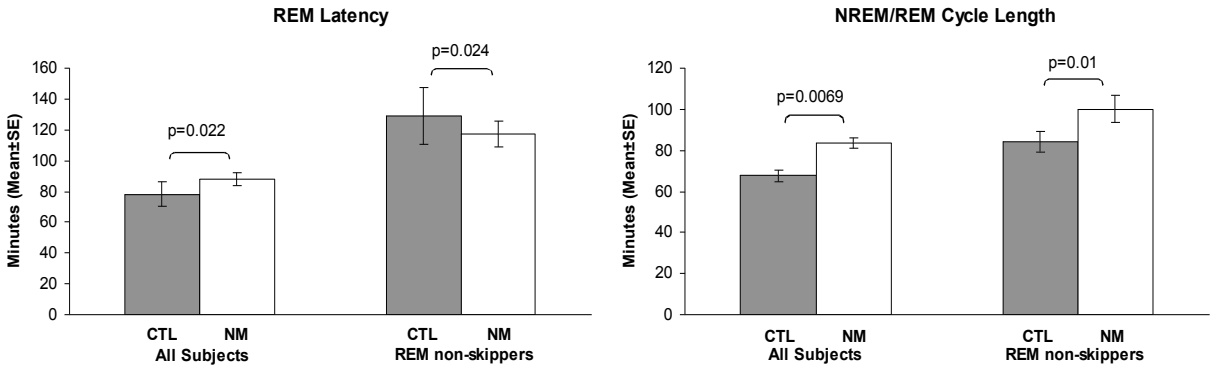
Paquette T.[1], Nielsen, T.A.[1,2], Stenstrom P.[1,3], Lara-Carrasco J.[1,3], Solomonova E.[1,4], Bourdeau, G.[1,5], Dallaire S.[1,4]
[1]Sleep Research Centre, Hôpital du Sacré-Coeur de Montréal; [2]Psychiatry Department, Université de Montréal; [3]Psychology Department, Université de Montréal; [4]Psychology Department, McGill University; [5]Psychology Department, Université du Québec à Montréal, Québec, Canada

Introduction: The pathophysiology of idiopathic nightmare disorder remains unknown. Non-traumatized nightmare sufferers were subjected to a partial REM deprivation protocol and attributes of their REM sleep compared with that of non-nightmare controls.

Methods: Nightmare sufferers (NM: n=13; 10W; M=26.5±9.5yrs) and controls without nightmare complaints (CTL: n=12; 8W; M=25.6±7.0yrs) slept in the laboratory for 3 consecutive nights. Only results for night 1 are reported here. REM sleep variables were assessed with standard criteria by a trained polysomnographer. REM latency, average REM/NREM cycle duration, REM fragmentation, REM efficiency, total time in REM, sleep onset latency, sleep efficiency, time to persistent sleep (10 min uninterrupted sleep) and # 'skipped' REM periods were compared for NM and CTL groups using chi-squares and one-way ANOVA.

Results: The NM group showed longer REM latency (M=129.3±66.7min) than the CTL group (M=78.2±28.6; F(1,23)=6.010, p=0.022) and longer REM/NREM cycle duration (M=117.1±30.7 min) than the CTL group (M=88.2±14.5min; F(1.23)=8.806, p=0.0069) but no differences on other assessed variables. However, these differences were potentially explained by the fact that more NM sufferers skipped early night REM periods (6/13) than did CTL subjects (2/12; chi-sq=2.49, p<.11). Therefore, analyses were repeated excluding all REM period 'skippers'. NM 'non-skippers' (n=7; 4W; 27.0±10.0yrs) nevertheless still scored higher on REM latency (NM: M=83.5±17.5min, CTL: M=67.8±8.1min; F(1,15)=6.258, p=0.024) and REM/NREM cycle duration (NM: M=99.95±12.98min, CTL: M=84.25±9.09min; F(1,15)=8.679, p=0.0100) than did CTL non-skippers (n=10; 7W; 24.4±5.6yrs).

Conclusion:REM sleep pressure, especially early in the night, is reduced in non-traumatized nightmare sufferers compared to controls. While such a reduction may enable skipping early-night REM periods, it may also—and independently—lengthen REM latency and cycle length. Such REM pressure dysregulation may also underlie occasional bouts of high REM pressure later in the night and contribute to nightmare formation at this time.



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Differential Impact of Two Prospective Log Measures on the Frequency of Nightmares

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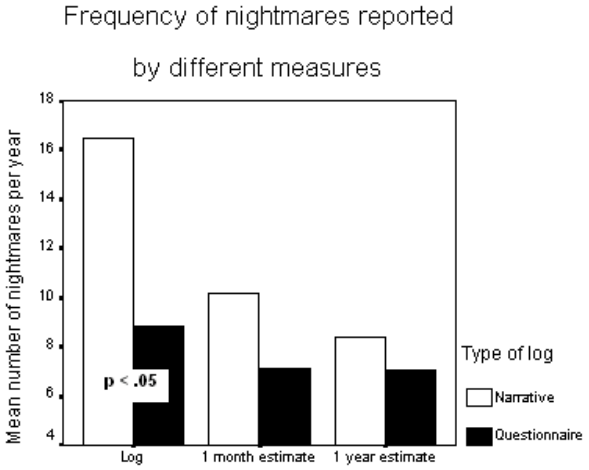
Objectives : The use of prospective dream logs has become the gold standard in assessing the nightmare frequency. Prospective logs have one or two formats: questionnaire (e.g., subjects indicate if there was dream recall and if so, the number and type of dreams recalled) and narrative (e.g., complete written transcript are provided for each dream recalled). The goal of this study was to compare the frequency of nightmares between these two types of logs.
Method : Participants were 260 undergraduates (228 women, 32 men, mean age = 22.8 ± 5.2 years) who completed a dream log for two to five consecutive weeks. Two hundred and three participants completed a questionnaire log and 57 a narrative log. Prior to completing their logs, all participants were required to estimate the number of nightmares experienced over the past month and past year. In their daily logs, participants noted if a recalled dream was a bad dream (very disturbing dreams that do not awaken the dreamer) or a nightmare (very disturbing dreams that awaken the sleeper). Given varying log durations, log frequencies were prorated to one year for comparative purposes. Total dream recall per week was also calculated.
Results: ANOVAS revealed that significantly greater frequencies of nightmares were reported in the prospective measure than by the two retrospective estimates (8.3 ± 10.0 for the 1 year estimate and 10.0 ± 12.9 for the 1 month estimate), but only when using a narrative log (*p*s < .05). No significant difference was found between the two estimates. *T*-tests revealed that significantly greater frequencies of nightmares were reported in the narrative logs (17.1 ± 20.2) than in the questionnaire logs (8.9 ± 15.6). Similarly, the mean number of dreams recalled per week was also significantly greater in the narrative logs (7.9 ± 2.8) as compared to the questionnaire logs (6.1 ± 3.5). All *p*s < .05. **Conclusions**: The results reported in this study are consistent with previous studies highlighting the underestimation of retrospective measures (estimates) of nightmare frequency. Moreover, this was the first study to contrast dream recall frequency data between two types of prospective logs. When compared to questionnaire logs, narrative logs consistently yield higher frequencies of everyday dreams and nightmares. This difference may be partially due to an attentional bias created by the greater involvement required to complete a narrative log.



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The Exploration of Dream's Emotional Content Using Automatic Analysis

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Objectives: Until now, most of the studies on dreams have used time consuming coding systems that depend on a rater's judgment. It is of great interest to develop more objective means of scoring dreams that can be used with large dream banks and that can be reproduced across laboratories. This study stands amongst the first steps toward the exploration of dream's emotional content using automatic analysis tools. The goal of this study is to create a system that can reliably replace human in judging dream emotions.
Methods: Dreams were gathered from a dream bank created during a normative study conducted at the Sleep Research Laboratory of the University of Ottawa. A sample of 100 dreams, reported by 29 individuals of varied age and sex, was used in this study. Their participation mainly consisted of completing a brief dream diary at home during a maximum of three weeks, and to write down all the dreams they remembered when waking up, until a maximum of four dreams. The dreams were rated by two judges using two 0-3 scales describing respectively the negative and the positive orientation of the dream content. The inter-judges agreement was 57.7% on the positive scale and 80.8% on the negative one. The agreement being low for the positive scale compared to the negative scale, and the score on the positive scale being not well differentiated from one dream to another, we dropped this scale for subsequent analysis. The automatic analysis of the negative orientation of dreams were performed using the online version of the General Inquirer (GI), the online version of the Linguistic Inquiry and Word Count (LIWC), the weighted GI and HM lexicons, and a bag-of-words approach making use of the Balie text pre-processing software. We compared everything with a baseline strategy that consists of always guessing the most common rating. In the case of negative scale, the most common rating is 2 on the 0-3 scale (33% of dreams were rated 2).
Results: We calculated classifiers accuracy (i.e. their performance at exactly finding the right 0-3 label) and their mean-squared error (i.e. difference with human judgment when incorrectly guessing). The GI and the LIWC outperformed other strategies accuracy and mean squared error. Both resources obtained an accuracy of approximately 50%, which is statistically better than the baseline accuracy (33%). The mean-squared error for both classifiers was approximately 0.60, meaning that most errors have only a difference of 1 on the scale.
Conclusions: These results constitute a small, but significant first step in the automatic analysis of dream emotions. In future work, we will extend our dataset and use dreamer's own rating of dreams' emotions instead of judges' ratings. Further development of this technology could facilitate the analysis and mining of a greater number of dreams from individuals of different age, sex, social status, thus improving our understanding of dreams.

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Sleep Paralysis and Nightmares Are Both Related To Affect Distress

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Introduction. Sleep paralysis (SP) and nightmares (NM) are both REM-related parasomnias that have been linked to psychopathologies such as anxiety. Affect distress, a proposed personality trait characterized by strong emotional reactivity, reliably predicts the effects of NMs on well-being. Here we introduce a measure of SP distress and investigate its links to NMs and affect distress.

Methods. 193 participants aged 18 to 87 (mean=31.8 yrs; SD=12.85; 33.7% male; 59.1% female; 7.6% unspecified), responded to a battery of online questionnaires. All subjects reported having had at least 1 SP episode. SP distress was measured by 3 items modeled after the NM Distress Questionnaire, NM distress was assessed with a single general question and social anxiety was assessed by the 4 Liebowitz Social Anxiety Scale (LSAS) subscales, including "social interaction", "public speaking", "being observed" (OBS) and "eating and drinking in public". The Other Experiences Questionnaire (OEQ), a new instrument for assessing a variety of social imagery and presence experiences, was also debuted.

Results. SP distress correlated positively with NM distress (r=.541; p<.001). Two multiple regression analyses with SP distress and NM distress as separate dependent measures and LSAS subscales and OEQ scores as predictor variables revealed similar factor solutions. SP distress had a 2-factor solution (p<.001, R=.359, 12% VAF), with the most sensitive predictor the OEQ score, followed by LSAS OBS score. NM distress also had a 2-factor solution (p<.001, R=.341, 11% VAF) with LSAS OBS score as the main predictor followed by OEQ score.

Conclusion. Distress following both SP and NM experiences is related to similar, social anxiety and social imagery, factors. More specifically, it is related to anxiety associated with being passively observed and a propensity to generate presence imagery in waking life. Thus, a common psychopathological factor may determine distress reactions central to two different REM sleep parasomnias.

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Dream Like Quality of Sleep Onset and REM Mentation is Reduced in REM Sleep-Deprived Nightmare Sufferers

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Objectives: Little is known about the effect of REM deprivation on sleep mentation imagery in participants with nightmares (NM). Previous work has shown that the dreamlike quality (DLQ) of sleep onset (SO) imagery is a sensitive indicator of prior REM sleep deprivation. In the present study, the DLQ of SO and REM imagery from NM sufferers was compared to that from non-nightmare controls (CTL) during and following a partial REM sleep deprivation procedure.
Methods: Participants with (NM: n=11; 8W; M=25.1±7.8 yrs) and without (CTL: n=11; 7W; M=25.4±6.3 yrs) nightmare complaints slept in the laboratory for 3 consecutive nights. Following an adaptation night, they were partially REM-deprived (Night 2) by forced awakenings after 5 min of REM had elapsed in each REM episode after the 2nd. On Night 3, they were awakened up to 8 times after at least 5 sec of Hori SO stages 4 and 5 had elapsed. At each REM (night 2) and SO (night 3) awakening, participants reported mentation and rated it for DLQ on 9 point scales. Scores were averaged for all trials and compared for NM and CTL groups using independent samples t-tests. Linear trends across trials were also examined.
Results: REM imagery was characterized as less dreamlike by the NM group (M=6.33±2.13) than by the CTL group (M=8.06±1.38; $t(10)=2.31$, p=.031). A similar trend was reported for SO awakenings for which the NM group reported lower DLQ scores (M=3.55±1.39) than did the CTL group (M=5.13±2.42; $t(10)=1.88$, p=.079). Across SO awakenings, DLQ scores increased linearly for the NM group (linear trend: $F(1,8)=8.27$, p=.005) but not for the CTL group ($F(1,8)=.186$, p=.667).
Discussion: Low DLQ of REM and SO imagery after REM deprivation may be one of several indicators of REM sleep pressure dysregulation observed for these NM participants.

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Logical Thought and the Detection of Bizarreness in Sleep Mentation

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Objectives: While many dream theorists depict dreaming cognition as diminished and deficient, others maintain that higher order cognition is commonplace. The present study tests the hypothesis that, although the ability to detect bizarreness within the dream environment is diminished, the ability to think logically is maintained.
Methods: Fourteen participants aged 21-32 years (M=23.4) slept an adaptation and 2 experimental nights in a sleep laboratory. On each experimental night participants were awakened four times after 10 minutes of stage REM or 2 sleep for mentation reports. They rated their mentation on 9-point Likert scales for the presence and awareness of bizarreness and logical rigor of thinking. For each participant, scores were averaged within stage to produce mean REM and mean Stage 2 scores.
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 scale of measurement (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, no awareness of this bizarreness during dreaming was reported for 18 (51%); in only 2 cases (6%) was it fully appreciated. Paired t-tests found no stage differences for ratings of the degree of awareness of bizarreness (REM: M=2.46, SD=1.72; Stage2: M=3.46, SD=3.22; $t(3)=-.471$, $p=.670$) or the logical rigor of thoughts (REM: M=8.42, SD=1.20; Stage2: M=8.06, SD=0.94; $t(5)=.55$, $p=.606$).
Discussion: The ability to detect bizarreness is either absent or diminished substantially in both stage REM and stage 2 sleep, while logical rigor is preserved. Neither type of cognition appears to be modulated differentially by sleep stage.

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The Effect of Napping on Mood in Women with Sever Premenstrual Symptoms

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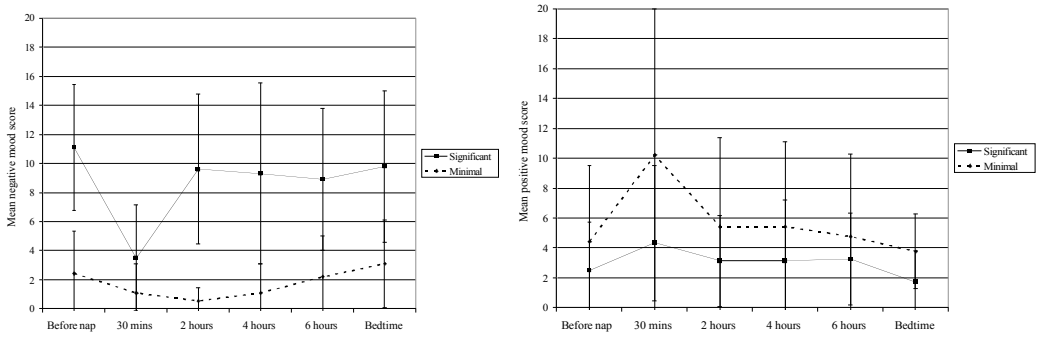
Introduction: Women with Premenstrual Syndrome tend to experience an increase in negative mood and a decrease in positive mood during the late-luteal phase of the menstrual cycle. Among the general population a daytime nap has been found to improve mood. We investigated the effects of a short mid-afternoon nap during the late-luteal phase of the menstrual cycle on mood among women with severe and minimal premenstrual symptoms.
Methods: Ten women with severe emotional/behavioral premenstrual symptoms and nine women with minimal symptoms (mean age 27 years) participated in the study. Participants came to the laboratory in the mid-afternoon for a nap of a maximum duration of 30 minutes. Positive and negative mood was measured before and 30 minutes after the nap, and at two-hourly intervals until nocturnal bedtime. The same procedure was repeated for the 'no nap condition' when participants engaged in a quiet activity rather than a nap. The nap and no nap conditions were counterbalanced.
Results: All women, with the exception of one participant with minimal symptoms, fell asleep; the mean sleep latency for both groups was less than 10 minutes. For both groups, compared to before napping, intensity of negative mood decreased 0.5, 2 and 4 hours after napping, ($p \leq .05$) and intensity of positive mood increased 0.5 hours after napping ($p < .05$). The improvement in negative mood 0.5 hours after napping was slightly higher in women with severe PMS ($p < .001$). No changes in intensity of negative mood were noted without a nap, while intensity of positive mood significantly decreased after 2 hours and before nocturnal bedtime ($p < .05$).
Conclusion: The results of this study suggest an increased sleep need in young women during the late-luteal phase of the cycle, and an improvement in mood with napping in young women, with potentially more benefit for those with more severe premenstrual symptoms.
Figure 1 Mean and SD negative mood score and Figure 2 positive mood score on the Nowlis Mood Adjective Checklist (Nowlis MACL) for women with significant and minimal premenstrual symptoms before and 30 minutes after the nap condition, as well as at 2-hourly intervals after the nap until bedtime.



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Subjective Alertness and Cognitive Performance Across the Menstrual Cycle

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Objectives: There are well-documented circadian variations in vigilance and cognitive performance, and these measures vary in association with the endogenous circadian rhythm of core body temperature[1,2]. The menstrual cycle is characterized by specific changes in both the hormone profile and thermoregulatory system[3]. The aim of the current study was to investigate the circadian and menstrual variations in subjective alertness and cognitive performance.

Methods: Seven healthy women were studied during the mid-follicular (MF) and mid-luteal (ML) phases of their menstrual cycle. Participants underwent a 72-hour multiple-nap procedure (36 cycles of 60-min wake episodes alternating with 60-min naps). During each waking episode, alertness was subjectively assessed by a 10-cm visual analog scale. Cognitive performance was measured by a 4-minute mental calculation (MC) test, during which participants completed as many additions as possible. Measures were z-transformed to account for inter-participant variability. Core body temperature (CBT) was continuously recorded by a rectal sensor and distal temperature (DT) was measured by skin sensors applied at the hands and feet.

Results: Dual-harmonic regression revealed a significantly reduced CBT amplitude in the ML vs. MF phase ($p=0.02$), while no significant difference in time of CBT minimum was found. ANOVA's revealed a significant main effect of menstrual phase for CBT ($p<0.001$) and DT ($p=0.025$), and a significant main effect of circadian phase for CBT and DT at both menstrual phases ($p<0.001$). Participants had significantly lower levels of alertness ($p=0.033$) and scored lower on measures of cognitive performance ($p=0.01$) throughout the experiment in the ML vs. MF phase. A significant main effect of circadian phase was found for alertness and MC in both menstrual phases ($p<0.01$). Pearson's correlation revealed significant positive correlations in both menstrual phases between CBT & alertness (MF: $r = 0.7462$; ML: $r = 0.7943$) and CBT & MC (MF: $r = 0.7551$; ML: $r = 0.7894$). Distal temperature, however, had significant negative correlations with alertness in both menstrual phases (MF: $r = -0.7168$; ML: $r = -0.7857$), and also with MC in both menstrual phases (MF: $r = -0.7743$; ML: $r = -0.6668$). Finally, subjective alertness was significantly and positively correlated with MC scores in both menstrual phases (MF: $r = 0.7706$; ML: $r = 0.7674$).

Conclusions: This study reinforced the finding that alertness and cognitive performance are highest at or near the CBT peak. It is surprising then, that subjective alertness levels and cognitive performance were found to be reduced in the ML phase, despite the consistently raised CBT seen during that menstrual phase. These results suggest that some other luteal phase-associated factor, probably progesterone, which has both sedative/soporific and thermogenic properties, causes reductions in subjective alertness and cognitive performance while simultaneously increasing body temperature.

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Symptom Pattern of Sleep-disorder Complaint Influences Physician Referral

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Objectives:
Older adults are under-referred to sleep clinics. To explore reasons for this, we surveyed (1) primary care patients to inquire about which sleep disorder-related symptoms they recently discussed with their family doctors, and (2) first-time sleep clinic patients to discover what symptom presentation resulted in a "successful" referral.

Method:
The Primary Care Sample comprised 191 older family practice patients and the Sleep Clinic Sample comprised 117 consecutive new patients.
Participants completed the Sleep Study Checklist (SSC) in their respective waiting areas. The SSC includes 21 symptoms of sleep disorder, insomnia, fatigue, sleepiness, psychological and health functioning. Respondents rate symptom severity and check which had been discussed with their family practice physician in the past year (Primary Care Sample) or their referring physician (Sleep Clinic Sample). All Primary Care participants were offered a sleep evaluation, including questionnaires, medical assessment, and polysomnography. Sleep Clinic subjects were awaiting their first appointment at the clinic. According to their participation, Primary Care subjects were designated: Refusers (completed SSC, refused further evaluation), Drop-outs (completed some evaluation steps, but not PSG), Completers (completed all evaluation steps).

Results:
Approximately 35% of the Primary Care Sample endorsed sleep disorder-related symptoms, but few had discussed these with their doctor within the past year. Significantly more Completers had discussed sleep disorder symptoms than Refusers or Drop-outs. The discussed symptoms of the Sleep Clinic Sample were much more focussed on sleep apnea-related symptoms (snoring, breathing interruption, non-refreshing sleep, daytime sleepiness, fatigue) than those of the Completers, who discussed a wider range of symptoms (insomnia, body pain, nocturnal urination and anxiety). On PSG, Completers had a high rate of apnea diagnosis (84%).

Conclusions:
- Sleep Clinic patients are more focussed in their presentation of apnea-related symptoms.
- Completers present a wider range of symptoms, possibly distracting from a sleep clinic referral.

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Can One or More Questionnaires be Used To Detect Sleep Apnea In Sleepy Individuals?

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Objectives: The Berlin Questionnaire (BQ) has been used to accurately identify patients at high risk for having sleep apnea in the primary care setting and in atrial fibrillation patients. Excessive daytime sleepiness (EDS) and/or complaints of unrefreshing nocturnal sleep are key features of sleep apnea but are also non-specific complaints in many medical and sleep disorders. The aim of this study is to determine if the BQ alone or in association with other questionnaires can be used to detect sleep apnea in patients presenting to a sleep clinic with EDS and/or unrefreshing sleep.
Methods: This is a retrospective chart review study of 130 sleep clinic patients who were referred for sleep assessment for EDS and/or unrefreshing sleep. All patients underwent 2 overnight polysomnographic (PSG) studies in the sleep laboratory and were administered a thorough questionnaire battery including the following: BQ, Epworth Sleepiness Scale (ESS), Fatigue Severity Scale (FSS), Center for Epidemiologic Studies in Depression Scale (CES-D) and the Athens Insomnia Scale (AIS). The RDI values were obtained from averaging the measurements from the two nights of recordings.
Results: Of the 130 charts reviewed, the BQ identified 75 (58%) as being at high-risk for sleep apnea but overnight PSG found only 30 of the 130 patients (23%) had an RDI>10. The BQ performed with 0.60 sensitivity and 0.43 specificity at the RDI>10 level and resulted in 40% false negatives and 57% false positives. A 3-questionnaire battery comprised of the BQ, ESS and FSS performed with sensitivity of 0.43 and specificity of 0.92. A 5-questionnaire battery including the BQ, ESS, FSS, CES-D and AIS performed with specificity of 0.85 and sensitivity of 0.7.
Conclusions: The results of the study demonstrate that a subject population that is comprised of a large proportion of sleepy individuals may reduce the ability of the BQ to detect sleep apnea. However, combining the BQ with other questionnaires improves the sensitivity and specificity of detecting sleep apnea in patients endorsing EDS and/or unrefreshing sleep. In clinical practice, especially for sleep clinics with long waiting lists, this 5-questionnaire battery could be used to help identify patients at greater risk for having sleep apnea and who may require more urgent sleep studies.

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At Home vs In Sleep Laboratory Diagnosis and Treatment of Obstructive Sleep Apnea: Effect on Mood, Anxiety Level and Cognitive Function

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Objectives: To compare the two methods of diagnosing and treatment of Obstructive sleep apnea (OSA) on mood, anxiety symptoms and cognitive function: Home monitoring with autoCPAP titration and in-lab diagnostic assessment and CPAP initiation.
Methods: One hundred consecutive adult subjects referred to the Sleep Medicine outpatient clinics at the Royal University Hospital in Saskatoon, SK for assessment for OSA were recruited to participate in this study, and randomized into two groups: Group 1: at home diagnosis using Embletta (Medcare Inc), and Group 2: In-laboratory PSG. Subjects with RDI > 15 underwent a titration study at home using AUTO-SET to determine optimal fixed CPAP pressure (group 1). The in-laboratory CPAP titration was followed by at home CPAP treatment based on the in-lab result (group 2).
All patients underwent baseline measurements of symptom severity and performance and at 2, 4 and 12 weeks including mood and anxiety assessment (Center for Epidemiological Studies for Depression scale and Spielberger State Trait Anxiety Inventory) and Neurocognitive performance (Trail-Making test, Symbol digit modalities test and Concentration endurance test).
Results: There was a significant improvement in depression scores in patients with RDI>15 within both groups over the first 12 weeks of follow-up (Group 1: N=22, p=0.02, F=3.36, Group 2: N=33, p=0.001, F=5.6). There was no difference between the change in the depression scores between the two groups over time.
Although the mean anxiety scores in our group of patients were not significantly different from the normal population (State anxiety: average 34.4, SD 9.70; Trait anxiety 40.2+9.43), a significant decline in the anxiety scores was observed over time with treatment in both groups. The treatment groups did not differ in change in anxiety scores over the first 12 weeks of treatment. Similarly, the results of the neurocognitive tests did not show a significant difference between the two groups.

Conclusions: The results of this study demonstrate similar outcomes on mood and cognitive function when OSA is diagnosed and treated following in home monitoring and CPAP initiation, as compared to the standard, in-lab evaluation. If replicated, implementation of the in home monitoring followed by auto-CPAP titration may allow faster access to diagnosis and treatment for patients waiting for assessment in the sleep laboratory.

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Jaw Position During Sleep

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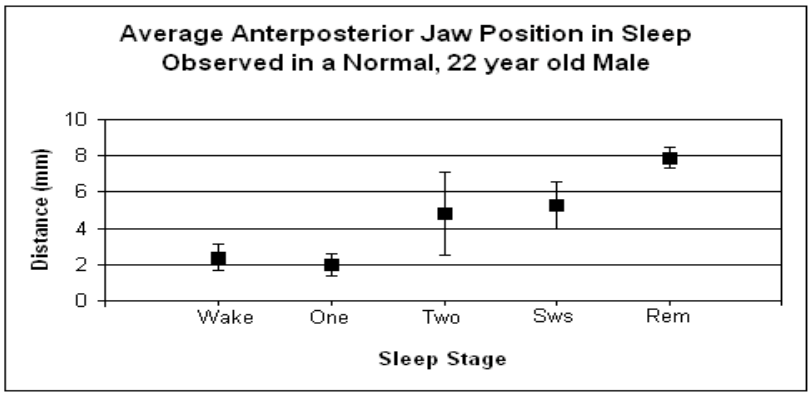
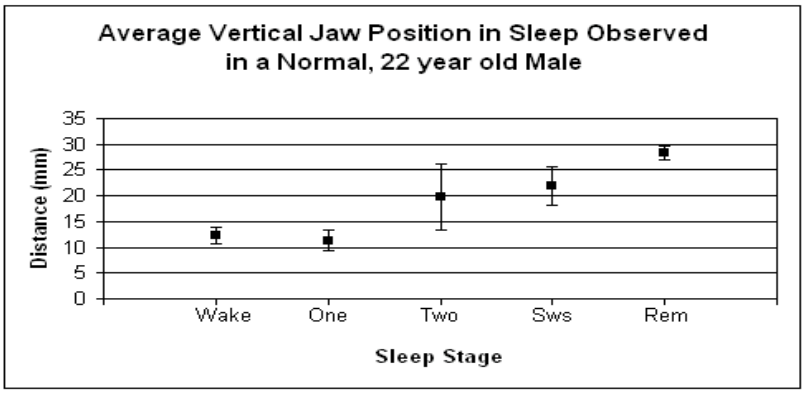
Objective: To describe change in jaw position during sleep in normal subjects. **Methods**: Mandibular position was mapped using spherical coordinates from data provided by magneto-resistive sensors strategically placed around 3 different moving joints on an external apparatus attached at the temples, nose and mandible. In this way, jaw position in each of the three measurement planes (vertical, anteroposterior, lateral) could be independently measured. We have studied 5 normal subjects to date (age, sex, BMI, nasal resistance). Subjects undergo overnight polysomnography (Channels: 4 EEG, 2 EOG, 1 chin EMG, oronasal airflow, thoracoabdominal movement, intercostal EMG, pulse oximetry, R&L ant tibialis EMG, ECG) in the supine position. **Results**: A tendency to greater jaw opening and jaw retraction was observed during REM and slow wave sleep as compared with other sleep stages. The maximum vertical jaw opening reached 3 cm during slow wave and REM sleep, and the greater jaw opening was, as expected, associated with greater jaw retraction. Arousals were accompanied by jaw closure and protrusion towards the clenched position. Detailed graphic data will be displayed at the conference meeting. **Conclusion**: Mandibular posture during sleep in healthy young adults is significantly influenced by sleep stage.





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Cognitive Difficulties in Sleep Apnea Syndrome: Vigilance and Attention are Impaired – A New Comparative Study of Younger And Older Patients

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Objective: Patients with obstructive sleep apnea syndrome (OSAS) present cognitive deficits similar to those observed with aging. The aim of the study was to assess the effects of age on cognitive functions in OSAS patients. It was hypothesized that older OSAS patients will exhibit significant cognitive dysfunction relative to younger OSAS patients and controls.
Methods: Younger and older OSAS patients were compared to younger and older control subjects (age cut-off set at 50 y). Participants underwent a PSG and neuropsychological evaluation. Variables were analyzed by two-way analyses of variance (ANOVAs) with 2 factors: Group (control and OSAS) and Age (younger and older). Additionally, we evaluated the contribution of attentional deficits to cognitive dysfunction for each sub-group of patients by using Spearman correlation coefficients.
Results: No Group by Age interaction was found for any neuropsychological variables ($p<0.05$). However, for vigilance and attentional testing, main Group ($28.3 > F > 4.5 = 0.04 > p > 0.000002$) and Age ($15.1>F>4.3 = 0.04>p>0.001$) effects were found. Similarly, memory (short-term, long-term and, procedural) and executive functions indicated main Group ($10.2 > F > 4.7 = 0.04 > p > 0.002$) and Age ($13.0>F>4.0 = 0.05>p>0.0008$) effects. Correlations indicated that attentional deficits contributed importantly to a poorer cognitive performance in younger OSAS patients only ($0.756>r>0.637 = 0.002>p>0.01$).
Conclusions: Our results are in agreement with those of the literature for both OSAS-related and aging-related cognitive deficits but shows age can add to the effect of the OSAS condition to worsen those cognitive deficits.

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Neonatal Caffeinc Has Long-Term Impact On Sleep and Breathing in Freely-Behaving Adult Rats

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Objectives. Neonatal caffeine is widely used to treat respiratory instabilities in premature newborns; however the use of an adenosine receptors antagonist during a sensitive period of development is always a matter of concern to clinicians (*Finer et al., 2006, Pediatrics*). Since adenosine plays a critical role in sleep (*Huang et al., 2005, Nat Neurosci*) and breathing (*Montandon et al., 2006, Am J Physiol Regul Integr Comp Physiol*), we tested the hypothesis that neonatal caffeine changes sleep architecture and modifies breathing across sleep-wake states.
Methods. Newborns received each day from postnatal day 3-12 a dose of caffeine (15 mg/kg) or water (control). At adulthood, sleep-wake states and ventilatory activity were evaluated using respectively an EEG/EMG telemetry system and whole-body plethysmography in freely-behaving adult male rats.
Results. Rats treated with neonatal caffeine presented a higher percentage of wakefulness (by 132%, $P<0.0001$) and a lower percentage of non-REM sleep (by 37%, $P<0.0001$) than controls, whereas REM sleep did not change. Number of bouts of wakefulness increased by 38% in caffeine-treated rats compared to controls ($P=0.042$). During wakefulness, treatment with neonatal caffeine increased minute ventilation and tidal volume (by 37% and 25%, $P=0.024$ and $P=0.034$, respectively), but decreased occurrence and duration of apneas (by 34% and 27% respectively). During non-REM sleep, neonatal caffeine increased by 41% minute ventilation ($P=0.024$), due mostly to an enhanced respiratory frequency (by 18%, $P=0.023$). During REM sleep, there was no changes of breathing due to neonatal caffeine.
Conclusions. These results show that treatment with neonatal caffeine in newborns persistently reduces, but also fractionates, sleep in adult rats and increases ventilation across sleep-wake states. This work was funded by CIHR and G.M. was awarded by the Foundation for the Research into Children' disease.

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Differentiation of Cheyne-Stokes Respiration From Obstructive Sleep Apnea By Pattern Recognition On Finger Oximetry

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Objectives: A pattern of saw-tooth desaturation on overnight finger oximetry has been used in hospital and at home to detect individuals with sleep-disordered breathing (SDB). The purpose of this study was to test the hypothesis that Cheyne-Stokes respiration (CSR) can be differentiated from obstructive sleep apnea (OSA) by identifying symmetric sawtooth desaturations on finger oximetry.
Methods: Overnight polysomnograms (Alice 2.2) of patients with CSR or OSA were stripped back to 2 channels : oxygen saturation by finger oximetry and heart rate printed at the same rate as usually obtained from downloads of home finger oximetry. Twenty-three one hour runs of SDB events (9 CSR, 14 OSA) from 10 patients were reviewed by six independent observers blinded to event type.
CSR/ OSA events were chosen to be of similar severity with respect to number of events/unit time and depth of desaturation. The reviewers were asked to identify whether each run was symmetric (CSR) or asymmetric (OSA).
Results: The six observers had an overall average accuracy of 70% +/- 5% (1 S.D.) (Range 61-78%) in correctly identifying the type of SDB events and an average accuracy of 76% +/- 11% (1 S.D.) in identifying CSR from finger oximetry patterns.The interobserver variability was high but independent of whether the observer was a sleep physician(2) a non-sleep physician(2) or a sleep technologist(2).
Conclusions: Symmetric desaturations can be identified with a moderate degree of accuracy simply from the symmetry of the signal. The identification of Cheyne-Stokes respiration prior to full polysomnography may lead to more aggressive therapy for CHF or earlier consideration of adaptive servoventilation.

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Clustered Obstructive Apneas Induce Long-Term Facilitation of Upper Airway Motor Outflow in Spontaneously Breathing Rats In-Vivo

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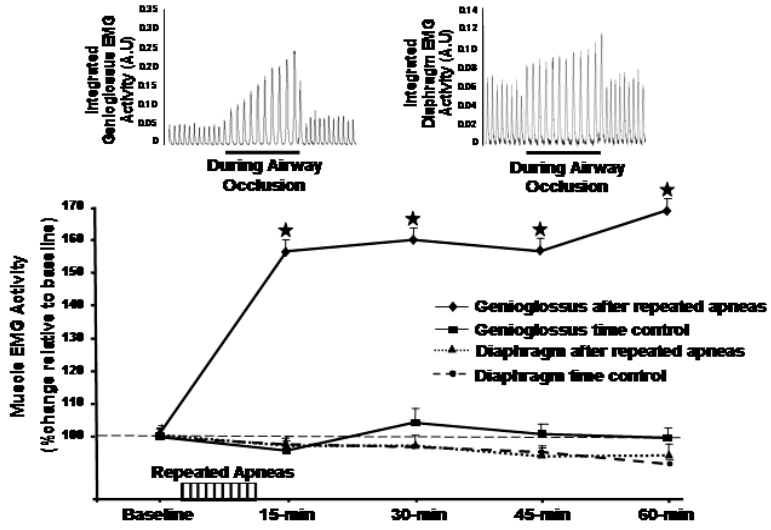
Objectives: Respiratory long-term facilitation (LTF) is a long-lasting increase in respiratory motor outflow induced by episodic but not continuous hypoxia. While three 5-minute periods of hypoxia have been shown to induce LTF, it is unknown if brief periods of asphyxia produced by apneas (as seen in obstructive sleep apnea) also induce LTF. Therefore, the aim was to determine whether clustered (repeated) airway occlusions evoke respiratory LTF. **Methods:** Experiments were performed on anesthetized, tracheostomized, spontaneously breathing adult male Sprague-Dawley rats (n=17). Respiratory motor activity was determined by recording the EMG activity of both diaphragm and genioglossus muscles. Airway occlusions (apneas) were induced by obstructing tracheal airflow using a specially-constructed device. Following a 30-45 minute stabilization period, one of 2 experimental protocols was executed. Protocol 1: rats were not exposed to apneas, and motor activity was recorded for 120 minutes (n=8). This group served as the control. Protocol 2: respiratory motor activity was recorded for 60 minutes before and after exposure to ten 15-second airway obstructions, each separated by 1 minute (n=9). **Results:** Respiratory frequency and amplitude of both the diaphragm and genioglossus muscle activities remained unchanged in the control experiments ($P>0.05$; fig. 1). During each occlusion, the inspiratory amplitude of both diaphragm and genioglossus muscles increased. After the ten occlusions, the amplitude of the genioglossus inspiratory activity transiently returned toward baseline levels and then over the subsequent 60 minutes, increased to levels significantly greater than baseline (168 ± 12%; $P<0.05$; fig. 1). Respiratory frequency and the amplitude of diaphragm inspiratory activity remained stable and were unchanged during the post-apneic period ($P>0.05$; fig. 1). **Conclusions:** Repeated airway obstructions evoke LTF in genioglossus motor activity. Accordingly, we suggest that respiratory LTF of upper airway muscles may be a protective mechanism for maintaining airway patency, which may play a role in obstructive sleep apnea.



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Impaired Gabaergic and Glycinergic Neurotransmission Induces REM-Sleep Behaviour Disorder (RBD) In Transgenic Mice

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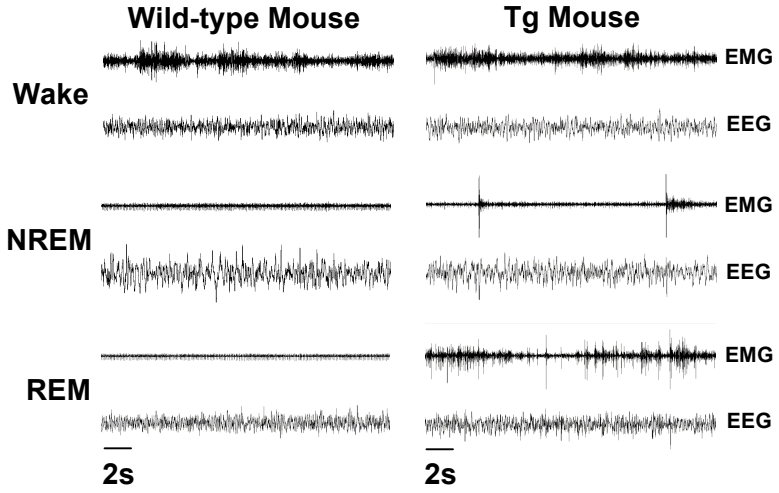
Introduction: Chronic RBD is a neurological disorder that is characterized by excessive phasic muscle activity in REM sleep, which often leads to disturbed sleep and physical injury. It is also a harbinger of neurodegenerative disorders, with 80-90% of RBD patients eventually developing Parkinson's disease or other synucleinopathies. Although its cause is unknown, RBD is effectively treated with the benzodiazepine clonazepam (a $GABA_A$ agonist). This suggests that dysregulation of the endogenous inhibitory processes that normally suppress phasic muscle activation in REM sleep may underlie the exaggerated motor activity in RBD. We therefore hypothesize that transgenic mice with impaired $GABA_A$ and glycine receptor transmission would have excessive motor activity in REM sleep and therefore exhibit an RBD phenotype.
Methods: To test this hypothesis, we used a transgenic mouse model in which both GABAergic and glycinergic neurotransmission is severely down-regulated (Becker et al., J. Neurosci, 22:2505-12, 2002). To characterize levels of somatic muscle activity, we recorded both EEG and neck EMG activity across the sleep-wake cycle in freely-behaving transgenic (Tg, n=4) and wild-type mice (Wt, n=4).
Results: While Tg mice have normal sleep-wake architecture, they have abnormal motor activity during sleep, and particularly in REM sleep. Using both videography and EEG/EMG activity, we observed that all Tg mice exhibited a clear RBD phenotype. They presented with overt periods of vigorous limb movements and jerks. Compared to Wt mice, Tg had a 217% (P=0.016) increase in muscle activity during REM sleep. Although basal levels of muscle activity were similar in Tg and Wt mice during both waking and NREM sleep, all Tg mice had regular myoclonic twitches in NREM sleep.
Conclusions: We conclude that: 1) GABAergic and glycinergic processes regulate motor suppression in both REM and NREM sleep; and, 2) impaired inhibitory neurotransmission may underlie RBD.



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Glutamatergic Control of Somatic Motoneurons In Freely-Behaving Rats

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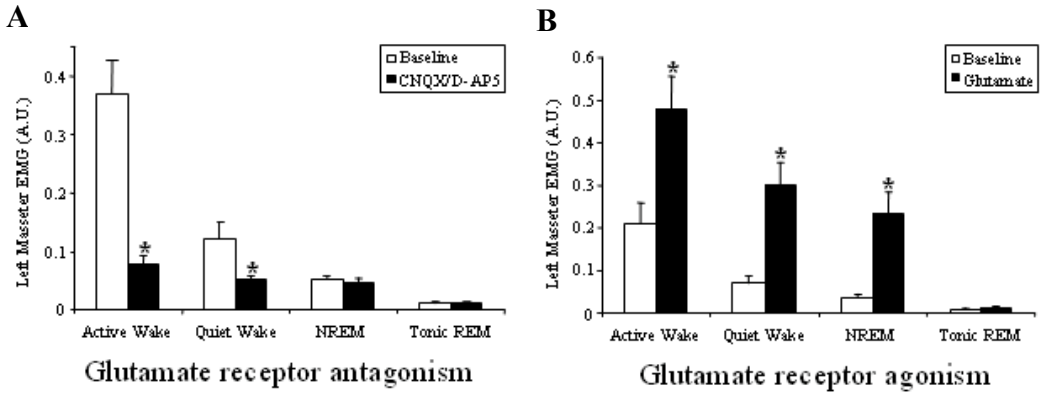
Introduction: Skeletal muscle activity is potently suppressed in sleep and particularly during REM sleep. Abnormalities in muscle tone underlie most of the major sleep disorders including narcolepsy, REM-sleep behaviour disorder and obstructive sleep apnea. The biochemical mechanisms that mediate suppression of muscle tone in sleep are unclear. In the current study, we hypothesize that withdrawal of excitatory glutamatergic inputs onto somatic motoneurons may be responsible for sleep-dependent reductions of muscle tone.

Methods: To test this hypothesis, we exogenously applied (using microdialysis probes) either glutamatergic agonists (glutamate, NMDA and AMPA) or antagonists (CNQX and D-AP5) onto trigeminal motoneurons in freely behaving rats (n=24) while recording masseter EMG activity across the natural sleep-wake cycle.

Results: Glutamate receptor antagonism led to significant decreases in masseter activity in waking (p=0.001), but had no effect during either NREM (p=0.879) or tonic REM sleep (p=0.939) (figure A). However, we found that blockade of non-NMDA receptors abolished phasic muscle activity during REM periods. While application of glutamatergic agonists significantly increased masseter activity during both waking (p=0.001) and NREM (p=0.002), they had no effect on EMG activity in tonic REM sleep (p=0.916; figure B); however, glutamatergic agonists significantly enhanced muscle activity during phasic REM periods.

Conclusions: We conclude that: 1) glutamatergic inputs play a predominate role in motoneuron excitation during wakefulness but play a minor role during NREM and tonic REM sleep; 2) since exogenous application of glutamate onto motoneurons is unable to overcome the atonia of REM sleep, other powerful inhibitory mechanisms must be involved; and, 3) phasic REM events are mediated by glutamate acting on non-NMDA receptors.



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REM Sleep Behavior Disorder Predicts Cognitive Impairment in Parkinsons's Disease

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Objective: Cognitive impairments affecting memory, executive functions, and visuospatial processing are well documented in Parkinson's disease (PD) regardless of the presence of dementia. Although more than one third of patients with PD have REM sleep behavior disorder (RBD), a parasomnia related to brainstem neuronal dysfunctions, very little is known about its association with cognitive impairment in PD. The aim of this study is to determine the impact of the presence of RBD on the cognitive profile of nondemented patients with PD.
Methods: Thirty-four nondemented patients with PD (18 with RBD and 16 without RBD) and 25 healthy controls matched for age and educational level underwent sleep laboratory recordings and a comprehensive neuropsychological assessment. Standardized tests assessing verbal memory (Rey auditory verbal learning test), executive functions (letter and semantic fluency test, trail making test parts A and B, and Stroop colour word test), visuospatial (Rey complex figure and block design from the WAIS-III) and visuoperceptual (Bells test) functions have been administrated. One-way ANOVAs were used to assess between-group differences.
Results: No main effect of Group was observed for either mini-mental state examination score, Beck-II depression score, PD clinical variables, or mean dopaminergic medication dosage. Patients with PD and concomitant RBD showed significantly poorer performance on tests measuring verbal memory, executive functions, as well as visuospatial and visuoperceptual processing compared to both PD patients without RBD and control subjects. Patients with PD without RBD had no detectable cognitive impairment compared to controls.
Conclusions: This study shows that cognitive impairment in nondemented patients with PD is closely related to the presence of RBD, a sleep disturbance that was not controlled for in previous studies assessing cognitive deficits in PD.

Financial support: FRSQ and CIHR

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EEG Spectral Analysis in Wakefulness REM and NREM Sleep Following Sport-Related Concussions

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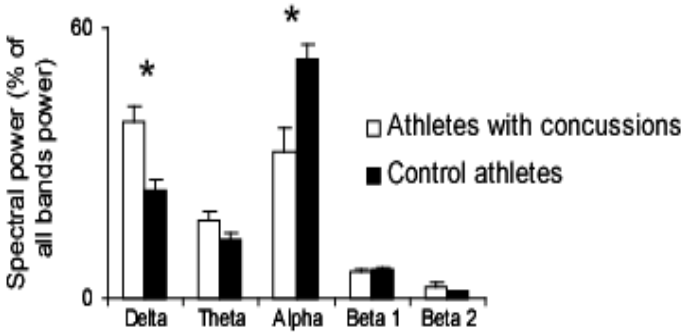
Objective: Athletes who sustain concussions generally suffer cognitive and neurobehavioral symptoms in the hours, weeks, or sometimes months, following their injury. Sleep and vigilance disorders are amongst the most reported symptoms; however, no polysomnographic study has been done in this population. The aim of this study was thus to investigate the effects of sport-related concussions on sleep architecture and on quantitative EEG (QEEG) in wakefulness, REM and NREM sleep.

Methods: Ten athletes who experienced at least one concussion during the last year (total history of 4.6 ± 2.1 concussions) and 11 non-concussed athletes were recorded for two consecutive nights in the laboratory and during a 10-minute period of wakefulness the next morning. They completed the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale. Spectral analysis (Fast Fourier transforms) of the EEG recorded during wakefulness, REM and NREM sleep was performed. Nighttime course of delta activity was also determined by using a nonlinear regression analysis on all-night NREM sleep. Group differences were measured with Student t-tests and analyses of variance.

Results: Concussed athletes reported worse sleep quality and more daytime dysfunctions than control athletes ($p<0.01$). These athletes had significantly more delta activity during wakefulness ($p<0.01$) and less alpha activity in comparison with controls ($p<0.05$; see Fig. 1). Their subjective sleep quality and daytime dysfunctions cannot be attributed to disturbed sleep, because no between-group difference was found on any polysomnographic variable or on sleep QEEG variables. Moreover, no group difference was obtained for the delta activity dissipation.

Conclusion: Concussions in athletes were associated with abnormal waking EEG and these anomalies may be associated with the daytime dysfunctions reported by these athletes. In spite of their subjective complaints in sleep quality, no change was observed in sleep parameters and in sleep QEEG. Sport-related concussions are thus associated with wakefulness problems rather than sleep disorders.

Figure 1. Spectral power for waking EEG in athletes with concussions and control athletes



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Strong Association Between Cyclic Alternating Pattern A1 and SWA in Sleep Bruxers and Control Subjects

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Objectives: The cyclic alternating pattern (CAP) is a spontaneous periodic EEG activity observed during NREM sleep. It is divided into phase A (A1, A2, A3) and phase B. It was previously reported that sleep bruxism (SB) is associated to CAP A3. This study aims to examine CAP phase A of SB subjects in comparison to control subjects.

Methods: 8 SB subjects (mean age 22.8 (min-max; 21-27); 5M, 3W) were compared to 8 control sleepers (mean age 23.0 (20-26); 4M, 4W). SB subjects were selected according to tooth grinding history ≥ 3 nights/week and clinical exam. Polysomnographic recordings were made for all subjects over 2 consecutive nights for SB diagnosis and to rule out other sleep disorders (e.g., apnea, periodic limb movement). Sleep variables, masticatory muscle tone of masseter and suprahyoid muscles were analysed using Harmonie software (Stellate, Canada). Statistical analysis was assessed for the first four sleep cycles of each study group using ANOVA and t-tests. Correlations were done between sleep (slow wave activity and micro-arousals) and CAP variables. Statistical analyses were done with SYSTAT (USA) and SPSS (USA).

Results: CAP A1 rate is strongly correlated to SWA in both SB ($r=0.70$, $p<0.001$) and control subjects ($r=0.79$, $p<0.001$). A similar distribution is observed between these two variables with a quadratic distribution of NREM sleep ($p<0.02$) and a linear decrease from the first to the fourth sleep cycle ($p<0.01$). Both CAP A2 and A3 rates peaked strongly and slightly during the transition phase between NREM and REM sleep within each cycle, respectively. However, only the CAP A1 rate was significantly different between SB and normal subjects.

Conclusions: The strong association between CAP A1 rate and SWA seem to suggest neurological synchronisation during NREM sleep. While CAP A1 should be further validated in SB, the higher value of CAP A1 rate observed in normal subjects in comparison to SB subject would suggest an altered sleep microstructure due to tooth grinding during sleep.

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The Prevalence of Headaches, Neck Pain and Fatigue in Sleep Bruxism

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Objectives: Sleep bruxism (SB) has been studied for over a decade in our sleep research laboratory. The objective of this retrospective analysis is to report the prevalence of headaches, neck or shoulder pain and morning fatigue in SB subjects.
Methods: SB subjects were selected according to tooth-grinding history (>3 nights/week), without trauma or chronic pain history. All subjects completed questionnaires for SB/pain diagnostic and sleep disorders (Canadian Sleep Society). SB diagnosis and absence of other sleep disorders were confirmed by 2 nights of polygraphic recordings. Sleep and SB variables were analyzed based on previously validated criteria. The following polygraphic criteria were used to identify SB subjects: > 4 SB episode/hour of sleep, > 25 SB bursts/hour of sleep and > 1 episode with grinding noise. SB subjects were divided in 2 groups based on afore mentioned criteria: low SB subjects failed in 2 out of the 3 criteria and high SB subjects met at least 2 criteria. A total of 21 controls (mean age±SEM: 22.90±0.65), 38 low SB (26.71±0.88) and 41 high SB (24.78±0.70) subjects were selected. Chi-square and odds ratios (OR), with their 95% confidence intervals were used to compare answers between both SB groups and between each SB groups and the control group.
Results: Headaches or migraines, occurring occasionally to frequently, were reported twice more often in SB groups (low SB: 52.6%; high SB: 51.2%) than in controls (19%), with OR of 4.7 [1.3-16.7] and 4.5 [1.3-15.6] respectively. Reported morning headaches were also significantly higher in SB groups (low SB: 32.4%; high SB: 17.6%) than in controls (0%). Furthermore, neck or shoulder pain were reported more frequently in both SB groups (low SB: 68.4%; high SB: 68.4%) in comparison to controls (23.8%), with high OR (low SB: 6.9 [2.1-23.3], high SB: 5.7 [1.8-18.9]). Fatigue upon awakening was reported 5 times more in SB groups (low SB: 43.8%; high SB: 41.2%) than in controls (8.3%), with nearly significant OR (low SB: 8.6 [0.98-74.4], high SB: 7.7 [0.89-66.6]).
Conclusion: High prevalence of reported headache/migraine and neck/shoulder pain in SB subjects support the need for further investigations to study possible common mechanisms and association.

Research supported by: **CIHR, FRSQ, CFI**

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Cognitive Impairments In REM Sleep Behavior Disorder Patients

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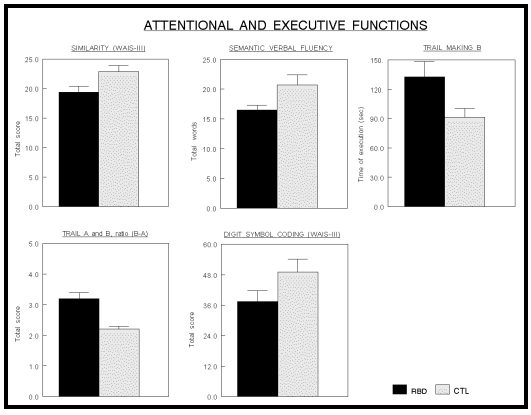
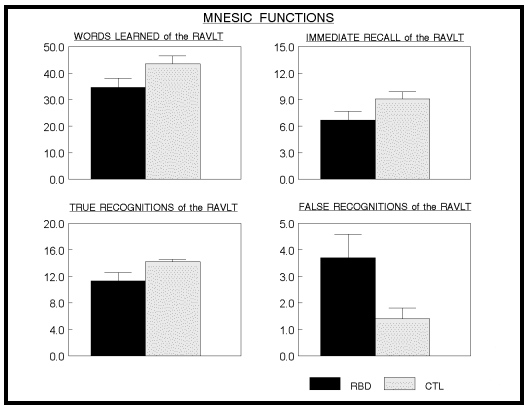
Introduction: A recent study performed in our laboratory showed a slowing of waking EEG in RBD patients compared to normal controls (Fantini *et al*., 2003). The aim of the present study was to verify the functional significance of this EEG slowing in relation to the cognitive performance of these patients.
Methods: Fourteen men (mean age: 66.64 ± 2.05y; mean educational level: 12.21 ± 1.08y) meeting the ASDA criteria for idiopathic RBD and fourteen healthy men matched for age and education (mean age: 65.73 ± 2.24y; mean educational level: 13.54 ± 1.05y) underwent one night of polysomnographic recording, a 10-minute waking EEG recording and a full neuropsychological assessment. Quantitative EEG analyses were performed for frontal (FP1 + FP2 + F3 + F4 + F7 + F8), central (C3 + C4), parietal (P3 + P4), occipital (O1 + O2) and temporal (T3 + T4 + T5 + T6) regions during wakefulness.
Results: Compared to controls, RBD patients had more Delta power ($p<0,005$) and more Theta power ($p<0.02$) in all cortical regions. These patients showed significantly poorer performance in Semantic Verbal Fluency ($p<0.01$), Similarity task ($p<0.02$), Digit Coding task ($p<0.05$) and Trail Making task ($p<0.0007$). Moreover, we found memory impairments in Word Memory Learning task (Rey Auditory Verbal Learning Test) ($p<0.05$), in immediate recall ($p<0.03$), and for the True ($p<0.01$) and False ($p<0.04$) recognition conditions. However, correlations between quantitative EEG and cognitive abnormalities were not statistically significant.
Conclusion: The present results show that both waking EEG and cognitive performance of male RBD patients are altered independently compared to controls. However we did not find visuospatial constructional or visuospatial learning dysfunction as reported previously in this patient population (Ferini-Strambi *et al*., 2004). Taken together, these results are congruent with the hypothesis that RBD is an early manifestation of a neurodegenerative disease.
Supports: Canadian Institutes of Health Research (CIHR) and Fonds de Recherche en Santé du Québec (FRSQ).





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Periodic Leg Movements During Sleep Are Associated With Cardiovascular Changes in healthy Normal Subjects

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Objectives

Several studies have shown a high prevalence of periodic leg movements during sleep (PLMS) in healthy normal subjects, especially in older populations. The clinical significance of PLMS in normal non-complaining subjects is still controversial. Recently, we have demonstrated that PLMS in patients with restless legs syndrome (RLS) are associated with a significant increase not only in heart rate (HR), but also in blood pressure (BP). The aim of this study was to assess HR and BP changes associated with PLMS with and without micro-arousals (MA) in healthy subjects.

Methods

We studied eight normal subjects (3 women; age=44.4±10.9 yrs) with normal BP at rest (systolic BP(SBP)=108.8±11.1mmHg, diastolic BP(DBP)=68.4±9.1mmHg). Beat-to-beat non invasive BP (Portapres) was continuously recorded during one night of polysomnography. Ten PLMS with MA and 10 PLMS without MA were analysed in each subject. Only movements which were separated by at least 20 seconds were selected for analysis, to avoid the overlapping of cardiovascular responses. For each movement, the increase in HR, SBP and DBP was calculated as the difference between the peak value and the baseline (mean value for beats –10 to –4, before the movement). The within-subject comparison of increments associated with PLMS with and without MA was assessed by paired t-tests.

Results

Significant increments of HR and BP were associated with all PLMS. PLMS with MA, compared to PLMS alone, were associated with a higher rise of HR(11.0±4.3 versus 6.5±2.3bpm,p=0.003), SBP(21.9±6.1 versus 14.2±4.5mmHg,p=0.0006) and DBP(12.3±4.7 versus 6.9±1.8mmHg,p=0.003).

Conclusion

These results show important increases of HR and BP in association with PLMS in normal non-complaining subjects, similar to what we observed in RLS patients. Studies have shown that enhanced BP variability is associated with the development of vascular and cardiac damage. PLMS-related repetitive BP fluctuations could thus be harmful to the cardiovascular system, even in normal subjects.

Supported by the Canadian Institutes of Health Research, "Fonds de la Recherche en Santé du Québec" and the Canadian Government Senior Chair on Sleep Disorders (J Montplaisir).

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Somnambulistic Episodes in Sleepwalkers With A High Periodic Leg Movements in Sleep (PLMS) Index are Rarely Associated With PLMS

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Objectives: Sleepwalking has been reported in association with other sleep disorders including periodic leg movements in sleep (PLMS). However, whether or not PLMS actually trigger somnambulism is unknown. We previously showed that sleep deprivation significantly increases sleepwalking frequency in predisposed individuals. The goal of the present study was to asses PLMS in relation to somnambulistic episodes during normal sleep and following sleep deprivation in sleepwalkers with a high PLMS index.
Methods: Seven sleepwalkers (6 males, 1 females; mean age: 30.7±9.9 years) with a PLMS index >10 were investigated during a baseline night and during recovery sleep following 25 hours of sleep deprivation. PLMS were defined as movements lasting 0.5 to 5 seconds, separated by intervals of 4 to 90 seconds and occurring in series of at least 4 consecutive movements. PLMS were analysed during sleep and in relation to each somnambulistic event.
Results: Seven somnambulistic episodes were recorded at baseline and 19 during recovery sleep. Sleep deprivation significantly increased the mean frequency of the episodes (1.0±0.8 vs 2.7 ± 1.4, $p<0.05$) while significantly decreasing subjects' mean PLMS index (19.5±5.5 vs 8.3±5.5, $p<0.05$). The proportion of somnambulistic episodes preceded within 90 seconds by a single leg movement part of a PLMS significantly decreased from baseline (3/7 or 43%) to recovery sleep (0/19 or 0%), $p< 0.03$. Of the 26 episodes, only 2 (both at baseline) were preceded within 30 seconds by an individual leg movement part of a PLMS and no leg movements were observed immediately prior to any of the episodes' onset.
Conclusion: The data indicate that most sleepwalking episodes in adults with a high PLMS index are not associated to or triggered by PLMS. Moreover, significant increases in the frequency of sleepwalking episodes after sleep deprivation are accompanied by significant decreases in PLMS frequency.
Funding Support: Research supported by the Canadian Institutes of Health Research, the Natural Sciences and Engineering Research Council of Canada and the Fonds de la Recherche en Santé du Québec.

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Does Melatonin Affect The Circadian Variation of RLS Symptoms?

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INTRODUCTION : A recent study conducted in our laboratory showed that melatonin secretion started approximately two hours before the worsening of restless legs syndrome (RLS) symptoms at night and could perhaps explain their circadian variation. The aim of this study was to verify the hypothesis of the direct involvement of melatonin in this phenomenon. The effects on RLS symptoms of the administration of melatonin and conversely those of its suppression by the exposure to bright light were studied.

METHODS : Eight RLS subjects (2 men, 6 women, mean age = 53.3 ± 9.1 years) were studied in three conditions. First, the control condition allowed to measure the PLMS index and the severity of the sensory and motor symptoms during the Suggested Immobilization Test (SIT). The second and third conditions were the administration of melatonin (3 mg at 7:00 pm) and the exposure in the bright light (3000 lux from 7:00 pm to midnight), respectively. These two experimental nights were separated by one week and the order of those conditions was inverted for half the subjects. The SIT was administered twice for each condition: before the habitual appearance of symptoms (from 7:30 pm till 8:30 pm) and after (from 11:00 pm till 00:00 am).

RESULTS : The administration of exogenous melatonin and exposure to bright light did not significantly influence sleep architecture, PLMW, PLMS or the sensory symptoms experienced during the SIT. However, a difference on motor symptoms during the 1st SIT was observed between the two experimental conditions (exogenous melatonin increased the number of leg movements whereas the bright light decreased it; $p = 0.004$). No effect was found for the second SIT.

CONCLUSION : Although melatonin may have a certain influence, notably on motor symptoms, other mechanisms could explain in a more direct way, the circadian variation of RLS symptoms.

Supported by the Canadian Institutes of Health Research (CIHR) and the Fonds de Recherche en Santé du Québec (FRSQ).

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Daytime Consequences of Insomnia: A factor Analysis

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Introduction: Although insomnia is frequently associated with complaints of significant daytime impairment, little is known regarding the nature of these consequences. This study aimed at defining the nature of self-reported daytime consequences of insomnia and at identifying predictors of these complaints.

Methods: Participants were 160 adults (aged 30-72, mean = 50.3; 60.6% women) meeting criteria for chronic insomnia recruited in the context of a larger treatment study. For the present study, only baseline data were used. Daytime consequences were measured by 14 indicators: five Multidimensional Fatigue Inventory (MFI) subscales, eight SF-36 Health Survey subscales, and the Insomnia Severity Index item asking to what extent sleep difficulties interfere with daily functioning. Potential predictors of daytime consequences were socio-demographic, sleep (continuity and architecture), health (number of medical conditions, frequency of physical activity), and clinical (psychiatric co-morbidities, depressive and anxiety symptoms assessed by the BDI and BAI) variables.

Results: A factor analysis using the promax oblique rotation method was performed on the 14 indicators. A three-factor structure was selected based on parsimony and interpretability. Variables included in each factor were: F1) all MFI subscales, SF-36 vitality subscale, and ISI interference item; F2) SF-36 physical functioning, role physical, bodily pain, and general health subscales; and F3) SF-36 social functioning, role emotional, and mental health subscales. Correlations between factors ranged from r = .22 (F2 vs. F3) to .55 (F1 vs. F2). Stepwise logistic regressions were then performed on the aggregate standardized score of each factor. Significant predictors were: for F1 (R^2 = .473): higher BDI, younger age, longer wake after sleep onset (diary), and higher BAI; for F2 (R^2 = .406): younger age, higher number of medical conditions, being unemployed, higher BDI, lower sleep efficiency (PSG), higher BAI, presence of past psychiatric diagnosis, and longer time in bed (diary); for F3 (R^2 = .450): higher BDI, younger age, higher number of medical conditions, more frequent physical activity, and lower sleep efficiency (PSG).

Conclusions: These results suggest that daytime impairment in individuals with insomnia can be classified as related to fatigue, physical health, and mental health. Furthermore, these different subtypes of daytime consequences are explained by distinct sets of variables. Confirmation of these findings with more objective data of daytime impairment is needed.

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Meta-Analysis of Sleep Changes During Placebo Administration Compared To Untreated Groups in Insomnia Treatment Trials

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Introduction. Insomnia is presumed to be a condition susceptible to placebo effects. However, few studies have systematically examined how sleep changes with the administration of a placebo. Accordingly, a meta-analysis was conducted to assess the magnitude of changes from baseline after psychological and pharmacologic placebo interventions and compared these changes to those of untreated groups.
Methods. Medline, PsychInfo and Current Contents databases (1990 –2004) were searched for primary insomnia treatment studies using a randomized controlled parallel-group design. Thirty-four studies (*n*=1392 subjects) met inclusion criteria; twenty-three used a pharmacological placebo (*n*=1163), four used a psychological placebo (*n*=81), and seven used a waitlist condition (*n*=148). Placebo treated and untreated groups were issued from different trials. Effect sizes were computed for each end-point variable based on subjective (patient-reported) and polysomnographic measures. Between-group comparisons were then performed using a random effects model analysis.
Results. Significant pre-post changes were observed in the pharmacological placebo condition on several sleep parameters, both on objectively and subjectively measured outcomes (objective and subjective SOL and TST and subjective WASO). Although a tendency was observed for objective SOL, only the changes on subjective SOL and TST in the pharmacological placebo condition were significantly different from the corresponding changes in the untreated group. No differences were significant for the psychological placebo groups.
Conclusions. Although the present findings suggest that sleep may significantly change in response to a pharmacological placebo, conclusions remain tentative because of possible confounds that may arise when comparing groups issued from different trials. This study highlights the complexity of studying placebo reactivity in insomnia treatment trials and stresses the need for further research addressing this question.

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Utilization of Hypnotic Medications Among Cancer Patients

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Insomnia affects between 30 to 50% of cancer patients. Hypnotic medications are the most frequently used treatment for sleep difficulties in the general population. However, only a few studies using convenient samples have assessed the frequency of utilization of these medications in cancer patients. Also, the numerous negative consequences associated with the utilization of hypnotics (e.g., diurnal effects, risk of dependence with chronic use) emphasize the importance of identifying the characteristics of patients at risk to use such medications to treat their insomnia.
Objectives. The goals of this study were: (1) to estimate the frequency of utilization of hypnotic medications in a large sample of randomly selected (from a provincial medical databank) patients recently treated for cancer and; (2) to identify the demographic, psychosocial and medical factors associated with the usage of hypnotic medication.
Methods. Five thousands patients who had been treated for breast, prostate, lung or colorectal cancer at L'Hôtel-Dieu de Québec were solicited by mail to take part in this study. Among them, 1 984 completed a battery of self-report scales assessing the utilization of hypnotic medications and some potential risk factors (e.g., physical symptoms, psychological distress).
Results. The results showed that 22.6% of the participants were currently using a hypnotic medication. The logistic regression revealed that older age, difficulties initiating sleep, major life events experienced in the past 6 months, higher levels of anxiety and urinary symptoms, past or present psychological difficulties, lower levels of role functioning, greater use of opiates and current or past chemotherapy were found to be associated with an increased likelihood to use a hypnotic medication.
Conclusions. This study shows that hypnotic medications are more frequently used by cancer patients than the general population (5.8%; Santé Canada, 1994) and it identifies some characteristics associated with their use that could be targeted in a prevention program.

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Sleep and Fatigue in Individuals with Insomnia

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Objectives : Although fatigue is the most consistent complaint among individuals with insomnia, its relation to sleep impairment remains poorly understood. The aim of this study was to describe different patterns of relations between sleep and fatigue among individuals with insomnia. **Methods :** The current analysis used baseline data collected in the context of a larger treatment study. Participants were 160 adults who met the diagnostic criteria for chronic insomnia (aged 30-72, mean = 50.3; 60.6% women). All underwent three nights of polysomnographic (PSG) recordings and completed the Multidimensional Fatigue Inventory (MFI). Sleep variables and scores obtained on the MFI were standardized into z-scores. For each participant, a composite score of sleep impairment averaging z-scores for PSG-defined sleep onset latency, wake after sleep onset and total sleep time was then derived. Similarly, a composite score of fatigue averaging z-scores for the five sub-scales of the MFI was computed. Participants also completed the Beck Depression Inventory (BDI), the Beck Anxiety Inventory (BAI) and the SF-36 Health Survey. **Results :** Composite sleep impairment and fatigue scores were submitted to a hierarchical cluster analysis using Ward's method. A 4-cluster solution was selected based on parsimony, interpretability and cluster sizes ($R2 = 0.68$). Individuals were thus classified as having either 1) both severe insomnia and severe fatigue (SI-SF; n = 15); 2) severe insomnia but mild fatigue (SI-MF; n = 15); 3) mild insomnia but severe fatigue (MI-SF; n = 68) or 4) both mild insomnia and mild fatigue (MI-MF; n = 61). Those with SI-SF had greater self-reported sleep problems compared to all other clusters, while those with SI-MF had significantly more impaired PSG-defined sleep. While general and mental fatigue were higher in both SF clusters compared to MF clusters, physical fatigue and decrease in activities were different across all 4 clusters, those with SI-SF being the most impaired, followed by those with MI-SF, those with SI-MF and finally those with MI-MF. Compared to both clusters with MF, those with SF had significantly higher BDI ($p < .01$) and lower SF-36 scores ($p < .01$). **Conclusion :** Results suggest different patterns of sleep-fatigue relations among individuals with insomnia, such that fatigue seems to occur independently of PSG-defined sleep difficulties and appears paralleled by more depressive symptoms and a greater decrease in quality of life.
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Cardiovascular Changes Associated with Micro-Arousals in Primary Insomniacs and Normal Subjects

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Introduction

Subjects with primary insomnia have higher sympathetic activity during steady sleep compared to controls. The aims of this study were, firstly, to compare heart rate (HR) and blood pressure (BP) changes associated with micro-arousals (MAs) in subjects with chronic primary insomnia and controls. Secondly, to measure the relationship between MA-related cardiovascular changes and demographic, clinical and sleep variables.

Methods

We studied 7 subjects with chronic primary insomnia (4 women; age 44.8 ± 6.6 years; BMI=25.1 ± 5.3 Kg/m^2) and 7 age, sex and BMI matched controls. Subjects underwent a measure of brachial arterial BP in sitting position in the evening, followed by full polysomnography, which also included beat-to-beat non invasive BP (Portapres). Ten MAs occurring in stage 2 NREM sleep were selected for cardiovascular analyses in each subject. For each MA the increase (Δ) in HR, systolic BP (SBP), diastolic BP (DBP) were calculated as the difference between the peak value and the baseline (i.e., the mean value for the beats -10 to -2, before the MA). The increments associated to MAs were compared between insomniacs and controls by unpaired t-test. Correlation analyses between cardiovascular changes (ΔHR, ΔSBP and ΔDBP) and age, BMI, clinical BP, and sleep measures (sleep latency, sleep efficiency, % sleep stages, MA index) were also assessed in all subjects pulled together.

Results

MA-related cardiovascular responses were not different between insomniacs and controls (ΔHR: 5±3 vs 6±3 bpm, p=ns; ΔSBP: 19±5 vs 16±6 mmHg, p=ns; ΔDBP: 9±3 vs 8±3 mmHg, p=ns). There was no correlation between cardiovascular changes and age and sleep variables. By contrast, ΔSBP correlated with BMI ($R=0.57$, $p<0.05$) and evening brachial SBP ($R=0.71$, $p<0.01$).

Conclusion

Cardiovascular responses associated with MAs do not differ between insomniacs and controls. BMI and clinical blood pressure appear to be important determinants of blood pressure responses to cortical arousals.

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Incidence and Risk Factors of Insomnia in a Population-Based Sample

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Objectives: This study estimated the incidence of insomnia and examined its risk factors in a population-based sample of self-defined good sleepers followed over a one-year period.
Methods: Participants were 394 self-defined good sleepers randomly selected from the adult population. They completed three postal evaluations over a one-year period (i.e, baseline, six months and twelve months later) which included assessment of sleep and insomnia, psychological and personality variables, stressful life events and coping skills, and health-related quality of life. Participants were categorized into three subgroups: (a) good sleepers (i.e., participants who remained good sleepers at the three evaluations), (b) incident cases of insomnia symptoms (i.e., good sleepers who developed insomnia symptoms either at six month or twelve month follow-up) and (c) incident cases of insomnia syndrome (i.e., good sleepers who developed an insomnia syndrome either at six month or twelve month follow-up).
Results: One-year incidence rates were 31.2% for insomnia symptoms and 8.6% for insomnia syndrome. When incidence rates were computed only for those individuals without any prior history of insomnia at baseline assessment, the rates decreased to 22.7% (symptoms) and 4.7% (syndrome) respectively. Incident cases of insomnia syndrome presented a pre-morbid psychological vulnerability, as evidenced by higher arousability predisposition and higher anxiety and depressive symptoms at baseline. A logistic regression indicated that five variables were significantly associated with a new onset of an insomnia syndrome (i.e., family history of insomnia, previous episode of insomnia, arousability, the NEO-FFI openness subscale and the SF-12 general health subscale). Incident cases also reported significantly more negative life events than good sleepers within the six months preceding the onset of their insomnia and an increase of bodily pain concomitant to the onset of insomnia.
Conclusions: These results provide evidence that several characteristics previously observed among individuals with insomnia in several cross-sectional studies may represent important predisposing factors to new onset of insomnia. Improved knowledge of those risk factors could guide the development of more effective public health prevention and intervention programs for insomnia.

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Development and Dissemination of an Insomnia Clinical Practice Guideline: The Alberta Experience

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Objectives:The objective of this study was to address the problem of inadequate training and guidance in the diagnosis and management of insomnia in primary care medicine. A collaborative effort between the University of Calgary, Department of CME/PD and the AMA Toward Optimized Practice (TOP) program was initiated to address this issue. The project was designed to develop an insomnia clinical practice guideline and an effective tool for disseminating the guideline. Interim results of this project and future plans for evaluating the efficacy and effectiveness of the guideline and dissemination process are reported here.
Methods:The AMA/TOP program recruited a group of sleep specialists, family physicians, a psychologist and clinical pharmacist to develop an insomnia clinical practice guideline. A three-hour, small group, case-based Insomnia course was developed and accredited by the College of Family Physicians of Canada. The guideline was embedded into the course content. The course was used as the tool for dissemination of the guideline. A standard self-report, of the participant's "commitment to change" clinical practice was performed at the end of the course. Evaluation of the participant's ability to maintain the commitment to change was assessed three months later.
Results: Seventy-five physicians participated in five courses. Forty-one of the participants (41/75) submitted their commitment to change (response rate 54.7%). Forty-one participants (41/41) submitted the 3-month follow-up commitment to change (100% response rate). Table 1 summarizes the frequency of individuals who maintained their commitment to change. Greater than 90% of those who completed the commitment to change task were partially or fully able to maintain their commitment to change.

Table 1
Commitment to Change Contract: Three Months Post-Change

	Percent (%)
No longer plan to implement change	00.72
Could not implement change	07.44
Partially implemented change	35.52
Implemented change	56.34

Conclusion: Insomnia is a common problem in primary care and family physicians have limited resources to guide assessment and management. The three-hour CME program with an embedded insomnia clinical practice guideline appears to be an effective strategy for the dissemination and implementation of the clinical practice guideline. There are substantial limitations to the generalizability of the conclusion based on the nature of the data and data collection process. However the results describe a tendency toward uptake and implementation that is worth pursuing in the future.

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Cerebral Asymmetry in Sleep: Relation with BDI and BAI Scores in Chronic Primary Insomnia

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Introduction

While central asymmetry is a characteristic of depressive symptoms during sleep, cerebral asymmetry in relation to anxious symptoms remains unknown. The objective of the present study is to document relationships between Beck Depression Inventory (BDI) and Beck Anxiety Inventory (BAI) sub-clinical scores with asymmetry during sleep in chronic primary insomnia sufferers (PI) and good sleepers (GS).

Methods

Thirteen GS and 19 INS completed both inventories before undergoing four consecutive nights of PSG recordings. The first four sleep cycles of Nights 2 and 3 were retained for spectral analysis. The frequency activity (ranging from 0.00 to 35.00 Hz) was computed (C3-C4, P3-P4, F3-F4 sites). Asymmetry scores resulted from the subtraction of the log of the left EEG power from the log of the right EEG power. Mean values of both nights were averaged.

Results

Significant Pearson correlations ($p<.05$) showed that in GS, only BAI scores correlated with REM central asymmetry in the 0.00-1.00-Hz range (r=-.56). In INS, asymmetry correlated with neither questionnaire score. The INS group was further divided in psychophysiological (Psy-I, n=13) and paradoxical (Par-I, n=6). Significant correlations were observed only for Par-I. BAI scores were related to frontal asymmetries in NREM and REM [(11.00-14.00-Hz (r=-.90; r=-.96) and 20.00-35.00-Hz (r=-.97; r=-.93) bands]. Frontal asymmetries were also observed for the 7.00-11.00-Hz band (r=-.93) in NREM and the 14.00-20.00-Hz band in REM (r=-.92). Central asymmetries were observed in the 20.00-35.00-Hz band in NREM (r=-.88) and the 11.00-14.00-Hz band in REM (r=-.88).

Conclusion

Surprisingly, BDI scores were not related to asymmetry. Asymmetry was only slightly related to BAI scores in good sleepers and not at all in Psy-I. However, in Par-I, as BAI scores increased, the asymmetry in higher EEG activity in the right central and frontal regions decreased. Cerebral asymmetry can still be detected with anxiety sub-clinical scores, and especially, in paradoxical insomnia sufferers.

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Mechanisms Explaining The Effect of Cognitive-Behavioural Therapy For Chronic Insomnia Comorbid with Breast Cancer

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Objectives. Our randomized controlled study supported the efficacy of cognitive-behavioural therapy (CBT) for chronic insomnia comorbid with breast cancer (Savard, Simard, Ivers & Morin, 2005). However, the mechanisms through which CBT leads to sleep improvements remain unknown. A secondary analysis of Savard et al.'s study (2005) was performed to investigate the potential mediating role of cognitive (i.e., decreased dysfunctional beliefs about sleep), behavioural (i.e., higher levels of treatment adherence to behavioural strategies) and non-specific factors (i.e., reduced psychological distress, higher levels of treatment expectancies and credibility, and greater levels of therapeutic alliance). **Methods.** Fifty-seven women with insomnia comorbid with breast cancer received CBT as part of the randomized controlled trial. The treatment consisted of eight weekly sessions of CBT administered in groups and combined the utilization of stimulus control, sleep restriction, cognitive therapy, sleep hygiene, and fatigue management. The participants were assessed at pre- and post-treatment, as well as at 6-month follow-up. The assessment protocol included polysomnography (PSG; 3 nights), a daily sleep diary (2 weeks), the Insomnia Severity Index (ISI), the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS), and the Hospital Anxiety and Depression Scale (HADS), a questionnaire assessing patients' treatment expectancies and credibility and a questionnaire assessing the the perception of the therapeutic alliance from both the patient's and clinician's points of view. A coding grid was also developed to assess the patient's adherence to behavioural treatment strategies. **Results.** CBT for insomnia was associated with a significant reduction of DBAS and HADS scores. Regression analyses revealed that higher initial treatment expectancies and credibility was the most consistent mediating factor of sleep improvements at post-treatment when assessed using subjective measures (sleep diary and ISI). PSG-assessed sleep improvements were best predicted by reduced DBAS scores and a better adherence to the instruction of avoiding day napping. At follow-up, subjectively-assessed sleep improvements were most importantly predicted by adherence to behavioural strategies (i.e., avoidance of day napping and arising from bed during nocturnal awakenings), while none of the potential mediators was significantly associated with sleep improvements as assessed with PSG. **Conclusions.** Results of this study suggest that cognitive and behavioural factors that are postulated to mediate the effect of CBT for insomnia are particularly influential in explaining objective sleep indices at post-treatment and subjective sleep indices at 6-month follow-up. Non-specific therapeutic ingredients, that have been associated with a so-called placebo effect, are particularly influential in explaining subjective measures of sleep improvements at post-treatment.

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Chronic Primary Insomnia: Relation Between Quality of Sleep and Cognitive Evoked Potentials Measures

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Introduction: There is intra and inter-individual variations in sleep quality in psychophysiological insomnia sufferers (Psy-I). Recently, our ERPs data (N1, P2 and N350) suggested inhibition deficits in addition to cortical arousal in Psy-I relative to good sleepers (GS). The objective of the present study is to investigate the relation between sleep quality (sleep efficiency) and the amplitudes and latencies of the different ERPs in a multi-assessment protocol.

Methods: Participants, 15 Psy-I and 16 GS, underwent four consecutive nights of PSG recordings (N1 to N4). ERPs in the evening and upon awakening were recorded on N3 and N4, with the addition of sleep-onset recordings on N4. Auditory stimuli consisted of 'standard' frequent (70 dB, 2000 Hz, .85 probability) and 'deviant' rare stimuli (90 dB, 1500 Hz, .15 probability). Sleep quality/efficiency was computed on each night. The amplitude and latency of each ERP component (N1, P2 and N350) were assessed on each recording.

Results: Pearson correlations revealed that in Psy-I sufferers, the amplitude of N1 before and during sleep-onset was negatively correlated ($p < .05$) with the objective sleep efficiency of the night preceding the ERPs recordings. As such, N1 amplitude decreased as sleep efficiency increased ($r = -.73$; $r = -.62$ respectively). In the morning, P2 amplitude significantly decreased as objective sleep efficiency increased ($r = -.56$). There were no relationships between sleep efficiency and N350 amplitude nor with the latency of any ERP component. In GS, N1 latency was longer as sleep efficiency was higher ($r = .508$, $p = .045$). No other significant relationships were observed.

Conclusion: These results suggest that as sleep efficiency decreases, arousal increases. This is especially true for Psy-I sufferers. These results highlight once again that when information processing and/or performance are assessed, the sleep quality of the night preceding the evaluation shall be documented.

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Perceived Health and Psychological Consequences Associated with Work Schedules for Regular Night and Rotating Shift Workers: The Moderating Effect of Insomnia Symptoms

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Introduction: The impact of shift work sleep disorder (SWSD) on physical and psychological functioning is poorly documented. Only a few studies demonstrated a high rate of depressive symptoms and low work productivity among night workers suspected of suffering from SWSD. This study aims at assessing the impact of the work schedule on perceived health and psychological variables of night and rotating shift workers.
Methods: The sample consisted of 192 adults (44% of women; age, M = 37.2), 20 permanent night workers, 76 rotating night shift workers, and 96 day workers selected from a larger epidemiological study. Each rotating shift worker and night worker was paired with a day worker based on gender, age, income, and insomnia symptoms. Each group of workers was further classified into a good sleepers or insomnia symptoms groups. Participants completed self-reported questionnaires about sleep variables (e.g., total sleep time), health related variables (e.g., self-perceived health, quality of life measured with the SF-36, medication use), and psychological variables (e.g., anxiety, depression, fatigue).
Results: Results suggested that there were differences mainly on sleep and health related variables for workers without insomnia complaint. Night workers in this subgroup reported a shorter sleep duration ($F_{(2,185)} = 5.72$, $p < .01$) and a lower perceived mental health on the SF-36 ($F_{(2,185)} = 3.48$, $p < .05$) than good sleepers working day or rotating shifts. These differences were no longer present for subgroups of workers with insomnia symptoms. For these workers, permanent night workers used more hypnotics than the other two insomnia symptoms subgroups ($F_{(2,185)} = 2.96$, $p < .05$). Also, workers with insomnia symptoms presented high levels of anxiety, depression, and fatigue regardless of work schedules.
Conclusion: These results suggest that the impact of a work schedule on perceived health and psychological variables appears less significant than has been previously reported. However, it seems that the presence of insomnia symptoms may better explain the impact on perceived health and psychological variables. Further analyses are needed to specify the proportion of perceived health and psychological consequences explained by insomnia symptoms for each group of workers.

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Insomnia and Increased REM Sleep Pressure in a Rat Model of Post Myocardial Infarction Depression

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Introduction:
Myocardial infarction (MI) is followed within a few months by major depression in 15-30 % of patients. We have replicated this phenomenon in a rat model in which post-MI behavioral signs of depression and limbic apoptosis were blocked by antidepressants (Wann et al., 2006, 2007). We now report preliminary results of a pilot study characterizing sleep patterns of rats following MI.
Methods:
Six adult Sprague-Dawley rats were implanted with chronic EEG and EMG electrodes. Five days after surgery, the rats were habituated to the recording equipment for two days and then baseline sleep was recorded for 24h. The following morning MI was induced by occluding the left coronary artery for 40 minutes in three rats; the three other rats were used as sham controls. Sleep was recorded again for 24h two weeks after MI. Comparisons between groups were made using t-tests for independent samples.
Results:
Compared to sham rats, MI rats displayed less total sleep time and more minutes in REM sleep. Analysis by fractions of recording time showed that sleep loss was more important during the light-dark transition and that REM sleep was particularly increased during the first part of the light period.
Conclusion:
These observations of signs of insomnia and increased REM sleep pressure in rats following MI are compatible with sleep in rat models of depression.

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Evolution of Sleep Disturbances and Other Concomitant Symptoms Following Mild Traumatic Brain injury: A Preliminary Report

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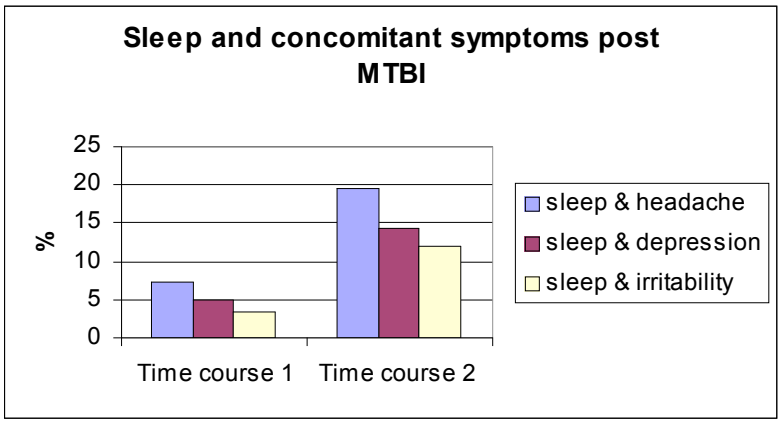
Introduction: Sleep disturbances, headaches, mood and cognitive alterations are reported in patients with mild traumatic brain injury (MTBI). However, the evolution in time of these symptoms appears variable. This study analyzed patient charts to determine the time course of the symptoms over a 4-month period.
Methods: Of 304 patients diagnosed with MTBI, 175 patient charts were randomly selected and reviewed for presence or absence of symptoms via the Rivermead Post-Concussion Symptom Assessment Questionnaire and self-report. Sample studied included 68.6% males and 31.4% female subjects with mean age of 48.9 (min 16 – max 93). Data were collected in a two time interval fashion (interval 1 = mean 10.7 days (response rate: 75%) vs. interval 2 = mean 6.3 weeks (response rate: 56%) and controlled for presence of pre-trauma symptoms. Odd Ratios were calculated.
Results: Sleep, headache and history of psychiatric complaints were present in only 1, 4 and 2 patients prior to trauma, respectively. For time intervals 1 and 2, sleep disturbances prevalence were 11.1% and 34.7% (p: 0.0001; OR: 1.6 [ns]). However, complaints of headache were reported by 46.6% and 40.6% (p: 0.37; OR 4.3 [1.7-10.3]), respectively. The co-existence of headache and sleep symptoms for time courses 1 and 2 were 7.2% (OR: 3.2 [ns]), and 19.6% (OR: 3.7 [1.5-9.1]), respectively; for sleep symptoms and depression 4.9% (OR: 26.8 [5.9-120.9]) and 14.3% (OR: 9.3 [2.9-29.9]) respectively; for sleep symptoms and irritability 3.3% (OR: 31.4 [4.9-202.2]) and 12.1% (OR: 5.7 [1.8-17.6]) respectively.
Conclusion: For sleep disturbances, the evolution of this complaint from time courses 1 to 2 was found to be not significant; however patients were 4.3 times more likely to report headaches in time course 2. Patients whom reported headaches were 3.7 times more likely to report concomitant sleep disturbances in the second time interval. For mood-related symptoms, despite significant ORs, conclusions regarding time course 1 cannot be drawn as patient sample size is limited. However, for time course 2, patients whom reported sleep disturbances were 9.3 and 5.7 times more likely to also report depressive symptoms and irritability, respectively. This data suggest the possibility of a time effect for these symptoms. We are currently reviewing charts of over 500 patients with assessment of one-year follow-up (Supported by CIHR/Pain M2C Training grant and Trauma hospital funds).



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Does Having Sleep Apnea Preclude The Diagnosis of Chronic Fatigue Syndrome

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Objectives

Chronic Fatigue Syndrome (CFS) is an illness characterized by disabling fatigue of at least 6 months. The pathophysiology and etiology of CFS is poorly understood and no single diagnostic test can confirm its presence. Traditionally, an individual cannot be diagnosed with CFS if sleep apnea is present: this is an exclusion criterion. The main objective of this study was to demonstrate that this exclusion criterion is inappropriate.

Methods

We evaluated 68 individuals who had been diagnosed with CFS with respect to fatigue, sleepiness, insomnia and psychological distress. No participant had ever been evaluated prior to our study in a sleep lab. Subsequent to overnight polysomnography, 45 were found to have sleep apnea; twenty-three were not. We offered CPAP treatment to individuals with apnea and examined changes pre and three months post CPAP treatment. We examined the findings for individuals who either fully complied with treatment or did not do so. Of interest for the present submission is the variable of fatigue, the core symptom of CFS.

Results

Results show that compliant participants were more fatigued pre-treatment than non-compliant participants; both compliant and noncompliant participants improved over time. After treatment, fatigue scores for all participants with apnea resembled those of individuals with CFS but no apnea.

Conclusion

Since the treatment itself did not appear to have any differential effect, these data suggest that sleep apnea is a *comorbidity* of CFS rather than an exclusionary criterion in the diagnosis.

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How To Make Insomnia Interventions Available To People With Cancer: Insights From Patients

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Objectives: Insomnia is reported by almost one third of cancer patients. Non-pharmacologic interventions can improve the sleep of people with cancer, yet such interventions are rarely offered to them. The study question was how to make these interventions available to cancer patients. This was an exploratory, qualitative study of patients' ideas and experiences in which we sought answers to *a priori* questions as well as emergent themes.

Methods: Participants with cancer and sleep difficulty (difficulty initiating or maintaining sleep, for at least one month, that interfered with functioning) were recruited through notices in newspapers and cancer centre waiting areas. Five focus groups and six one-to-one interviews were conducted. The questions included: What would be the best way for you to find out about a service for insomnia treatment? What would make it easy/difficult for you to participate? Transcripts were examined independently by three readers who identified participants' answers, as well as themes that emerged from participants' reflections on their experience with cancer and sleep difficulty. The readers then worked to reach consensus on a final classification for the content. The analysis followed established criteria for rigour in qualitative research including: explicit, systematic techniques to analyse the data; multiple coders; identifying deviant cases; and supporting themes with direct quotations from the source.

Results: Twenty-six people (15 women and 11 men) with a variety of cancer types participated. The age range was 44-79 years (mean = 57.7 yrs, SD = 11.2 yrs). Participants provided many practical answers to the questions (see Table for a partial list). In addition, the following themes emerged: sleep difficulty needs greater recognition by health professionals; patients wish to receive more sleep-related information; and that although patients perceive sleep as being important, they are reluctant to report sleep problems to doctors. Furthermore, participants recommended that help for sleep difficulty be integrated into the health care system.

Conclusion: This information is helpful for providing access to insomnia interventions.

Supported, in part, by a grant from the Dering Cancer Research Fund, Kingston General Hospital.
Aspects of this study were presented at the 2005 APSS meeting.

Table. Factors that would make it easy to participate in an insomnia intervention

Topic	Description
Scheduling of the program	Choice of time; Avoid holidays, bad weather, work hours and mornings.
Consideration of energy levels of the participants	Receive intervention after recovery from cancer treatment or during part of chemotherapy cycle when person has sufficient energy to attend.
Credibility	Recommendation or referral from credible person and or organization (e.g., cancer centre staff). Access to research.
Recognition by the health care system	Diagnosis of sleep problem. Orders for treatment and follow-up.
Ease of participation	Easy parking & little paper work. Short & sweet.
No cost	Covered by provincial health care system & no cost for parking.
Group composition	All male or all female (if there are gender-specific issues like menopausal symptoms); groups specific to cancer diagnosis; open to all diagnosis and/or open to those with & without cancer.

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Sleepiness And Depression Contribute To Fatigue In Systemic Lupus Erythematosus (SLE)

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Objectives: Patients with SLE often complain of being tired. This distressing and disabling symptom persists outside of periods of disease flares. Reported in the SLE literature as "fatigue", we prefer to describe it as tiredness, encompassing the physical symptoms of exhaustion, weakness, and deconditioning as well as low mood, poor sleep, and daytime sleepiness. The aim of the study is to characterize the contribution of disturbed sleep, sleepiness and depression to the symptom of tiredness in SLE.

Methods: We identified a group of 35 SLE patients (31 women, 4 men) who complained of disabling tiredness (mean Yoshitake fatigue symptom score of 17.6 ± 4.5 out of maximum of 30). 70% of these patients met the ACR diagnostic criteria for fibromyalgia. Their sleep was evaluated by overnight polysomnography and compared to 17 healthy controls. Daytime multiple sleep latency testing (MSLT), the Beck Depression Inventory and Epworth Sleepiness Scale were also completed.

Results: In comparison to healthy controls, the overnight sleep studies of SLE patients showed impaired sleep efficiency, high frequency of movement arousals, and the majority (77%) had high levels of alpha EEG non-REM sleep. 23% of patients had periodic limb movement disorder and 26% of patients had obstructive sleep apnea. One patient met diagnostic criteria for narcolepsy-cataplexy and 18/35 of the patients were excessively sleepy on MSLT (mean sleep latency < 10 minutes). There was no association between daytime sleepiness and SLE disease features such as neuropsychiatric SLE, medications, fibromyalgia, or disease activity. As a whole, the study group reported mild to moderate depression (mean BDI = 15.8 ± 9.9).

The non-sleepy patients, however, were more depressed with significantly higher BDI scores than the sleepy patients.

Conclusion: The study shows that most tired SLE patients have disordered sleep. A significant proportion of these tired SLE patients are objectively sleepy and can be distinguished from depressed non-sleepy SLE patients. As each require different approaches to management, sleep pathology, sleepiness, and depression should be assessed in tired SLE patients.

Table 1. Overnight polysomnography: sleep architecture.

	Healthy controls (n=17)	SLE patients (n= 35)	p*
Total sleep time, (min.)	381 ± 46	377 ± 98	NS
Sleep efficiency, (%)	85.7 ± 10	75.0 ± 19	p < 0.02
Awakenings /hour sleep	2.61 ± 1.7	4.26 ± 2.6	p < 0.01
Stage 1 sleep (%)	7.44 ± 4.2	11.3 ± 6.6	p < 0.02
Stage 2 sleep (%)	54.2 ± 4.6	57.8 ± 8.5	NS
Stage 3/4 sleep (%)	16.2 ± 6.0	11.0 ± 8.2	p < 0.02
REM sleep (%)	20.9 ± 5.1	19.2 ± 8.6	NS
REM onset latency (min.)	88.1 ± 36	92.3 ± 60	NS
Alpha-EEG score ≥3 (# of patients)	4	27	p < 0.001

Values are mean ± standard deviation except where indicated. * Controls vs. SLE, NS= not significant

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Tobacco Smoking, Sleep and Clinical Symptoms In Patients With Schizophrenia

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Objectives: Tobacco smoking is highly prevalent in schizophrenia. The aim of the present study was to investigate the relationship between smoking habits, sleep and clinical scales in chronic schizophrenia.
Method: Twenty-one persons diagnosed with schizophrenia and clinically stable under atypical neuroleptic treatment spent two consecutive nights in a sleep laboratory. All patients were treated with atypical neuroleptics except one. Sleep stages were determined for night two. The Positive and Negative Syndrome Scale (PANSS) and the Fagerstrom scale for nicotine dependence were administered on the morning following night two. Fifteen patients with chronic smoking habits (12 M, 3 W, mean age: 27.5 ± 6.1) were compared to six non-smokers (4 M, 2 W, mean age: 32.5 ± 10.3) using t-tests. Spearman's rho assessed the relationship between sleep variables, smoking habits, and PANSS scores.
Results: Smokers showed less SWS (Stages 3+4: 6.8% ± 9.0 versus 17.7% ± 14.8; $p<.05$), more REM sleep (20.3% ± 5.4 versus 13.0% ± 5.2; $p<.01$) and more REM sleep efficiency (85.7% ± 8.0 versus 69.7% ± 8.4; $p<.0007$) than non smokers. Smokers also had more positive symptoms (20.5 ± 2.8 versus 17.1 ± 3.8; $p<.04$) and a higher total PANSS score (77.9 ± 8.2 versus 70.2 ± 7.8; $p<.06$) than non-smokers while they performed better than non-smokers on the RBANS. REM sleep was negatively correlated with positive symptoms ($r = -0.52$, $p< .05$), negative symptoms ($r = -0.57$, $p<.03$) and PANSS total score ($r = -0.67$, $p<.006$).
Conclusion: The present results show that tobacco smoking, clinical symptoms and REM sleep are interrelated in schizophrenia. This support the notion that a cholinergic mechanism related to REM sleep underlie clinical symptoms of schizophrenia.
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Medication, Perceptions Of Sleep And Polysomnography Results

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Objectives
Medications can affect sleep parameters, sleep quality, and daytime functioning. Such side effects, however, are rarely examined in the context of patients reporting sleep problems or daytime fatigue and sleepiness. In this investigation we examined patterns in medication profiles in terms of stimulating and sedating effects in two patient groups: (1) consecutive referrals to a sleep laboratory and (2) older adults in primary care. In addition, we explored the relationship between stimulating and sedating medications and sleep/wake variables for each group.

Methods
The Primary Care sample comprised 191 older (55+) family practice patients and the Sleep Clinic sample comprised 125 consecutive new patients. All participants completed the Sleep Study Checklist (SSC) in their respective waiting areas. The SSC includes 21 symptoms of sleep disorder, insomnia, fatigue, sleepiness, and psychological and health functioning. We also obtained written information about current medication use and illnesses.
To date, 35 of the Sleep Clinic participants have completed their PSG evaluation. All Primary Care participants were offered a sleep evaluation, including questionnaires, medical assessment, and polysomnography. Some declined further evaluation, but 26 participants completed all measures, including PSG.

Results
We found that the medication profile, in terms of potential stimulating or sedating effects, differed according to group as well as to experienced sleep/wake difficulties.

Conclusion
Medication profiles permit a better understanding of sleep disturbances and daytime complaints in patient groups. This may alert physicians to possible iatrogenic sleep disorders.

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The Relationship Between Insomnia and Depression In Prostate Cancer Patients Receiving Androgen-Deprevation Therapy

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Objectives. Insomnia and depression are highly interrelated. Studies conducted in the general population suggest that patients with clinical depression are very likely to suffer from insomnia. Longitudinal studies also suggest that insomnia can represent a risk factor for the subsequent development of depressive disorders. Although insomnia and depression are common in cancer patients, no study has yet investigated the longitudinal relationships between these two disturbances. The goal of this study was to assess to what extent depression represents a risk factor for insomnia as opposed to insomnia being a risk factor for depression.

Methods. Twenty-nine patients about to begin long-term ADT for advanced prostate cancer participated in this longitudinal study. The first psychological evaluation was conducted a few days following recruitment (prior to the introduction of ADT) and was followed by six additional evaluations conducted at 2-month intervals. At each time assessment, the participants completed a battery of semi-structured interviews and self-report questionnaires including the Structured Interview for DSM-IV (SCID), the Hamilton Depression Rating Scale (HDRS), the depression subscale of the Hospital Anxiety and Depression Scale (HADS-D), the Beck Depression Inventory (BDI) and the Insomnia Severity Index (ISI).

Results. Patients with a score of 8 or greater on the ISI at one time point had a significantly increased risk of having clinical depression at the subsequent evaluation (SCID: OR = 5.94; HADS-D: OR = 4.83; BDI: OR = 21.3; HDRS: OR = 3.60). Conversely, patients with clinical depression at one time point were even more likely to have clinical levels of insomnia at the subsequent assessment (SCID: OR = 10.83; HADS-D: OR = 9.28; BDI: OR = 19.96; HDRS: OR = 7.45). To further explore cross-sectional and longitudinal relationships between insomnia and depressive symptoms, four structural equation models were completed, for each of the continuous depression measures. Results revealed modest but significant paths between the ISI score obtained at one point and: (a) the sum of major depressive symptoms assessed with the SCID ($B = 0.18$), (b) BDI scores ($B = 0.13$, $p < .10$), and (c) HDRS scores ($B = 0.22$) obtained at the previous evaluation. Only one path between depressive symptoms assessed at one point and the ISI score obtained at the previous evaluation was significant ($B = 0.20$).

Conclusions. Results of this study suggest that, although the relationship between insomnia and depression goes both ways, depression is a more important risk factor for insomnia than insomnia is a risk factor for depression in men treated for prostate cancer with ADT.

This study was supported in part by a National Alliance for Research on Schizophrenia and Depression Young Investigator award and research scientist awards from the Canadian Institutes of Health Research and the *Fonds de la recherche en santé du Québec* held by the first author.

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Insomnia and Androgen-Deprevation Therapy for Prostate Cancer

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Objectives. Because of the testosterone decline associated with its use, ADT causes numerous side effects including the occurrence of hot flashes. Previous studies conducted in other populations have shown that nocturnal hot flashes are associated with sleep disturbances. ADT is therefore likely to increase the risk for developing insomnia in prostate cancer patients, although this hypothesis has never been investigated using validated insomnia assessment tools. The goals of this study were to: (1) assess the longitudinal course of insomnia symptoms in men prior to and while receiving ADT for prostate cancer; and to (2) assess the contribution of the introduction of ADT in the development of insomnia symptoms.

Methods. Twenty-nine patients about to begin long-term ADT for advanced prostate cancer participated in this longitudinal study. The first psychological evaluation was conducted a few days following recruitment (prior to ADT introduction) and was followed by six additional evaluations conducted at 2-month intervals. At each time assessment, the participants completed a battery of self-report questionnaires including the Insomnia Severity Index (ISI).

Results. The participants obtained a mean ISI score of 5.14 (*SD* = 4.87) at baseline, 5.75 (*SD* = 5.22) at 2 months, 5.44 (*SD* = 4.59) at 4 months, 5.93 (*SD* = 4.75) at 6 months, 5.50 (*SD* = 4.50) at 8 months, 6.19 (*SD* = 4.73) at 10 months, and 5.50 (*SD* = 5.42) at 12 months. A normal linear mixed model analysis using a randomized block design revealed no significant overall time effect, F (6, 154) = 0.89, p = .51 and an *a priori* pairwise comparison revealed no significant time effect from baseline to the 2-month evaluation (p = .50, Cohen's d = 0.13). The proportion of patients having an ISI score of 8 or greater, which indicates clinical levels of insomnia, was 31.0% at baseline, 39.3% at 2 months, 33.3% at 4 months, 37.0% at 6 months, 30.8% at 8 months, 34.6% at 10 months, and 34.6% at 12 months. A generalized linear mixed model revealed no overall significant time effect, F (6, 154) = 0.28, p = .95 and no significant effect from baseline to the 2 month evaluation (p = .32, Cohen's d = 0.16).

Conclusions. Results of this study suggest that the introduction of ADT has a minimal role in the development of insomnia symptoms in men with prostate cancer.

This study was supported in part by a National Alliance for Research on Schizophrenia and Depression Young Investigator award and research scientist awards from the Canadian Institutes of Health Research and the *Fonds de la recherche en santé du Québec* held by the first author.

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REM Sleep is Reduced on an Ultra-Rapid Sleep-Wake Cycle Procedure in Bipolar Affective Disorder

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Objectives: A disturbed sleep/wake cycle and/or circadian regulation of sleep may participate in the sleep disturbances observed in bipolar affective disorder. The aim of the present study was to explore the circadian variation of sleep in bipolar patients.
Methods: Four bipolar patients (3 men, 1 woman; 40-47yrs), stabilized on lithium and/or risperdal, and 3 healthy controls (2 men, 1 woman; aged 20-42yrs) were recruited. Following two 8-hour sleep episodes, participants underwent a 48-hour ultra-rapid sleep-wake cycle procedure (60 min wake-60 min nap opportunities). The reliable physiological correlate of the circadian clock core body temperature (CBT) was recorded using a rectal sensor every 60 sec. Sleep was polysomnographically recorded and quantified during 20-sec epochs. One-factor analysis (ANOVA) was used to assess the circadian variation of sleep parameters. Correlations were calculated between REM sleep and CBT. Results are reported as mean ± SEM.
Results: Baseline sleep recordings revealed that bipolar subjects had less total sleep than controls (TST=340.4 min/baseline night ± 26.0 min, and 444.6 min/baseline night ± 5.5 min, respectively), and less REM sleep (patients=71.1 min ± 5.7 min; controls=98.8 min ± 12.3 min), but had similar amounts of REM sleep as a percentage of TST, with patients achieving 21.2%±1.9% and controls 22.2%±2.9%. Interestingly, REM sleep was drastically reduced during the ultra-rapid sleep-wake cycle procedure in bipolar patients (REM=5.6% TST ±4.0%), compared to controls (REM=18.2% TST ±6.2%) and to their own baseline sleep recording at night. ANOVA revealed a significant circadian rhythm of REM sleep in both patients [$F(23,120)=3.57$, $p<0.01$] and controls [$F(23,72)=1.87$, $p=0.02$], but a significant correlation between REM and CBT was found only in controls ($r=-0.60$, $p<0.01$).
Conclusions: The present study revealed reduced REM sleep in bipolar patients on an ultra-rapid sleep-wake schedule, despite relatively normal baseline sleep episodes. This is markedly different from what was observed in healthy control subjects. These results support a disruption of sleep production and/or the circadian regulation of sleep in bipolar affective disorder. An influence of lithium and/or risperdal administration is not excluded.

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